Australian Government
Department of Health and Ageing

Communicable Diseases Intelligence

Volume 32 Number 3 Quarterly report September 2008

Annual reports

Articles

Short reports

Quarterly reports

Administrative

© Commonwealth of Australia 2008

ISSN 0725-3141
ISSN 1445-4866 Online

This work is copyright. Apart from any use as permitted under the *Copyright Act 1968,* no part may be reproduced by any process without prior written permission from the Commonwealth. Requests and inquiries concerning reproduction and rights should be addressed to the Commonwealth Copyright Administration, Attorney General's Department, Robert Garran Offices, National Circuit, Barton ACT 2600 or posted at http://www.ag.gov.au/cca

Communicable Diseases Intelligence aims to diseminate information on the epidemiology and control of communicable diseases in Australia. *Communicable Diseases Intelligence* invites contributions dealing with any aspect of communicable disease epidemiology, surveillance or prevention and control in Australia. Submissions can be in the form of original articles, short reports, surveillance summaries, reviews or correspondence. Instructions for authors can be found in *Commun Dis Intell* 2008;32:130–133.

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

Editor

Ian McKay

Editorial and Production Staff

Conan Liu, Alison Milton

Editorial Advisory Board

Jeremy McAnulty (Chair), Scott Cameron, John Kaldor, Peter McIntyre, Paul Van Buynder, Charles Watson

Website

http://www.health.gov.au/cda

Subscriptions and contacts

Communicable Diseases Intelligence is produced every quarter by:
Surveillance Branch
Office of Health Protection
Australian Government Department of Health and Ageing
GPO Box 9848, (MDP 6)
CANBERRA ACT 2601;
Telephone: +61 2 6289 2717
Facsimile: +61 2 6289 2600
Email: cdi.editor@health.gov.au

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

Disclaimer

Opinions expressed in *Communicable Diseases Intelligence* are those of the authors and not necessarily those of the Australian Government Department of Health and Ageing or the Communicable Diseases Network Australia. Data may be subject to revision.

Printed by Union Offset, Canberra

Annual reports

Annual report of the Australian Meningococcal Surveillance Programme, 2007

The Australian Meningococcal Surveillance Programme

Abstract

In 2007 there were 242 laboratory-confirmed cases of invasive meningococcal disease analysed by the National Neisseria Network, a nationwide network of reference laboratories. The phenotypes (serogroup, serotype and serosubtype) and antibiotic susceptibility of 127 isolates of *Neisseria meningitidis* from invasive cases of meningococcal disease were determined and an additional 115 cases were confirmed by non-culture based methods. Nationally, 192 (85%) confirmed cases where a serogroup was determined were infected with serogroup B and 14 (6.2%) with serogroup C meningococci. The total number of confirmed cases was 29 fewer than the 271 cases identified in 2006. The only jurisdiction to record a substantial increase in laboratory confirmed cases was New South Wales and this was in sporadic cases of serogroup B infection. Typical primary and secondary disease peaks were observed in those aged 4 years or less and in adolescents and young adults respectively. Serogroup B cases predominated in all age groups and jurisdictions. The common phenotypes circulating in Australia were B:15:P1.7, B:4:P1.4 and C:2a:P1.5. No evidence of meningococcal capsular 'switching' was detected. About three-quarters of all isolates showed decreased susceptibility to the penicillin group of antibiotics (minimum inhibitory concentration [MIC] 0.06–0.5 mg/L). All isolates remained susceptible to rifampicin. A single serogroup B isolate had decreased susceptibility to ciprofloxacin (MIC 0.06 mg/L). This was the first local isolate of this type since the original report of this phenomenon in Australia in 2000. *Commun Dis Intell* 2008;32:299–307.

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

Introduction

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

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

Methods

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

Isolate based invasive meningococcal disease cases

Each case confirmation was based upon isolation of a meningococcus from a normally sterile site and defined as IMD according to Public Health Laboratory Network criteria.[3] Information on the site of infection, the age and sex of the patient and the outcome (survived/died) of the infection was sought. The isolate-based subset of the program categorised cases on the basis of site of isolation of the organism. Where an isolate was grown from both blood and cerebrospinal fluid (CSF) cultures

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

Phenotyping of invasive isolates of meningococci by serotyping and serosubtyping was based on the detection of outer membrane protein (porin) antigens using a standard set of monoclonal antibodies obtained from the National Institute for Public Health, The Netherlands. Increasingly, sequencing of products derived from amplification of the porin genes *porA* and *porB* has been used to supplement and supplant serotyping analyses based on the use of monoclonal antibodies. For the purposes of continuity and comparability, the typing data from both approaches has been unified in the accompanying tables by converting sequence data to the more familiar serotyping/serosubtyping nomenclature.

Antibiotic susceptibility was assessed by determining the minimal inhibitory concentration (MIC) to antibiotics used for therapeutic and prophylactic purposes. This program uses the following parameters to define the various levels of penicillin susceptibility/resistance when determined by a standardised agar plate dilution technique.[4]

sensitive, MIC ≤ 0.03 mg/L

less sensitive, MIC 0.06 – 0.5 mg/L

relatively resistant MIC ≤ 1 mg/L

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

Non-culture-based laboratory-confirmed cases

Additional laboratory confirmation of suspected cases of IMD was obtained by means of non-culture based methods including nucleic acid amplification testing (NAAT) and serological techniques. NAAT testing is essentially by polymerase chain reaction (PCR) techniques[5] and has been progressively introduced in the different jurisdictions. Data from the results of these investigations were included for the first time in the 1999 report.[1] The serological results are based on results of tests performed using the methods and test criteria of the Manchester Public Health Laboratory Service reference laboratory, United Kingdom as assessed for Australian conditions.[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. This separation is not possible for cases diagnosed serologically.

Results

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

Number of laboratory confirmed cases

There were 242 laboratory confirmed cases of IMD in 2007 (Table 1) compared with 271 in 2006, 345 in 2005 and 361 in 2004. In 127 (52.4%) cases, a positive culture was obtained with or without a positive non-culture based test and 115 cases were confirmed by a non-culture based method alone. The total number of all laboratory confirmed cases decreased in most jurisdictions in 2006 when compared with 2005 data. The largest decrease in

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

State or territory	Serogroup						Total
	B	C	A	Y	W135	NG*	
ACT	4	0			1		5
NSW	78	7		5	1	10	101
NT	1	1					2
Qld	30	3		1	1	1	36
SA	11	1		1	1		14
Tas	3	0		1	1		5
Vic	46	2		4	3	4	59
WA	19	0				1	20
Australia	192	14	0	12	8	16	242

* Not serogrouped.

numbers were in Queensland (to 36 from 68) and Victoria (to 59 from 75). Small or no numerical differences were noted in other jurisdictions with the exception of New South Wales where numbers detected increased to 102 from 84 after a decrease in 2006.

Seasonality

Forty cases occurred between 1 January and 31 March, 44 between 1 April and 30 June, 77 between 1 July and 30 September and 81 between 1 October and 31 December. A winter peak of meningococcal disease is more usual.

Age distribution

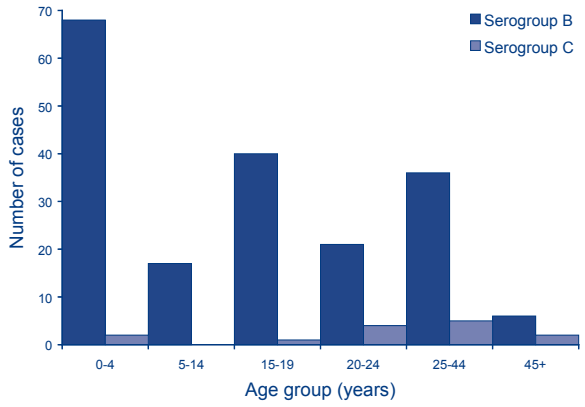
Nationally, the peak incidence of meningococcal disease was again in those aged 4 years or under (Table 2, Figure). Those aged less than one year or in the 1–4 years age group together accounted for 83 cases (34.3% of the total) in 2007. There were 100 cases confirmed in these age groups (37%) in 2006. A secondary disease peak is also usual in the adolescent/young adult age group. The total of 47 cases (19.4% of all confirmed cases) in those aged 15–19 years was much the same as the number and proportion of cases in this age group in 2006 (49, 18%). Those aged 15–24 years together accounted for 74 cases (30.6%) in 2007 and 79 cases (29%) in 2006.

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

State or territory	Serogroup	Age group										Total
		<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	NS	
ACT	B	1	2						1			4
	C											0
	Total	2	2						1			5
NSW	B	10	21	3	4	7	9	11	8	4	1	78
	C	1	1				1		3	1		7
	Total	15	24	3	6	8	10	14	12	8	1	101
NT	B						1					1
	C					1						1
	Total					1	1					2
Qld	B	3	7	3	1	8	1	3	4			30
	C						2			1		3
	Total	3	9	3	2	8	3	3	4	1		36
SA	B	2	3	1		4				1		11
	C								1			1
	Total	2	3	1		5	1		1	1		14
Tas	B		1		1						1	3
	C											0
	Total		1		1	1				1	1	5
Vic	B	8	6	1	3	17	7	0	3	1		46
	C						1		1			2
	Total	9	8	2	3	20	9	3	4	1		59
WA	B	2	2			4	3	2	4		2	19
	C											0
	Total	2	3			4	3	2	4		2	20
Australia	B	26	42	8	9	40	21	16	20	6	4	192
	C	1	1	0	0	1	4	0	5	2		14
	Total B+C	27	43	8	9	41	25	16	25	8	4	206
	Other	6	7	1	3	6	2	6	1	4		36
	Total	33	50	9	12	47	27	22	26	12	4	242
	% of all	13.6	20.7	3.7	5	19.4	11.2	9	10.7	5	1.7	

NS Not stated. Totals include cases due to other serogroups (20) and cases where the serogroup was not determined (16).

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



Serogroup data

The serogroup of the meningococci causing disease was determined in 226 of the 242 laboratory confirmed cases of IMD. Of these 226 cases where a serogroup was determined, 192 (85%) were serogroup B and 14 (6.2%) serogroup C. In 2006, 217 (83.8%) were serogroup B and 26 (10%) serogroup C. In 2007 an additional 8 cases (3.5%) were of W135 and 12 (5.3%) of serogroup Y. With the continuing decline in numbers of serogroup C infections, serogroup B meningococci predominated in all age groups and jurisdictional differences in serogroup distribution were not evident. Seven of the 14 cases of serogroup C disease in 2007 were in those aged 45 years or more, a single case was recorded in those aged 15–19 years and a further 4 in those aged 20–24 years. Seven serogroup C cases were identified in those aged 15–24 years in 2006. Seven of the 14 serogroup C cases were in New South Wales.

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

Phenotypes of invasive meningococcal isolates

The typical heterogeneity of serogroup B meningococci was again seen in 2007 when the phenotypes of invasive isolates, based on a determination of their serogroup, serotype and serosubtype were analysed. The predominant serotypes/serosubtypes in each state and territory are shown in Table 4. Serogroup B meningococci are in general also more difficult to characterise by serological methods and a number could not be phenotyped. A total of 27 isolates were of serotype 4 and 12 of these from New South Wales (8), Victoria (3) and the Australian Capital Territory (1) were of serosubtype P1.4.

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

Year	Serogroup	Age group									
		< 4 years		5–14 years		15–19 years		20–24 years		25+ years	
		n	%	n	%	n	%	n	%	n	%
2007	B	68	86.0	17	90.0	40	91	21	81.0	42	74.0
	C	2	2.5	0	0.0	1	2.3	4	15.0	7	12.0
	All*	79		19		44		26		57	
2006	B	93	93.0	21	84.0	40	82.0	21	70.0	38	61.3
	C	2	2	3	12.0	4	8.2	7	23.0	10	16.1
	All	100		25		49		30		62	
2005	B	99	90.0	38	75.0	39	81.0	22	67.0	51	50.0
	C	6	5.5	5	10.0	4	8.0	8	24.0	27	27.0
	All	110		51		48		33		101	
2004	B	97	88.0	27	77.0	40	65.0	20	57.0	59	50.0
	C	6	5.5	5	14.0	17	28.0	11	31.0	32	27.0
	All	110		35		61		35		117	

* All cases where a serogroup was determined.

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

State or territory	Serogroup B				Serogroup C			
	Serotype	n	Serosubtype	n	Serotype	n	Serosubtype	n
Australian Capital Territory	4	1	1.4	1				
New South Wales	4	20	1.4	8	2a	4	1.5	2
			1.15	4			1.2	1
			1.7	2			1.4	1
			1.14	2				
			1.5	1				
			1.5,2	1				
			1.22,14	1				
			nst	1				
	15	5	1.5	5				
	1	3	1.4	1				
			nst	2				
	nt	14	1.9	3				
			1.14	1				
			1.15	1				
			1.16	1				
			1.4	1				
			nst	7				
Northern Territory					2a	1	1.5	1
Queensland	1	3	1.14	3	2a	3	1.5	1
	15	1	1.7,16	1			nst	2
	nt	7	1.14	2				
			1.15	1				
			nst	4				
Tasmania	nt	1	1.17	1				
Victoria	4	6	1.4	3	2a	2	1.16	2
			1.15	2				
			nst	1				
	15	6	1.7,16	4				
			1.4	1				
			1.5,10	1				
	nt	9	1.5,2	3				
			1.14	3				
			1.5,15	1				
			1..15	2				
			1..16	1				
			nst	2				
Western Australia	14	1	1.5	1				
	15	1	nst	1				
	1	1	1.14	1				
	nt	6	diverse					

nt Not serotypeable

nst Not serosubtypeable

Fourteen serogroup B strains with this subtype/serosubtype were seen in 2006. This phenotype has been circulating in New Zealand at high rates for many years. Another 13 serogroup B isolates were of serotype 15 and included 5 each of sero-subtypes 1.5 and 1.7,16. The latter phenotype has been circulating in Australia for many years.

There is continuing interest in the presence of any serogroup B or serogroup C meningococci of serotypes that indicate the possibility of genetic recombination events. Among serogroup C strains, phenotype C:2a:P1.4 is of particular interest. This phenotype has figured prominently in Victorian data in former years. In 2003 there were 29, in 2004, 21 and in 2005, 8 serogroup C isolates of this serotype/serosubtype detected nationally. Only a single isolate with this phenotype were seen in 2007 (in New South Wales). All of the serotypeable serogroup C isolates were of serotype 2a.

Outcome data for invasive meningococcal disease for all laboratory confirmed cases

Outcome data (survived or died) were available for 96 (40%) of the 242 laboratory confirmed cases (Table 5). Four deaths were recorded in this group (4.2%). Outcomes were available for 73 of 192 of serogroup B infections and 5 of 14 of serogroup C infections. There was a single death from each of serogroup B and serogroup Y infections and 2 attributable to serogroup C.

There was 1 death in 38 patients with meningitis (due to a serogroup B meningococcus). Three deaths were recorded in 57 bacteraemic patients. There were 39 cases of serogroup B meningococcal bacteraemia with no deaths. The single fatality with serogroup Y disease was in a group of 6 bacteraemic cases where outcomes were recorded and the 2 septicaemic fatalities due to serogroup C meningococci were recorded in 5 instances of bacteraemia with this serogroup.

Anatomical source of samples for laboratory confirmed cases

Table 6 shows the source of clinical samples by which laboratory confirmation of IMD was obtained. Those diagnoses shown as culture positive may have had positive PCR and/or serology, those shown as PCR positive were culture negative with or without positive serology and those shown as serologically positive were culture and PCR negative. There were 29 isolates from CSF either alone or with a blood culture isolate and 93 from blood cultures alone. There were 4 other isolates from synovial fluid and one, most unusually from the peritoneal fluid of a patient undergoing peritoneal dialysis.

Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillin

One hundred and twenty-seven isolates were available for determination of their susceptibility to penicillin and other antibiotics. Using defined criteria, 101 isolates (79%) were less sensitive to penicillin in the MIC range 0.06–0.5 mg/L and the remainder (21%) fully sensitive (MIC 0.03 mg/L or less). The proportion of less sensitive strains is higher than that reported in recent years (67% in 2006). Seven isolates had MICs of 0.5 mg/L. Six of these were found in New South Wales. Four were of serogroup B and 3 of serogroup Y.

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

Disease type	Outcome	Serogroup					Total
		B	C	Y	W135	NG	
Meningitis	Survived	32	0	2	2	1	37
	Died	1	0	0	0	0	1
	Total	33	0	2	2	1	38
Septicaemia	Survived	39	3	5	2	5	54
	Died	0	2	1	0	0	3
	Total	39	5	6	2	5	57
All cases*	Survived	72	3	7	3	7	92
	Died	1	2	1	0	0	4
	Total	73	5	8	3	7	96

NG Not groupable.

* Includes 3 cases of joint infection, 1 each of serogroup B and W135 and 1 non-serogroupable case all of whom survived.

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

Specimen type	Isolate of MC	Polymerase chain reaction positive*	Total
Blood	93	63	156
Cerebrospinal fluid +/– blood	29	45	74
Other†	5	2	7
Serology alone‡			5
Total	127	110	242

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

† Joint and fluid samples (4 isolates from joints and 2 by PCR of joint fluid; 1 culture from peritoneal fluid).

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

Other antibiotics

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

Discussion

There has been a further decline in the number of laboratory confirmed cases of IMD in Australia in 2007. Much of the interpretation of these surveillance data needs to be in the context of the recently completed program of vaccination of children and adolescents with the serogroup C conjugate vaccine. The only jurisdiction to show a rise in numbers of laboratory confirmed cases was New South Wales. Cultures were obtained from sterile sites in 127 cases, the lowest number of isolates detected over the duration of the program that commenced in 1994 and a further decline from the 166 cases from whom isolates were obtained in 2006. Non-culture based diagnoses were used to confirm a further 115 (47%) of cases IMD.

Only 14 serogroup C infections were identified nationally in 2007, half of these in New South Wales, so that serogroup B disease accounted for 85% of all infections where a serogroup was determined. Only small numbers of infections due to serogroups Y and W135 were encountered, and this is usual for Australia. A primary peak in IMD infection rates was again evident in younger age groups with a secondary peak in adolescents and young adults. In contrast to data from the earlier years of this program, serogroup C disease was infrequently encountered in the latter age group in 2007. Also of interest is the continuing decline in numbers of IMD in those aged 25 years or more (Table 3). A decrease in serogroup C cases in essentially unvaccinated age groups has been noted elsewhere. It is attributed to the secondary benefit of herd immunity accruing to the wider community following vaccination of those age groups where disease was formerly highly concentrated.[10]

The continuing absence of any substantial numbers of meningococci showing evidence of genetic recombination in phenotyping and genotyping data is reassuring and also consistent with data from the United Kingdom.[10] Analysis of meningococcal subtypes and any evidence for the expansion of 'new' subtypes will continue as part of the NNN program. Mortality data were assessable in only a low proportion of cases and must be interpreted with caution. The NNN does not attempt collection of morbidity data associated with IMD.

NNN trend data show an upward shift in penicillin MICs insofar as the proportion of invasive isolates with reduced susceptibility to penicillins increased from 67% to 79% in 2007. However, penicillins remain a suitable treatment for IMD in Australia. All isolates were susceptible to the third generation cephalosporins and to the 'clearance' antibiotic rifampicin. Of particular interest was a serogroup B isolate from New South Wales with reduced susceptibility to ciprofloxacin (0.06 mg/L). The first ever reported case of an invasive *N. meningitidis* with reduced susceptibility to fluoroquinolones and where the molecular basis of the resistance mechanism involved was also described arose from surveillance conducted by this program.[11] Subsequently, other sporadic cases of meningococci with reduced quinolone susceptibility have been reported in several countries, and, more recently, clusters of quinolone less-susceptible meningococci have also been described in India[12] and the United States of America.[13] Serogroups A, B, C and Y have all exhibited this decreased quinolone susceptibility and a number of different resistance mechanisms are

now known to be involved. Invasive meningococci possess the potential to develop full resistance to quinolones (similar to MIC levels now seen in quinolone-resistant *Neisseria gonorrhoeae*)[14] so that antimicrobial resistance surveillance remains an important component of AMSP activities.

Acknowledgments

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

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

Participants in the 2007 Australian Meningococcal Surveillance Programme (to whom isolates and samples should be referred and enquiries directed) are listed below.

Australian Capital Territory

Dr P Collignon/Ms S Bradbury
Microbiology Department
The Canberra Hospital
PO Box 11
Garran ACT 2606
Telephone: +61 6 244 2425
Email: peter.collignon@act.gov.au

New South Wales

J Tapsall/A Limnios/TR Shultz
Microbiology Department
SEALS
The Prince of Wales Hospital
Randwick NSW 2031
Telephone: +61 2 9382 9079
Facsimile: +61 2 9398 4275
Email: j.tapsall@unsw.edu.au

J Mercer/R Porritt
Department of Microbiology and Infectious Diseases
SWAPS
Locked Mail Bag 90
Liverpool NSW 2179
Telephone: +61 2 9828 5128
Facsimile: +61 2 9828 5129
Email: Joanne.Mercer@swsahs.nsw.gov.au or Robert.Porritt@swsahs.nsw.gov.au

Northern Territory

Paul Southwell and staff
Microbiology Laboratory, NTGPS
Royal Darwin Hospital Campus
Tiwi NT 0810
Telephone: +61 8 8922 8034
Facsimile: +61 8 8980 0714
Email: paul.southwell@nt.gov.au

Queensland

John Bates/Denise Murphy/Helen Smith
Public Health Microbiology
Queensland Health Scientific Services
39 Kessels Road
Coopers Plains Qld 4108
Telephone: +61 7 3274 9101
Facsimile : +61 073274 9175
Email: john_bates@health.qld.gov.au

Tasmania

Dr A McGregor/Mr Mark Gardam/
Mrs Belinda Chamley
Department of Microbiology and Infectious Diseases
Royal Hobart Hospital
GPO Box 1061L
Hobart Tasmania 7001
Telephone: +61 36222 8656
Email: mark.gardam@dhhs.tas.gov.au

South Australia

Mr A Lawrence
Microbiology Department
Women's and Children's Hospital
72 King William Road
North Adelaide SA 5006
Telephone: +61 8 8161 6376
Facsimile: +61 8 8161 6051
Email: andrew.lawrence@cywhs.sa.gov.au

Victoria

Geoff Hogg/Julia Griffith/Angelo Zaia
Microbiological Diagnostic Unit Public Health Laboratory (MDU PHL)
Department of Microbiology and Immunology
The University of Melbourne
Parkville Victoria 3052
Telephone: +61 3 8344 5701
Facsimile: +61 3 8344 7833
Email: g.hogg@mdu.unimelb.edu.au

Western Australia

Mr P Campbell/Dr AD Keil
Department of Microbiology
Princess Margaret Hospital for Children
1 Thomas Street
Subiaco WA 6008
Telephone: +61 8 9340 8273
Facsimile: +61 8 9380 4474
Email: tony.keil@health.wa.gov.au;
peter.campbell@health.wa.gov.au

The Australian Meningococcal Surveillance members in 2007 were (in alphabetical order): John Bates, Denise Murphy, Helen Smith, Public Health Microbiology, Queensland Health Scientific Services, Coopers Plains, Queensland, Athena Limnios, Sanghamitra Ray, Tiffany Hogan and John Tapsall, Department of Microbiology, The Prince of Wales Hospital, Randwick, New South Wales; Jo Mercer and Robert Porritt, Department of Microbiology and Infectious Diseases, SWAPS, Liverpool, New South Wales; Geoff Hogg, Julia Griffith and Angelo Zaia, The Microbiological Diagnostic Unit, PHL, Department of Microbiology and Immunology, University of Melbourne, Parkville, Victoria; Andrew Lawrence, Microbiology Department, Women's and Children's Hospital, North Adelaide, South Australia; Peter Campbell and Tony Keil, Department of Microbiology, Princess Margaret Hospital for Children, Subiaco, Western Australia; Mark Gardam and Belinda Chamley, Department of Microbiology and Infectious Diseases, Royal Hobart Hospital, Hobart, Tasmania; Gary Lum and Microbiology Staff, Microbiology Laboratory, Royal Darwin Hospital, Casuarina, Northern Territory; Susan Bradbury and Peter Collignon, Microbiology Department, Canberra Hospital, Garran, Australian Capital Territory.

Author details

Corresponding author: Associate Professor John Tapsall, Department of Microbiology, SEALS, The Prince of Wales Hospital, High Street, RANDWICK, NSW 2031.

References

1. National Neisseria Network. Meningococcal Isolate Surveillance Australia 1994. *Commun Dis Intell* 1995:19:286–289.
2. The Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 2006. *Commun Dis Intell* 2007;31:185–194.
3. Public Health Laboratory Network meningococcal long case definition: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-phlncd-mening.htm
4. Tapsall J and members of the National Neisseria Network of Australia. Antimicrobial testing and applications in the pathogenic *Neisseria*. In: Merlino J, ed. *Antimicrobial susceptibility testing: methods and practices with an Australian perspective*. Australian Society for Microbiology, Sydney, 2004. pp 175–188.
5. Porritt RJ, Mercer JL, Munro R. Detection and serogroup determination of *Neisseria meningitidis* in CSF by polymerase chain reaction (PCR). *Pathology* 2000;32:42–45.
6. Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 1999. *Commun Dis Intell* 2000;24:181–189.
7. Gray SJ, Borrow R, Kaczmarski EB. Meningococcal serology. In: Pollard AJ, Martin MCJ, eds. *Meningococcal disease methods and protocols*. Humana Press, Totawa, New Jersey, 2001 pp 61–87.
8. Robertson PW, Reinbott P, Duffy Y, Binotto E, Tapsall JW. Confirmation of invasive meningococcal disease by single point estimation of IgM antibody to outer membrane protein of *Neisseria meningitidis*. *Pathology* 2001:33:375–378.
9. Lahra MM, Robertson PW, Whybin R, Tapsall JW. Enhanced serological diagnosis of invasive meningococcal disease by determining anti-group C capsule IgM antibody by enzyme immunoassay. *Pathology* 2005;37:239–241.
10. Maiden MCJ, Ibarrz-Pavón AB, Urwin R, Gray SJ, Andrews NJ, Clarke SC, et al. Impact of meningococcal serogroup C conjugate vaccines on carriage and herd immunity. *J Infect Dis* 2008;197:737–743.
11. Shultz TR, Tapsall JW, White PA, Newton PJ. An invasive isolate of *Neisseria meningitidis* showing decreased susceptibility to quinolones. *Antimicrob Agents Chemother* 2000;44:1116.
12. Singhal S, Purnapatre KP, Kalia V, Dube S, Nair D, Deb M, et al. Ciprofloxacin-resistant *Neisseria meningitidis,* Delhi, India. *Emerg Infect Dis* 2007;13:1614–1616.
13. Rainbow J. Boxrud D, Glennen A, Danila R, Juni B, Mariotti J, et al. Emergence of fluoroquinolone-resistant *Neisseria meningitidis*—Minnesota and North Dakota, 2007–2008. *MMWR Morb Mortal Wkly Rep* 2008;57:173–175.
14. Shultz TR, White PA, Tapsall JW. An in-vitro assessment of the further potential for development of quinolone resistance in *Neisseria meningitidis*. *Antimicrob Agent Chemother* 2005;49:1753–1760.

Annual report of the Australian National Poliovirus Reference Laboratory, 2007

Jason A Roberts, Kristina A Grant, Aishah Ibrahim, Bruce R Thorley

Abstract

In July 2007, wild poliovirus type 1 was isolated from a patient suffering poliomyelitis in Melbourne, Australia with onset in Pakistan. The imported case of polio demonstrates the ongoing risk faced by polio-free countries until the global certification of polio eradication. The poliovirus was detected by the National Poliovirus Reference Laboratory (NPRL) for Australia; accredited by the World Health Organization (WHO). The NPRL acts as the national laboratory for the Pacific Islands, Brunei Darussalam and Papua New Guinea. Additionally, the NPRL functions as a regional reference laboratory for the WHO Western Pacific Region. The NPRL, in collaboration with the Australian Paediatric Surveillance Unit, co-ordinates surveillance for acute flaccid paralysis (AFP), a major clinical presentation of poliovirus infection. After classification of AFP cases by the Polio Expert Committee, the non-polio AFP rate for Australia in 2007 was 0.65 per 100,000 children aged less than 15 years, below the performance indicator of 1.0 per 100,000 set by the WHO. Adequate faecal sample collection totalled 48% (13/27) of eligible AFP notifications, below the 80% performance indicator recommended by the WHO. During 2007, 119 specimens were referred to the NPRL, 70 from AFP cases and 49 from other sources, including contacts of the wild poliovirus importation, all negative for poliovirus infection. Coxsackievirus A4 was isolated from 1 case and adenovirus from 2 cases. During 2007, 1,313 cases of poliomyelitis due to wild poliovirus infection were reported world-wide: 1,207 occurring in the 4 remaining polio endemic countries and 106 cases reported in 5 non-endemic countries. *Commun Dis Intell* 2008;32:308–315.

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

Introduction

In 1988 the World Health Assembly, which is the governing body of the World Health Organization (WHO), passed a resolution to eradicate polio by the year 2000.[1] Although that date has now passed, great progress has been made toward the goal of global polio eradication. There are 4 remaining endemic countries, Afghanistan, India, Nigeria and Pakistan with a further 8 countries reporting importation of poliovirus from endemic regions in June 2008.[2]

Established in 1994 by the Australian Commonwealth Government, the National Poliovirus Reference Laboratory (NPRL), based within the Victorian Infectious Diseases Reference Laboratory (VIDRL), has played a major role in Australia's commitment to the WHO polio eradication program. The NPRL is accredited by WHO as the national laboratory for the isolation and characterisation of poliovirus from clinical specimens within Australia, the Pacific Islands, Papua New Guinea and Brunei Darussalam. The NPRL is also designated as a Regional Reference Laboratory for the WHO Western Pacific Region (WPR) and receives poliovirus isolates for further characterisation from National Polio Reference Laboratories of the WPR.

The Australian Commonwealth Government initiated a surveillance program for acute flaccid paralysis (AFP) in 1995 based on the guidelines recommended by WHO focussing on AFP cases in children aged less than 15 years. Co-ordination of the AFP surveillance program is undertaken at the NPRL in collaboration with the Australian Paediatric Surveillance Unit (APSU). Suspected cases of poliomyelitis and notified cases of AFP, regardless of age, are subjected to review by the Australian Polio Expert Committee (PEC), a sub-committee of the Communicable Diseases Network Australia.

The current recommendation for polio vaccination in Australia is at 2, 4 and 6 months and 4 years of age. A 10 yearly supplemental vaccination for 'at-risk' groups such as health-care workers and travellers to countries known to contain active transmission of wild poliovirus is also recommended. Primary vaccination in adults should include 3 doses at 1 to 2 month intervals.[3] As of November 2005, the Australian National Immunisation Program moved to the exclusive use of inactivated poliovirus vaccine (IPV), in place of the live attenuated Sabin oral poliovirus vaccine (OPV).[4] The use of OPV reduces the incidence of vaccine associated paralytic poliomyelitis (VAPP), estimated to occur in one in 2.4 million doses of OPV distributed. After administration of OPV, the recipient may shed live poliovirus intermittently for up to 6 weeks. In immunocompromised persons who receive OPV, virus excretion can persist in excess of 6 weeks.[5] The exclusive use of IPV in the vaccination schedule eliminates the potential for VAPP in vaccine recipients. Virology laboratories are no longer expected to routinely isolate OPV-derived polioviruses from

clinical specimens and any poliovirus isolated within Australia should now indicate an importation event and requires complete investigation.

This report summarises the activities of the Australian National Poliovirus Reference Laboratory in 2007 and includes a summary of the laboratory testing of the wild poliovirus importation. A comparison of AFP surveillance in Australia against performance indicators established by WHO is also presented.

Methods

The current system of AFP surveillance used by the NPRL in collaboration with the APSU is as follows:

- Clinicians reviewing patients presenting with AFP are advised to notify the NPRL by telephone.
- In keeping with WHO guidelines, the AFP surveillance program requires that all AFP cases involving children less than 15 years of age be notified. However, the NPRL tests specimens from cases of suspected poliomyelitis involving patients of all ages. AFP cases in children aged less than 15 years are notified on monthly report cards/emails submitted by paediatricians to the APSU.
- 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 for testing.
- Reporting 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 involves a patient aged greater than 15 years, an overseas resident, 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
- A follow-up questionnaire is sent to notifying clinicians 60 days after the onset of paralysis, if the PEC requires more information regarding the AFP case, before a final classification can be made.
- 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/).
- At the end of each calendar year a small number of AFP notifications remain unclassified by the PEC as no clinical and laboratory data are available from the notifying clinician to enable a final classification by the committee.

Upon receipt at the NPRL, faecal specimens are treated with Minimum Essential Medium containing Earle'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. In keeping with WHO requirements, cell lines used for the isolation of poliovirus are L20B (a transgenic mouse epithelial cell line expressing the human poliovirus receptor, CD155)[6] and RD-A (human rhabdomyosarcoma). The NPRL utilises two additional cell lines for the isolation of poliovirus and non-polio enteroviruses: Hep2 (human epidermoid carcinoma) and HEL (human embryonic lung). Laboratories throughout Australia are encouraged to refer enteroviruses of unknown serotype to the NPRL for further characterisation as poliovirus can be clinically involved with non-paralytic conditions such as aseptic meningitis.

All polioviruses, whether isolated from AFP cases or other sources, undergo a process known as intratypic differentiation to distinguish between wild and vaccine strains of poliovirus. With the approval of WHO, the NPRL tests all polioviruses by diagnostic polymerase chain reaction (PCR) and sequencing of the VP1 genomic region. Current WHO PCR protocols allow the determination of poliovirus serotype (1, 2 or 3) and whether the poliovirus is Sabin-like.

Two regions of the poliovirus genome are routinely sequenced from all poliovirus isolations. The more important of these regions is the VP1 genomic region, which is the virus capsid encoding region containing a major antigenic determinant. One per cent or more change in this region compared to the prototype OPV strain is, by definition, a vaccine-derived poliovirus. The second region of interest is the 3D genomic region, which is sequenced in order to determine whether the virus has undergone a recombination event with another poliovirus serotype or non-polio enterovirus.

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

Results

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

A total of 53 cases of AFP with onset of symptoms in 2007, were notified to the APSU or the NPRL. Of these, 27 cases involved patients aged less than 15 years and were therefore considered eligible for reporting to the WHO. Thirteen cases were either ineligible according to WHO criteria as the patient was aged 15 years or over, or due to an error of notification, 5 duplicate notifications of AFP cases were received. Eight cases remain pending classification due to insufficient information.

Eligible acute flaccid paralysis cases

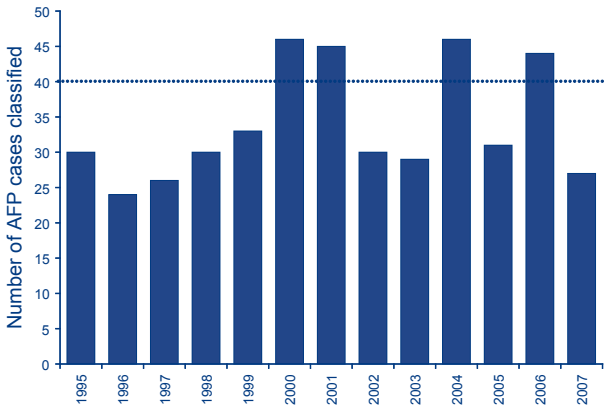
Sufficient data were available to classify 26 of the eligible cases as non-polio AFP. One case, with onset in June 2007, could not be discarded as non-polio AFP on the available information and was reported to WHO as polio compatible.

The annual rate for notification of AFP cases was 0.88 per 100,000 (35/40) children aged less than 15 years. The annual rate for cases classified as non-polio AFP by the PEC was 0.65 per 100,000 (26/40) children aged less than 15 years, in 2007 (Table 1). The classification of eligible AFP cases from 1995 to 2007 is presented in the Figure.

Ineligible cases

A total of 13 ineligible cases were notified with 8 cases considered ineligible due to the patients being older than 15 years of age. A further case involved the importation of wild poliovirus by a 22-year-old student from Pakistan.[7] Four notifications were later reported as non-AFP; 3 cases involved children less than 15 years of age and 1 case involved a 69-year-old returned traveller.

Figure. Classification of eligible acute flaccid paralysis cases from 1995 to 2007



World Health Organization acute flaccid paralysis surveillance performance indicator for Australia = 40 cases per year.

Notifications of acute flaccid paralysis by state and territory

In 2007, AFP cases were notified from all states and territories in Australia, with the exception of the Northern Territory (Table 2). The AFP notification rates for all states and territories exceeded the AFP surveillance performance indicator of 1 case per 100,000 children except for New South Wales and the Northern Territory (Table 2). The 3 most populous states, New South Wales, Queensland and Victoria, which account for more than 75% of

Table 1. AFP surveillance in Australia 2007, compared with WHO indicator targets for children less than 15 years

WHO indicator target for AFP cases of children less than 15 years*	Australia's surveillance for AFP cases with onset in 2007	Australia's AFP surveillance rates for 2007
Non-polio AFP case rate of 1.0 per 100,000 children (40 cases for Australia in 2007).	35 unique cases of AFP notified 26 cases classified by the PEC as non-polio AFP and one classified as polio compatible	AFP notification rate: 0.88/ 100,000 children. Non-polio AFP case rate: 0.65/ 100,000 children.
More than 80% of notified AFP cases with 2 adequate faecal specimens collected at least 24 hours apart, within 14 days of onset of paralysis.	14 AFP cases with 2 or more adequate specimens	Referral of adequate specimens from AFP cases: 52% (14/27) of the eligible cases.

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

AFP Acute flaccid paralysis.

WHO World Health Organization.

expected AFP cases, did not meet surveillance performance indicators based on final classification of cases by the PEC.

Faecal specimen collection from acute flaccid paralysis cases

A performance indicator set by WHO stipulates that adequate faecal specimens be collected from 80% of eligible AFP cases. The WHO defines adequate specimens for poliovirus culture, as 2 faecal specimens collected 24 to 48 hours apart and within 14 days of onset of symptoms.

Eligible acute flaccid paralysis cases

In 2007, faecal specimens from 18 of 27 eligible cases were tested at the NPRL:

- fourteen (52%) cases had adequate specimens;
- three (11%) cases had one specimen collected within 14 days of onset of symptoms;
- one (4%) case had 2 specimens collected after 14 days of onset of symptoms;
- no faecal specimens were received from the 9 (33%) remaining eligible cases.

The proportion of eligible cases with adequate faecal specimen collection was 52% (14/27) which represents the best result for this WHO criterion since AFP surveillance commenced in Australia. However, this result does not satisfy the faecal collection performance indicator set by WHO of 80% of eligible AFP cases.

Ineligible cases

Faecal specimens were referred to the NPRL from 8 of the 13 ineligible cases. Wild poliovirus was isolated from 2 faecal specimens, collected in Melbourne on days 15 and 17 post-onset of symptoms, from an imported case of poliomyelitis from Pakistan.[7] No enteroviruses were isolated from the faecal specimens of the remaining cases.

Laboratory testing of specimens

Acute flaccid paralysis cases

Between 1 January and 31 December 2007, a total of 68 specimens were referred from patients of all ages and nationalities with AFP.

Forty-seven specimens were received from 21 cases of AFP that involved Australian children less than 15 years of age, as per the WHO criterion for AFP surveillance. A non-polio enterovirus, identified as coxsackievirus A4 by sequencing of a portion of the VP1 genomic region, was isolated from 2 specimens of 1 case. Adenovirus was isolated from 1 specimen of 1 case and from 2 specimens of a further case of AFP. No enterovirus was isolated from the remaining 42 faecal specimens (Table 3).

A total of 9 faecal specimens were referred from 6 AFP cases involving Australian patients greater than 15 years of age. Two nasogastric aspirates and a rectal tube specimen were also received from one of the cases. Enterovirus was not isolated from any of the specimens.

Table 2. Unique notifications of eligible AFP cases with onset of symptoms between 1 January and 31 December 2007, by state or territory of residence

State or territory	Estimated population aged <15 years*	Expected number of cases/year	Total number of notifications	Eligible cases classified by PEC 1 January to 31 December 2007	Non-polio AFP rate per 100,000 population aged <15 years
ACT	62,430	0.5	2	1	2.00
NSW	1,309,104	13	13	5	0.39
NT	50,674	0.5	0	0	0.00
Qld	816,566	8	10	4	0.5
SA	283,763	3	4	2	0.67
Tas	96,318	1	3	2	2.07
Vic	961,410	10	17	9	0.9
WA	404,349	4	4	3	0.75
Australia	3,984,614	40	53	26	0.65

* Australian Bureau of Statistics, estimated resident population, preliminary – 30 June 2006. ABS publication 3201.0, December 2006.

AFP Acute flaccid paralysis.

Six faecal specimens, 1 cerebrospinal fluid (CSF) and a throat swab were received from the index case of the wild poliovirus importation. Wild poliovirus was isolated from the first 2 faecal specimens, while the remaining specimens were negative for virus isolation. Adenovirus was isolated from a faecal specimen received from a Papua New Guinea national referred by an Australian hospital; the WHO regarded this case as originating from Papua New Guinea due to the patient's nationality.

Table 3. Results from specimens referred to the Australian National Poliovirus Reference Laboratory from within Australia, 1 January to 31 December 2007

Result	Isolations from AFP cases*	Isolations from non-AFP referred samples†	Total
Poliovirus wild type 1	2	0	2
Poliovirus sabin-like	0	0	0
NPEV‡	2	30	32
Adenovirus	4	0	4
No virus isolated	60	51	111
Total	68	81	149

AFP Acute flaccid paralysis.

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

† Includes specimens from close contacts of the index case of the importation of wild poliovirus type 1 from Pakistan.

‡ NPEV: non-polio enterovirus. Molecular sequence results of NPEV from AFP and non-AFP sources identified coxsackieviruses A4, A16, coxsackievirus B1, echoviruses 3, 6, 7, 9 18, 20, 30 and enterovirus 71.

Sources other than acute flaccid paralysis

Fifty-one specimens were received from sources other than AFP in the reporting period (Table 3), and no enterovirus was isolated from any of the specimens.

Forty-four faecal specimens were from contacts of the polio importation case. Seven faecal specimens were received from 3 cases that were initially reported as AFP but later identified as notification errors.

Thirty untyped enterovirus isolates were received for serotype identification from a virology laboratory in Australia.

A summary of enterovirus testing at the NPRL for the period 1995 to 2007 can be found in Table 4.

Importation of wild poliovirus in Australia from Pakistan, July 2007

A 22-year-student from Pakistan who had been vaccinated with at least three doses of OPV as a child,

Table 4. Summary of enterovirus testing at the Australian National Poliovirus Reference Laboratory, 1995 to 2007

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

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

† Includes specimens from patients of all ages, relating to the importation of wild poliovirus type 1 from Pakistan.

returned to Pakistan in March 2007. Upon return to Australia in July, the patient attended the emergency department of a metropolitan Victorian Hospital.

A faecal specimen, throat swab and CSF were received by the Polio Reference Laboratory on Monday 9 July and prepared as described in the methods. The NPRL reported a poliovirus type 1 positive result on Friday 13 July, with confirmation of wild-type 1 serotype on 16 July. The Communicable Diseases Network Australia, the Australian Health Protection Committee and the Australian Government Department of Heath and Ageing were consulted throughout the confirmation process. The case was reviewed by the PEC and classified as poliomyelitis, wild-poliovirus infection, based upon clinical and laboratory evidence.

The NPRL sequenced the VP1 genomic region of the poliovirus isolate and performed phylogenetic analysis using cognate sequences. The Australian isolation showed a high sequence identity with type 1 wild poliovirus isolates from Pakistan in the year 2000. Poliovirus ELISA was also performed on 16 July with a result of non-Sabin-like poliovirus type 1. The wild poliovirus 1 isolate was referred to the WPR WHO Global Specialised Laboratory, National Institute of Infectious Diseases Japan, for sequence confirmation, according to WHO protocol.

A second faecal specimen was collected on 9 July and a poliovirus isolated from this second faecal specimen was positive by pan-enterovirus and pan-poliovirus reverse transcription (RT) PCR. The VP1 genomic region of the second wild poliovirus isolate was 100% identical to the isolate from the first faecal specimen.

The patient was isolated at the hospital until 2 consecutive faecal specimens, collected 7 days apart, were negative in cell culture.

Laboratory testing of faecal specimens from close contacts of the index case

The Department of Human Services investigated persons considered at risk of poliovirus infection through close contact with the index case. Two faecal specimens, collected 24 to 48 hours apart, were requested from each person for testing by the NPRL. All specimens were negative for enterovirus isolation by cell culture. Specimens were also referred to the VIDRL Viral Identification Laboratory for rapid screening and result confirmation by RT-PCR.

Overseas born health care workers who had possible contact with the index case upon admission to hospital and who could not provide evidence of recent polio immunisation were requested to provide 2 faecal specimens for testing. Specimens were received from 10 health care workers and all were negative for virus isolation by cell culture.

The index case remained the only positive isolation with no evidence of transmission of the wild poliovirus in Australia. WHO regarded the case as being from Pakistan, irrespective of the residency status of the patient, as the onset of illness occurred in that country. Therefore, Australia maintained its polio-free status. The Chief Medical Officer of Australia released a statement in September 2007, requesting all public health officials to maintain awareness for cases of AFP after the importation and issued a reminder for containment of wild poliovirus and potentially infectious material.

Polio serology

Acute and convalescent sera from the index case of the polio importation were tested for polio antibodies. No significant rise in titre was observed. The time between onset of symptoms in Pakistan and collection of acute-phase serum in Melbourne would significantly influence this result.

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

Regional reference laboratory activities

In addition to the Australian samples, 156 specimens and isolates were received from countries of the Western Pacific Region in 2007. The specimens referred for testing included 24 faecal specimens from 12 cases of AFP from Pacific Island countries and Brunei Darussalam, 85 specimens and isolates from Malaysia, 22 specimens and isolates from the Philippines and 23 specimens and isolates from Papua New Guinea from both AFP cases and non-AFP sources.

Two poliovirus isolates were received from a non-AFP case from Singapore in October 2007. The isolates were sequenced in parallel with the National Polio Reference Laboratory of Singapore, based at the Singapore General Hospital, to assist the laboratory with establishing poliovirus genome sequencing as a routine procedure.

Quality assurance program

The laboratory retained full accreditation status as a WHO Regional Reference Laboratory after an on-site review by WHO. In July 2006, the NPRL implemented a reduced period for cell culture incubation from 14 days to 10 days according to a

new WHO test algorithm. Other elements of the new test algorithm, already performed in the polio endemic countries, were implemented by the NPRL from September 2007.

The NPRL passed an annual PCR proficiency panel containing 10 samples from the Centers for Disease Control and Prevention in the United States of America.

An annual proficiency panel for poliovirus isolation and characterisation was passed as part of the accreditation procedure for a WHO National Polio Reference Laboratory. The proficiency panel was subsequently distributed to laboratories throughout the Western Pacific Region by the NPRL, as part of the terms of reference as a regional reference laboratory.

Discussion

The importation of wild poliovirus from Pakistan in July 2007 represented the first case of wild poliovirus infection in Australia for 30 years. The previous case in 1977 was also an importation, involving a child recently returned from Turkey.[8] While the focus of the WHO surveillance system for eradication of poliomyelitis is primarily on cases of AFP in children less than 15 years of age, the NPRL tests specimens from patients of any age with a clinical suspicion of poliomyelitis. This case highlights the need for continued vigilance for polio-like illness in persons of any age within Australia as described previously.[9]

The index case was a 22-year-old student and was notified as suspected poliomyelitis. There had been speculation as to whether a case of polio would be diagnosed in Australia[10] given the gaps in the AFP surveillance system that focuses on children aged less than 15 years and that a clinically confirmed case of polio had not been diagnosed in Australia since the 1970s. Maintaining high levels of polio vaccine coverage is crucial to Australia's status as polio-free and this may have contributed to the importation not extending beyond the index case.

From the experience of the wild poliovirus importation, the NPRL recognised the strengths and weaknesses of virus culture and nucleic acid tests performed on virus isolates and directly on specimens. The main concern is the length of time required for cell culture testing of clinical specimens. For example, the length of time required to obtain a negative cell culture result for the index case extended the period of isolation to 34 days. As a result the NPRL is investigating alternative procedures that will complement the methods currently recommended by WHO.

From 2001, the NPRL established routine nucleotide sequencing of all poliovirus isolates from AFP cases in Australia. The establishment of sequencing and in-house phylogenetic analysis combined with access to the WHO global polio laboratory network, enabled the rapid identification of wild poliovirus originating in Pakistan, providing an epidemiological link with the patient's travel history.

In 2007, Australia was unable to reach the WHO AFP surveillance performance indicator of 1 non-polio AFP case per 100,000 children less than 15 years of age. On previous occasions whenever Australia achieved the AFP surveillance performance indicator, at least 2 of the 3 most populous states, New South Wales, Queensland and Victoria, reached the performance indicator for AFP notifications. In 2007, the 3 most populous states each failed to attain the AFP surveillance performance indicator; this was only the second time that New South Wales had not achieved the performance indicator since 1997. Even if sufficient clinical information were available to enable a final case classification of the 8 pending cases, the total number would be 36 and the non-polio AFP rate would increase to 0.90, still below the performance indicator of 1.0 non-polio AFP case per 100,000 children less than 15 years of age.

Queensland is the only state in Australia to make AFP a notifiable condition and was unable to meet the AFP surveillance performance indicator in 2007. This is the third time that this has occurred in Queensland since AFP was declared a notifiable condition in that state in 2001.

While the late notification of AFP cases to the APSU or the NPRL is valuable data to satisfy the criterion for the annual notification rate of cases, the collection of faecal specimens within 14 days of the onset of symptoms requires a more immediate response by clinicians. Australia has never met the WHO criterion of testing 2 faecal specimens from 80% of eligible AFP cases, with 2007 producing the best result of 52%. A 'polio compatible' case was reported to WHO in 2007. The case was notified as AFP and insufficient clinical and laboratory data were available to discard the case as non-polio AFP. The case had onset of paralysis in June 2007 and was not related to the wild poliovirus importation.

The removal of OPV from the immunisation schedule in November 2005, means that poliovirus should no longer be isolated from clinical specimens in Australia. The isolation of either a wild or vaccine strain of poliovirus is significant as it would represent an importation into Australia. Any poliovirus isolation in Australia now needs to be fully investigated to determine the source of the virus and to ensure there is no person-to-person transmission.

The NPRL anticipates that other studies, such as an appropriate nation-wide enterovirus surveillance scheme, will need to be considered for Australia to be confident that it is polio-free prior to global certification of polio eradication. Such schemes are already in use in other developed nations, such as France, Japan, Germany, New Zealand and the United States of America. The feasibility of such a scheme in Australia is currently under investigation.

Acknowledgements

The Australian National Poliovirus Reference Laboratory is funded by the Australian Government Department of Health and Ageing and the Victorian Government Department of Human Services. We would like to thank Dr Andrew Stewardson, Dr Carolyn Beckett, Dr Poh-Sien Loh, Dr Hayden Prime, Dr John Daffy and the VIDRL Viral Identification laboratory for their assistance during the investigation of the polio importation. We also thank the Australian Paediatric Surveillance Unit for their ongoing collaboration with AFP surveillance and members of the Polio Expert Committee. We would also like to acknowledge the notifying clinicians and laboratories (national and international) for their assistance throughout 2007.

Author details

Mr Jason A Roberts, Medical Scientist
Ms Kristina A Grant, Medical Scientist
Mrs Aishah Ibrahim, Medical Scientist
Dr Bruce R Thorley, Head, Poliovirus Reference Laboratory

Corresponding author: Mr Jason Roberts, Victorian Infectious Diseases Reference Laboratory, Locked Bag 815, Carlton South, VIC 3053. Telephone: +61 3 9342 2607. Facsimile: +61 3 9342 2665. Email: Jason.Roberts@mh.org.au

References

1. World Heath Organisation. Global eradication of poliomyelitis by the year 2000 Resolution WHA41.28. Available from www.who.int/csr/ihr/polioresolution4128en.pdf
2. The global eradication of polio. Wild poliovirus. Case count by country. Available from: http://www.polioeradication.org/casecount.asp Accessed 18 June 2008.
3. Australian Government Department of Health and Ageing, National Health and Medical Research Council. *The Australian Immunisation Handbook*, 9th Edn. 2008:251–256.
4. Australian Government Department of Health and Ageing. Replacement of oral polio vaccine (OPV) with inactivated polio vaccine (IPV). Available from: http://www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/opvrep-qapub Accessed 2 August 2007.
5. Kew OM, Sutter RW, Nottay BK, McDonough MJ, Prevots DR, Quick L, et al. Prolonged replication of a type 1 vaccine-derived poliovirus in an immunodeficient patient. *J Clin Microbiol* 1998;36:2893–2899.
6. Wood DJ, Hull B. L20B cells simplify culture of polioviruses from clinical samples. *J Med Virol* 1999;58:188–182.
7. Thorley B, Kelly H, Roberts J. Importation of wild poliovirus into Australia, July 2007. *Commun Dis Intell* 2007;31:299
8. Kennett ML, Brussen KA, Wood DJ, van der Avoort HG, Ras A, et al. Australia's last case of wild poliovirus infection. *Commun Dis Intell* 1999;23:76.
9. Thorley BR, Brussen KA, Elliott EJ, Kelly HA. Vigilance is required for Australia to remain polio free. *Med J Aust* 2006;184:474–475.
10. Durrheim DN, Massey IP, Kelly H. Re-emerging poliomyelitis – is Australia's surveillance adequate? *Commun Dis Intell* 2006;30:275–277.

Articles

Trends in invasive *Haemophilus influenzae* type b disease in Australia, 1995–2005

Han Wang, Shelley Deeks, Amy Glasswell, Peter McIntyre

Abstract

The epidemiology of invasive *Haemophilus influenzae* type b (Hib) disease and its prevention by vaccination is reviewed for the period 1995 to 2005, comparing surveillance data for 1995–2000, when both PRP-OMP and HbOC vaccines were used, with 2000–2005, when only PRP-OMP vaccine was used. Over the whole time period, notifications of invasive Hib disease have declined dramatically. In the second time period, a greater decline in Hib cases was seen. This could be due to either the different vaccines being used, differences in vaccine coverage or both. Although disease incidence has decreased markedly in both Indigenous and non-Indigenous populations, Indigenous people are still at significantly greater risk. It is also concerning that almost 60% of invasive Hib cases in children are preventable, in that they are occurring in unimmunised or incompletely immunised children among whom the incidence of Hib disease is estimated to be about 15 times that of fully immunised children. Australia is now in the third era of Hib vaccine use, during which both PRP-T and PRP-OMP vaccines are used, depending on ethnicity or jurisdiction of residence. Continued enhanced surveillance for invasive Hib disease is important for optimal monitoring of trends into the future. *Commun Dis Intell* 2008;32:316–325.

Keywords: disease surveillance, disease control, *Haemophilus influenzae* type b, immunisation, vaccination, Indigenous

Introduction

Invasive *Haemophilus influenzae* type b disease in Australia

Haemophilus influenzae type b (Hib) is a bacterium that causes a broad spectrum of illnesses ranging from local respiratory infection to serious invasive disease, including meningitis, epiglottitis, septic arthritis and septicemia.[1] Among the six encapsulated strains that have been identified (designated as types a to f), Hib is the most virulent.[1–3] Non-encapsulated strains, classified as non-typeable, have also been identified and are usually associated with non-invasive infections, however they are capable of causing invasive disease, including neonatal sepsis.[2–4] Invasive Hib disease has been notifiable in Australia in most jurisdictions since 1990 and in all by 1993; non-type b invasive *H. influenzae* is reportable only in South Australia and the Northern Territory.

Hib is predominantly a disease of childhood with over 80% of cases worldwide occurring in children aged less than 5 years.[5] Prior to the introduction of immunisation, Hib was the most common cause of bacterial meningitis in Australian children.[6,7] However, since the introduction of Hib capsular polysaccharide-protein conjugate vaccines, the incidence of invasive Hib disease has declined dramatically.[8] Indigenous children are known to be at increased risk,[9–13] and have remained at increased risk despite vaccination programs.[14] Non-Indigenous children in central Australia also had a relatively high incidence in the pre-vaccine era.[15]

The first comprehensive review of the impact of Hib vaccines in Australia between July 1993 and June 2000 reported that in the four years 1996–97 to 1999–00, the average annual incidence of invasive Hib disease in children aged less than five years was 1.7 cases per 100,000 population,[14] a reduction of 87%–95%. Although the reduction was most marked in the target population, reduced incidence was seen in older age groups not eligible for immunisation, consistent with a herd immunity effect.[14] In July 2000, the recommended Hib vaccine for non-Indigenous children changed from one conjugated to mutant diphtheria toxin (HbOC) to one conjugated to the outer membrane protein of *Neisseria meningitidis* (PRP-OMP). Universal use of PRP-OMP ceased at the end of 2005, with some jurisdictions continuing with PRP-OMP and others adopting combination vaccines with the Hib component conjugated to tetanus protein (PRP-T). This article documents invasive Hib disease and vaccine coverage during the period January 1995 to December 2005, with particular emphasis on the period of universal PRP-OMP use from 2000 to 2005.

Haemophilus influenzae type b vaccine in Australia

A number of conjugated Hib vaccines have been used in Australia (Table 1). Vaccines using the polyribosylribitol phosphate (PRP) polysaccharide of the Hib capsule were first developed in the 1970s

Table 1. Conjugated *Haemophilus influenzae* type b vaccines previously and currently registered for use in Australia

Generic name	Trade name	Hib antigen	Conjugating protein	Currently available
PRP-D	ProHIBit*	Hib capsular polysaccharide	Diphtheria toxoid protein	No
PRP-T	ActHib*	Hib capsular polysaccharide	Tetanus toxoid protein	No
HbOC	HibTITER†	Hib capsular oligosaccharide	Mutant diphtheria toxoid protein (CRM 197)	No
PRP-OMP	Liquid PedvaxHIB‡	Hib capsular polysaccharide	Outer membrane protein of group B meningococcus	Yes
Hib (PRP-OMP)-hepatitis B	COMVAX‡	Hib capsular polysaccharide	Outer membrane protein of group B meningococcus, hepatitis B surface antigen	Yes
PRP-T	Infanrix hexa,§ Hiberix§	Hib capsular polysaccharide	Tetanus toxoid protein	Yes

* Manufactured by Aventis Pasteur.

† Manufactured by Wyeth.

‡ Manufactured by CSL Biotherapies/Merck & Co Inc.

§ Manufactured by GlaxoSmithKline.

but these vaccines produced a T-cell independent immune response that was not effective in protecting aged children less than 18 months.[16] Linking the PRP polysaccharide to a protein (i.e. conjugation) enhanced the immunogenicity of the vaccine by enabling T-cell stimulation. Conjugate Hib vaccines were first introduced in Australia in May 1992 for children over 18 months of age. Vaccines licensed for use in children aged more than 6 weeks became available in January 1993.[14]

There have been several key milestones in Hib immunisation practice in Australia since vaccine introduction, highlighted in Table 2. Between July 1993 and June 2000, a different Hib schedule was recommended for Indigenous and non-Indigenous children due to the increased risk of disease in Indigenous children and an earlier mean age of onset (Table 3). Serological evidence suggested that in young infants a single dose of the PRP-OMP vaccine elicited a better immune response than a single dose of the HbOC vaccine.[7,17] Therefore PRP-OMP vaccine (at 2 and 4 months and a booster dose at 12 months) was provided for all Indigenous children and all children in the Northern Territory, while HbOC at 2, 4 and 6 months of age with a booster at 18 months was provided for non-Indigenous children in other jurisdictions. In July 2000, the Hib vaccine provided for the funded program changed with PRP-OMP used for all infants commencing vaccination from this date. A final change occurred relatively recently. From November 2005 onwards, Queensland, Victoria, South Australia, the Northern Territory and Indigenous children in Western Australia continue to use PRP-OMP in a combined vaccine with Hepatitis B (PRP-OMP-

Table 2. Significant events in *Haemophilus influenzae* type b immunisation practice in Australia, 1992 to 2005

Year	Month	Event
1992	May	PRP-D (ProHIBit) approved for vaccination of infants aged at least 18 months.
1993	January	HbOC (HibTITER) and PRP-OMP (PedvaxHIB) marketed for use in children aged at least 2 months.
1993	April	PRP-T (ActHib) marketed for use in children aged at least 2 months.
1993	May	Reimbursement of vaccine cost for children born after February 1993.
1993	July	Fully funded national infant immunisation programme.
1993	August	Fully funded one dose catch up campaign for children aged less than 5 years.
2000	February	Combined Hib (PRP-OMP)-hepatitis B vaccine approved.
2000	May	PRP-OMP recommended for all infants (administered separately or in combination with hepatitis B vaccine).
2005	November	PRP-OMP recommended for all infants in Northern Territory, South Australia, Queensland, Victoria and Indigenous children in Western Australian
		PRP-T recommended for all infants in New South Wales, Australian Capital Territory, Tasmania and non-Indigenous children in Western Australian

HepB, COMVAX), in their funded programs. A PRP-T containing combination (Infanrix hexa) is being used for non-Indigenous children in Western Australia and all children in New South Wales, the Australian Capital Territory and Tasmania, using a 2, 4 and 6 month primary series with a booster dose at 12 months. Therefore, for non-Indigenous children in Australia there have been 3 vaccine eras (Table 3).

Methods

Study period

The two relevant time periods or vaccine eras covered by this article are between July 1993 and June 2000, when both HbOC vaccine and PRP-OMP were used depending on Indigenous status and residence in the Northern Territory (era 1) and July 2000 to November 2005 when PRP-OMP was used universally (era 2, see Table 3). To account for transition issues and to ensure relatively equal time periods, the two time periods examined for the purposes of this article are January 1995 to June 2000 and July 2000 to December 2005.

Data sources

Surveillance systems

Invasive Hib disease has been part of the National Notifiable Diseases Surveillance System (NNDSS) since its inception in 1990. The national invasive Hib disease case definition changed in 2004, during the period under surveillance.[18,19] As of 1 January 2004 only confirmed cases of invasive Hib disease were reported (Table 4). Information collected through the NNDSS includes date of diagnosis, age, gender, postcode, state or territory, and Indigenous status. Data on notified cases of invasive Hib disease were obtained from the NNDSS as of March 2007.

The Hib Case Surveillance Scheme (HCSS) is an enhanced surveillance system designed to collect supplementary information not routinely available from the NNDSS on cases of laboratory-confirmed invasive Hib disease. The HCSS was established in January 1994 with data collected retrospectively to 1 July 1993. Designated state and territory health department staff complete an enhanced surveillance form for each case of invasive Hib disease. Data on cases with an onset during the period under surveillance were obtained from the HCSS as of January 2007. Data collected include detailed information on immunisation status and disease, including some information on past medical history of the case, outcome of the case and laboratory confirmation method. Although immunisation status is a data field in NNDSS, it is rarely completed and therefore the enhanced system is needed for reliable analysis of this variable.

Data from the Australian Childhood Immunisation Register (ACIR), which contains information on the vaccination status of all Australian children born since 1996 and registered with Medicare, was used to assess Hib coverage for the time period of interest. A second or third dose assumption was used when estimating coverage. If the second PRP-OMP or third HbOC was recorded as having been given, it was assumed that the preceding doses had also been given. If a child was recorded as having received either a second or third dose of Hib-containing vaccine the child was categorised as being fully immunised at 12 months of age. If a child was recorded as having received either a third or fourth dose of Hib-containing vaccine, the child was categorised as being fully immunised at 24 months of age.[14]

In this article Indigenous status consists of two categories, 'Indigenous' which measures whether a person is identified as being of Aboriginal or Torres

Table 3. Recommended *Haemophilus influenzae* type b for Indigenous and non-Indigenous Australian children, July 1993 to December 2005 – 'The 3 eras of *Haemophilus influenzae* type b vaccination in Australia'

Time period	*Haemophilus influenzae* type b recommended	
Era 1 July 1993–June 2000	PRP-OMP* All Indigenous children and non-Indigenous children in the Northern Territory	HbOC† Non-Indigenous children outside the Northern Territory
Era 2 July 2000–November 2005	PRP-OMP All children	PRP-OMP All children
Era 3 From November 2005	PRP-OMP All children in the Northern Territory, Queensland, Victoria, South Australia and Indigenous children in Western Australia	PRP-T‡ All children in New South Wales, the Australian Capital Territory, Tasmania and non-Indigenous children in Western Australia

* PRP-OMP (COMVAX, Liquid PedvaxHIB): 2 doses at 2 and 4 months, booster dose at 12 months.

† HbOC (HibTITER): 3 doses at 2, 4 and 6 months, booster dose at 18 months.

‡ PRP-T (Hiberix, Infanrix hexa): 3 doses at 2, 4 and 6 months, booster dose at 12 months.

Table 4. Case definition for notification of invasive *Haemophilus influenzae* type b disease to the National Notifiable Diseases Surveillance System

Time period	Case definition*
Prior to 2004[18]	a) A clinically compatible illness (meningitis, epiglottitis, cellulitis, septic arthritis, osteomyelitis, pneumonia, pericarditis or septicaemia) *and* either: • the isolation of *Haemophilus influenzae* type b (Hib) from blood *or* • detection of Hib antigen in a clinical case *or* • detection of Gram negative coccobacilli where the organism fails to grow in a clinical case or b) A confident diagnosis of epiglottitis by direct vision, laryngoscopy or x-ray.
January 2004 onwards[19]	a) Isolation of *Haemophilus influenzae* type b (Hib) from a normally sterile site where typing has been confirmed at an approved reference laboratory or b) Detection of Hib antigen in cerebrospinal fluid when other laboratory parameters are consistent with meningitis.

* Only confirmed cases should be notified from January 2004 onwards (by laboratory definitive evidence).

Strait Islander origin and a composite category, 'other' which includes those recorded as non-Indigenous and those listed as 'not stated/inadequately described'. The latter group will be described as 'non-Indigenous' throughout this report. Coverage by Indigenous status has been accurately recorded on the ACIR since 2002.[20]

Population denominators and death data

For the years 1991 to 2005 resident populations were derived from both the Australian Bureau of Statistics (ABS) and the Australian Institute of Health and Welfare (AIHW). Death data for cases were cross matched between NNDSS and HCSS databases.

Estimating per cent reduction between the two vaccine eras

The formula used for estimating per cent reduction between the two eras was:

Percentage reduction = ((R1–R2)/R1)x100 = (1–R2/R1)x100

where R1 and R2 are the January 1995–June 2000 and July 2000–December 2005 Hib incidence rate, respectively. The 95% confidence interval (CI) of reduction was calculated using the Taylor Series for Rate Ratio.[21]

Definitions of vaccine eligibility and vaccination status

A number of definitions were applied to identify eligibility for vaccination and vaccination status of cases (Table 5). Doses of vaccine given less than 15 days prior to disease onset were not considered to count towards the immunisation status of the case.

Results

National Notifiable Diseases Surveillance System

Between 1993 and 2005 a total of 1,046 invasive Hib cases were reported to the NNDSS (Table 6). The number of Hib notifications per quarter between is shown in Figure 1. For the 30-month pre-vaccination period between January 1991 and June 1993, the average number of overall Hib notifications to NNDSS per quarter was 120 (range 81–142). This can be compared with notifications during the two vaccine periods between January 1995 and June 2000, and July 2000 to December 2005, where the average reported notifications per quarter was 14 (range 3–33) and 6 (range 1–12), respectively. These figures reveal that since the introduction of vaccination in mid-1993, there has been a dramatic decrease in the number of invasive Hib cases, both in the overall population and among those eligible for vaccine.

Table 6 shows the rate of invasive Hib disease by Indigenous status and the rate ratio between Indigenous and non-Indigenous Australians. It can be seen that despite universal vaccination and large decreases in overall incidence in both populations, Indigenous people continue to remain at significantly higher risk of invasive Hib disease compared to non-Indigenous Australians, with rate ratios ranging from 2.7 in 1993–94 to 17.6 in 2002–03. These ratios exceeded unity for all time periods under surveillance.

There were 309 notifications of invasive Hib diseases with onset between January 1995 and June 2000 and 120 notifications between July 2000 and

Table 5. Definitions of vaccine eligibility and vaccination status

Eligible for vaccination	• Children born after 31st July 1988.
Eligible for routine infant immunisation	• Children born from 1 March 1993 onwards.
Fully immunised for age	• Two doses of PRP-OMP before the age of one year and > 14 days before disease onset among children 4 to 12 months of age (i.e. eligible to have completed the primary series) • Three doses of HbOC before the age of one year and > 14 days before disease onset among children 6 to 12 months of age (i.e. eligible to have completed the primary series) • Two doses of PRP-OMP before the age of one year AND a booster dose at 12 months of age and > 14 days before disease onset among children at least 12 months of age (i.e. eligible to have completed the primary series and booster) • Three doses of HbOC before the age of one year AND a booster dose at 12 months of age and > 14 days before disease onset among children at least 12 months of age (i.e. eligible to have completed the primary series and booster) • One dose of any Hib vaccine given at 15–59 months of age and > 14 days before disease onset • One dose of any Hib vaccine given at 12–14 months of age AND a booster dose given at least 2 months after and > 14 days before disease onset
Partially immunised for age	• One or more doses of any Hib vaccine but not fully immunised for age (see above). • Fully immunised for age but with disease onset within 14 days of receipt of last dose
Unimmunised	• No Hib immunisations • First dose of primary series Hib vaccine given within 14 days of disease onset

Table 6. Invasive *Haemophilus influenzae* type b disease incidence in Indigenous and non-Indigenous Australians and incidence rate ratios, 1993 to 2005

Date of diagnosis	Australia		Indigenous		Non-Indigenous		Indigenous: non-Indigenous	
	n	Rate per 100,000	n	Rate per 100,000	n	Rate per 100,000	Rate ratio	95%CI
Jan 93–Dec 94	617	1.74	35	4.48	582	1.68	2.67	(1.84, 3.76)
Jan 95–Dec 96	167	0.46	11	1.34	156	0.44	3.06	(1.50, 5.63)
Jan 97–Dec 98	92	0.25	11	1.29	81	0.22	5.77	(2.77, 10.89)
Jan 99–Jun 00	50	0.18	9	1.35	41	0.15	9.17	(3.92, 19.16)
Jul 00–Dec 01	38	0.13	4	0.59	34	0.12	4.87	(1.26, 13.66)
Jan 02–Dec 03	50	0.13	15	1.59	35	0.09	17.54	(8.90, 32.96)
Jan 04–Dec 05	32	0.08	5	0.51	27	0.07	7.48	(2.25, 19.70)

December 2005. Figure 2 compares the Hib incidence rates by jurisdiction during the two Hib vaccine eras described previously. Both Indigenous and non-Indigenous cases have been included in these calculations. Disease rates have decreased in all jurisdictions, with the Northern Territory continuing to have the highest incidence of Hib disease. The overall rate of disease has declined between the two time periods by 63.6% from 0.30 to 0.11 cases per year per 100,000 population. The decrease ranged from 23% in the Northern Territory to 100% in the Australian Capital Territory. The decline in invasive disease has been greatest among children under 5 years of age; from 2.12 to 0.55 per 100,000 in children aged 1 to 4 years, and from 5.32 to 1.52 cases per 100,000 in infants aged under 1 year (Figure 3).

Deaths

There were 16 deaths recorded in NNDSS due to invasive Hib disease between January 1995 and June 2000 with an overall case fatality rate (CFR) of 5.2%, and 7 deaths between July 2000 and December 2005 with a CFR of 5.8%. For the first time since surveillance began, no deaths from invasive Hib disease were reported in 2005. The CFR did not differ by Indigenous status.

When limiting the analysis to the vaccine-eligible population, 6 cases died between January 1995 and June 2000 with an overall CFR of 3.2% and 2 cases died between July 2000 and December 2005 with a CFR of 2.8%. Of note, both of the deaths in the

Figure 1. Number of *Haemophilus influenzae* type b notifications per quarter and publicly funded immunisation, Australia, 1992 to 2005

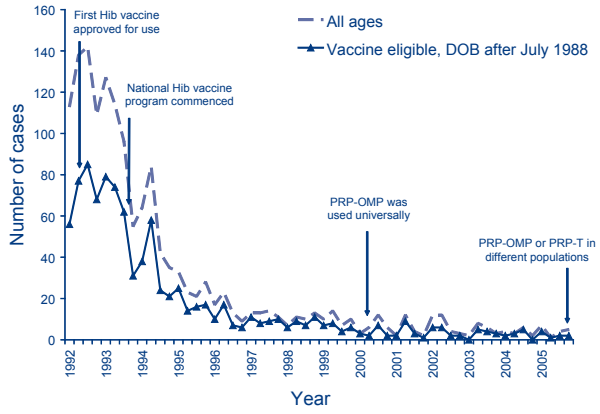


Figure 2. Incidence of invasive *Haemophilus influenzae* type b disease per 100,000 population, by state or territory and percentage reduction in illness between the two vaccines eras

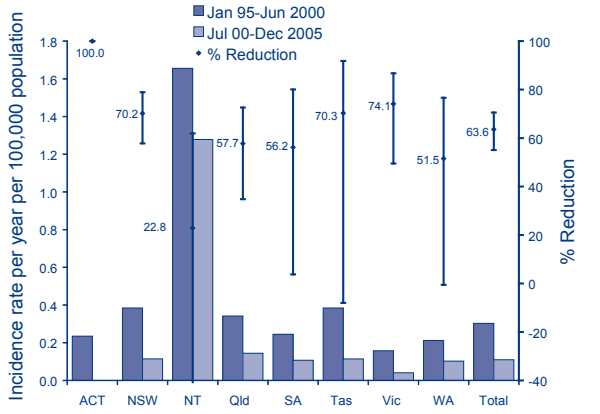
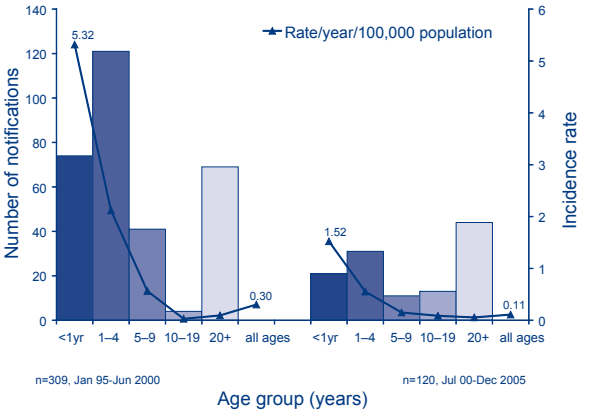


Figure 3. Number of *Haemophilus influenzae* type b cases, Australia, by age and age-specific incidence rates during the two vaccine eras

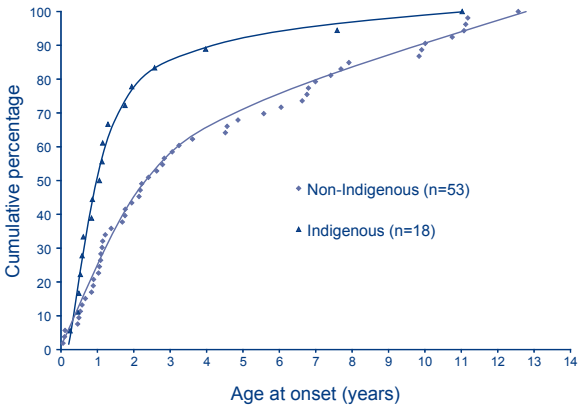


second time period were among infants with septicaemia aged under 12 months, one of whom was too young to receive vaccine, the other of whom had only received one dose of their primary series.

Haemophilus influenzae type b Case Surveillance Scheme

All Hib cases reported to HCSS with onset dates between July 1993 and December 2005 were examined by age to determine eligibility for vaccine according to birth date (definitions are outlined in Table 5). A total of 643 cases were identified in the HCSS; 491 were vaccine-eligible as they were born after July 1988. This included 60 Indigenous or Northern Territory residents for whom PRP-OMP was recommended in all time periods, with 20 having disease onset between July 2000 and December 2005 (i.e. era 2). Of the remaining 431, 163 had disease onset between January 1995 and June 2000 and 51 had disease onset between July 2000 and December 2005. Therefore during era 2 a total of 71 cases were reported. Indigenous children had a significantly earlier age of illness onset compared to non-Indigenous children (log-rank test p=0.02) (Figure 4). The median age of Indigenous cases was 14 months compared with a median age of 30 months among non-Indigenous cases (non-parametric test for median p=0.02).

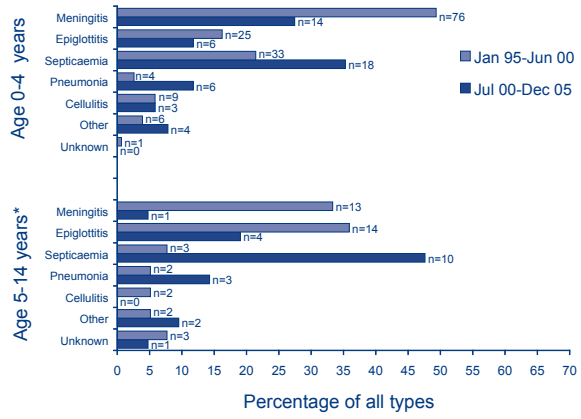
Figure 4. Age at onset of *Haemophilus influenzae* type b cases occurring between July 2000 to December 2005 among vaccine-eligible Indigenous and non-Indigenous children (n=71)



Clinical presentation of Hib disease varied with age and between the two time periods (Figure 5). While overall disease was less common during the second time period, the relative frequency of clinical presentations varied. Hib meningitis and epiglottitis among children under 5 years of age decreased between the two time periods by 81.6% and 76%,

respectively, whereas the proportion coded as septicaemia and pneumonia increased among older children.

Figure 5. Clinical presentation of *Haemophilus influenzae* type b cases, by age group and time period

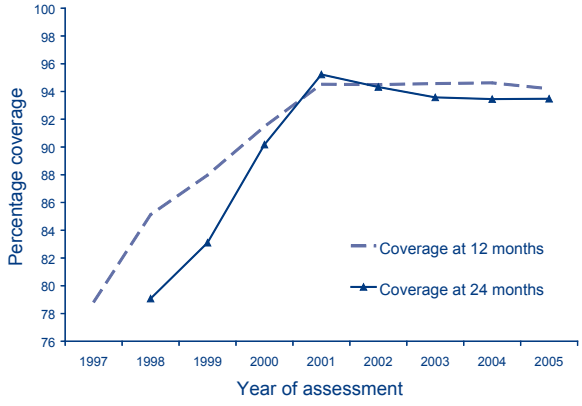


* Included children born before July 1988.

Estimating vaccine coverage

Since 2000, every state and territory in Australia has achieved Hib vaccine coverage rates above 90% for the primary series, as recorded by ACIR and assessed at 12 months of age (Figure 6). Vaccine coverage for the primary series and booster, assessed at 24 months of age is slightly lower, but above 90%. Since 2002, when ACIR Indigenous data quality has been acceptable, coverage in Indigenous children has been slightly lower than that of non-Indigenous children (by about 1%), but has also been above 90% for both the primary series and primary series with booster. Vaccine coverage varied minimally by state and territory. Given the high coverage, a large proportion of cases occurring among vaccinated individuals would be expected even with a highly effective vaccine.

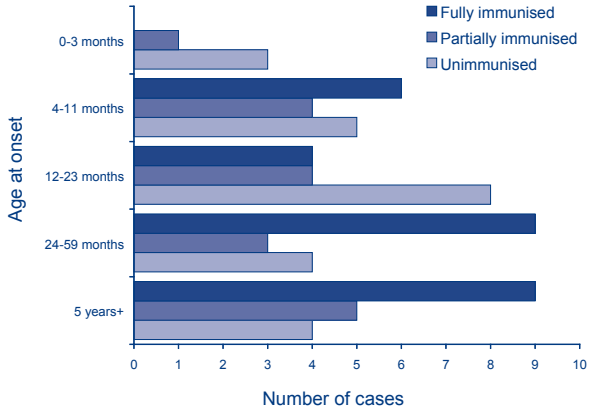
Figure 6. *Haemophilus influenzae* type b vaccine coverage at 12 and 24 months of age, Australia, 1997 to 2005



Preventable cases between July 2000 and December 2005

Of the 71 invasive Hib disease cases occurring among vaccine-eligible children during era 2 when PRP-OMP was used throughout Australia, 2 did not have completed information on vaccination status and 4 occurred in children too young to have completed the primary series (i.e. less than 4 months of age). The latter 4 were not preventable by vaccination (Figure 7). Out of the remaining 65 cases (Figure 8), 15 (23%) occurred in children 4–11 months of age (eligible for primary series) and 50 (77%) occurred in children 12 months of age and older (eligible for primary and booster). There was a total of 28 Hib cases that were fully immunised according to their age and were therefore vaccine failures.

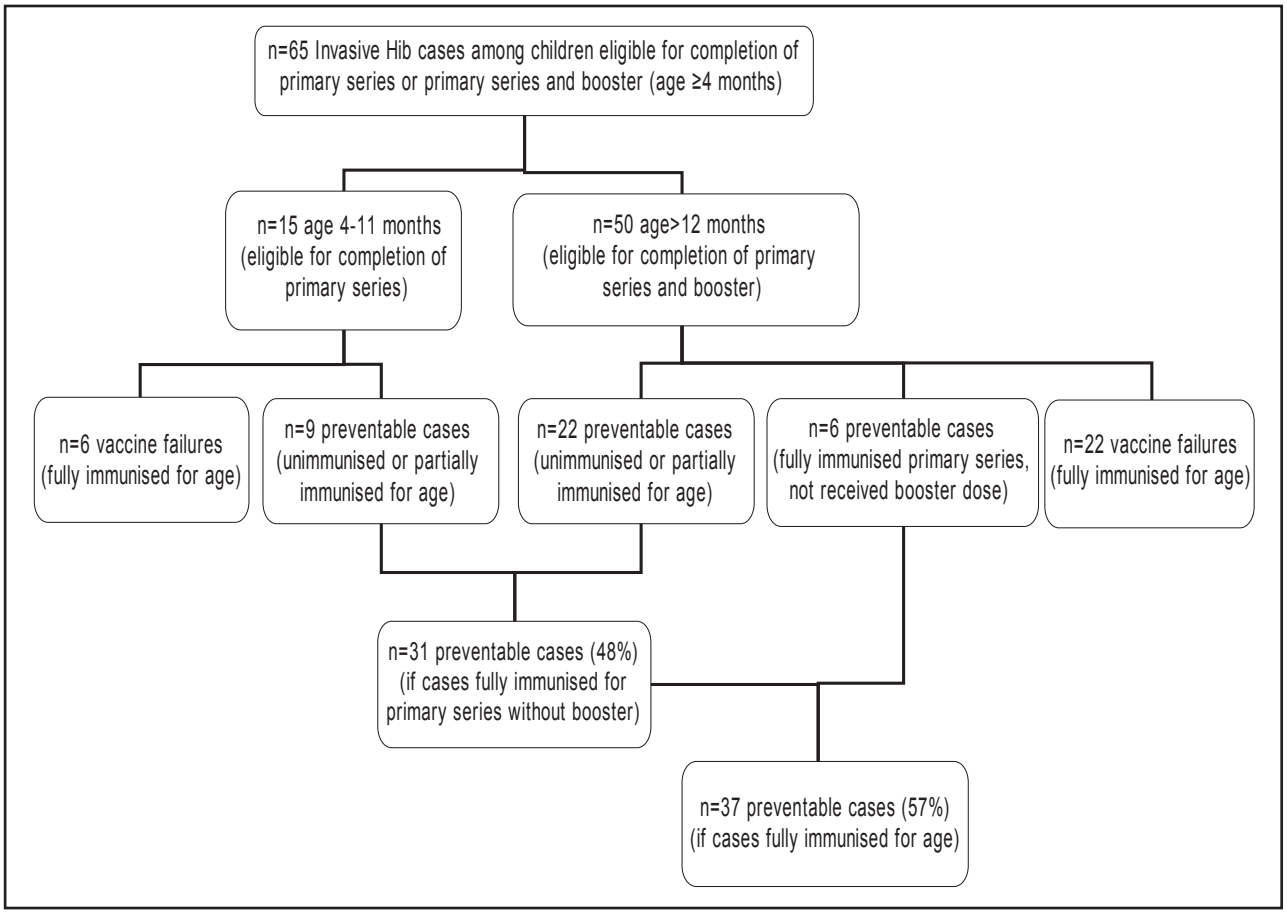
Figure 7. Vaccine status among vaccine-eligible *Haemophilus influenzae* type b cases (n=69*), July 2000 to December 2005, by age



* Two cases were removed as their vaccine status was unknown.

In children aged 4–11 months, 9 (60%) were either unimmunised or had only partially completed their primary series vaccination. Of the 50 cases in children aged 12 months or older, 28 (56%) were either unimmunised or had received only partial immunisation for age according to the definition in Table 5. Within this latter group, 6 cases were fully immunised for the primary series but had not received their booster dose. Therefore, 37 of 65 cases (57%) might have been prevented had they been fully immunised for age. The proportion of

Figure 8. Preventable cases of invasive *Haemophilus influenzae* type b disease in vaccine eligible children, July 2000 to December 2005



preventable cases would decrease to 48% if primary series alone (i.e. without booster) was considered. The proportion of cases identified as preventable, did not differ significantly between Indigenous and non-Indigenous Australians (59% versus 56%, respectively).

The data were further analysed to estimate the incidence of disease in children who had been fully immunised for age versus those who were not, including cases with disease onset between July 2000 and December 2005 who were older than 12 months (Figure 7). The denominators were obtained by selecting from the ACIR a cohort of children born between July 1995 and December 2004. The 'fully immunised' denominator was calculated using the ACIR criteria for fully immunised at 24 months, as the Hib booster is due at 12 months of age. The estimated incidence of invasive Hib disease in children aged over 12 months who were fully immunised for age was 0.9 per 100,000 (22/2,343,878), compared with an estimated incidence of 14.0 per 100,000 (28/199,697) in children who were not fully immunised.

Discussion

Immunisation with Hib vaccine has resulted in a dramatic and sustained reduction in the incidence of invasive Hib disease in Australia. The data demonstrate that the rate of the decline in disease was significantly greater between 2000 and 2005, in all jurisdictions excluding the Northern Territory, when the entire country was using the PRP-OMP vaccine, than during the earlier time period when HbOC was the primary vaccine in use. However it is important to note that some people with disease onset between 2000 and 2005 may have been immunised with HbOC if they were born when this vaccine was being used in their jurisdiction of residence, but their disease onset was during the latter time period. The increased rate of disease decline in the most recent 5-year period may therefore be related to differences in the vaccine, increasing vaccine coverage (though under-reporting to the ACIR would have to be taken into account especially prior to 2000) or a combination of factors.

Although disease incidence has decreased markedly in both Indigenous and non-Indigenous

populations, the continued disparity in incidence is concerning. This disparity is not unique to Australia, having been observed among Indigenous populations in both the United States of America and Canada.[11,22,23] Studies in Alaskan populations suggest that continued low-level nasopharyngeal colonisation facilitates transmission to susceptible children.[24] Environmental and housing conditions, including overcrowding, are also potential contributing factors to these health disparities.[25,26]

Higher rates of invasive Hib disease among Indigenous children have continued throughout the time period under surveillance despite high vaccine coverage among both Indigenous and non-Indigenous children in the latter vaccine era. ACIR data have shown that infant vaccination is more frequently delayed in Indigenous children, stressing the importance of continual emphasis on timely receipt of immunisation.[27,28] Given the earlier onset of disease among Indigenous people, it is appropriate to continue the use of PRP-OMP in jurisdictions where there are a large proportion of Indigenous people.

It is concerning that almost 60% of invasive Hib cases are preventable, in that they are occurring among people who are either not immunised or not fully immunised for age. These children remain at risk for serious invasive disease, despite herd immunity effects. Until all children are immunised with Hib vaccine in a timely manner, preventable cases will continue. No vaccine is 100% effective and vaccine failures are expected. However, the total number of true vaccine failures among infants continues to be small. Despite some protection from herd immunity, the rate of disease among children who were not fully vaccinated for age was about 15 times higher than in fully immunised children, a potentially powerful incentive for continued Hib immunisation despite Hib disease having become rare.

A recent publication from Scheifele and colleagues has suggested that children with Hib vaccine failure are more likely to be immunosuppressed.[13] Of the 28 Hib cases occurring among fully immunised children, 2 (7%) were known to have had immunocompromising conditions and 17 (61%) were reported to have no underlying illness. However, detailed information was not available on all children in order to assess this systematically.

Australia is now in the third era of Hib vaccine, during which both PRP-T and PRP-OMP are being used, depending on ethnicity or jurisdiction of residence. Continued surveillance will allow monitoring of the impact of this change but the small number of cases now occurring mean that any change, should it occur, will take many years to detect. The data presented in this report suggest that the regional changes in recommended Hib vaccine are appropriate given current invasive Hib disease epidemiology in Australia. One needs to balance the benefits of improved compliance associated with giving multiple antigens in combination vaccines that use PRP-T with the benefits of earlier protection with PRP-OMP and this will vary depending on the setting.

Acknowledgements

We thank the state and territory Hib enhanced surveillance contacts: Anne Murphy from the Department of Human Services in Melbourne, Caroline Giele from the Health Department of Western Australia, Craig Davis from the Communicable Diseases Unit of Queensland Health, David Coleman from the Department of Community and Health Service in Tasmania, Lesley Scott from the Department of Health and Community Services in the Northern Territory, Robin Gilmour from the Communicable Diseases Branch of the New South Wales Health Department and Ros Holland from the Department of Human Services Communicable Disease Branch in Adelaide.

We also wish to acknowledge Brynley Hull for providing Australian Childhood Immunisation Register data and the data contributed by laboratories throughout Australia and the following organisations for provision of additional data for this article: the Surveillance Policy and Systems Section of the Office of Health Protection, Australian Government Department of Health and Ageing, for data from the National Notifiable Diseases Surveillance System; and the Australian Institute of Health and Welfare, Hospitals and Mental Health Services Unit and Population Health Unit, for data from the National Hospital Morbidity and Mortality Databases.

Author details

Ms Han Wang,[1] Statistician
Dr Shelley L Deeks,[1] Deputy Director
Dr Amy D Glasswell,[1] Research Fellow
Professor Peter B McIntyre,[1,2] Director

1. National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, Children's Hospital at Westmead, Westmead, New South Wales
2. Staff Specialist in Infectious Diseases, Royal Alexandra Hospital for Children, Sydney, New South Wales

Corresponding author: Ms Shelley Deeks, NCIRS, Children's Hospital at Westmead, Locked Bag 4001, WESTMEAD NSW 2145. Telephone: +61 2 9845 1440. Facsimile: +61 2 9845 1418. Email: ShelleD2@chw.edu.au

References

1. Peltola H. *H. influenzae* in the post-vaccination era. *Lancet* 1993;341:864–865.
2. Gilsdorf JR. *Haemophilus influenzae* non-type b infections in children. *Am J Dis Child* 1987;141:1063–1065.

3. Heath PT, Booy R, Azzopardi HJ, Slack MP, Fogarty J, Moloney AC, et al. Non-type b *Haemophilus influenzae* disease: clinical and epidemiologic characteristics in the *Haemophilus influenzae* type b vaccine era. *Pediatr Infect Dis J* 2001;20:300–305.
4. Brook I, Gillmore JD. Expression of capsules by *Haemophilus influenzae* in mixed infections. *J Infect* 1995;30:219–222.
5. Peltola H. Worldwide *Haemophilus influenzae* type b disease at the beginning of the 21st century: global analysis of the disease burden 25 years after the use of the polysaccharide vaccine and a decade after the advent of conjugates. *Clin Microbiol Rev* 2000;13:302–317.
6. Hanna JN, Wild BE. Bacterial meningitis in children under five years of age in Western Australia. *Med J Aust* 1991;155:160–164.
7. McIntyre PB, Leeder SR, Irwig LM. Invasive *Haemophilus influenzae* type b disease in Sydney children 1985–1987: a population-based study. *Med J Aust* 1991;154:832–837.
8. Brotherton J, Wang H, Schaffer A, Quinn H, Menzies R, et al. Vaccine preventable diseases and vaccination coverage in Australia, 2003 to 2005. *Commun Dis Intell* 2007;31 Suppl:viii–S152.
9. Bisgard KM, Kao A, Leake J, Strebel PM, Perkins BA, Wharton M. *Haemophilus influenzae* invasive disease in the United States, 1994–1995: near disappearance of a vaccine-preventable childhood disease. *Emerg Infect Dis* 1998;4:229–237.
10. Hammitt LL, Block S, Hennessy TW, DeByle C, Peters H, Parkinson A, et al. Outbreak of invasive *Haemophilus influenzae* serotype a disease. *Pediatr Infect Dis J* 2005;24:453–456.
11. Millar EV, O'Brien KL, Levine OS, Kvamme S, Reid R, Santosham M. Toward elimination of *Haemophilus influenzae* type B carriage and disease among high-risk American Indian children. *Am J Public Health* 2000;90:1550–1554.
12. Millar EV, O'Brien KL, Watt JP, Lingappa J, Pallipamu R, Rosenstein N, et al. Epidemiology of invasive *Haemophilus influenzae* type A disease among Navajo and White Mountain Apache children, 1988–2003. *Clin Infect Dis* 2005;40:823–830.
13. Scheifele D, Halperin S, Law B, King A, Halperin S, Morris R, et al. Invasive *Haemophilus influenzae* type b infections in vaccinated and unvaccinated children in Canada, 2001–2003. *CMAJ* 2005;172:53–56.
14. Horby P, Gilmour R, Wang H, McIntyre P. Progress towards eliminating Hib in Australia: an evaluation of *Haemophilus influenzae* type b prevention in Australia, 1 July 1993 to 30 June 2000. *Commun Dis Intell* 2003;27:324–341.
15. Hanna JN. The epidemiology of invasive *Haemophilus influenzae* infections in children under five years of age in the Northern Territory: a three-year study. *Med J Aust* 1990;152:234–6, 238, 240.
16. Peltola H, Käyhty H, Sivonen A, Mäkelä H. *Haemophilus influenzae* type b capsular polysaccharide vaccine in children: a double-blind field study of 100,000 vaccinees 3 months to 5 years of age in Finland. *Pediatrics* 1977;60:730–737.
17. National Health and Medical Research Council. *The Australian Immunisation Handbook*. 8th edn. Canberra: Australian Government Department of Health and Ageing; 2003.
18. Public Health Committee, National Health and Medical Research Council. Surveillance case definitions. Canberra: Australian Government Printing Service; 1994.
19. Communicable Diseases Network Australia. Interim surveillance case definitions for the Australian National Notifiable Diseases Surveillance System. Version 1, 1 January 2004. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda_surveil-nndss-dislist.htm/$FILE/casedef.pdf Accessed March 2008.
20. Rank C, Menzies RI. How reliable are Australian Childhood Immunisation Register coverage estimates for indigenous children? An assessment of data quality and coverage. *Commun Dis Intell* 2007;31:283–287.
21. Sahai H, Khurshid A. *Statistics in Epidemiology: Methods, Techniques, and Applications*. 1st edn. CRC-Press; 1996.
22. Degani N, Navarro C, Deeks SL, Lovgren M. Invasive bacterial diseases in northern Canada. *Emerg Infect Dis* 2008;14:34–40.
23. Galil K, Singleton R, Levine OS, Fitzgerald MA, Bulkow L, Getty M, et al. Reemergence of invasive *Haemophilus influenzae* type b disease in a well-vaccinated population in remote Alaska. *J Infect Dis* 1999;179:101–106.
24. Singleton R, Hammitt L, Hennessy T, Bulkow L, DeByle C, Parkinson A, et al. The Alaska *Haemophilus influenzae* type b experience: lessons in controlling a vaccine-preventable disease. *Pediatrics* 2006;118:e421–e429.
25. Banerji A, Bell A, Mills EL, McDonald J, Subbarao K, Stark G, et al. Lower respiratory tract infections in Inuit infants on Baffin Island. *CMAJ* 2001;164:1847–1850.
26. Kovesi T, Creery D, Gilbert NL, Dales R, Fugler D, Thompson B, et al. Indoor air quality risk factors for severe lower respiratory tract infections in Inuit infants in Baffin Region, Nunavut: a pilot study. *Indoor Air* 2006;16:266–275.
27. Kolos V, Menzies R, McIntyre P. Higher pertussis hospitalization rates in indigenous Australian infants, and delayed vaccination. *Vaccine* 2007;25:588–590.
28. Menzies R, McIntyre P, Beard F. Vaccine preventable diseases and vaccination coverage in Aboriginal and Torres Strait Islander people, Australia, 1999 to 2002. *Commun Dis Intell* 2004;28:127–159.

An evaluation of the Australian Rotavirus Surveillance Program

April R Roberts-Witteveen, Mahomed S Patel, Paul W Roche

Abstract

The Australian Rotavirus Serotyping Program (ARSP) serotypes rotavirus isolates obtained from stool samples sent from Australian laboratories. In collaboration with ARSP, the Australian Government Department of Health and Ageing evaluated the program for its utility and capacity to monitor effectiveness of the rotavirus vaccines recently introduced into the Australian National Immunisation Program. The system was described using ARSP annual reports and staff interviews. The attributes of the system were assessed by adapting standard guidelines for evaluating a surveillance system. Email surveys or face to face interviews were conducted with staff of ARSP, participating laboratories, rotavirus vaccine manufacturing companies and representatives of the Communicable Diseases Network Australia. The ability of the ARSP to monitor changes in rotavirus serotype epidemiology was assessed. ARSP serotypes rotavirus isolates received from participating laboratories at least bi-annually, with results being reported at least as often. Serotype analyses have informed formulation of rotavirus vaccines and contributed to forecasting the extent of outbreaks caused by novel serotypes. The ARSP will be able to monitor changes in rotavirus serotype epidemiology and identify probable vaccination failures. Enhancement of the representativeness and sensitivity of the system are needed for the data to remain useful in the public health context. Methods for transferring data between the program and state and territory health departments need to be developed. *Commun Dis Intell* 2008;32:326–332.

Keywords: evaluation, rotavirus, vaccination

Introduction

Rotavirus is the most common cause of hospitalisations of children with diarrhoea worldwide.[1] The majority of infections occur in children under 5 years.[2] In both developing countries and developed countries rotavirus incidence is high in infants.[3,4] Worldwide, human infections are most often caused by Group A rotaviruses, which consist of a genome encased by three protein layers. The outer capsid layer is made of the VP7 protein which contains VP4 protein 'spikes'. The VP7 glycoprotein and VP4 protease-sensitive protein carry the G serotype and P serotype specific antigens respectively. The middle capsid layer, the VP6 protein, expresses an antigen which determines the group and subgroup of the virus.[5] The Australian Rotavirus Surveillance Program (ARSP) performs serotyping of VP4 and VP7 proteins of rotavirus isolates sent from laboratories in several Australian states and territories. Serotyping rotavirus isolates is important to monitor the emergence of new rotavirus serotypes.

In Australia in 2006, rotavirus infections were estimated to cause approximately 10,000 hospitalisations annually at an estimated cost of $19 million. An additional 22,000 emergency department (ED) visits and 150,000 general practitioner (GP) visits were attributed to acute rotavirus gastroenteritis, costing over $11 million.[6] Rotavirus vaccines, Rotarix and Rotateq, were licensed for use in Australia in 2006. From 1 July 2007, all children will receive either Rotarix or RotaTeq vaccination as part of the national childhood vaccination schedule. The vaccines have been designed to provide protection against severe diarrhoea caused by serotypes G1, G2, G3 and G4. These serotypes cause at least 90% of infections worldwide.[5] Rotarix (developed by GlaxoSmithKline) is based on a live attenuated monovalent virus (serotype P1A[8]G1). It has an overall clinical efficacy of 95.8% (95% C.I. 89.6–98.7) against severe rotavirus disease caused by serotypes P[8]G1, P[4]G2, P[8]G3, P[8]G4 and P[8]G9. RotaTeq (developed by Merck) is a live pentavalent bovine-human reassortant strain containing G1, G2, G3, G4 and P1A antigens.[7] Trials in 11 countries including the United States of America, Finland and South American countries[1] demonstrated a clinical efficacy of 98.2% (95% C.I. 89.6–100) against severe rotavirus disease caused by serotypes G1, G2, G3, G4 and G9. Both Rotarix and Rotateq are administered orally and unlike the earlier vaccine RotaShield, neither were associated with increased risk of intussusception in phase 3 trials.[7,8]

The ARSP and the Australian Government Department of Health and Ageing agreed to a collaborative evaluation of the ARSP to describe the surveillance system, to assess its attributes and to determine if the ARSP provides surveillance data appropriate for the vaccine era. This paper reports key findings of the evaluation.

Methods

Face-to-face interviews with key ARSP staff were used to gather information about serotyping

rotavirus isolates and reporting. A flow chart was constructed to describe how samples are received by the ARSP, how stool samples are serotyped in the ARSP, and how data are managed. This was verified as accurate by the laboratory director (Figure).

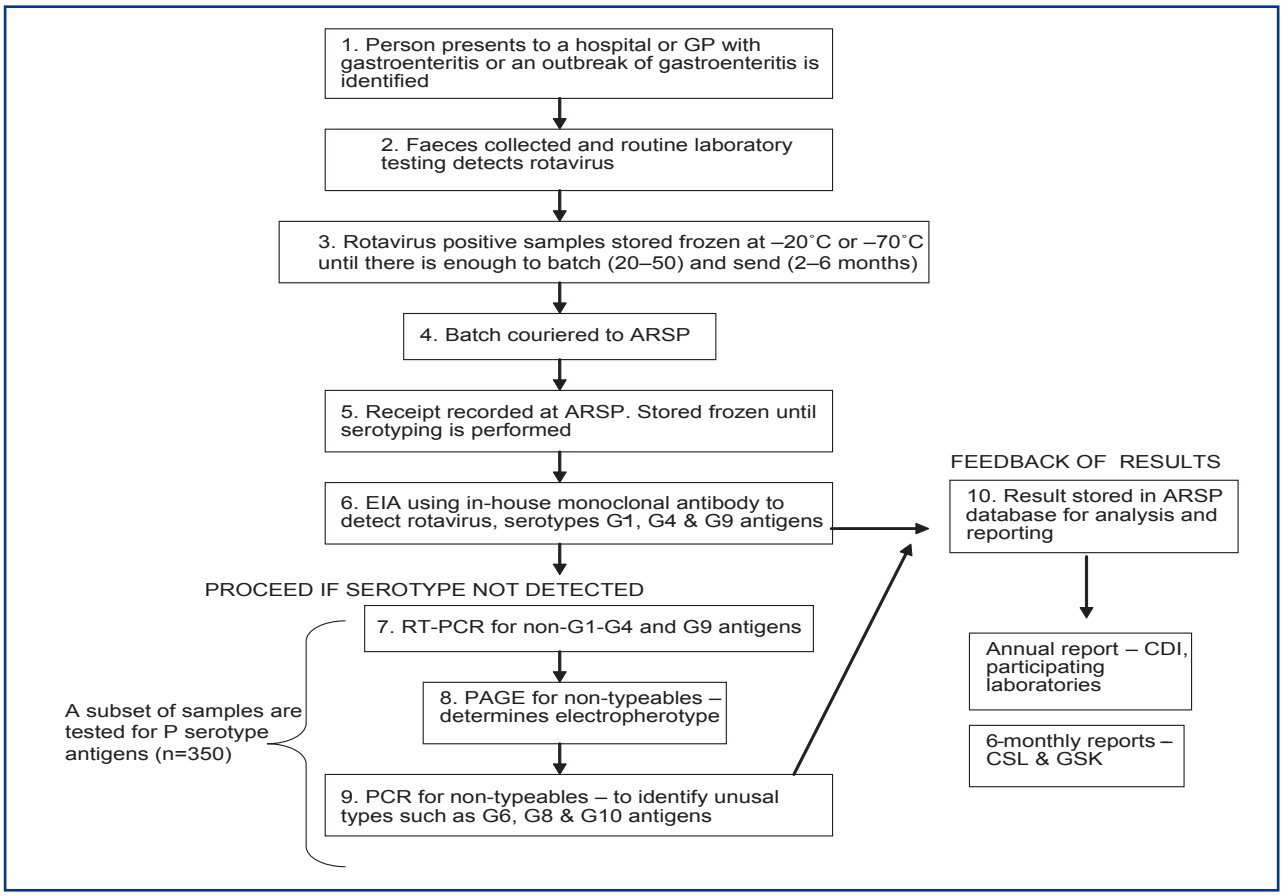
The assessment of the system attributes was adapted from the guidelines for the evaluation of the surveillance systems produced by the US Centers for Disease Control and Prevention.[9] Eleven attributes of the system were assessed. In this paper, the flexibility, sensitivity, representativeness, timeliness and usefulness of the ARSP will be reported, because of their relevance to the current situation of introduction of the rotavirus vaccines being introduced.

The 5 attributes of the ARSP system were defined as follows.

- Flexibility: the ability of the system to adapt to changing operating conditions and information and policy needs.
- Sensitivity: assessed for 3 different aspects of the ARSP:
  - the proportion of gastroenteritis stool samples that contained rotavirus;
  - the proportion of all rotavirus positive stool samples collected by each participating laboratory and which are sent to the ARSP for serotyping;
  - the ability of the ARSP to detect outbreaks, new or unusual strains, and infections acquired overseas.

  (Note: the ability of the ARSP to detect all rotavirus infections was not assessed as it was not a goal of the system.)
- Representativeness: how representative the isolates serotyped by the ARSP were of isolates from across Australia in terms of the age and location of the case from which the isolate was obtained and by comparing ARSP data to the:
  - published estimates of the number of cases of rotavirus occurring in Australia from hospitalisation data; and
  - number and age of rotavirus cases notified in the Northern Territory each year.
- Timeliness: the ability of the ARSP to produce results and reports in a manner which was judged as timely by stakeholders.
- Usefulness: the contribution of the ARSP to the prevention and control of rotavirus in Australia.

Figure. Flow chart of the system used by the Australian Rotavirus Surveillance Program to serotype rotavirus isolates and report on results



All stakeholders (Table) except ARSP staff were invited to participate in the evaluation by completing email surveys. Australian stakeholders were telephoned to ascertain if they would participate 1–2 days after the survey was emailed. If, after a specified return date, surveys were not completed, Australian participants were telephoned to determine if assistance was needed to complete the survey. International participants were contacted again via email.

Results

Description of the system

Participating laboratories sent stool samples to the ARSP with a unique sample code and the sex and age of the case from which it was obtained. This code allows samples to be linked to hospital data by the sending laboratory if needed.

Participating laboratories detect rotavirus using enzyme immunoassay (EIA) or latex agglutination tests. Samples of stool (0.05 mL–1.0 mL) containing rotavirus are sent to the ARSP. Nearly 60% (355/628) of samples are obtained from patients hospitalised with gastroenteritis though samples have been sent from non-hospitalised cases in outbreaks in the Northern Territory.

Upon receipt, the ARSP confirm that rotavirus is in the stool sample using an in-house monoclonal antibody (MAb) EIA, which also identifies common serotypes G1, G2, G3, G4 and G9. If rotavirus is not identified in the stool by the MAb EIA, there is no further testing. If rotavirus is detected but common serotypes are not identified, samples are genotyped by reverse-transcriptase polymerase chain reaction (RT-PCR).[10] If the serotype is not identified using EIA or RT-PCR, the RNA of the virus is analysed using polyacrylamide gel electrophoresis (PAGE) to determine if the electrophoretic pattern is similar to patterns of known serotypes.

The age and sex of a case, the date of specimen collection, the code associated with the sending laboratory and the EIA, RT-PCR and/or PAGE results are stored an Excel database. Stool samples are stored in locked freezers at the Royal Children's Hospital, Melbourne. The results of serotyping are published annually in *Communicable Diseases Intelligence* (CDI). The report is also forwarded to staff of vaccine manufacturing companies and participating laboratories. Data reported in the annual reports include the:

- number of stool samples received by ARSP (by month of receipt, and by collaborating laboratory);
- proportion and number of isolates of each serotype;
- age and gender of cases;
- geographic distribution of G serotypes in Australia, by state or territory; and
- whether isolates were associated with an outbreak.

Attributes

The proportion of staff from laboratories which contributed to the ARSP in 2004–05 who participated in the evaluation is shown in the Table. The low response rate from staff of laboratories who had previously participated in the program reflects that the questionnaire was sent to a retired laboratory staff or an expired email address, and that an appropriate participant from that laboratory could not be located.

Table. Stakeholders of the Australian Rotavirus Surveillance Program who were invited and surveyed, and attributes they assessed in the evaluation, 2006

Stakeholder	Number invited	Number participated	Attributes assessed
Staff from laboratories which currently participate	8	7	Flexibility, representativeness, timeliness, usefulness, sensitivity
Staff from laboratories which participated previously	5	2	Flexibility, representativeness, timeliness, usefulness, sensitivity
Vaccine manufacturing companies	2	2	Timeliness, usefulness
CDNA representatives of some states and territories*	3	3	Flexibility, representativeness, timeliness, usefulness, sensitivity
International experts in rotavirus surveillance	11	1	Flexibility, representativeness, timeliness, usefulness, sensitivity
ARSP staff (or annual reports)	NA	NA	Representativeness, flexibility, sensitivity, timeliness

* Communicable Diseases Network Australia representatives were asked to participate in states and territories where rotavirus was notifiable in 2006.

Flexibility

Based on experience, the ARSP laboratory director considers the current system as being flexible to adapt to changes in the number of laboratories participating, the number of samples serotyped, and the amount of funding received.

Between 1999 and 2004, the greatest number of laboratories participating in the ARSP was in 1999–2000 with 17 laboratories and 1,126 samples serotyped. The least number of laboratories participating was 7 in 2002–03 when 573 samples were serotyped. In the evaluation, 5 of 7 laboratories reported that ARSP can process as many samples as are sent, indicating that the participating laboratories perceived ARSP to be flexible.

Samples from outbreaks can be serotyped by ARSP whenever they are received. They are reported faster than results from routine serotyping, at no additional cost. The system is able to serotype new or unusual serotypes within 3 months by MAbs if these exist for the serotype, or by RT-PCR. The emergence of small numbers of the unusual serotypes G9 and G12 in 2001–02 and 2005–06 respectively was detected by the program.[4,10]

Representativeness

In 2004–05 only 5 of 8 Australian states and territories contributed to the ARSP. Representativeness of rural and remote locations could not be assessed in this study because the residential addresses of cases are not recorded by the ARSP. Stool specimens of cases occurring in rural and remote areas may be less likely to be tested by participating laboratories, which are mainly located in larger towns and cities.

The ARSP does not collect information on Indigenous status so neither the prevalence of serotypes, nor the burden of rotavirus disease in Indigenous populations could be estimated.

The proportion of hospital in-patients and out-patients from whom rotavirus samples were obtained was ascertained from laboratories involved in the Program in 2005. Of 7 laboratories, an average of 60% of rotavirus isolates came from in-patients. The proportion of in-patients and out-patients with samples tested by the ARSP was compared to estimations of the proportion of rotavirus hospital in-patients and out-patients in Australia. Galati estimated that for every case hospitalised for rotavirus, 2.2 visited an emergency department as an out-patient.[6] In the sample of rotavirus cases who had isolates serotyped by the ARSP, the ratio of hospitalisations to ED visits was 1:0.75. The ARSP therefore serotype a greater proportion of isolates from in-patients and may not be representative of non-hospitalised cases.

The age distribution of the hospitalised cases based on ARSP data differs from that of ICD-coded hospital separation data. In hospital separation data, most of the hospitalised cases (39.5%) were children aged between 12 and 23 months[6] while in ARSP data, most children (46.6%) were aged between 0 and 11 months. ARSP may not serotype a representative sample of children older than one year, who may be less likely to be hospitalised.

Differences reported by the ARSP and Galati[6] about the proportion of rotavirus cases that are hospitalised and the age distribution of cases, may be explained by differences in methods used to obtain the results and the populations sampled. Galati's estimates were based on hospitalisation and pathology data which were linked to obtain the rotavirus attributable fraction of all hospitalised gastroenteritis cases. This rotavirus attributable fraction was used to make estimations of the number of rotavirus cases, their ages, and the severity of their infection. This methodology may introduce misclassification biases associated with ICD coding. In contrast, the sample population of the ARSP is cases hospitalised, in mainly public hospitals, which are laboratory-confirmed.

The representativeness of the isolates received by the ARSP of all notified cases was assessed for cases in the Northern Territory. In 1999 and 2000, the proportion of notified rotavirus cases that had isolates serotyped by the ARSP ranged from between 18% and 20%, and from 2001 to 2005, between 39% and 60%.

The ARSP sample is representative of rotavirus from hospitalised cases in the areas where participating laboratories are located. It is not known if the isolates serotyped are representative of isolates causing disease in Indigenous populations, rural and remote areas and areas where there is no participating laboratory.

Sensitivity

Laboratories participating in 2004–05 detected rotavirus in between 3.3% and 17.6% of all stool samples they collected. Four of seven collaborating laboratories sent at least 90% of all stool samples that were positive for rotavirus to the ARSP for serotyping. One laboratory sent only 14% of stool samples in which rotavirus was detected. Overall, the ARSP received isolates from 63% of all rotavirus positive samples collected by participating laboratories in 2005. It is possible that a selection bias

was introduced into the sample of isolates sent by laboratories to be serotyped by the ARSP. It is not clear how this impacts the sensitivity of ARSP.

The sensitivity of the system to detect new or unusual serotypes of rotavirus is difficult to quantify because the true number of unusual serotypes circulating in Australia is not known. Nonetheless, in the last 5 years small numbers of serotypes G9 and G12 have been detected by the ARSP. These serotypes had not previously been observed in Australia.

The sensitivity of the ARSP surveillance system to provide information about whether a serotype was acquired overseas or is endemic in a particular Australian sub-population is limited. The ARSP does not collect data about travel, place of residence or Indigenous status for rotavirus cases. The ARSP does not have the sensitivity to detect rotavirus outbreaks independent of notification by public health or laboratory staff, as expected of laboratory surveillance. Outbreaks may be detected retrospectively by sorting data by date of collection and sending laboratory.

Timeliness

Timeliness of serotyping

As serotyping results do not affect the clinical management of patients, the time taken for results to reach the participating laboratory was not an important issue for stakeholders. On the other hand, the time taken to serotype samples collected during an outbreak increased the usefulness of results. The ARSP serotyped rotavirus isolates from two outbreaks in 2004–05 and reported on the results within 6–7 days. The relevant jurisdiction could then anticipate the extent and severity of the outbreak based on the uniqueness of the serotypes, and plan an appropriate response.

Timeliness of reporting

The ARSP annual report is published in CDI approximately 6 months after the end of the reporting period. A summary of serotyping results is prepared every 6 months and distributed to vaccine manufacturing companies.

Stakeholders were asked how frequently they would like reports of serotyping results after the introduction of the rotavirus vaccination program. Staff of participating laboratories who review the results only for information would like reports every 6 months. Communicable Diseases Network Australia representatives had different opinions about the requirements for reporting; 1 jurisdiction was satisfied with receiving serotyping results annually, 1 would find 6 monthly reports useful. Representatives of vaccine companies were satisfied with 6 monthly reports. The annual report was viewed as an important means of communication with international stakeholders.

Usefulness

In terms of usefulness in the control and prevention of the rotavirus in Australia, the ARSP has had relatively little impact to date. In the vaccine era however, the baseline prevalence data of rotavirus serotypes circulating in Australia collected by the ARSP in previous years will be used when vaccine effectiveness is assessed.

The perceived usefulness of ARSP results to stakeholders varies. One participating laboratory reported using the results to validate their own routine diagnostic test. One participant mentioned the usefulness of serotyping at the beginning of outbreaks to forecast the potential impact and extent of the outbreak according to if the serotype is new in a population. For vaccine companies, the most important use of ARSP data has been to inform the formulation of vaccines for use in Australia by determining if vaccine strains match circulating serotypes.

Discussion

The evaluation showed that, in the pre-vaccine era, the ARSP has provided baseline data on the serotypes of rotavirus causing hospitalisation in children in Australia with a sufficiently timely, flexible and sensitive system. Since mid-2007 vaccines have been available for use in preventing rotavirus infections in Australian children. In the vaccine era, national surveillance of rotavirus will provide notification data and information that could be used to assess the impact of vaccination, the rate of vaccination failures, changes in rotavirus epidemiology and the emergence of replacement serotypes. Data provided by the ARSP will contribute to the latter functions of national surveillance and will provide epidemiologists in Australia the unique opportunity to evaluate the impact of both Rotarix and RotaTeq in a single country.

Based on past experience of adapting to changing participation rates and new stakeholders, the ARSP will adapt to meet changing needs of stakeholders in the vaccine era and the greater demand for serotyping. Collaboration with new stakeholders such as public health personnel and staff of health departments will create new demands on the ARSP.

In order to provide valid data that could be used to assess the impact of rotavirus vaccines, representative sampling is required. Approaches to increasing the number of laboratories participating in the pro-

gram should implemented. Representative sampling of all states and territories, rural and remote areas, and Indigenous populations should be an aim of the program.

There was only limited patient demographic information available to the ARSP in the pre-vaccine era but data collected after rotavirus becomes a notifiable condition, will increase the value of serotyping data. Travel history information may explain the origin of unusual strains, locality information may be used to identify rotavirus outbreaks, and Indigenous status data will provide information data on the strains circulating in Indigenous populations. These data will also enable the effectiveness of vaccines to be determined in Australian sub-populations.

In the vaccine era, interest in rotavirus serotyping results will increase so results should be reported more often than currently. Isolates obtained from vaccinated children should be rapidly serotyped to enhance investigation of suspected vaccination failure. Timely identification of the serotypes in outbreaks will be important as rotavirus vaccine coverage increases, the incidence of disease decreases, and the risk of outbreaks caused by non-vaccine serotypes becomes increasingly important.

A key challenge is for the role of the ARSP to be clarified, clearly communicated to all stakeholders, and for the data it provides to be integrated into surveillance systems. Data flow from the ARSP to either public health units or state health departments should be developed in collaboration with each jurisdiction. If serotype data are matched to notification data, both the sensitivity of the ARSP system and the usefulness of the data for public health action will increase.

Conclusions

Australia is in the unique position of being able to evaluate the impact of two licensed vaccines in a population where the baseline epidemiology of rotavirus serotypes has been documented. The system is flexible, and can perform timely serotyping and reporting of data. The ARSP should strengthen the representativeness of its data in collaboration with state and territory public health systems to increase its sensitivity.

Acknowledgements

Many thanks to Professor Graeme Barnes and Dr Carl Kirkwood of the Murdoch Childrens Research Institute, Parkville, Victoria for their time, input and enthusiasm.

Thank you to those who provided data used in the evaluation.

From the following organisations

Alice Springs Pathology Laboratory, Northern Territory

Microbiology Department, Royal Brisbane Hospital, Queensland

Microbiology Department, Royal Darwin Hospital, Northern Territory

Pathology Department, Gove District Hospital, Northern Territory

PathWest, Western Australia

The New Children's Hospital, Westmead, New South Wales

Virology Department, South Eastern Area Laboratory Services, New South Wales

Women's & Children's Hospital, Perth, Western Australia

Women's and Children's Hospital Adelaide, South Australia

Communicable Diseases Network Australia representatives from Queensland, the Northern Territory and Western Australia

Staff of vaccine manufacturing companies

CSL

GlaxoSmithKline

Jon Gentsch, Division of Viral Diseases, Centers for Disease Control and Prevention, USA

Rosalie Schultz, Centre for Disease Control, Northern Territory

This work was performed when April Roberts-Witteveen was a Masters of Applied Epidemiology student at the National Centre of Epidemiology and Population Health, Australian National University. Her funding was a scholarship provided by the Australian Government Department of Health and Ageing.

Author details

Ms April R Roberts-Witteveen[1]
Dr Mahomed S Patel[2]
Dr Paul W Roche[3]

1. Master of Applied Epidemiology Scholar, Australian National University, Acton, Australian Capital Territory
2. Assoc Professor, National Centre for Epidemiology and Population Health, Australian National University, Australian Capital Territory
3. Senior Epidemiologist, Surveillance Policy and Systems Section, Australian Government Department of Health and Ageing, Canberra, Australian Capital Territory

Corresponding author: Mrs April Roberts-Witteveen, GPO Box 9848, Canberra, ACT 2601. Telephone: +61 2 6289 2721. Facsimile: +61 2 6289 2600. Email: lirpa_r@yahoo.com

References

1. Parashar UD, Gibson CJ, Bresse JS, Glass RI. Rotavirus and severe childhood diarrhea. *Emerg Infect Dis* 2006;12:304–306.
2. Carlin JB, Jackson T, Bishop RF, Barnes GL. Cost effectiveness of rotavirus vaccination in Australia. *Aust N Z J Public Health* 1999;23:611–616.
3. Villa S, Guiscafre H, Martinez H, Munoz O, Gutierrez G. Seasonal diarrhoeal mortality among Mexican children. Bull World Health Organization 1999;77:375–380.
4. Kirkwood C, Bogdanovic-Sakran N, Barnes G, Bishop R. Rotavirus serotype G9P[8] and acute gastroenteritis outbreak in children, Northern Australia. *Emerg Infect Dis* 2004;10:1593–1600.
5. Gentsch J, Liard AR, Biefelt B, Griffin DD, Banyai K, Ramachandran M, et al. Serotype diversity and reassortment between human and animal rotavirus strains: implications for rotavirus vaccine programs. *J Infect Dis* 2005;192 Suppl 1:S146–S159.
6. Galati JC, Harsley S, Richmond P, Carlin JB. The burden of rotavirus-related illness among young children on the Australian health care system. *Aust N Z J Public Health* 2006;30:416–421.
7. Vesikari T, Matson DO, Dennehy P, Van Damme P, Santosham M, Rodriguez Z, et al. Safety and efficacy of a pentavalent human-bovine (WC3) reassortant rotavirus vaccine. *New Engl J Med* 2006;354:23–33.
8. Ruiz-Palacios GM, Pérez-Schael I, Velázquez FR, Abate H, Breuer T, Clemens SC, et al. Safety and efficacy of an attenuated vaccine against severe rotavirus gastroenteritis. *N Engl J Med* 2006;354:11–22.
9. German RR, Lee LM, Horan JM, Milstein RL, Pertowski CA, Waller MN: Guidelines Working Group Centers for Disease Control and Prevention. Updated guidelines for evaluating public health surveillance systems: recommendations from the Guidelines Working Group. *MMWR Recomm Rep* 2001;50(RR-13):1–35; quiz CE1–7.
10. Kirkwood CD, Cannan D, Bogdanovic-Sakran N, Bishop RF, Barnes GL. National Rotavirus Surveillance Program annual report 2005–06. *Commun Dis Intell* 2006;30:434–438.

Short reports

Notifications of enteric diseases in returning travellers who visit friends and relatives overseas: A call for action

Vicki G Slinko, Kari AJ Jarvinen, Frank H Beard, Bradley J McCall

The health risks of travelling to developing countries are generally well appreciated in the Australian community. With the increasing frequency of global travel, travel medicine is acknowledged as an essential aspect of health services and travellers medical clinics in large cities assist overseas travellers with their health requirements.

A common purpose for overseas travel by foreign-born Australian residents and their children is 'visiting friends and relatives' (VFR), usually in their country of origin. It has been noted in other developed countries[1–5] that these travellers account for a disproportionate amount of illness from malaria, hepatitis A and typhoid on return to their country of residence. A study in New South Wales identified VFR travel as a risk factor for hepatitis A.[6] Considerable clinical and public health resources are required to control these introduced communicable diseases. However, the prevention of these illnesses in VFR travellers receives limited attention.

We recently examined case reports for notifications of hepatitis A, hepatitis E, typhoid and paratyphoid infections reported to the Brisbane Southside Population Health Unit (BSPHU) between 1 January 2006 and 3 April 2008. Cases where there was a clear record of visiting friends and relatives overseas were categorised as 'definite' VFR travellers. In the absence of a clear record, cases with a history of at least one month's stay in a country with plausible associations to the name of the case were categorised as 'probable' VFR travellers.

The number and proportion of cases notified to the BSPHU with a history of VFR travel are shown in the Table. At least one quarter of hepatitis A notifications and half of the enteric fever notifications were in VFR travellers.

When staying with friends or relatives, VFR travellers may adopt the living conditions of the local community; their diet will be dictated by local circumstances, they are more likely to drink untreated water, spend time in crowded conditions and in markets, have sexual contact with local residents and use local medical and dental services.[2]

It is possible that VFR travellers may be less likely to seek out and adhere to travel recommendations. Their health care provider may incorrectly assume that VFR travellers are aware of travel related health issues. Australian-born children of VFR travellers may be particularly vulnerable to vaccine preventable diseases such as hepatitis A[7,8] while visiting their parent's country of origin if the family do not seek pre-travel advice.

Travel health care should be accessible and culturally appropriate and language barriers may need to be addressed. Travel medicine is an important component of primary care with the health care provider requiring access to appropriate and up-to-date resources. Then, the message for these primary care providers is that travel related health recommendations apply to all travellers regardless of their ethnic origins or presumed immunity. The new edi-

Table. Notifications of hepatitis A, hepatitis E, typhoid and paratyphoid infection associated with overseas travel and travellers visiting friends and relatives and reported to the Brisbane Southside Population Health Unit, 1 January 2006 to 3 April 2008

Disease	Number notified	Overseas travel		Overseas travellers visiting friends and relatives	
		n	% of notifications	n	% of notifications
Hepatitis A	31	15	48	7 (+ 2 probable)	23 (29)
Hepatitis E	2	2	100	1 (+ 1 probable)	50 (100)
Typhoid	12	11	92	7 (+ 2 probable)	58 (75)
Paratyphoid	8	8	100	3 (+ 1 probable)	38 (50)

tion of The Australian Immunisation Handbook9 emphasises this message. Vaccination and health education must be combined to ensure adequate protection for VFR and other travellers.

Multicultural organisations and councils could also be engaged to enhance communication with VFR travellers about the need to protect their health when travelling overseas and how to access appropriate medical services before departure. Travel health messages of this nature could be incorporated into existing health services and programs for people from culturally and linguistically diverse backgrounds.

However, challenges for communicable disease control will remain while there is a disproportionate amount of communicable disease in developing countries and increased global population movement. In the meantime, efforts should be made to improve awareness of the health issues for VFR travellers amongst primary care providers and the multicultural community.

Acknowledgements

Dr Christine Selvey, Communicable Diseases Branch, Queensland Health

Author details

Dr Vicki G Slinko, Public Health Registrar[1]
Dr Kari AJ Jarvinen, Public Health Medical Officer[1]
Dr Frank H Beard, Senior Medical Officer[2]
Dr Bradley J McCall, Public Health Medical Officer[1]

1. Brisbane Southside Population Health Unit, Queensland Health
2. Communicable Diseases Branch, Queensland Health

Corresponding author: Dr Vicki Slinko, Public Health Registrar, Brisbane Southside Population Health Unit, PO Box 333, ARCHERFIELD, QLD 4108. Telephone: +61 7 3000 9128. Facsimile: +617 3000 9130. Email: Vicki_Slinko@health.qld.gov.au

References

1. Fulford M, Keystone JS. Health risks associated with visiting friends and relatives in developing countries. *Curr Infect Dis Rep* 2005;7:48–53.
2. Angell SY, Cetron MS. Health disparities among travelers visiting friends and relatives abroad. *Ann Intern Med* 2005;142:67–72.
3. Ackers ML, Puhr ND, Tauxe RV, Mintz ED. Laboratory-based surveillance of *Salmonella* serotype Typhi infections in the United States: antimicrobial resistance on the rise. *JAMA* 2000;283:6668–6673.
4. Behrens RH, Collins M, Botto B, Heptonstall J. Risk for British travellers of acquiring hepatitis A. *BMJ* 1995;311:193.
5. Basnyat B, Maskey AP, Zimmerman MD, Murdoch DR. Enteric (typhoid) fever in travelers. *Clin Infect Dis* 2005;41:1467–1472.
6. Ward K, McAnulty J. Hepatitis A: who in NSW is most at risk of infection? *N S W Public Health Bull* 2008;19:32–35.
7. Yung A, Ruff T, Torresi J, Ledger K, O'Brien D. *Manual of Travel Medicine: A Pre-travel Guide for Health Care Practitioners* 2nd edn. Melbourne: IP Communications, 2004.
8. Fenner L, Weber R, Steffen R, Schalagenhauf P. Imported infectious disease and purpose of travel, Switzerland. *Emerg Infect Dis* 2007;13:217–222.
9. Australian Technical Advisory Group on Immunisation, Australian Government Department of Health and Ageing. *The Australian Immunisation Handbook* 9th edn. Canberra: Australian Government Department of Health and Ageing and the National Health and Medical Research Council, 2008.

Quarterly reports

OzFoodNet quarterly report, 1 April to 30 June 2008

The OzFoodNet Working Group

Introduction

The Australian Government Department of Health and Ageing established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease. This quarterly report documents investigations of outbreaks of gastrointestinal illness and clusters of disease potentially related to food, occurring in Australia from 1 April to 30 June 2008.

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

During the second quarter of 2008, OzFoodNet sites reported 397 outbreaks of enteric illness, including those transmitted by contaminated food. Outbreaks of gastroenteritis are often not reported to health agencies or the reports are delayed, meaning that these figures under-represent the true burden of enteric illness. In total, these outbreaks affected 6,295 people, of which 202 were hospitalised and 18 people died. The majority (84.6%, n=335) of outbreaks were due to person-to-person transmission (Table 1).

Foodborne disease outbreaks

There were 25 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as the primary mode of transmission (Table 2). These outbreaks affected a total of 393 people and resulted in 21 being admitted to hospital. There were 3 deaths. This compares with 34 outbreaks for the second quarter of 2007[1] and 29 outbreaks in the first quarter of 2008.[2]

Table 1. Mode of transmission for outbreaks of gastrointestinal illness reported by OzFoodNet sites, 1 April to 30 June 2008

Transmission mode	Number of outbreaks	Per cent of total
Foodborne	25	6.3
Person-to-person	335	84.6
Unknown	30	7.6
Salmonella cluster	5	1.3
Other pathogen cluster	2	0.5
Total	397	100.0

Salmonella was implicated in 11 outbreaks during this quarter, with *S.* Typhimurium being the most common serotype. There were 2 outbreaks each due to *S.* Typhimurium phage types 135a, 44 and U290, and 1 each due to phage type 9 and phage type 135. One outbreak was due to *S.* Johannesburg.

There were 4 foodborne outbreaks of norovirus during this quarter. There were 3 foodborne toxin-related outbreaks during the quarter, including 2 *Clostridium perfringens* outbreaks and 1 ciguatera fish poisoning outbreak. There was 1 outbreak due to hepatitis A. The remaining 6 outbreaks were caused by unknown aetiological agents.

Thirteen outbreaks reported in this quarter were associated with food prepared in restaurants, 3 with food prepared in private residences, 3 with food prepared by a commercial caterer, 2 in aged care facilities and 1 outbreak each in a correctional facility and a school. One outbreak was associated with primary produce, and 1 was a community outbreak.

To investigate these outbreaks, sites conducted 6 cohort studies and collected case series data for 18 investigations. Investigators obtained analytical epidemiological evidence in 4 outbreaks and microbiological evidence in 3 outbreaks. For the remaining 18 outbreaks, investigators obtained descriptive evidence implicating the food vehicle or suggesting foodborne transmission.

The following jurisdictional summaries describe key outbreaks and public health actions which occurred in this quarter. The Australian Capital Territory and the Northern Territory did not report any foodborne outbreaks during this quarter.

New South Wales

New South Wales reported 11 foodborne outbreaks during this quarter.

Table 2. Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 April to 30 June 2008 (n=25)

State	Month of outbreak	Setting prepared	Infection	Number affected	Evidence	Responsible vehicles
NSW	March†	Commercial caterer	*Salmonella* Typhimurium 135	5	D	Suspected chicken rissoles
	March†	Private residence	*Salmonella* Typhimurium 135	4	D	Suspected eggs
	April	Restaurant	Norovirus	25	D	Unknown
	April	Restaurant	*Salmonella* Typhimurium U290	4	M	Variety of Chinese dishes
	April	Commercial caterer	*Clostridium perfringens* toxin type A	31	A	Gravy
	April	Restaurant	*Salmonella* Typhimurium U290	7	D	Chilli beef dish
	April	Restaurant	Unknown	7	D	Rice, or salt & pepper prawns
	May	Restaurant	Unknown	17	A	Fattouch salad – Lebanese bread salad
	May	Restaurant	Unknown	2	D	Stir fry beef
	May	Community	*Salmonella* Typhimurium 9	14	D	Suspected chicken and eggs
	June	Correctional facility	Unknown	14	D	Lasagne meal
Qld	March†	Primary produce	Ciguatera	6	D	Black kingfish
	April	Restaurant	*Clostridium perfringens*	2	M	Refried Mexican beans
SA	June	Aged care facility	*Salmonella* Typhimurium 135	38	A	Vitamised foods
Tas	April	Private residences	*Salmonella* Typhimurium 135a	3	D	Suspected eggs
Vic	April	Restaurant	*Salmonella* Typhimurium 44	4	D	Suspected desserts
	May	Restaurant	*Salmonella* Johannesburg	14	M	Roast pork
	May	Commercial caterer	Unknown	21	A	Chicken curry
	May	Restaurant	Norovirus	14	D	Breakfast meals
	May	Restaurant	Hepatitis A	12	D	Salads and sandwiches
	May	School	*Salmonella* Typhimurium 44	26	D	Unknown
	June	Restaurant	Unknown	9	D	Multiple ready to eat foods
	June	Private residence	*Salmonella* Typhimurium 135a	4	D	Suspect egg/custard dessert
WA	April	Restaurant	Norovirus	75	D	Unknown
	April	Aged care facility	Norovirus	42	D	Unknown

* No foodborne outbreaks were reported in Australian Capital Territory, the Northern Territory or South Australia during the quarter.

† Outbreak first detected in April 2008, first case of illness onset in March 2008.

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

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

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

In an outbreak of *S.* Typhimurium phage type 135 with multi-locus variable number tandem repeat analysis (MLVA) pattern 3-12-9-10-550, 5 cases reported eating Thai chicken rissoles from 5 different food premises that were supplied by a common gourmet food producer. A swab from the floor of the kitchen of the gourmet food producer was positive for a molecular strain of *S.* Typhimurium indistinguishable from that of the human cases. In another outbreak, 4 cases of *S.* Typhimurium MLVA 3-12-9-10-550 were linked to a small scale local egg producer. Samples collected from the egg farm were negative for the outbreak strain but positive for 3 other *Salmonella* serovars. The gourmet food producer involved in the first outbreak may have purchased eggs from the small scale egg farm involved in the second outbreak. These 2 outbreaks were linked to a community-wide cluster of at

least 135 cases of *S.* Typhimurium with the same MLVA-type, with 83% of isolates identified as phage type 135 and the remaining isolates phage types 135a, 197, 6, 6 var 1 and U307.

An outbreak of *Clostridium perfringens* toxin type A affected 31 of 100 staff at a local high school who had attended a catered lunch at the school. The lunch was provided by a commercial caterer/ takeaway store. Cases reported symptoms of diarrhoea with abdominal pains (median duration of 18 hours), with a median onset of 14 hours after the catered lunch. A retrospective cohort study showed that consumption of gravy was independently associated with illness (RR=5.0, 95% CI 1.7–14.8). Two of the 3 faecal specimens submitted were positive for *C. perfringens* toxin type A. It is likely that inadequate cooling and reheating of the gravy prior to consumption contributed to the outbreak.

Two outbreaks of *S.* Typhimurium U290 (MLVA 3-12-10-12-523) affected 2 separate groups eating at Chinese restaurants. The first group of 4 ill people (1 laboratory-confirmed *S.* Typhimurium U290) had a shared meal which included a wide variety of Chinese foods. Symptoms of diarrhoea, abdominal cramps, vomiting and fever developed 36 hours after the shared meal. One case was hospitalised. An environmental investigation identified *S.* Typhimurium U290 from the kitchen preparation bench and a wooden chopping block on the bench. The second group of 7 ill people (5 laboratory-confirmed *S.* Typhimurium U290) shared a meal from a Chinese restaurant on the same street as the Chinese restaurant implicated in the first group of ill persons. Four cases became ill after eating at the restaurant, and 3 cases became ill after eating leftovers from that meal. While not confirmed, it is highly likely that the same establishment was responsible for both outbreaks.

An outbreak of *S.* Typhimurium 9 (MLVA 3-10-14-11-496) affected 14 people in the community. Consumption of egg and/or chicken products was suspected as the source of illness for these cases.

A probable foodborne outbreak due to norovirus affected 25 of 45 people on a bus tour in April. A cohort study did not identify any specific food items associated with illness; but an ill food handler who prepared food for the tour group at one of the premises visited on the tour was the likely source of the infection. Recommendations about food safety principles including exclusion of workers with gastroenteritis was made to the premises.

New South Wales also reported 4 suspected foodborne outbreaks with an unknown aetiology. A point source outbreak affected 14 of 49 people in a correctional facility following the consumption of a lasagne meal. The other 3 outbreaks occurred in restaurant settings and affected a total of 26 people.

Queensland

Queensland reported 2 outbreaks of foodborne illness during this quarter. In the first outbreak, 6 cases of ciguatera fish poisoning were associated with the consumption of black kingfish caught by recreational fishermen. Symptoms included reverse temperature sensation, numbness of hands, mouth and feet, skin rash and muscle pain. In the second outbreak, 2 males became ill with diarrhoea and stomach cramps approximately 12 hours after consuming chicken enchiladas with refried beans and rice from a Mexican restaurant in Brisbane. Food samples collected for testing included chicken enchiladas, refried beans and rice. High spore counts of *Clostridium perfringens* were detected in one faecal specimen and the refried bean food sample. Other food samples were negative for *C. perfringens*. Results suggested that time-temperature abuse of the refried beans was the contributing factor for this outbreak.

South Australia

South Australia reported one outbreak of suspected foodborne illness this quarter. An investigation was undertaken in an aged care facility with 21 confirmed cases of *S.* Typhimurium phage type 135. An association was found between illness and the consumption of vitamised and soft food diets. Advice on infection control measures and kitchen hygiene practices was provided to the facility.

Tasmania

Tasmania reported an outbreak of *S.* Typhimurium 135a that affected 3 people in southern Tasmania. Illness was associated with the consumption of uncooked eggs from the same egg supplier. Two of the cases were children from the same household. The children consumed uncooked cake batter containing raw egg in their home the day prior to onset of their illness. The third case was a female who had also eaten uncooked muffin mix containing raw egg in her home approximately 36 hours prior to onset of illness. No eggs were available for sampling from either household. The implicated egg supplier is not the same as the supplier implicated in outbreaks of *Salmonella* in Tasmania during 2005, 2007 and in the first quarter of 2008.[2]

Victoria

Victoria reported 8 outbreaks of foodborne illness this quarter.

Two outbreaks of *S.* Typhimurium 44 were reported during the quarter. In the first outbreak, 4 confirmed cases of *S.* Typhimurium 44 were diagnosed in people from 2 separate groups who dined at the same restaurant on the same night. The source of illness was suspected to be an undercooked egg-containing dessert (chocolate mousse, ice cream or chocolate pudding). In the second *S.* Typhimurium 44 outbreak, 26 of 66 people from a rural boarding school were affected. The descriptive epidemiology of the outbreak suggested a point source foodborne outbreak. A retrospective cohort study was conducted, but a specific source could not be identified.

An outbreak of *S.* Johannesburg was linked to contaminated pork that was prepared at a restaurant. Twelve cases (10 laboratory confirmed *S.* Johannesburg) ate the pork during a 3 week period, most of them on a single weekend. The outbreak was first reported after 2 people became ill after consuming takeaway pork purchased from the restaurant. Leftover pork from the meal was positive for *S.* Johannesburg. Faecal specimens from 2 food handlers at the restaurant (one of whom was asymptomatic) were also positive for *S.* Johannesburg. A sample of raw pork obtained from the pork supplier was positive for *S.* Johannesburg.

An outbreak of gastrointestinal illness was reported amongst 21 people from approximately 240 guests at a commercially catered function where a selection of curries and various other types of Sri Lankan foods were served. The suspected aetiology was *Clostridium perfringens* based on the incubation period, symptoms and duration of illness, but faecal specimens, collected over a week after symptoms resolved, were negative for bacterial and viral pathogens. There was a statistically significant association with consumption of chicken curry and illness (RR 4.6, 95% CI 1.23–17.21; p=0.004). Temperature abuse of food was suspected but this could not be confirmed.

An outbreak of norovirus was notified in 2 separate groups of people who attended a café. The first group reported 8 people ill from a group of 9 and the second reported 6 ill from a group of 7 people. Norovirus was detected in 6 of 8 faecal specimens collected from cases. No illness was reported amongst food handlers at the café. It is suspected that the outbreak was foodborne, based on 2 separate groups being affected, and the incubation periods.

Routine follow-up of a case of hepatitis A notified in May revealed that the case was a part owner of a café. In late May another case was notified and the case mentioned eating at this café during their incubation period. In total, 10 notified cases of hepatitis A had eaten foods such as sandwiches and salads from this café during their incubation period. In addition, there was one confirmed case notified in a household contact of one of these 10 cases.

An outbreak of *S.* Typhimurium 135a affected 4 of 5 family members who consumed a dessert made with lightly cooked eggs that had been left at room temperature overnight. No leftover foods were available for testing and *Salmonella* was not detected in or on the surface of eggs purchased from the same food premises as those used in preparation of the dessert.

An outbreak of suspected viral gastroenteritis affected 9 people from 14 separate groups who dined at the restaurant on the same day. During the investigation it was discovered that a food handler who was responsible for preparing mixed ready to eat foods worked at the restaurant preparing food whilst he was symptomatic with vomiting and diarrhoea.

Western Australia

Western Australia reported 2 outbreaks of suspected foodborne illness this quarter.

In April, an outbreak of norovirus occurred in staff and residents in an aged care facility. The index case was a chef who had prepared food while he was ill with gastroenteritis. Other staff and residents subsequently became ill over a 24 hour period. Faecal specimens from 8 residents were positive for norovirus. The epidemiological picture was consistent with foodborne transmission.

Another outbreak of norovirus occurred amongst patrons who had eaten a buffet meal at a restaurant at 2 different sittings in 1 weekend. A total of 366 people were reported to have eaten at these buffet meals, and 92 of them were interviewed in a cohort study. Thai fish curry was the only food with significantly increased relative risk (RR=1.30, p<0.05), but this food was consumed by only 28% of cases. Six faecal specimens obtained were positive for norovirus. An inspection of the premises did not identify any major deficiencies and there were no reports of staff illness. It is likely that 1 or more foods served at the buffet during the 2 affected meal sittings were contaminated.

Other reports and activities of note

Increase in listeriosis in New South Wales

New South Wales reported a continuing increase in listeriosis cases, with 8 cases this quarter, giving a total of 22 cases for the year to date (the 5-year average of listeriosis in New South Wales is 27 cases per year). Of the 8 cases this quarter, 2 were in pregnant women. Epidemiological investigations were unable

to establish any links between cases. All isolates (n=17) tested by pulse-field gel electrophoresis were distinct from each other with the exception of a maternal-neonatal isolate pair, indicating that cases were not linked to a common source.

Shiga toxin-producing *Escherichia coli* in a visiting group of students from Japan

Japanese public health officials identified an outbreak of Shiga toxin-producing *Escherichia coli* O26 in Japanese students who had visited Sydney in late February. All cases were diagnosed and notified in Japan. Japanese public health officials contacted NSW Health and local investigators assisted in an environmental investigation at venues visited by the students including a wildlife park and several restaurants. No association was found.

Salmonella Paratyphi B biovar Java linked to sandpits

New South Wales also reported a non-foodborne outbreak of *S.* Paratyphi B biovar Java affecting at least 27 cases this quarter. Epidemiological and environmental investigations identified an association between illness and playing in council playgrounds. *S.* Paratyphi B biovar Java was isolated from sand samples taken from implicated playgrounds. Implicated playgrounds with positive samples have been closed and clean-up efforts have commenced. Animal sampling and further investigations are continuing to determine the source of contamination of the sand.

Comments

Outbreaks associated with eggs and egg-containing products

Eggs and egg-containing foods continue to be a frequent source of foodborne outbreaks of gastrointestinal illness. During this quarter, 25% (6/25) of reported foodborne outbreaks were suspected to be due to eggs or egg-containing foods. *S.* Typhimurium was found to be the aetiological agent in all of these outbreaks, with 4 different phage types (135, 135a, 9 and 44) involved. In most of these outbreaks the consumption of foods containing raw or undercooked eggs was implicated, highlighting the need for thorough cooking of eggs and egg-containing foods or alternatively, substituting pasteurised eggs in dishes that are intended to be consumed uncooked or are only lightly cooked.

Outbreaks of *Clostridium perfringens*

Two outbreaks during the quarter were due to *Clostridium perfringens* enterotoxin. It was thought that temperature abuse was a contributing factor in these outbreaks and highlights the need for ongoing education of food handlers about correct methods of cooling, reheating and hot-holding of foods.

Outbreak of *Salmonella* in an aged care facility

The outbreak of *Salmonella* Typhimurium 135 in an aged care facility, reported by South Australia, highlights the potential for outbreaks in aged care settings. Foodborne outbreaks in aged care facilities are rare, about 98% of gastrointestinal outbreaks in aged care facilities are viral gastroenteritis, spread by person-to-person transmission (OzFoodNet Working Group, unpublished data 2008).

Outbreak of *Salmonella* Paratyphi B biovar Java linked to a sandpit

Of interest in this quarter's report is the outbreak of *S.* Paratyphi B biovar Java in New South Wales associated with sand in local playgrounds. *Salmonella* has been associated with sandpits and playgrounds in other countries,[3] but this is the first known outbreak of salmonellosis in Australia associated with sandpits. The strain of *S.* Paratyphi B biovar Java in this outbreak is fully sensitive to antibiotics and therefore different from the multi-drug-resistant strain that has previously been associated with contact with tropical fish tanks.[4]

Harmonising systems for the molecular typing of *Salmonella* species

A community wide cluster of *S.* Typhimurium in New South Wales with 135 cases (including 2 separate point-source outbreaks discussed under foodborne outbreaks) was first detected using MLVA, which has been used to routinely type *S.* Typhimurium isolates in New South Wales in addition to phage typing since October 2007. This cluster highlights one of the challenges associated with the introduction of a new surveillance tool. Experience has shown that a single phage type of *S.* Typhimurium can be associated with multiple MLVA patterns,[2] however, in this cluster, 1 MLVA pattern was associated with at least 6 different phage types. Australia continues to work towards understanding the epidemiological concordance of the different molecular typing methods currently in use by the states and territories and towards a harmonised typing scheme.[5]

Acknowledgements

OzFoodNet thanks the investigators in the public health units and state and territory departments of health, as well as public health laboratories and local government environmental health officers who provided data used in this report. We would also like to

thank laboratories conducting serotyping, molecular typing and phage typing of *Salmonella* for their continuing work during this quarter.

The OzFoodNet Working Group is *(in alphabetical order)*: Mary Barker (WA), Robert Bell (Qld), Therese Carroll (NSW), Craig Dalton (Hunter New England), Gerard Fitzsimmons (DoHA), Kathleen Fullerton (DoHA), Robyn Gibbs (WA), Joy Gregory (Vic), Gillian Hall (NCEPH), Michelle Harlock (NT), Geoff Hogg (MDU), Rebecca Hundy (ACT), Martyn Kirk (DoHA), Karin Lalor (Vic), Katrina Leslie (DoHA), Michelle McPherson (SA), Tony Merritt (Hunter New England), Jennie Musto (NSW), Lillian Mwanri (SA), Nevada Pingault (WA), Jane Raupach (SA), April Roberts-Witteveen (Hunter New England), Mark Salter (FSANZ), Craig Shadbolt (NSWFA), Frances Sheehan (Qld), Russell Stafford (Qld), Nicola Stephens (Tas), Barbara Telfer (NSW), Hassan Vally (NCEPH), Mark Veitch (MDU), Tory Worgan (Hunter New England).

Correspondence

Ms Katrina Leslie, Epidemiologist, OzFoodNet, Office of Health Protection, Australian Government Department of Health and Ageing, GPO Box 9848, MDP 14, CANBERRA ACT 2601. Telephone: +61 2 6289 2739. Facsimile: +61 2 6289 2500. Email: katrina.leslie@health.gov.au

References

1. OzFoodNet Working Group. OzFoodNet quarterly report, 1 April to 30 June 2007. *Commun Dis Intell* 2007;31:314–318.
2. OzFoodNet Working Group. OzFoodNet quarterly report, 1 January to 31 March 2008. *Commun Dis Intell* 2008;32:267–271.
3. Doorduyn Y, Van Den Brandhof WE, Van Duynhoven YT, Wannet WJ, Van Pelt W. Risk factors for *Salmonella* Enteritidis and Typhimurium (DT104 and non-DT104) infections in The Netherlands: predominant roles for raw eggs in Enteritidis and sandboxes in Typhimurium infections. *Epidemiol Infect* 2006;134:617–626.
4. Musto J, Kirk M, Lightfoot D, Combs BG, Mwanri L. Multidrug resistant *Salmonella* Java infections acquired from tropical fish aquariums, Australia, 2003–04. *Commun Dis Intell* 2006;30:222–227.
5. Ammon A. Molecular typing for public health purposes. *Euro Surveill* 2008;13(19):pii=18864. [online] Available from: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=18864

Communicable diseases surveillance

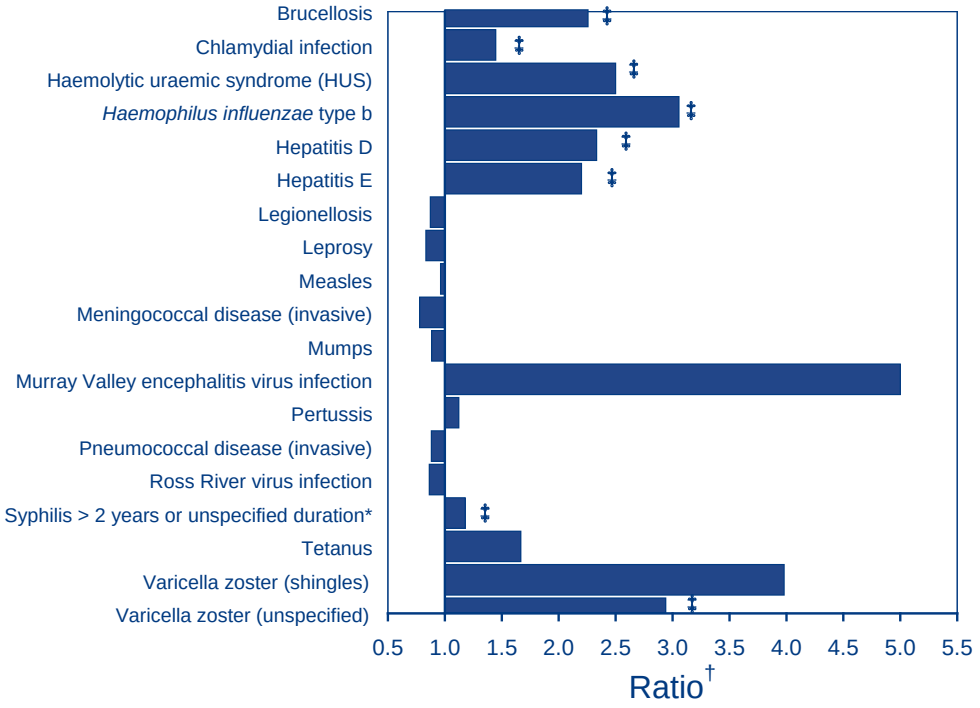
Highlights for 2nd quarter, 2008

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

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

Figure 1 shows the changes in selected disease notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a diagnosis in the first quarter (April to June) 2008, in comparison with the five-year mean for the same period. Notifications were above the five year mean for the same period and exceeded two standard deviations from the five-year mean for: brucellosis, chlamydial infection, haemolytic uraemic syndrome, *Haemophilus influenzae* type b, hepatitis D, hepatitis E, syphilis (greater than 2 years or unspecified duration) and varicella zoster (unspecified).

Figure 1. Selected* diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 April to 30 June 2008 with historical data*



* Selected diseases are chosen each quarter according to current activity. Five year averages and the ratios of notifications in the reporting period in the five year mean should be interpreted with caution. Changes in surveillance practice, diagnostic techniques and reporting, may contribute to increases or decreases in the total notifications received over a 5 year period. Ratios are to be taken as a crude measure of current disease activity and may reflect changes in reporting rather than changes in disease activity. See Table 1 for all diseases.

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

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

§ Ratio for syphilis of less than 2 years duration based on 4 years data.

Notifications were above the five-year mean, but less than 2 standard deviations from the five-year mean for pertussis, tetanus, Murray Valley encephalitis virus infection and varicella zoster (shingles). Notifications were equal to or below the five-year mean for measles, mumps, invasive pneumococcal disease, legionellosis, Ross River virus infection, leprosy, and invasive meningococcal disease.

Bloodborne viruses

Hepatitis D

Hepatitis D infection requires the presence of the hepatitis B virus to replicate and can occur as an acute co-infection with hepatitis B virus, or as a super-infection with chronic hepatitis B infection. The modes of hepatitis D transmission are similar to those for hepatitis B through exposure to infected blood and serous body fluids. Hepatitis D infection can be misdiagnosed as an exacerbation of chronic hepatitis B.[1] Preventative measures for hepatitis D infection are essentially through hepatitis B immunisation in order to prevent hepatitis B infection and hence hepatitis D co-infection.

Hepatitis D occurs worldwide and is most prevalent in countries that have a high incidence of hepatitis B. The highest incidence occurs in parts of Russia, Romania, Southern Italy and the Mediterranean countries, Africa, South America and the islands of the Western Pacific. However, despite high rates of hepatitis B in China the incidence of hepatitis D is disproportionately lower.[2] In Australia, hepatitis D infection is uncommon.

During the second quarter of 2008 there were 14 cases of hepatitis D virus infection notified to the NNDSS. Cases were reported from New South Wales (n=5), Western Australia (n=4), Queensland (n=2), Victoria (n=2) and the Northern Territory (n=1).

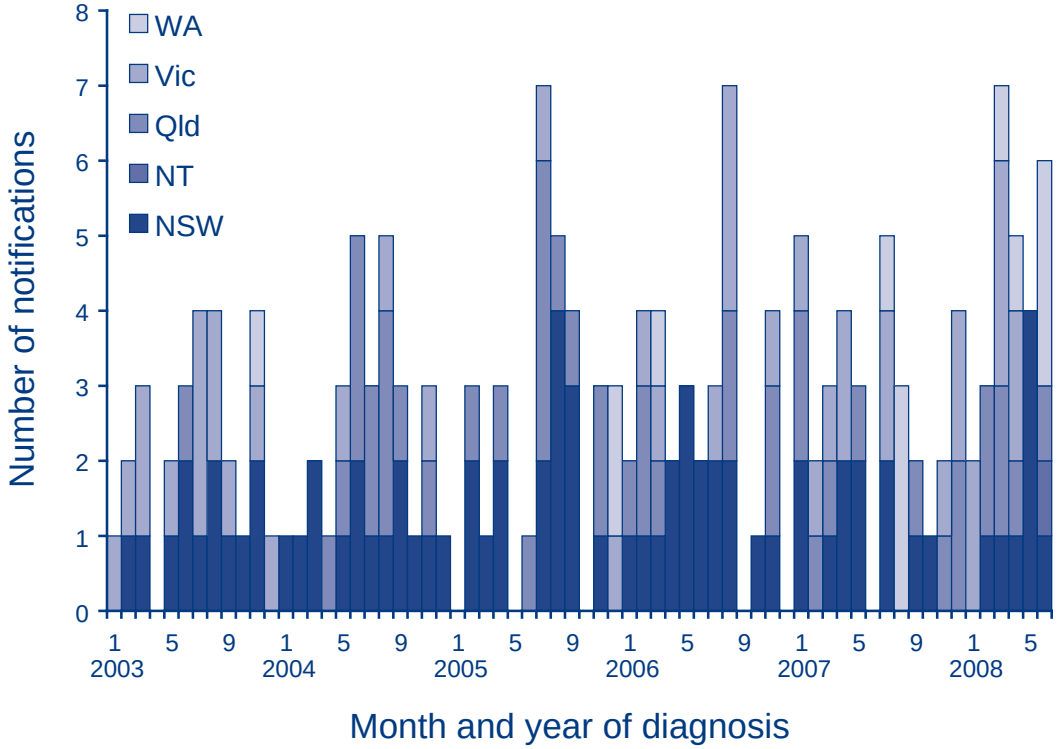
Figure 2 shows the number of notifications of hepatitis D reported to the NNDSS since 2003. For the first half of 2008 there were 25 cases of hepatitis D virus infection reported nationally, 92% higher than the 5 year-mean for the previous corresponding periods (n=13.0). The recent increase in hepatitis D virus infection notifications may be associated with increased co-testing for both hepatitis B and D viruses due to an increased awareness of the hepatitis D virus and co-infection issues.

Gastrointestinal diseases

Haemolytic uraemic syndrome

An association between infection with Shiga toxin-producing *Escherichia coli* (named for their similarity to toxins produced by *Shigella*) and the post diarrhoeal haemolytic uraemic syndrome (HUS) was first described in 1983.[3] Only confirmed cases

Figure 2. Notifications of hepatitis D, Australia, 1 January 2003 to 30 June 2008, by month of diagnosis



of HUS are reported to NNDSS. A confirmed case requires acute microangiopathic anaemia on peripheral blood smear (schistocytes, burr cells or helmet cells), and at least one of acute renal impairment (haematuria, proteinuria or elevated creatinine level), or thrombocytopaenia, particularly during the first 7 days of illness.

Figure 3 shows the number of notifications for HUS received by NNDSS between 1 January 2003 and 30 June 2008. During the second quarter of 2008, there were 6 notifications of HUS, including 2 fatal cases. Five cases were notified in New South Wales and 1 in the Northern Territory. In the year-to-date to 30 June 2008 there were 14 cases of HUS, exceeding the five-year mean of year to date notifications (7.2 notifications) by more than 2 standard deviations. The number of HUS notifications in the quarter represents an increase over the same period of 2007, when 3 cases were notified, with a total of 10 cases in the year-to-date in 2007.

Vaccine preventable diseases

Haemophilus influenzae type b

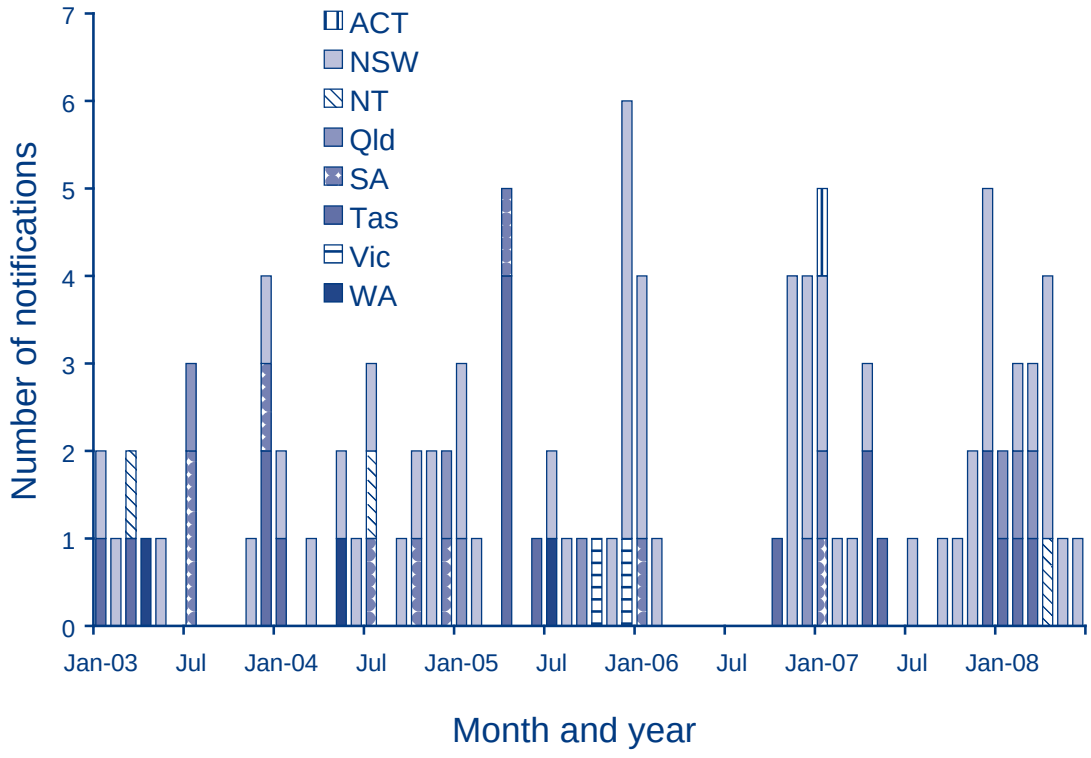
Haemophilus influenzae are Gram-negative coccobacilli that are classified into serotypes a through to f on the basis of the antigenic characteristics of the polysaccharide capsule, if present. The unencapsulated strains are nontypeable. *H. influenzae* serotype b is the most pathogenic. Humans are the reservoir of the bacteria, and transmission is via droplet infection and discharges from the nose and throat during the infectious period. The National Immunisation Program Schedule provides for a total of 4 doses of Hib vaccine, given at 2, 4, 6 and 12 months of age if using the PRP-Hib containing vaccine. The PRP-OMP Hib containing vaccine is indicated for use in Aboriginal and Torres Strait Islander children in areas of higher risk with doses provided at 2, 4 and 12 months.

Between 1 April and 30 June 2008, a total of 11 notifications of Hib were diagnosed and reported to the NNDSS. This was an increase from the 5 cases notified in the first quarter of 2008 and the 5 year-to-date mean for this period (n= 3.6). There have been 16 notifications for the year-to-date, which was 2.3 times the year-to-date five-year mean (7.0).

The annualised notification rate for this quarter was 0.2 cases per 100,000 population, an increase from 0.1 cases per 100,000 population in the first quarter of 2008.

The majority of cases were reported from New South Wales (n=6) and Queensland (n=3), with 1 each being notified from both Victoria and the Northern Territory. Five cases were in children, with 2 being infants aged less than 12 months. Both infants were reported to be unvaccinated for

Figure 3. Notifications of haemolytic uraemic syndrome, Australia, 1 January 2003 to 30 June 2008, by month of diagnosis



the disease. The majority of cases (n=10) were in non-Indigenous Australians, with 1 case being of unknown Indigenous status. Cases were approximately evenly divided between the sexes, with 5 males and 6 females affected.

Measles

Measles is an acute, highly communicable viral disease that can lead to serious complications such as pneumonia (lung infection), encephalitis (inflammation of the brain) or otitis media (middle ear infection). In the past, measles infection was a common childhood illness, but as a result of national immunisation campaigns measles is now rare in Australia, except for occasional outbreaks of limited duration that are generally linked to an imported case.[4] The current National Immunisation Program Schedule recommends 2 doses of the measles-mumps-rubella vaccine (MMR) at 12 months of age and at 4 years of age, unless there is a contraindication. High-level vaccination coverage is imperative to enable measles elimination, requiring rates for each new birth cohort of greater than 95% for a single dose and greater than 90% for 2 doses.[5]

Between 1 April and 30 June 2008, 26 cases of measles were reported to the NNDSS compared to the 33 cases reported in the first quarter of 2008. The majority of cases in this quarter were from New South Wales (n=23), with Western Australia (n=2) and Queensland (n=1) also reporting cases. The number of cases in the second quarter of 2008 was comparable to the five-year mean (n=27). The annualised notification rate has decreased this quarter to 0.5 cases per 100,000 population compared with the first quarter of 2008 when it was 0.6 cases per 100,000 population.

Fifty-four per cent of cases (n=14) were male and 46% (12) were female with ages ranging from less than 1 year to 48 years. Of the 26 cases, vaccination status was known for 21 of the cases, with 9 (35%) reported as fully vaccinated for age and 12 (46%) reported as not vaccinated.

Of the 26 cases, 1 unvaccinated infant aged less than 1 year acquired measles overseas. The annualised rate of locally acquired measles was estimated at 4.8 cases per million population. NSW Health has reported 4 generations of transmission of measles in a localised community.

Genotyping data was available for 7 cases, all from New South Wales. The majority (n=5) were D5, with 1 each being D4 and D9.

Mumps

The mumps virus is a member of the Paramyxovirdae family, genus *Rubulavirus*. Infection with the virus causes an acute disease characterised by fever, swelling, and tenderness of one or more salivary glands. Testicular atrophy occurs in about one-third of patients, but sterility is rare. Transmission is airborne, via droplet spread or by direct contact with the saliva of an infected person. In the absence of immunisation, mumps is endemic. In Australia, immunisation is included as part of the MMR vaccine provided at 12 months and 4 years of age.

Between 1 April and 30 June 2008, 52 cases of mumps were reported to the NNDSS. This was a decrease from the previous quarter (n=144) and was also less than the 5 year-to-date mean for this quarter of 59 cases. However, total case numbers to date in 2008 (n=199) were 1.4 times higher than for the same period in 2007 (n=139) and were twice the 5 year-to-date mean of 96 cases. The annualised notification rate for this quarter was 1.0 cases per 100,000 population, a decrease from 2.7 for the first quarter of 2008.

Cases for this quarter were reported from Western Australia (n=27), the Northern Territory (n=9), New South Wales (n=8), Queensland (n=5), Victoria (n=2) and Tasmania (n=1). Fifty-four per cent of cases were male (n=28) and 46% female (n=24) with ages ranging from 8 to 70 years. Of the 52 cases, 18 were fully vaccinated for age, 1 was partially vaccinated for age, 10 were not vaccinated and in 23 cases vaccination status was unknown.

Of the 52 cases, 4 were imported from overseas of which 1 had a history of partial vaccination and the other 3 were reported as having unknown vaccination status.

Pertussis

Pertussis (whooping cough) is an acute bacterial infection of the respiratory tract cause by *Bordetella pertussis*. The initial catarrhal stage has an insidious onset with an irritating cough that gradually becomes paroxysmal, usually within 1–2 weeks and lasting for 1–2 months or longer. Paroxysms can be followed by a characteristic high-pitched inspiratory whoop. In vaccinated populations, the number of fatalities from pertussis is low. Infants under 6 months are at most risk of death being too young to have completed primary immunisation. Transmission is by direct contact with discharges from respiratory mucous membranes of infected persons by the airborne route. In vaccinated populations, bacteria are frequently brought home by an older sibling and sometimes a parent.

Between 1 April and 30 June 2008, 1,935 cases of pertussis were reported to the NNDSS. The majority of cases were reported in New South Wales (n=881) followed by Queensland (n=337) and Victoria (n=321) with South Australia (n=168), the Northern Territory (n=129), Western Australia (n=67), the Australian Capital Territory (n=24) and Tasmania (n=8) also reporting cases in this quarter. These case numbers were 1.5 times more than in the same period in 2007 (n=1,271) but only 1.2 times the year-to-date five-year mean for this quarter.

The annualised notification rate for this quarter of 37 cases per 100,000 population was higher than that for the first quarter (29) and for the same period in 2007 (23 per 100,000). Notifications for the year-to-date (n=3,448) exceeded both the same period in 2007 (n=2,309) and the year-to-date five-year mean (n=3,271).

Fifty-eight per cent of cases were female (n=1,125) and 42% male (n=810). The average age in this quarter was 38 cases with ages ranging from less than 1 year to 92 years.

Tetanus

Tetanus is an acute disease induced by an exotoxin of the bacteria *Clostridium tetani*, which grows anaerobically at the site of a puncture wound injury. Direct person-to-person transmission is not possible. The disease is characterised by painful muscular contractions and has a case mortality rate of between 10% and 80%. Active immunity is induced by tetanus toxoid and persists for at least 10 years after full immunisation. The current National Immunisation Program schedule provides for immunisation at 2, 4 and 6 months, 4 years and 15–17 years.

Between 1 April and 30 June 2008, there was 1 new case of tetanus reported to the NNDSS. In the previous quarter, there were 3 reported cases. The number of cases in the second quarter of 2008 was comparable with the five-year mean (n=0.6). However, the year-to-date of 4 cases exceeded the five-year mean (n=1.6). The annualised rate for the second quarter of 2008 was 0.02 cases per 100,000 population, compared with 0.00 cases per 100,000 population for the same period in 2007.

All 4 cases reported for the year-to-date in 2008 were from elderly people, with the cases having an age range of 70 through to 87 years and a mean of 80 years. Vaccination status for all cases was either unknown (n=3) or partially vaccinated with 1 dose of vaccine (n=1). No deaths resulted from illness.

Vectorborne diseases

There are currently 9 notifiable mosquito-borne diseases under national surveillance in Australia. These include alphaviruses (Barmah Forest virus and Ross River virus), flaviviruses (dengue, Japanese encephalitis, Kunjin, Murray Valley encephalitis and flavivirus infection not elsewhere classified), yellow fever and malaria.

Murray Valley encephalitis virus infection

On 29 April 2008, the Western Australian Department of Health issued a media release reminding people living and holidaying in Western Australia's north to continue to take precautions against mosquito bites following the death of a 49-year-old Kimberley resident from Murray Valley encephalitis virus (MVEV) infection.

MVEV was first isolated from patients who died from encephalitis in the Murray Valley in Victoria and South Australia in 1951. Retrospectively, the first epidemics of disease caused by this virus are thought to have occurred in 1917 and 1918 (initially named Australian X disease). It was previously included as one of the causative agents in the disease called Australian encephalitis, which also included disease caused by Kunjin virus, another flavivirus. These viruses are now accepted as causing 2 separate diseases. The last Australia-wide outbreak of MVEV disease was in 1974. Since then almost all cases have been in northern and central Australia (with a few cases reported in the Midwest and Murchison regions, less than 500 km north of Perth, in 2000).

Acknowledgements

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

References

1. Heymann DL. *Control of Communicable Diseases Manual*. 18th edn. Washington: American Public Health Association; 2004.
2. World Health Organization. Hepatitis Delta. Geneva: World Health Organization; 2001. Available from: http://www.who.int/csr/disease/hepatitis/HepatitisD_whocdscsrncs2001_1.pdf Accessed on 20 August 2008.
3. Mead PS, Griffin PM. *Escherichia coli* O157:H7. *Lancet* 1998;352:1207–1212.
4. National Health and Medical Research Council. *The Australian Immunisation Handbook*. 9th edn. Canberra: Commonwealth of Australia; 2008. p.201. Available from: http://www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/5335A7AB925D3E39CA25742100194409/$File/handbook-9.pdf Accessed on 11 May 2008.
5. Heath T, Burgess M, McIntyre P, Catton M. The national measles surveillance strategy. *Commun Dis Intell* 1999;23:41–50.

Tables

National Notifiable Diseases Surveillance System

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 36,787 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 April and 30 June 2008 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1. Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis (NEC)	All jurisdictions
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (incident)	All jurisdictions except Qld
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except NSW
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Salmonellosis	All jurisdictions
STEC, VTEC	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 (all)	
Syphilis <2 years duration	All jurisdictions
Syphilis >2 years or unspecified duration	All jurisdictions
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 NSW & Vic
Varicella zoster (shingles)	All jurisdictions except NSW & Vic
Varicella zoster (unspecified)	All jurisdictions except NSW & Vic
Vectorborne diseases	
Arbovirus infection (NEC)†	All jurisdictions
Barmah Forest virus infection	All jurisdictions
Dengue virus infection	All jurisdictions
Japanese encephalitis virus infection	All jurisdictions
Kunjin virus infection	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus infection	All jurisdictions
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssaviruses (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Tularaemia	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

* Notifiable in South Australia as of 1 May 2008.

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

NEC Not elsewhere classified.

Table 2. Notifications of diseases received by state and territory health authorities in the period 1 April to 30 June 2008, by date of diagnosis*

Disease	State or territory								Total 2nd quarter 2008†	Total 1st quarter 2008	Total 2nd quarter 2007	Last 5 years mean 2nd quarter	Year to date 2008	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.2	0.0
Hepatitis B (incident)	0	12	0	10	2	3	19	13	59	59	79	76.6	122	155.0	0.8
Hepatitis B (unspecified)	17	786	17	194	85	13	450	148	1,710	1,735	1,747	1528.8	3,499	3,095.2	1.1
Hepatitis C (incident)	2	4	0	NN	11	6	32	24	79	82	82	89.0	165	182.2	0.9
Hepatitis C (unspecified)	40	1,189	59	656	121	85	629	320	3,099	3,316	2,898	3011.4	6,469	6,289.2	1.0
Hepatitis D	0	5	1	2	0	0	2	4	14	12	7	6.0	27	13.2	2.3
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0.4	0	1.0	0.0
Campylobacteriosis§	88	NN	66	932	506	105	1,398	332	3,427	4,675	3,893	3465.2	8,112	7,768.8	1.0
Cryptosporidiosis	2	111	21	173	10	8	120	33	478	761	617	625.4	1,243	1,613.4	0.8
Haemolytic uraemic syndrome	0	5	1	0	0	0	0	0	6	8	3	2.4	14	7.2	2.5
Hepatitis A	2	9	2	27	4	0	35	7	86	85	39	77.0	172	169.4	1.1
Hepatitis E	0	2	3	2	0	0	2	2	11	14	7	5.0	25	13.8	2.2
Listeriosis	0	6	0	1	0	0	5	2	14	26	8	13.8	40	32.0	1.0
Salmonellosis	28	530	131	457	170	32	433	173	1,954	2,903	2,360	1948.8	4,876	4,897.0	1.0
STEC, VTEC‖	0	3	0	4	10	0	2	0	19	32	14	16.8	51	37.2	1.1
Shigellosis	0	17	33	25	39	1	18	49	182	243	135	139.8	427	313.2	1.3
Typhoid	0	11	1	5	0	0	7	1	25	35	19	14.2	60	40.2	1.8
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	0	1	1.0	0	1.8	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

Table 2. Notifications of diseases received by state and territory health authorities in the period 1 April to 30 June 2008, by date of diagnosis,* *continued*

Disease	State or territory								Total 2nd quarter 2008†	Total 1st quarter 2008	Total 2nd quarter 2007	Last 5 years mean 2nd quarter	Year to date 2008	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection¶	236	3,535	744	3,847	971	335	3,211	2,274	15,153	14,549	13,219	10475.0	29,804	21,033.4	1.4
Donovanosis	0	0	1	0	0	0	0	0	1	0	0	2.0	1	5.2	0.5
Gonococcal infection	4	303	515	402	204	6	234	676	2,344	2,081	2,320	2084.0	4,441	4,118.6	1.1
Syphilis (all)	3	344	61	76	15	3	210	80	792	828	796	615.8	1,629	1,226.6	1.3
Syphilis < two years duration	2	61	28	32	0	0	101	62	286	356	395	226.8	664	350.8	1.3
Syphilis >two years or unspecified duration	1	283	33	44	15	3	109	18	506	472	401	429.4	965	875.8	1.2
Syphilis - congenital	0	1	0	1	0	0	0	0	2	1	3	5.0	3	8.2	0.4
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Haemophilus influenzae type b	0	6	1	3	0	0	1	0	11	5	4	3.6	16	7.0	3.1
Influenza (laboratory confirmed)	27	161	4	299	11	16	77	82	677	436	569	411.4	1,147	634.6	1.6
Measles	0	23	0	1	0	0	0	2	26	33	3	27.0	59	39.0	1.0
Mumps	0	8	9	5	0	1	2	27	52	144	93	58.8	199	95.6	0.9
Pertussis	24	881	129	337	168	8	321	67	1,935	1,515	1,271	1724.2	3,524	3,274.8	1.1
Pneumococcal disease (invasive)	11	156	12	81	35	9	89	36	429	208	381	486.8	642	754.4	0.9
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Rubella	0	5	0	0	0	0	4	0	9	4	15	13.4	13	25.8	0.7
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	2	0.6	0	0.8	0.0
Tetanus	0	0	0	1	0	0	0	0	1	3	0	0.6	4	1.6	1.7
Varicella zoster (chickenpox)	8	NN	18	64	98	8	2	76	274	260	295	154.7	537	500.0	1.8
Varicella zoster (shingles)	3	NN	18	80	170	33	0	129	433	533	370	108.8	974	298.5	4.0
Varicella zoster (unspecified)	27	NN	0	731	104	8	2	188	1,060	976	934	360.6	2,031	751.4	2.9
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	0	3	0	0	0	0	3	7	6	7.8	10	25.0	0.4
Barmah Forest virus infection	1	142	20	270	9	0	8	41	491	836	574	549.2	1,329	979.4	0.9
Dengue virus infection	0	30	4	21	10	1	2	25	93	151	82	102.4	244	267.4	0.9
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0.4	0.0
Kunjin virus infection	0	0	0	0	0	0	0	0	0	1	0	1.4	1	6.0	0.0
Malaria	3	32	4	37	3	2	27	20	128	125	161	165.8	255	367.0	0.8
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	1	1	1	0	0.2	2	0.8	5.0
Ross River virus infection	3	236	56	535	31	8	47	242	1,158	2,797	1,290	1341.0	3,962	3,040.0	0.9

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 April to 30 June 2008, by date of diagnosis,* *continued*

Disease	State or territory								Total 2nd quarter 2008†	Total 1st quarter 2008	Total 2nd quarter 2007	Last 5 years mean 2nd quarter	Year to date 2008	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.4	0.0
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Brucellosis	0	1	0	13	0	0	0	0	14	10	7	6.2	24	16.4	2.3
Leptospirosis	0	5	0	25	0	0	1	0	31	42	28	40.8	73	91.8	0.8
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Ornithosis	0	14	0	1	0	0	14	3	32	23	24	43.2	56	84.8	0.7
Q fever	1	23	2	29	6	0	2	2	65	109	115	110.0	177	234.4	0.6
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Other bacterial infections															
Legionellosis	3	22	0	9	2	0	22	15	73	63	86	83.6	138	170.4	0.9
Leprosy	0	1	0	0	0	0	1	0	2	3	4	2.4	5	5.4	0.8
Meningococcal infection**	1	20	2	15	5	0	18	5	66	43	59	84.8	107	160.2	0.8
Tuberculosis	6	96	5	38	9	2	86	26	268	293	246	244.0	579	497.2	1.1
Total	540	8,735	1,940	9,412	2,809	693	7,533	5,125	36,787	40,066	34,866		72,649		0.5

* 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 date of notification.

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

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

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

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

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

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

NN Not notifiable.

NEC Not elsewhere classified.

NDP No data provided.

Table 3. Notification rates of diseases, 1 April to 30 June 2008, by state or territory. (Annualised rate per 100,000 population)

Disease*	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (incident)	0.0	0.7	0.0	1.0	0.5	2.4	1.5	2.5	1.1
Hepatitis B (unspecified)	20.0	45.6	31.6	18.6	21.5	10.5	34.6	28.1	32.6
Hepatitis C (incident)	2.4	0.2	0.0	NN	2.8	4.9	2.5	4.6	1.9
Hepatitis C (unspecified)	47.1	69.0	109.8	62.8	30.6	68.9	48.3	60.8	59.0
Hepatitis D	0.0	0.3	1.9	0.2	0.0	0.0	0.2	0.8	0.3
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis†	103.6	NN	122.8	89.2	127.8	85.1	107.4	63.1	97.1
Cryptosporidiosis	2.4	6.4	39.1	16.5	2.5	6.5	9.2	6.3	9.1
Haemolytic uraemic syndrome	0.0	0.3	1.9	0.0	0.0	0.0	0.0	0.0	0.1
Hepatitis A	2.4	0.5	3.7	2.6	1.0	0.0	2.7	1.3	1.6
Hepatitis E	0.0	0.1	5.6	0.2	0.0	0.0	0.2	0.4	0.2
Listeriosis	0.0	0.3	0.0	0.1	0.0	0.0	0.4	0.4	0.3
Salmonellosis	33.0	30.8	243.8	43.7	42.9	25.9	33.3	32.9	0.4
STEC, VTEC‡	0.0	0.2	0.0	0.4	2.5	0.0	0.2	0.0	37.2
Shigellosis	0.0	1.0	61.4	2.4	9.8	0.8	1.4	9.3	3.5
Typhoid	0.0	0.6	1.9	0.5	0.0	0.0	0.5	0.2	0.5
Quarantinable diseases									
Cholera	0.0	0.0	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§	277.8	205.3	1,384.6	368.0	245.2	271.6	246.8	431.9	288.5
Donovanosis	0.0	0.0	1.9	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	4.7	17.6	958.5	38.5	51.5	4.9	18.0	128.4	44.6
Syphilis (all)	3.5	20.0	113.5	7.3	3.8	2.4	16.1	15.2	15.1
Syphilis <2 years duration	2.4	3.5	52.1	3.1	0.0	0.0	7.8	11.8	5.4
Syphilis >2 years or unspecified duration	1.2	16.4	61.4	4.2	3.8	2.4	8.4	3.4	9.6
Syphilis - congenital	0.0	0.1	0.0	0.1	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	0.3	1.9	0.3	0.0	0.0	0.1	0.0	0.2
Influenza (laboratory confirmed)	31.8	9.3	7.4	28.6	2.8	13.0	5.9	15.6	12.9
Measles	0.0	1.3	0.0	0.1	0.0	0.0	0.0	0.4	0.5
Mumps	0.0	0.5	16.7	0.5	0.0	0.8	0.2	5.1	1.0
Pertussis	28.3	51.2	240.1	32.2	42.4	6.5	24.7	12.7	36.8
Pneumococcal disease (invasive)	13.0	9.1	22.3	7.7	8.8	7.3	6.8	6.8	8.2

Table 3. Notification rates of diseases, 1 April to 30 June 2008, by state or territory. (Annualised rate per 100,000 population), *continued*

Disease*	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
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	0.3	0.0	0.0	0.0	0.0	0.3	0.0	0.2
Rubella - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Varicella zoster (chickenpox)	9.4	NN	33.5	6.1	24.7	6.5	0.2	14.4	7.8
Varicella zoster (shingles)	3.5	NN	33.5	7.7	42.9	26.8	0.0	24.5	12.3
Varicella zoster (unspecified)	31.8	NN	0.0	69.9	26.3	6.5	0.2	35.7	30.0
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.0	0.1
Barmah Forest virus infection	1.2	8.2	37.2	25.8	2.3	0.0	0.6	7.8	9.3
Dengue virus infection	0.0	1.7	7.4	2.0	2.5	0.8	0.2	4.7	1.8
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	3.5	1.9	7.4	3.5	0.8	1.6	2.1	3.8	2.4
Murray Valley encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0
Ross River virus infection	3.5	13.7	104.2	51.2	7.8	6.5	3.6	46.0	22.0
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	1.2	0.0	0.0	0.0	0.0	0.3
Leptospirosis	0.0	0.3	0.0	2.4	0.0	0.0	0.1	0.0	0.6
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.8	0.0	0.1	0.0	0.0	1.1	0.6	0.6
Q fever	1.2	1.3	3.7	2.8	1.5	0.0	0.2	0.4	1.2
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial infections									
Legionellosis	3.5	1.3	0.0	0.9	0.5	0.0	1.7	2.8	1.4
Leprosy	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Meningococcal infection‖	1.2	1.2	3.7	1.4	1.3	0.0	1.4	0.9	1.3
Tuberculosis	7.1	5.6	9.3	3.6	2.3	1.6	6.6	4.9	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; Northern Territory and Queensland, which exclude ocular specimens; and Western Australia, which excludes ocular and perinatal infections.

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

NN Not notifiable.

NEC Not elsewhere classified.

NDP No data provided.

Laboratory Serology and Virology Reporting Scheme

There were 6,910 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 April to 30 June 2008 (Tables 4 and 5).

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 April to 30 June 2008, and total reports for the year†

	State or territory								This period 2008	This period 2007	Year to date 2008	Year to date 2007
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	–	8	–	1	1	–	1	–	11	6	24	12
Mumps virus	1	–	1	2	5	–	4	2	15	12	34	21
Rubella virus	–	1	–	1	–	–	1	–	3	6	9	14
Hepatitis viruses												
Hepatitis A virus	–	1	–	6	4	–	1	–	12	11	35	21
Hepatitis D virus	–	2	–	–	7	–	–	–	9	9	18	16
Hepatitis E virus	–	1	–	1	–	1	1	–	4	–	6	1
Arboviruses												
Ross River virus	–	9	17	191	20	–	6	19	262	422	1,058	723
Barmah Forest virus	–	14	1	108	4	–	1	–	128	158	378	297
Flavivirus (unspecified)	–	1	–	11	–	–	–	–	12	31	42	56
Adenoviruses												
Adenovirus not typed/ pending	–	50	–	60	251	2	4	–	367	210	716	390
Herpesviruses												
Herpes virus type 6	–	–	–	–	–	–	1	–	1	–	1	1
Cytomegalovirus	2	31	–	99	109	–	11	–	252	313	618	600
Varicella–zoster virus	3	79	–	301	148	2	29	–	562	625	1,322	1,322
Epstein–Barr virus	1	6	35	219	169	3	5	126	564	700	1,219	1,377
Other DNA viruses												
Parvovirus	–	2	–	28	8	–	5	–	43	73	123	165
Picornavirus family												
Echovirus type 11	–	3	–	–	–	–	–	–	3	–	3	–
Rhinovirus (all types)	1	44	–	–	3	–	–	–	48	95	85	160
Enterovirus not typed/ pending	1	6	1	6	1	2	2	–	19	55	100	82
Picornavirus not typed	–	–	–	–	–	6	–	–	6	1	7	1
Ortho/paramyxoviruses												
Influenza A virus	2	21	1	28	32	–	5	–	89	66	138	118
Influenza B virus	2	20	–	7	38	–	2	–	69	7	91	12
Parainfluenza virus type 1	–	23	–	7	44	–	12	–	86	7	149	11
Parainfluenza virus type 2	–	5	–	2	2	–	1	–	10	36	20	41
Parainfluenza virus type 3	–	7	–	5	5	–	–	–	17	48	24	81
Respiratory syncytial virus	–	406	1	114	154	7	56	–	738	604	943	775
Other RNA viruses												
HTLV–1	–	–	–	–	14	–	–	–	14	6	18	9
Rotavirus	–	24	–	–	63	–	1	–	88	58	197	103
Norwalk agent	–	11	–	–	–	–	–	–	11	98	28	238

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 April to 30 June 2008, and total reports for the year,† *continued*

	State or territory								This period 2008	This period 2007	Year to date 2008	Year to date 2007
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other												
Chlamydia trachomatis not typed	2	272	1	1,099	639	12	14	–	2,039	2,057	4,397	4,221
Chlamydia pneumoniae	–	–	–	–	–	–	1	–	1	–	1	–
Chlamydia psittaci	1	2	–	5	–	–	25	–	33	15	55	36
Mycoplasma pneumoniae	–	6	6	83	47	6	54	15	217	309	423	622
Mycoplasma hominis	–	2	–	–	–	–	–	–	2	1	4	4
Coxiella burnetii (Q fever)	4	31	–	15	7	1	14	–	72	75	152	103
Orientia tsutsuganushi	–	–	–	1	1	–	1	–	3	4	8	7
Rickettsia – spotted fever group	–	9	–	12	2	4	33	2	62	69	90	72
Streptococcus group A	–	5	25	147	–	–	1	–	178	263	450	495
Brucella species	–	–	–	9	–	–	–	–	9	2	17	3
Bordetella pertussis	1	35	1	68	131	–	13	–	249	216	446	396
Legionella pneumophila	–	1	–	–	1	–	2	–	4	17	11	21
Legionella longbeachae	–	–	–	–	1	–	–	–	1	3	5	6
Cryptococcus species	–	1	–	3	4	–	–	–	8	11	15	20
Leptospira species	–	–	–	20	4	–	–	–	24	15	54	38
Treponema pallidum	1	44	14	193	286	–	6	–	544	722	1,108	1,257
Entamoeba histolytica	–	–	–	1	–	–	–	–	1	1	4	5
Toxoplasma gondii	–	–	–	2	1	–	2	–	5	7	6	15
Echinococcus granulosus	–	–	–	–	6	–	9	–	15	11	22	14
Total	22	1,183	104	2,855	2,212	46	324	164	6,910	7,455	14,674	13,982

* 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 2008*

State or territory	Laboratory	April 2008	May 2008	June 2008	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	65	84	130	279
	New Children's Hospital, Westmead	85	126	125	336
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	41	54	49	144
	South West Area Pathology Service, Liverpool	87	108	16	211
Queensland	Queensland Medical Laboratory, West End	1,483	946	663	3,092
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	655	824	730	2,209
Tasmania	Northern Tasmanian Pathology Service, Launceston	9	9	22	40
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Australian Ricketsial Reference Laboratory	41	42	47	130
	Monash Medical Centre, Melbourne	14	6	54	74
	Royal Children's Hospital, Melbourne	12	6	–	18
	Victorian Infectious Diseases Reference Laboratory, Fairfield	49	52	62	163
Western Australia	PathWest Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	90		124	214
Total		2,631	2,257	2,022	6,910

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

– No data received this period.

Additional reports

Australian Sentinel Practice Research Network

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

The network consists of general practitioners who report presentations on a number of defined medical conditions each week. ASPREN was established in 1991 to provide a rapid monitoring scheme for infectious diseases that can alert public health officials of epidemics in their early stages as well as play a role in the evaluation of public health campaigns and research of conditions commonly seen in general practice. Electronic 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 2008, four conditions are being monitored. They include influenza like illness, 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.*

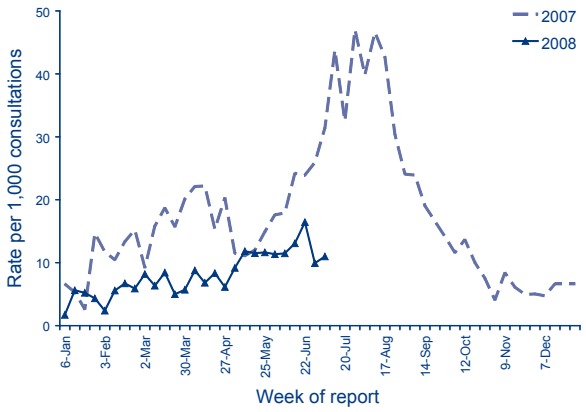
Data on influenza-like illness, gastroenteritis, chickenpox and shingles from 1 April to 30 June 2008 compared with 2007, are shown as the rate per 1,000 consultations in Figures 1, 2, 3 and 4, respectively.

Reporting period 1 April to 30 June 2008

Sentinel practices contributing to ASPREN were located in all jurisdictions other than the Northern Territory. A total of 96 general practitioners contributed data to ASPREN in the second quarter of 2008. Each week an average of 68 general practitioners provided information to ASPREN at an average of 7,160 (range 5,307 to 7,850) consultations per week.

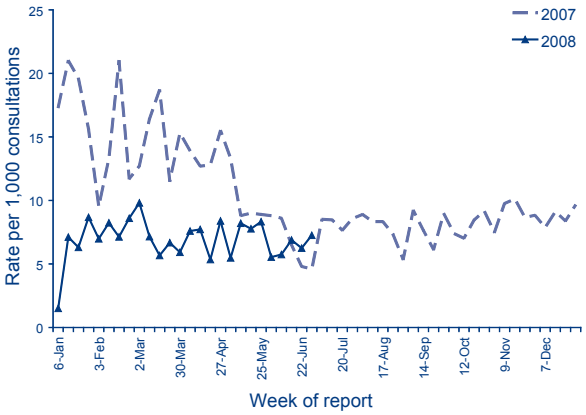
In the first quarter of 2008, influenza like illness (ILI) rates ranged from 6 to 17 cases per 1,000 consultations. For the same reporting period in 2007 reported rates were higher at 11 to 26 cases per 1,000 consultations (Figure 1).

Figure 1. Consultation rates for influenza-like illness, ASPREN, 1 January 2007 to 30 June 2008, by week of report



Reports of gastroenteritis from 1 April to 30 June 2008 were lower compared to the same period in 2007 (Figure 2). During this reporting period, consultation rates for gastroenteritis ranged from 5 to 8 cases per 1,000 consultations.

Figure 2. Consultation rates for gastroenteritis, ASPREN, 1 January 2007 to 30 June 2008, by week of report



Reports of varicella infections were reported at a lower rate for the second quarter of 2008 compared with the same period in 2007. From 1 April to 30 June 2008, recorded rates for chickenpox were between 0 to 0.4 cases per 1,000 consultations (Figure 3).

In the second quarter of 2008, rates for shingles fluctuated between less than 1 to 1.5 cases per 1,000 consultations (Figure 4).

Figure 3. Consultation rates for chickenpox, ASPREN, 1 January 2007 to 30 June 2008, by week of report

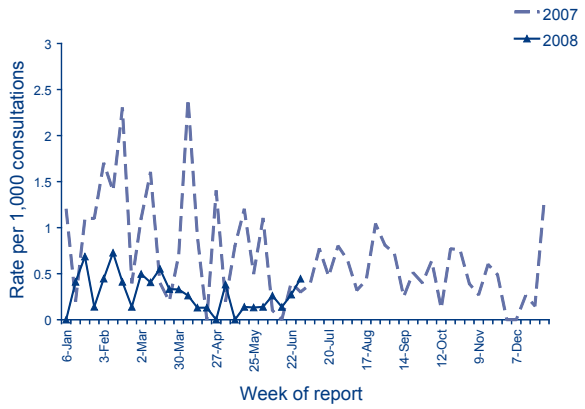
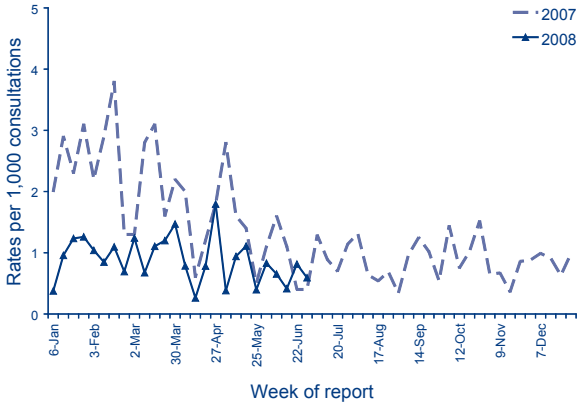


Figure 4. Consultation rates for shingles, ASPREN, 1 January 2007 to 30 June 2008, by week of report



Australian childhood immunisation coverage

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

The data show the percentage of children fully immunised at 12 months of age for the cohort born between 1 January and 31 March 2007, at 24 months of age for the cohort born between 1 January and 31 March 2006, and at 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 1 dose of a measles, mumps

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 2007; assessment date 30 June 2008

Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,156	24,042	960	15,365	4,880	1,596	17,530	7,558	73,087
Diphtheria, tetanus, pertussis (%)	93.7	91.6	92.4	91.6	91.8	91.4	92.8	90.5	91.8
Poliomyelitis (%)	93.8	91.5	92.3	91.6	91.8	91.3	92.8	90.5	91.8
Haemophilus influenzae type b (%)	95.8	94.7	95.6	93.9	94.3	94.0	94.8	94.2	94.5
Hepatitis B (%)	95.6	94.8	96.2	93.8	94.2	94.0	94.7	93.9	94.4
Fully immunised (%)	93.5	91.3	91.6	90.8	91.0	91.0	91.8	90.1	91.2
Change in fully immunised since last quarter (%)	-0.4	-0.3	+1.5	-0.4	+0.6	-1.7	-0.1	+1.1	-0.1

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 marginally by 0.1 percentage point to 91.2 (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 did not change and remained at 92.8% (Table 2). There were also no important changes in coverage for any individual vaccines due at 24 months of age or by jurisdiction.

Immunisation coverage for 'fully immunised' at 5 years of age for Australia decreased for the second consecutive quarter, by 0.9 percentage points, to 87.2% (Table 3). For 'fully immunised' and all individual vaccines, there were important decreases of greater than 1.5 percentage points in South Australia, New South Wales and the Australian Capital Territory, with a 2 percentage decrease in MMR coverage in New South Wales and the Australian Capital Territory. This decrease in coverage is likely due to the change in the coverage calculation algorithm, which, since the beginning of 2008, now calculates coverage for vaccines due at 4 years of age at the 5-year milestone, not the 6-year milestone. This means late immunisations given to a child aged between 5 and 6 years are no longer included in the assessment.

Figure 5 shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and 6 years, although the rate of increase has slowed over the past few years for all age groups. However, there is a noticeable dip in recent coverage at 6 years of age after a second consecutive quarterly decrease. 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

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 2006; assessment date 30 June 2008*

Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,204	22,941	932	14,687	4,608	1,601	16,598	7,182	69,753
Diphtheria, tetanus, pertussis (%)	96.7	94.9	96.1	94.9	95.4	95.1	95.9	93.8	95.1
Poliomyelitis (%)	96.6	94.8	96.1	94.8	95.3	95.1	95.8	93.7	95.1
Haemophilus influenzae type b (%)	96.6	95.4	95.4	93.9	94.4	95.4	94.6	93.6	94.6
Measles, mumps, rubella (%)	95.5	93.7	96.4	94.0	94.7	94.5	95.0	92.9	94.2
Hepatitis B (%)	97.2	95.7	97.3	95.6	96.2	96.2	96.4	94.8	95.9
Fully immunised (%)	94.8	92.5	94.7	92.6	93.3	93.4	93.6	91.2	92.8
Change in fully immunised since last quarter (%)	+0.8	-0.2	+0.8	+0.1	+0.6	-0.7	+0.0	-0.5	-0.0

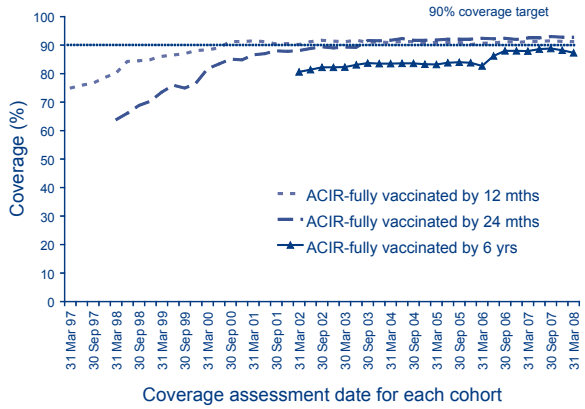
* The 12 months age data for this cohort was published in *Commun Dis Intell* 2007;31:333.

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 2003; assessment date 30 June 2008

Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,028	21,494	942	13,525	4,293	1,420	15,502	6,615	64,819
Diphtheria, tetanus, pertussis (%)	89.9	87.4	88.5	88.0	84.7	90.5	91.1	85.2	88.1
Poliomyelitis (%)	89.6	87.2	88.5	87.8	84.7	90.5	91.0	84.9	88.0
Measles, mumps, rubella (%)	89.0	86.9	88.3	87.8	84.7	90.3	90.8	85.1	87.8
Fully immunised (%)	88.9	86.4	87.9	87.3	84.2	89.8	90.5	84.1	87.3
Change in fully immunised since last quarter (%)	-1.7	-1.9	-0.4	-1.1	-1.5	+3.4	+0.1	+0.2	-0.9

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 2008, by age cohorts



Gonococcal surveillance

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

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

Reporting period 1 January to 31 March 2008

The AGSP laboratories received a total of 799 isolates in the first quarter of 2008 of which 783 underwent susceptibility testing. This approximates the 856 isolates reported in this period in 2007. Approximately 28% of this total was from New South Wales, 17.5% from Victoria, 16.1% from Queensland, 12.8% from Western Australia and South Australia and 11.7% from the Northern Territory. Small numbers of isolates were also received from Tasmania and the Australian Capital Territory.

Penicillins

In this quarter, 350 (44.7%) of all isolates examined were penicillin resistant by one or more mechanisms. Ninety-six (12.3%) were penicillinase producing *Neisseria gonorrhoeae* (PPNG) and 254 (32.4%) were penicillin resistant by chromosomal mechanisms, (CMRP). The proportion of all strains resistant to the penicillins by any mechanism ranged from nil in the Northern Territory and Australian Capital Territory to 75% in South Australia. In the corresponding quarter in 2007, 38.7% of isolates were penicillin resistant by any mechanism. The increase in penicillin resistant strains was in gonococci with chromosomally mediated resistance.

Figure 6 shows the proportions of gonococci fully sensitive (MIC ≤ 0.03 mg/L), less sensitive (MIC 0.06–0.5 mg/L), relatively resistant (MIC ≥ 1 mg/L) or else PPNG aggregated for Australia and by state and territory. A high proportion of those strains classified as PPNG or else resistant by chromo-

Figure 6. Categorisation of gonococci isolated in Australia, 1 January to 31 March 2008, by penicillin susceptibility and region

FS	Fully sensitive to penicillin, MIC ≤0.03 mg/L.
LS	Less sensitive to penicillin, MIC 0.06–0.5 mg/L.
RR	Relatively resistant to penicillin, MIC ≥1 mg/L.
PPNG	Penicillinase producing *Neisseria gonorrhoeae.*

somal 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 46 (21%) PPNG and 94 (43%) CMRP. South Australia had the highest proportion of penicillin resistant strains with 5 (5%) PPNG and 70 (70.7%) CMRP. Victoria had 63 (46%) CMRP and 11 (8%) PPNG. Queensland had higher numbers of PPNG, 21 (16.5%), but fewer CMRP, 11 (9%). Western Australia had equal numbers of PPNG and CMRP, each 12 (12%). No penicillin resistant strains were found in the Northern Territory or the Australian Capital Territory. There were 4 CMRP and 1 PPNG reported from Tasmania.

Ceftriaxone

Eight isolates with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected; 6 in New South Wales and 1 each in Western Australia and Queensland. A similar number was seen nationally in the first quarter of 2007.

Spectinomycin

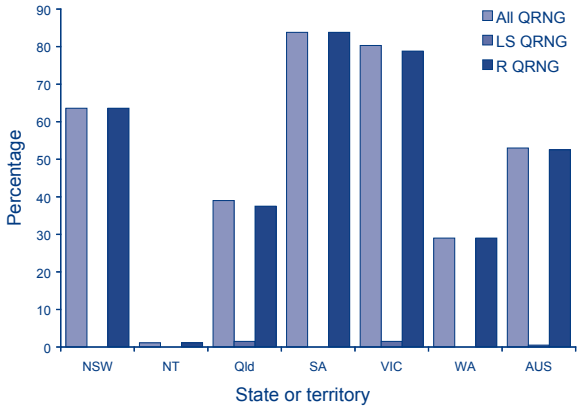
All isolates were susceptible to this injectable agent.

Quinolone antibiotics

The total number (415) and proportion (53%) of quinolone resistant *N. gonorrhoeae* (QRNG) was consistent with data reported in recent quarters showing high levels of resistance to this group of antibiotics. In the equivalent period in 2007, there were 436 (51.6%) QRNG. All but 4 of the 415 QRNG detected in this quarter had ciprofloxacin MICs of 1 mg/L or more and 379 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. 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 except the Australian Capital Territory (Figure 7). The highest number of QRNG was found in New South Wales (140) and this represented 63.6% of all isolates. The 83 (83.8%) QRNG in South Australia was the highest proportion of QRNG by jurisdiction. The 110 QRNG in Victoria also represented a high (80.3%) proportion of all isolates there. In Queensland, there were 28 (22%), and in Western Australia 24 (24%) QRNG. A single QRNG was detected in the Northern Territory and 3 in Tasmania.

Figure 7. The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 January to 31 March 2008, by jurisdiction



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

High level tetracycline resistance

Nationally, the number (135) and proportion (17.2%) of high level tetracycline resistance (TRNG) detected increased when compared with the 2007 data (125 TRNG, 14.8%). TRNG were found in all states and territories except the Australian Capital Territory and elsewhere represented between 8% (South Australia and the Northern Territory) and 24% of isolates (Western Australia) in mainland states.

Reference

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

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 three 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 *2005;29:91–92.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 October to 31 December 2007, as reported to 31 March 2008 are included in this issue of Communicable Diseases Intelligence *(Tables 4, and 5).*

Table 4. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 October to 31 December 2007, 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 2007	This period 2006	YTD 2007	YTD 2006
HIV diagnoses	Female	0	14	0	5	3	0	11	3	36	45	140	146
	Male	0	84	1	50	7	1	58	12	213	238	910	858
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	0	98	1	55	10	1	59	15	249	284	1,051	1,007
AIDS diagnoses	Female	0	3	0	0	0	0	3	0	6	4	15	20
	Male	0	14	0	4	1	0	10	0	29	50	137	193
	Total*	0	17	0	4	1	0	13	0	35	54	153	216
AIDS deaths	Female	0	1	0	0	0	0	0	0	1	2	8	7
	Male	0	2	1	2	0	0	5	2	12	17	45	74
	Total*	0	3	1	2	0	0	5	2	13	19	53	83

* Totals include people whose sex was reported as transgender.

Table 5. Cumulative diagnoses of HIV infection, AIDS, and deaths following AIDS since the introduction of HIV antibody testing to 31 December 2007, and reported by 31 March 2008, by sex and state or territory

	Sex	State or territory								Australia
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
HIV diagnoses	Female	32	928	23	298	111	12	400	221	2,025
	Male	261	13,823	137	2,897	979	112	5,503	1,275	24,987
	Not reported	0	228	0	0	0	0	22	0	250
	Total*	293	15,009	160	3,204	1,091	124	5,947	1,503	27,331
AIDS diagnoses	Female	10	262	4	73	32	4	116	42	543
	Male	92	5,509	45	1,055	412	55	2,049	434	9,651
	Total*	102	5,789	49	1,130	445	59	2,178	478	10,230
AIDS deaths	Female	7	138	1	43	20	2	64	29	304
	Male	73	3,597	30	676	280	33	1,426	299	6,414
	Total*	80	3,746	31	721	300	35	1,499	329	6,741

* Totals include people whose sex was reported as transgender.

National Enteric Pathogens Surveillance System

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

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

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

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

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

Reports to the National Enteric Pathogens Surveillance System of Salmonella *infection for the period 1 April to 30 June 2008 are included in Tables 6 and 7. Data include cases reported and entered by 18 July 2008. Counts are preliminary, and subject to adjustment after completion of typing and reporting of further cases to NEPSS. For more information see* Commun Dis Intell *2008;32:137.*

Reporting period 1 April to 30 June 2008

There were 1,712 reports to NEPSS of human *Salmonella* infection in the second quarter of 2008, approximately 25% fewer than in the first quarter of 2008. Limited second quarter data from Western Australia were available at the time of preparing this report. Taking this into account, the overall count of cases for the remainder of Australia appears to be around 10% more than the recent historical mean incidence of salmonellosis at this time of each year.

During the second quarter of 2008, the 25 most common *Salmonella* types in Australia accounted for 1,103 cases, 64% of all reported human *Salmonella* infections. Twenty of the 25 most common *Salmonella* infections in the second quarter of 2008 were also among those most commonly reported in the preceding quarter.

Increases above the historical average of *S.* Typhimurium phage type 135 (particularly in Victoria, New South Wales and South Australia) and *S.* Typhimurium phage type 44 (in Victoria and New South Wales) account for the greatest proportion of the overall national increase in salmonellosis.

Smaller, more localised increases during the second quarter of 2008 included *S.* Typhimurium phage types 126 and 120, and *S.* Johannesburg (all in Victoria), *S.* Paratyphi B biovar Java phage type Dundee, *S.* Typhimurium phage type U290, *S.* Montevideo and *S.* Wangata (in New South Wales), and *S.* Typhimurium phage type 9 (in the Australian Capital Territory).

Cases of *S.* Virchow phage type 8 were largely confined to Queensland during the second quarter of 2008. This contrasts with the first quarter when this typically Queensland *Salmonella* was reported widely from the other states and territories.

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

Table 6. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 April to 30 June 2008, as reported to 18 July 2008

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total all *Salmonella* for quarter	28	505	86	417	144	38	455	39	1,712
Total contributing *Salmonella* types	13	104	33	101	48	14	99	23	210

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

Table 7. Top 25 *Salmonella* types identified in Australia, 1 April to 30 June 2008, by state or territory

National rank	*Salmonella* type	State or territory								Total 2nd quarter 2008	Last 10 years mean 2nd quarter	Year to date 2008	Year to date 2007
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
1	*S.* Typhimurium PT 135	4	115	0	35	22	5	86	0	267	144	649	446
2	*S.* Typhimurium PT 9	8	40	1	14	18	0	46	0	127	124	260	558
3	*S.* Typhimurium PT 44	1	24	0	9	3	3	76	0	116	25	224	294
4	*S.* Typhimurium PT 170	3	31	0	14	0	0	21	0	69	79	167	199
5	*S.* Saintpaul	0	5	8	34	3	0	3	4	57	94	162	227
6	*S.* Birkenhead	0	14	0	27	0	0	0	0	41	62	126	143
7	*S.* Infantis	0	16	5	3	9	0	7	0	40	38	118	97
8	*S.* Typhimurium PT 126	2	17	0	1	0	0	15	0	35	27	87	28
9	*S.* Chester	0	4	2	15	5	2	3	3	34	41	93	111
10	*S.* Mississippi	0	0	0	2	1	18	4	0	25	21	83	110
11	*S.* Aberdeen	0	0	0	22	0	0	1	0	23	35	52	91
12	*S.* Muenchen	0	2	2	11	2	0	3	3	23	35	68	88
13	*S.* Stanley	0	8	0	0	3	0	9	3	23	14	49	67
14	*S.* Montevideo	2	11	1	9	0	0	0	0	23	12	55	83
15	*S.* Anatum	0	6	5	6	2	0	1	2	22	22	46	41
16	*S.* Hvittingfoss	0	1	4	15	0	0	0	1	21	36	54	80
17	*S.* Waycross	0	7	0	14	0	0	0	0	21	31	58	68
18	*S.* Paratyphi B bv Java PT Dundee	1	19	0	0	1	0	0	0	21	3.1	31	7
19	*S.* Virchow PT 8	0	0	1	18	0	1	0	0	20	71	116	161
20	*S.* Typhimurium PT 197	2	6	0	5	3	1	1	0	18	25	73	135
21	*S.* Typhimurium PT U290	1	16	0	1	0	0	0	0	18	13	40	26
22	*S.* Agona	0	6	0	2	2	0	3	2	15	17	28	40
23	*S.* Weltevreden	0	1	7	4	1	0	1	1	15	14	42	40
24	*S.* Typhimurium PT 135a	0	0	2	0	13	0	0	0	15	8	31	36
25	*S.* Ball	0	1	13	0	0	0	0	0	14	13	25	20

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

Meningococcal surveillance

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

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 *2008;32:135.*

Laboratory confirmed cases of invasive meningococcal disease for the period 1 April to 30 June 2008, are included in this issue of Communicable Diseases Intelligence *(Table 8).*

Table 8. Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 April to 30 June 2008, 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	08			2	2									2	2
	07			0	1						1			0	2
New South Wales	08			9	13	2	3	1	2	1	1			13	19
	07			5	17	3	6	2	2	1	1	3	4	14	30
Northern Territory	08					1	2							1	2
	07				1	1	1							1	2
Queensland	08			25	41	0	2			1	1			26	44
	07			8	19	1	1					1	1	10	21
South Australia	08			5	7									5	7
	07			3	4									3	4
Tasmania	08													0	0
	07									1	1			1	1
Victoria	08			20	24			1	1			3	3	24	28
	07			15	21	2	2	3	3	1	1	1	1	22	28
Western Australia	08			5	8								1	5	9
	07			4	7									4	7
Total	08			65	95	3	7	2	3	2	2	3	4	76	111
	07			35	70	7	10	5	5	3	3	5	6	55	94

OVERSEAS BRIEF

Reporting period 1 April to 30 June 2008

The Overseas brief highlights disease outbreaks during the quarter that were of major public health significance world-wide or those that may have important implications for Australia.

Chikungunya in India

A large outbreak of chikungunya was reported in early April 2008 in the districts of Sullia, Puttur and parts of Bantwal in Karnataka State and by the end of May had reached the neighbouring Kasaragod district and northern Kerala State.[1,2] Chikungunya is endemic in India and periodic outbreaks are reported. Unconfirmed reports put suspected cases at more than 50,000.[3] Large case numbers are common, with the last notable outbreak in 2006 affecting 8 states across India with approximately 1,427,700 cases (758,458 of these from Karnataka State and 80,000 from Kerala State).[4]

Cholera in Viet Nam

Between 5 March and 22 April 2008, the Ministry of Health of Viet Nam reported 2,490 cases of severe acute watery diarrhoea including 377 that were positive for *Vibrio cholerae*. The serotype was identified as 01 Ogawa.[5] By the end of 4 July, 143 acute diarrhoea cases and 649 cholera cases had been reported.[6] In 2007, the World Health Organization (WHO) reported 1,946 cases of cholera in Viet Nam.[7]

Dengue and dengue haemorrhagic fever

Pacific update

The dengue virus infection outbreak which began in late December 2007, continued into April throughout Fiji, with nearly 24,000 suspected cases and 1,600 hospitalisations reported by the Ministry of Health. There were also 2 isolated reports of travellers to Fiji being diagnosed with dengue virus infection in Australia and the Cook Islands. Dengue virus infection has continued to spread around the Western Pacific Region, with cases reported from the Cook Islands, Kiribati and Tonga.[8]

South East Asia update

Dengue outbreaks in South East Asia continued into the second quarter this year. In particular, Laos and the Philippines were reported to be having severe dengue seasons.

As of 16 May 2008, health officials in Laos reported 581 cases of dengue virus infection including 3 deaths. Abundant rainfall and a high incidence of the dengue virus in neighbouring countries were thought to contribute to the increase in case numbers.[9]

Philippines health officials reported significantly increased numbers of dengue fever (33.9% increase) in the first half of 2008 compared with 2007. The increase in cases is thought to be the result of precipitation from the recent typhoon Frank, which is thought to have favoured mosquito breeding. Dengue virus infection is endemic in the Philippines and cases are expected to rise during the rainy season between June and November.[10]

Hand, foot and mouth disease in Asia

Hand, foot and mouth disease (HFMD) is commonly caused by infection with coxsackievirus A16 and occurs world-wide as individual cases and in outbreaks. Enterovirus strains can also cause HFMD, including enterovirus 71 (EV71). Since March 2008, a growing number of HFMD cases caused by EV71 have been reported in parts of Asia, mainly affecting children.[11] Although the disease is usually self-limiting, cases have been fatal in infants.[12]

China

As of mid-June 2008, the Chinese Center for Disease Control and Prevention (China CDC) has stated that the HFMD outbreak is declining after a peak in case numbers on 14 May 2008. While over 176,000 cases were reported last month, Ministry of Health officials stated that the number of daily reported cases decreased from 11,501 during the outbreak's peak to 3,922 by 5 June 2008. The provinces most affected by this outbreak are Guangdong, Zhejiang, Hebei, Shandong, and Hunan (other provinces were also affected including the municipalities of Beijing and Chongqing).[13] Of the HFMD cases reported in China, most were in children 5 years of age and younger, with the majority of laboratory confirmed cases caused by EV71. The Chinese government has enhanced its surveillance, prevention, and control activities, including implementing a public awareness campaign and monitoring water quality.[11]

China (Hong Kong SAR)

As at the end of June 2008, 100 cases of HFMD had been reported (66% caused by EV71).[14] The number of EV71 reported cases for 2008 was higher than the annual total reported cases in the past decade.[11]

Mongolia

As of 7 July 2008, health officials have reported 2,618 cases of HFMD due to EV71 (up from 1,988 cases at 3 June 2008) since the outbreak started in May 2008. Children aged less than 10 years constitute 83% of cases with 10% of cases in infants under 12 months of age. No deaths have been reported even though approximately 25% of cases have required hospitalisation.[15]

Singapore

As of 2 July 2008, health officials have reported 15,776 cases (none fatal) of HFMD (although cases have been declining since the end of May 2008). As of 3 June 2008, 32% of cases tested as part of the Ministry's sentinel surveillance system were positive for EV71.[14]

Taiwan

As of 8 July 2008, the Centers for Disease Control, R.O.C. Taiwan, (Taiwan CDC) reported a total of 311 confirmed HFMD cases this year, including 10 deaths. The majority of cases were caused by EV71. The number of cases also declined towards the end of the reporting period.[11] Taiwan has experienced 2 EV71 outbreaks over the last 10 years—in 1998, 405 cases (case fatality rate (CFR) 19.3%) and in 2005, 145 cases (CFR 10.3%).[16] Of note, only cases of enterovirus infection with severe complications are required to be reported. Estimates from sentinel surveillance data and the national health insurance database indicate that case numbers are more likely to be around 200,000.[17]

Viet Nam

The Ho Chi Minh City Pasteur Institute has reported 2,357 HFMD cases (including 10 deaths) from the southern provinces of Viet Nam so far in 2008. The majority of these (1,018 cases) occurred in Ho Chi Minh City. In 2007, 2,988 HFMD cases (including 16 deaths) were reported across the country.[18]

Influenza (avian)

During the second quarter of 2008, WHO confirmed 6 cases of human H5N1, 4 of which were fatal, giving a case fatality rate of 67%. This is lower than the number of confirmed cases during the same period in 2007 (15 cases, CFR 60%). The WHO confirmed cases were reported from 3 countries: Bangladesh (1 case), Egypt (2 cases including 1 death) and Indonesia (3 fatal cases). This was Bangladesh's first human H5N1 avian influenza case (retrospectively confirmed by the WHO), and occurred in a 16-month-old boy who was exposed to poultry and slaughtered chickens, and has since fully recovered. There was no evidence of human-to-human transmission of avian influenza during the reporting period.[19]

Influenza (seasonal)

Oseltamivir resistance

From the last quarter 2007 to 13 June 2008, 52 countries have reported to WHO on oseltamivir resistance. A total of 6,978 H1N1 virus isolates were tested, 1,077 (15%) were found to be oseltamivir resistant.[20]

The percentages of H1N1 isolates that tested resistant to oseltamivir during the Northern Hemisphere 2007–2008 season were: Europe 24.3%; USA 10.9%; Canada 26%. All the H1N1 oseltamivir resistant isolates collected in Europe and the USA retained their sensitivity to zanamivir. Genetic analysis of some of the oseltamivir resistant isolates exhibited the H274Y substitution.[21]

Marburg haemorrhagic fever in the Netherlands ex Uganda

A 40-year-old Dutch tourist who developed Marburg haemorrhagic fever (MHF) after recent travel to Uganda, died in the Netherlands on 11 July 2008.[22] This was the first MHF case reported in the Netherlands. While visiting Uganda in June, the woman had entered caves on 2 occasions, and was reportedly exposed to fruit bats during a visit to the 'python cave' in the Maramagambo Forest. This cave is thought to harbour bat species suspected to be reservoirs for filoviruses including Marburg and Ebola.[22]

Measles in Europe

Outbreaks of measles have continued in 2008 with new outbreaks reported across Europe (including from Austria, Germany, Norway, the United Kingdom and Spain), Canada, the United States of America and Nigeria.

Fourteen years after local transmission of measles was halted in the United Kingdom (UK), the UK Health Protection Agency (HPA) announced that almost 10 years of low measles-mumps-rubella (MMR) vaccination coverage across the UK has resulted in sufficient numbers of susceptible children to support the continuous spread of measles.[23] The UK HPA reported 656 confirmed cases of measles as of 30 June 2008. The majority of these cases (450) were in London where school age children are most at risk. Of the 656 measles cases, 75% (494 cases) were in children between one and 18 years of age with approximately 95% in those with documented vaccination. The majority of cases were associated with an identical genotype D4 measles strain (MVs/

Enfield. GBR/14.07) that has now been circulating in the UK for more than a year.[24]

Research conducted by the UK HPA indicated that approximately 1.9 million school children and 300,000 pre-school children are not fully vaccinated for measles, creating the potential risk of a large scale outbreak with tens of thousands of cases. In response to this research and the increasing measles case numbers, the Chief Medical Officer has announced an urgent catch-up measles vaccination campaign to be implemented by Primary Care Trusts across the country and supported by the Department of Health. The offer of MMR vaccination will be prioritised to those aged 13 months to 18 years who have not previously been vaccinated, followed by primary school and then secondary school children who have received only a single dose of MMR vaccine. Those young adults over 18 years who are leaving school to attend higher education will be targeted later.[24]

Polio in Nigeria

During this reporting period the World Health Organization reported a new outbreak of wild poliovirus type 1 (WPV1) in the northern states of Nigeria. Between 1 January and 24 June 2008, 318 cases of wild poliovirus have been reported (287 WPV1 and 31 WPV3). Eight key states (Kano, Katsina, Jigawa, Borno, Sokoto, Bauchi, Kaduna and Zamfara) account for the vast majority of cases and are where approximately 20% of children remained unimmunised in 2007 (an improvement from the more than 50% unimmunised reported in 2006).[25] According to the Polio Expert Review Committee this outbreak is due to failure to immunise with only 42% of children in high polio burden states receiving greater than or equal to 3 doses of vaccine compared with 87% in the polio-free states.[26] Transmission outside the north of Nigeria has been limited but includes sporadic cases in the middle-belt states and small outbreaks of both WPV1 and WPV3 in the previously polio-free southern states.[26]

Nigeria accounts for more than 90% of WPV1 in the world this year[26] and is the only country in Africa where endemic wild poliovirus circulation continues. From 2003 to 2006, polio from northern Nigeria re-infected 20 countries causing outbreaks in countries as far away as Indonesia and Yemin.[27]

The current outbreak has increased the risk of renewed international spread of the virus with new polio cases in neighbouring Benin and western Niger genetically linked to viruses from northern Nigeria. Benin reported its first polio infection in 4 years in April this year, which was confirmed to have spread from the western border of Nigeria. As of 24 June 2008, 9 cases of wild poliovirus, all WPV1, have been reported from Niger (previously polio-free for the past 3 years) with the most recent case (from Maradi) having onset of symptoms on 12 April.[25]

The risk of polio re-infecting countries neighbouring Nigeria is potentially increased with the upcoming rainy season and large-scale population movements expected for the Hajj in the second half of the year.[25,27] The World Health Assembly specifically called on Nigeria to reduce the risk of international spread of polio by stopping the outbreak. In response, the Expert Review Committee for Polio Eradication in Nigeria has proposed 12 months of intensified eradication activities[26] including 2 large-scale rounds of emergency polio immunisation in July and August. In addition, a multi-country immunisation campaign was held across West Africa in mid-June along with heightened surveillance in at-risk countries including those re-infected in 2003 to 2006.[25,27]

Rift Valley fever in Madagascar

On 17 April 2008, the Ministry of Health (MOH) reported an outbreak of Rift Valley fever in humans in 5 regions across Madagascar. The MOH reported 418 suspected cases of Rift Valley fever (including 17 deaths), 59 of which have been laboratory confirmed. The onset of human cases of Rift Valley fever was preceded by cases in animals in early April 2008. Rift Valley fever is endemic in animals in Madagascar with outbreaks occurring from time to time.[28]

Yellow fever

In Africa, the yellow fever endemic zone includes the areas that lie within a band from 15°N to 10°S of the equator, from the Sahara desert to northern Angola, the Democratic Republic of the Congo and the United Republic of Tanzania.[12] During this reporting period cases have been reported in the Central African Republic (2 cases)[29] and Liberia (2 cases including 1 fatal),[30] both of which are situated in this endemic zone.

References

1. ProMED-mail [online]. Chikungunya (19): India (Karnataka). 20 May 2008. Available from: http://promedmail.org
2. Global Pacific Health Information Network. [Online]. (The Hindu). Full alert against chikungunya in northern districts. 24 May 2008. Available from: http://www.gphin.net
3. ProMED-mail [online]. Chikungunya (25): India (Karnataka, Kreala). 26 June 2008. Available from: http://promedmail.org
4. Gideon [online]. Chikungunya. India and the Maldives. Accessed June 2008.

5. WHO Disease Outbreak News [online]. Severe watery diarrhoea with *V. cholerae* positive cases in Viet Nam. 22 April 2008. Available from: http://www.who.int/csr/don/2008_04_22/en/index.html
6. ProMED-mail [online]. Cholera, diarrhoea and dysentery update 2008 (31); 30 July 2008. Available from: http://promedmail.org
7. World Health Organization. Cholera, 2007. *Wkly Epidemiol Rec* 2008;83:269–283.
8. ProMED-mail [online]. Dengue – Fiji (19). 16 April 2008. Available from: http://promedmail.org
9. ProMED-mail [online]. Dengue/DHF update 2008 (21). 26 May 2008. Available from: http://promedmail.org
10. Department of Health National Epidemiology Centre; Republic of the Phillipines [online]. Disease Surveillance report. Morbidity week 14 (30 March to 5 April 2008). Available from: http://www.doh.gov.ph/NEC
11. United States Centers for Disease Control and Prevention. Travellers health, outbreak report. Hand, foot and mouth disease in Asia. Available from: http://wwwn.cdc.gov/travel/contentHandFootMouthAsia.aspx Accessed on 20 August 2008.
12. Heymann DL, ed. *Control of Communicable Diseases Manual* 18th edn. Washington: American Public Health Association, 2004.
13. Chinese Center for Disease Control and Prevention and the Office of the World Health Organization in China. Press Release. China's HFMD toll rises to 43 as girl in Jiangxi succumbs. 19 May 2008. Available from: http://www.chinacdc.net.cn/n272562/n276003/23910.html
14. ProMED-mail [online]. Hand, foot and mouth disease – Asia (19). Center for Disease Control Notice. 3 July 2008. Available from: http://promedmail.org
15. ProMED-mail [online]. Hand, foot and mouth disease – Asia (21): Mongolia. 14 July 2008. Available from: http://promedmail.org
16. Global Pacific Health Information Network [Online]. Central News Agency English News. 16 July 2008. Available from: http://www.gphin.net
17. ProMED-mail [online]. Taiwan IHR Focal Point. Hand, foot and mouth disease – Asia (20): Taiwan. 8 July 2008. Available from: http://promedmail.org
18. ProMED-Mail [online]. Hand, foot and mouth disease – Asia (11). 17 May 2008. Available from: http://promedmail.org
19. World Health Organization. Epidemic and Pandemic Alert Response [online]. Cumulative number of confirmed human cases of avian influenza A/(H5N1) reported to WHO. Available from: http://www.who.int/csr/disease/avian_influenza/country/cases_table_2008_06_19/en/index.html Accessed on 14 August 2008.
20. World Health Organization, Influenza A(H1N1) virus resistance to oseltamivir, Last quarter 2007 to first quarter 2008. Update 13 June 2008. Available from: http://www.who.int/csr/disease/influenza/oseltamivir_summary/en/index.html Accessed 9 September 2008.i
21. European Centre for Disease Prevention and Control, Antivirals and Antiviral Resistance Influenza, updated 28 August 2008. Available from: http://ecdc.europa.eu/en/Health_topics/influenza/antivirals.aspx Accessed 9 September 2008.
22. European Centre for Disease Prevention and Control [online]. Imported Marburg fever case in the Netherlands. updated 16 July 2008. Available from: http://ecdc.europa.eu/en/Health_Topics/ebola_marburg_fevers/Article_20080805.aspx
23. United Kingdom Health Protection Agency [online]. *Health Protection Report* 2008;2 (25): 20 June 2008. Available from: http://www.hpa.org.uk/hpr/archives/2008/hpr2508.pdf Accessed on 20 June 2008.
24. United Kingdom Health Protection Agency [online] *Health Protection Report* 2008;2 (32): 8 August 2008. Available from: http://www.hpa.org.uk/hpr/archives/2008/hpr3208.pdf
25. Global Polio Eradication Initiative [online]. *Monthly situation report* June 2008. Available from: http://www.polioeradication.org/content/general/poliositrepJune2008.asp Accessed on 20 August 2008.
26. Global Polio Eradication Initiative [online]. Urgent operational improvements critical to stop Nigeria outbreak, says Expert Review Committee. 15 July 2008. Available from: http://www.polioeradication.org/meeting_detail.asp?day=10&month=7&year=2008. Accessed on 20 August 2008.
27. World Health Organization [Online]. Event Information site. (IHR National Focal Points). Nigeria/Poliomyelitis; 17 June 2008 and updated 15 August 2008. Available from: http://www.who.int/csr/alertresponse/ihreventinfo Accessed on 17 June 2008.
28. World Health Organization. Epidemic and Pandemic Alert Response [online]. Rift Valley fever in Madagascar. 18 April 2008. Available from http://www.who.int/csr/don/2008_04_18a/en/index.html Accessed on 21 April 2008.
29. World Health Organization. Epidemic and Pandemic Alert Response [online]. Yellow fever in the Central African Republic. Available from: http://www.who.int/csr/don/2008_05_20/en/index.html
30. World Health Organization. Epidemic and Pandemic Alert Response [online]. Yellow fever in Liberia – update. Available from: http://www.who.int/csr/don/2008_04_25/en/index.html

Correction

The article school-based vaccinations of a new human papilloma virus vaccination published in *Communicable Diseases Intelligence* (Reeve C, De La Rue S, Pashen D, Culpan M, Cheffins T. School-based vaccinations delivered by general practice in rural north Queensland: an evaluation of a new human papilloma virus vaccination program. *Commun Dis Intell* 2008;32:94–98.) listed 'fainting' as a 'significant adverse event'. Fainting is actually a non-significant event and is not uncommon in mass vaccination programs of adolescent girls.

In addition, the authors would like to acknowledge Queensland Health as the funding source for the school-based vaccination program.

Staff changes

Many thanks from the Communicable Disease Network Australia

Paul Roche has been with the Surveillance Unit (in various forms) for over seven years and has been in many ways the backbone for much of surveillance through the years. He has always been unswervingly generous, calm and professional in giving of his time and expertise. He is generous with his words too – as you only have to look at most any issue of *CDI* to affirm. He has supported several subcommittees – NTAC and the IPD Working Group are the two I am most familiar with – and his support has been invaluable. He has also done some succession planning and for that we thank him – and welcome his successors – to big shoes.

So, many and sincere thanks to you, Paul. I know you are moving on to lofty places – where your knowledge, skills-and language will be well appreciated and you will also find many challenges. A fond farewell.

Sincerely
Vicki Krause
Chair, CDNA