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Annual reports

Surveillance of antibiotic resistance in *Neisseria gonorrhoeae* in the WHO Western Pacific and South East Asian Regions, 2007–2008

The WHO Western Pacific and South East Asian Gonococcal Antimicrobial Surveillance Programmes

Abstract

Long-term surveillance of antimicrobial resistance in *Neisseria gonorrhoeae* has been conducted in the World Health Organization (WHO) Western Pacific Region (WPR) to optimise antibiotic treatment of gonococcal disease since 1992. In 2007 and 2008, this Gonococcal Antimicrobial Surveillance Programme (GASP) was enhanced by the inclusion of data from the South East Asian Region (SEAR) and recruitment of additional centres within the WPR. Approximately 17,450 *N. gonorrhoeae* were examined for their susceptibility to one or more antibiotics used for the treatment of gonorrhoea by external quality controlled methods in 24 reporting centres in 20 countries and/or jurisdictions. A high proportion of penicillin and/or quinolone resistance was again detected amongst isolates tested in North Asia and the WHO SEAR, but much lower rates of penicillin resistance and little quinolone resistance was present in most of the Pacific Island countries. The proportion of gonococci reported as 'resistant', 'less susceptible' or 'non-susceptible' gonococci to the third-generation cephalosporin antibiotic ceftriaxone lay in a wide range, but no major changes were evident in cephalosporin minimal inhibitory concentration (MIC) patterns in 2007–2008. Altered cephalosporin susceptibility was associated with treatment failures following therapy with oral third-generation cephalosporins. There is a need for revision and clarification of some of the *in vitro* criteria that are currently used to categorise the clinical importance of gonococci with different ceftriaxone and oral cephalosporin MIC levels. The number of instances of spectinomycin resistance remained low. A high proportion of strains tested continued to exhibit a form of plasmid mediated high level resistance to tetracyclines. The continuing emergence and spread of antibiotic resistant gonococci in and from the WHO WPR and SEAR supports the need for gonococcal antimicrobial resistance surveillance programs such as GASP to be maintained and potentially expanded. *Commun Dis Intell* 2010;34(1):1–7.

Keywords: annual reports; antimicrobial resistance; *Neisseria gonorrhoeae*

Introduction

The World Health Organization (WHO) Western Pacific (WPR) and South East Asian Regions (SEAR) have a continuing high incidence of gonorrhoea, but treatment and public health management of gonococcal disease in both regions have been severely compromised over many years by increasing antimicrobial resistance (AMR) in *Neisseria gonorrhoeae*. Currently, treatment of gonorrhoea in the public sector of 'Asian' countries in the WHO WPR and in the WHO SEAR is substantially based on the use of third-generation cephalosporin agents, most notably the injectable ceftriaxone, although there are a wide range of dosing regimens used.[1] The oral third-generation cephalosporin most commonly used is cefixime, but dosing regimens are more uniform.[1] These antibiotics are employed as single-dose treatments. Other injectable and oral cephalosporins are also used in some jurisdictions.[1] There is also widespread resistance to penicillins, early generation cephalosporins and quinolones in the 'Asian' group of WPR and in SEAR countries.[2,3] In the 'Pacific Island' or 'Oceania' group of countries within the WHO WPR, the penicillin group of agents remains the recommended treatment in a number of settings.[2] Other antibiotics such as spectinomycin and azithromycin are also recommended and used in some countries, although drug availability and cost limit their wider use. There are few reliable data on antibiotic usage and availability in the private sector in the WHO WPR and SEAR, but anecdotally, a wide variety of antibiotics are used, often in suboptimal doses.[1]

The WHO[4] and others[5,6] recommend that treatment options be refined by data from surveillance of AMR in *N. gonorrhoeae* and that use of an antibiotic for routine treatment be discontinued when therapeutic failure and/or AMR reaches a level of 5%. The WPR Gonococcal Antimicrobial Surveillance

Programme (GASP) has documented the emergence and spread of AMR in *N. gonorrhoeae* in the WHO WPR since 1992[2,7] to provide information for action and to optimise the antibiotic treatment for gonorrhoea. The WHO SEAR GASP has published similar data intermittently.[3] Considerable concerns have been expressed following the appearance and spread of gonococci 'non-susceptible' to the later-generation cephalosporins in the WHO WPR.[8–11] Their recognition followed documentation of treatment failures with several oral third-generation cephalosporins.[8,10,12] The gonococci involved were usually also resistant to other antibiotics, including penicillins and quinolones and would be classified as 'multi-drug resistant gonococci' by recently proposed criteria.[4]

This report provides an analysis of antimicrobial resistance in *N. gonorrhoeae* in the WHO WPR derived from the results of the WPR GASP surveillance for the calendar years 2007 and 2008, and is augmented by equivalent data in a number of centres in the WHO SEAR. The difficulties currently experienced with reliable detection and reporting of cephalosporin 'non-susceptible' gonococci[4] are discussed.

Methods

The methods used by the WHO WPR GASP have been published[7] and provide full details of the source of isolates, sample populations, laboratory test methods and quality assurance programs (EQAS) used to generate data. These general principles were unaltered in 2007–2008 and were also applied to centres in the WHO SEAR. However, there has been a continuing expansion of the *N. gonorrhoeae* comprising the panel strains used in WHO WPR and SEAR EQAS programmes so as to reflect the impact of emerging resistance initially to the quinolones and, latterly, the third-generation cephalosporins and issues related to the detection of these forms of resistance.[13,14]

Results and discussion

A total of 17,458 *N. gonorrhoeae* isolates were examined for their susceptibility to one or more antibiotics used for the treatment of gonorrhoea by EQAS controlled methods in the 2 years 2007–2008 in 24 reporting centres in 20 countries and jurisdictions: 16 in the WHO WPR and four in SEAR countries. There are important limitations that apply to data generated from surveys of this kind. Inevitably, low sample numbers only were available in some centres. This is for several reasons, including abandonment of laboratory-based diagnostic culture facilities where syndromic management is used and, more recently, substitution of diagnostic nucleic amplification assays for culture based approaches. Additionally, resource limitations restrict the capacity for susceptibility testing based on minimal inhibitory concentration (MIC) methodology, even when gonococcal isolates are available, so that disc testing procedures remain the only practical means of *in vitro* assessment of gonococcal antibiotic susceptibility in many situations.[14] Despite these limitations, in the absence of other data sources, and when surveillance is conducted over extended periods under the same conditions, this series has provided reliable trend data for the WHO WPR as a whole.

The consistent results that have been obtained over time in similar countries in the WPR reinforce the significance of the findings, and these data now include the addition of quality controlled information from the WHO SEAR. This allows inferential extrapolation of the data obtained to countries that are unable to participate fully in each surveillance period.

Tables 1–4 show the patterns of resistance to the quinolone and penicillin groups of antibiotics by jurisdiction for each year of the surveillance period. The WHO recommendation that an antibiotic should be removed from standard treatment schedules when the proportion of resistant isolates reaches 5% or more provides guidance for the interpretation of these data. The previously described patterns of resistance to these groups of antibiotics across the WHO WPR[2,7] were again evident in 2007–2008. A high proportion of both penicillin and/or quinolone resistance was detected amongst isolates tested in North Asia and the WHO SEAR, but much lower rates of penicillin resistance and little quinolone resistance was present in most of the Pacific Island countries. In 2007, quinolone resistance or reduced susceptibility was in excess of 90% of all *N. gonorrhoeae* isolates examined in China, the Hong Kong SAR, Mongolia, India, Thailand and Sri Lanka and between 75% and 90% of all isolates in Brunei, Japan, Korea, Malaysia, Singapore and Vietnam. Similarly, high proportions of quinolone resistant gonococci (QRNG) were found in these centres and also in Myanmar in 2008. Lower, but still substantial, proportions of QRNG were present in Australia, the Lao PDR, New Zealand and the Philippines in both years. Penicillin resistance rates were lower than those for the quinolone antibiotics, but followed a similar pattern in WPR and SEAR centres in both years. Not all jurisdictions monitored penicillin resistance because treatment of gonorrhoea with this group of antibiotics has long been discontinued, and even where this surveillance was performed, it was sometimes limited to detection of beta-lactamase production.

N. gonorrhoeae in the WPR and SEAR have also been shown to have decreased susceptibility to third-generation cephalosporins for a number of

years.[4,7–12] This altered susceptibility was accompanied by treatment failures following therapy with oral third-generation cephalosporins in a significant number of cases.[6,8,10,12] No major changes were evident in these patterns over the 2 years of surveillance reported here. There are however concerns in regard to assessments of the proportion of *N. gonorrhoeae* that display altered susceptibility to the third-generation cephalosporin antibiotics in the WHO WPR and SEAR. Surveillance of gonococcal susceptibility to 'third-generation' cephalosporins has emphasised the assessment of ceftriaxone susceptibility because of its wide use throughout both regions[1] so that the MIC data reported here were based mostly on assessment of the *in vitro* susceptibility of gonococcal isolates to the injectable agent ceftriaxone. Recent investigations have shown that the mechanisms of resistance to the third-generation cephalosporins are multiple and complex and involve the aggregation and expression of a number of different genes within *N. gonorrhoeae*.[15–17] The effects of this polygenic involvement on *in vitro* susceptibility of the injectable agents such as ceftriaxone and on the oral cephalosporins such as cefixime and ceftibuten differ considerably, meaning that susceptibility data for ceftriaxone cannot be used to predict reliably the outcomes of treatment with the oral drugs.[4,12] Further, it would also appear that there is a need for revision and clarification of some of the *in vitro* criteria that are currently used to categorise and report on the different MIC levels that arise with both the injectable and oral cephalosporins as the various resistance mechanisms aggregate over time in *N. gonorrhoeae*.[4] This process is currently in train through WHO working groups.[4] It is also now known that other important mechanisms of gonococcal cephalosporin resistance also exist, but are yet to be fully elucidated.[16] In 2007 and 2008, these limitations were evident in reporting and in EQAS data.[14] In 2009, a revised panel of WHO control strains was further developed and distributed in the WPR and SEAR. It is anticipated that more widespread use of these controls from 2010 onwards will better define 'decreased susceptibility', 'non-susceptibility' and 'resistance' to the different third-generation cephalosporin antibiotics.[13,14,18] This is not an easy task because of the need to define 'clinical' as opposed to *in vitro* resistance through improved and more complete examination of gonococci isolated from documented treatment failures. Additionally, different jurisdictions may employ different treatment doses, especially for ceftriaxone[1]

Table 1: Quinolone resistance in 8,376 strains of *Neisseria gonorrhoeae* in the World Health Organization Western Pacific Region and the South East Asia Region, 2007

Country	n	Less susceptible		Resistant		All QRNG	
		n	%	n	%	n	%
Western Pacific Region *(n = 7,507)*							
Australia	3,042	37	1.2	1,456	47.9	1,493	49.1
Brunei	208	50	24.0	120	57.7	170	81.7
China	1,163	41	3.5	1,108	95.3	1,149	98.8
Fiji	320	0	0.0	3	0.9	3	0.9
Hong Kong SAR	1,478	15	1.0	1,437	97.2	1,452	98.2
Japan	329	16	4.9	241	73.3	257	78.1
Korea	56	9	16.1	37	66.1	46	82.1
Lao PDR	9	NS	NS	3	33.0	3	33.0
Malaysia	41	5	12.2	29	70.7	34	82.9
Mongolia	10	4	40.0	6	60.0	10	100.0
New Caledonia	108	0	0.0	0	0.0	0	0.0
New Zealand	301	1	0.3	48	15.9	49	16.3
Papua New Guinea	54	0	0.0	0	0.0	0	0.0
Philippines	99	1	1.0	71	71.7	72	72.7
Singapore	160	12	7.5	122	76.3	134	83.8
Vietnam	129	45	34.9	70	54.3	115	89.1
South East Asian Region *(n = 869)*							
India	36	8	22.2	28	77.8	36	100.0
Sri Lanka	115	12	10.4	94	81.7	106	92.2
Thailand	718	217	30.2	480	66.9	697	97.1

NS Not specified

Table 2: Quinolone resistance in strains of *Neisseria gonorrhoeae* isolated in the World Health Organization Western Pacific Region and the South East Asia Region, 2008

Country	n	Less susceptible		Resistant		All QRNG	
		n	%	n	%	n	%
Western Pacific Region							
Australia	3,110	34	1.1	1,651	53.1	1,685	54.2
Brunei	353	92	26.1	168	47.6	260	73.7
China	1,403	53	3.8	1,348	96.1	1,401	99.9
Hong Kong SAR	1,393	12	0.9	1,362	97.8	1,374	98.6
Japan	328	14	4.3	240	73.2	254	77.4
Korea	141	29	20.6	106	75.2	135	95.7
Lao PDR	9	NS	NS	1	11.0	1	11.0
Malaysia	43	6	14.0	29	67.4	35	81.4
Mongolia	91	35	38.5	34	37.4	69	75.8
New Caledonia	152	2	1.3	3	2.0	5	3.3
New Zealand	258	2	0.8	53	20.5	55	21.3
Papua New Guinea	32	0	0.0	0	0.0	0	0.0
Philippines	84	4	4.8	68	81.0	72	85.7
Singapore	160	10	6.3	119	74.4	129	80.6
Vietnam	153	5	3.3	147	96.0	152	99.3
South East Asian Region							
India	60	10	16.7	50	83.3	60	100.0
Myanmar	12	4	33.3	6	50.0	10	83.3
Sri Lanka	34	0	0.0	26	76.5	26	76.5
Thailand	754	162	21.5	570	75.6	732	97.1

NS Not specified

that may alter MIC/outcome correlates. It is also established that elimination of *N. gonorrhoeae* from some infected sites is also more difficult, e.g. extragenital tract infections are harder to eradicate.[19] The following data are therefore indicative of a well documented increase in the MICs of cephalosporins in gonococci found in both regions. Sixteen centres examined *N. gonorrhoeae* for cephalosporin susceptibility in 2007 and 15 in 2008. The proportions of 'resistant', 'less susceptible' or 'non-susceptible' gonococci lay over a wide range in both years. A large number of centres including Australia, Fiji, India, Japan, Hong Kong, Korea, Laos, Malaysia, New Zealand, Papua New Guinea, the Philippines, Singapore, Thailand, Tonga and Vietnam reported no or very low proportions of strains with altered ceftriaxone susceptibility when tested in large numbers. Most of these centres tested isolates for susceptibility to ceftriaxone only, and it is not surprising that very few strains exhibited altered susceptibility to this antibiotic. Brunei, China, Myanmar and Mongolia all reported ceftriaxone 'resistant' or 'less susceptible' gonococci in much larger proportions. The number of strains tested in the countries and jurisdictions mentioned above approximates those shown in Tables 1–4. Very few isolates were tested separately for their susceptibility to the oral cephalosporin agents. It is thus not possible at present to interpret the *in vitro* data in terms of likely clinical outcome other than in general terms.

Spectinomycin resistance has been only infrequently found in earlier reports in this series. A form of high level resistance due to a single-step ribosomal mutation has been described,[20] and other reports of unexplained low level resistance or decreased susceptibility also occur. Fourteen centres examined gonococci for spectinomycin susceptibility in each year. Only a few sporadic cases of resistance to spectinomycin were found and in a limited number of settings in 2007–2008. Low numbers of isolates (10 or less) with *in vitro* resistance or decreased susceptibility to spectinomycin were found in Brunei, China, Japan, Laos, New Caledonia, Papua New Guinea and Thailand. The number of strains tested in the countries and jurisdictions mentioned above approximates those shown in Tables 1–4. The availability of spectinomycin as a treatment option has been significantly reduced following lack of reliable supplies of the drug. However, spectinomycin is still used as a first line and second line treatment in a number of WPR jurisdictions. Korea is one such

Table 3: Penicillin resistance in strains of *Neisseria gonorrhoeae* isolated in the World Health Organization Western Pacific Region and the South East Asia Region, 2007

Country	n	PPNG		CMRP		All Pen R	
		n	%	n	%	n	%
Western Pacific Region							
Australia	3,042	369	12.1	796	26.2	1,165	38.3
Brunei	308	119	51.3	79	25.6	198	64.3
China	1,163	435	37.4	NS	ND	NS	NS
Fiji	345	22	6.4	12	3.4	34	9.8
Hong Kong SAR	1,478	498	33.7	384	26.0	882	59.7
Japan	329	4	1.2	53	16.1	57	17.3
Korea	56	7	12.5	24	42.9	31	55.4
Lao PDR	9	NS	NS	NS	NS	7*	78.0
Malaysia	41	11	26.8	5	12.2	25	61.0
Mongolia	10	0	0.0	7	70.0	7	70.0
New Caledonia	108	0	0.0	0	0.0	0	0.0
New Zealand	301	5	1.7	60	19.9	65	21.6
Papua New Guinea	54	40	74.1	0	0.0	40	74.1
Philippines	99	89	89.9	0	0.0	89	89.9
Singapore	160	83	51.9	7	4.4	90	56.3
Tonga	55	NS	NS	NS	NS	9*	16.4
Vietnam	129	48	37.2	0	0.0	48	37.2
South East Asian Region							
India	36	13	36.1	4	11.1	17	47.2
Sri Lanka	39	24	61.5	2	5.1	26	66.7
Thailand†	815	701	86.0	16/22	72.7	NS	NS

ND Gonococci in China were examined for penicillinase production only.

NS Not specified

* Laos, Tonga – mechanism of penicillin resistance not specified.

† Thailand, a subset of 22 non-PPNG strains were tested for chromosomal resistance.

country, and an outbreak of spectinomycin resistant *N. gonorrhoeae* was reported there many years ago. Notably, no spectinomycin resistance has been detected there for many years and overall resistance has remained low to this antibiotic in both regions.

Tetracyclines are not a recommended treatment for gonorrhoea in the WHO WPR or SEAR, but historical data on the spread of 1 form of tetracycline resistance, namely a high level plasmid mediated type (TRNG), continues to be monitored in some countries. Eleven centres tested gonococci for this form of resistance in 2007 and 12 in 2008. In 2007 and 2008, up to 50% of gonococci examined exhibited this form of resistance. The proportion of TRNG has been high in some parts of the WPR for many years and between 35% and 55% of all strains in China, Hong Kong, Malaysia, the Philippines, Papua New Guinea, Singapore, Sri Lanka and Vietnam were TRNG, with proportions between 10% and 34% in Australia, India, Korea and New Zealand. The number of strains tested in the countries and jurisdictions mentioned above approximates those shown in Tables 1–4.

The complexities associated with surveillance in the WHO WPR and SEAR GASP have increased as the need for more and better quality surveillance of gonococcal antibiotic resistance has become more obvious.[4–6] Resistance to other antibiotics, such as azithromycin, that are being used either as a primary treatment for gonorrhoea or as adjunctive treatment for other pathogens, is known to occur in the WHO WPR, but substantive data are not yet available. Of concern are recent reports elsewhere of high level azithromycin resistance following widespread use of this antibiotic.[21]

Given the past history of emergence and spread of antibiotic resistant gonococci identified in the WHO WPR and SEAR to other parts of the world,[4] there is a high likelihood that, unless better disease

Table 4: Penicillin resistance in strains of *Neisseria gonorrhoeae* isolated in the World Health Organization Western Pacific Region and the South East Asia Region, 2008

Country	n	PPNG		CMRP		All Pen R	
		n	%	n	%	n	%
Western Pacific Region							
Australia	3,110	373	12.0	994	32.0	1,367	44.0
Brunei	351	201	70.5	44	12.5	245	69.8
China	1,403	543	38.7	ND	NS	NS	NS
Fiji	320	20	6.3	11	3.4	31	9.7
Hong Kong SAR	1,393	434	31.2	169	12.1	603	43.3
Japan	328	2	0.6	88	26.8	90	27.4
Korea	141	18	12.8	77	54.6	95	67.4
Lao PDR	9	NS	NS	NS	NS	7*	78.0
Malaysia	43	23	53.5	0	0.0	23	53.5
Mongolia	91	NS	NS	3	3.3	3	3.3
New Caledonia	152	0	0.0	2	1.3	2	1.3
New Zealand	258	6	2.3	57	22.1	63	24.4
Papua New Guinea	32	20	62.5	2	6.3	22	68.8
Philippines	84	76	90.5	0	0.0	76	90.5
Singapore	160	90	56.3	12	7.5	102	63.8
Tonga	14	1	7.1	0	0.0	1	7.1
Vietnam	153	40	26.1	9	5.9	49	32.0
South East Asian Region							
India	60	20	33.3	5	8.3	25	41.7
Myanmar	12	2	16.7	8	66.7	10	83.3
Sri Lanka	34	18	52.9	1	2.9	19	55.9
Thailand†	733	592	80.8	45/53	84.9	NS	NS

ND Gonococci in China were examined for penicillinase production only.

NS Not specified

* Laos – mechanism of penicillin resistance not specified.

† Thailand, a subset of 53 non-PPNG strains were tested for chromosomal resistance.

control becomes a reality, new forms of resistance will continue to appear and spread. A suggested approach to the closely related issues of gonococcal disease control and AMR control in *N. gonorrhoeae* has recently been published from WHO sources.[4] Implicit in these recommendations is the availability of reliable and verifiable antibiotic resistance surveillance data.

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Annual report of the National Influenza Surveillance Scheme, 2008

Marlena Kaczmarek, Rhonda Owen, Ian G Barr

Abstract

The 2008 influenza season was moderate overall, with fewer laboratory-confirmed cases and influenza-like illness (ILI) presentations than in 2007, which was the most severe influenza season since national reporting of influenza began in 2001. In 2008, the number of laboratory-confirmed notifications for influenza was 1.9 times higher than the 5-year mean. High notification rates were reflected in an increase in presentations with ILI to sentinel general practices and emergency departments. Notification rates were highest in the 0–4 year age group. Unusually, the season was predominantly due to influenza B, with 54% of notifications being influenza B and 43% being influenza A (3% type unknown). The rate of influenza B was higher among the younger age groups, compared with influenza A, which was more common in the older age groups. Of influenza viruses circulating during the 2008 season, A(H3) viruses were predominant and were antigenically similar to the 2008 A(H3) vaccine strain, while the majority of A(H1) strains showed significant drift away from the 2008 A(H1) vaccine strain. There were approximately equal proportions of viruses from the 2 influenza B lineages B/Yamagata and B/Victoria. *Commun Dis Intell* 2010;34(1):8–22.

Keywords: influenza, surveillance, vaccine, influenza-like illness, sentinel surveillance

Introduction

Influenza or 'the flu' is a common, highly infectious respiratory viral disease. The virus spreads from person to person by airborne droplets of exhaled respiratory secretions, especially by coughing or sneezing.[1] Typical symptoms include sudden onset of fever, sore throat, runny nose, cough, fatigue, headache, and aches and pains.

Influenza causes annual epidemics of respiratory disease. Influenza epidemics usually occur during the winter months in temperate climates, causing an increase in hospitalisations for pneumonia, an exacerbation of chronic diseases and also contributing to increased mortality. Those most susceptible include the elderly and very young people, or people of any age who have a higher risk of complications (e.g. pneumonia, heart failure) due to certain chronic medical conditions, e.g. heart, lung, kidney, liver, immune, or metabolic diseases. Most healthy children and adults only have minor symptoms.

Laboratory-confirmed influenza is a nationally notifiable disease in all states and territories and data are reported from each state or territory health department to the National Notifiable Diseases Surveillance System (NNDSS).

In temperate zones of Australia, the annual influenza season runs from May to October, with a peak in notifications around the middle of August. The severity of seasons varies from year to year. Australia experienced moderate to severe seasons in 2003 and 2007 but mild seasons in other years. In recent years, influenza A has been the predominant type circulating in Australia, most commonly the A(H3) subtype.

Surveillance methods

The surveillance of influenza during 2008 was based on the following sources of data:

- notifications of laboratory-confirmed influenza required by legislation in all states and territories, and notified to the NNDSS;
- subtype and strain data of circulating influenza viruses provided by the World Health Organization (WHO) Collaborating Centre for Reference and Research on Influenza;
- consultation rates for influenza-like illness (ILI) identified by sentinel general practitioners;
- consultation rates for ILI identified by hospital emergency departments (EDs);
- testing rates for influenza by New South Wales sentinel laboratories;
- absenteeism data from a national employer; and
- mortality data from the New South Wales Registry of Births, Deaths and Marriages and Australian Bureau of Statistics (ABS).

* Publication of this 2008 *Annual Report of the National Influenza Surveillance Scheme* has been delayed as a result of the response to the H1N1 2009 pandemic. The 2009 annual report will be published in *CDI* later this year.

National Notifiable Diseases Surveillance System

In 2008, laboratory-confirmed influenza was a notifiable disease under state and territory legislation in all jurisdictions. Laboratory notifications were sent to NNDSS for national collation. In this report, data were analysed by the date of diagnosis; the best substitute for the date of onset. The date of diagnosis was set as the date of onset, or where not supplied, the earliest of specimen collection, notification or notification received date. Age, sex, method of laboratory diagnosis and postcode or locality of patient residence were included in NNDSS notifications. Maps were produced using ArcGIS software.

Sentinel general practitioner surveillance

Sentinel general practitioner surveillance schemes for influenza monitor clinical consultations for ILI. In Australia, there are three such schemes: the Australian Sentinel Practices Research Network (ASPREN), which collects data at a national level from approximately 80 general practitioners from 7 states and territories (Australian Capital Territory, New South Wales, Queensland, South Australia, Tasmania, Victoria, and Western Australia), the Victorian Infectious Diseases Reference Laboratory General Practice Sentinel Surveillance Program (VIDRL GPSS), and the Northern Territory Tropical Influenza Surveillance Scheme. ASPREN and the Northern Territory Tropical Influenza Surveillance Scheme report ILI rates throughout the year, while VIDRL GPSS reported from late April to early November in 2008. The national case definition of ILI is: presentation with fever, cough and fatigue. All sentinel surveillance schemes, including ASPREN, used the national case definition for ILI in 2008.

Emergency department surveillance

Rates for influenza-like illness presentation were collected from 29 EDs across New South Wales and up to 9 EDs in Perth, Western Australia. Data were provided to the Surveillance Branch, Office of Health Protection within the Australian Government Department of Health and Ageing (DoHA) on a weekly basis, through the New South Wales Influenza Surveillance Report, and the West Australian Emergency Department Sentinel Surveillance News Report.

Laboratory surveillance

WHO Collaborating Centre for Reference and Research on Influenza

The WHO Collaborating Centres for Reference and Research on Influenza located in Australia, Japan, the United Kingdom and the United States of America (USA), are responsible for analysing influenza viruses collected through an international surveillance network involving 122 national influenza centres in 94 countries. The Melbourne centre analyses viruses received from Australia and from laboratories throughout Oceania, the Asian region and beyond. All virus isolates are analysed antigenically and a geographically and temporally representative number of viruses, together with any strains demonstrating uncharacteristic reactions during antigenic characterisation, are further analysed by genetic sequencing of the viral haemagglutinin gene and the neuraminidase gene. Together with serological and epidemiological data, these form the basis from which WHO makes recommendations in February (for the Northern Hemisphere) and in September (for the Southern Hemisphere) for the vaccine formulation to be used in the following winter. WHO vaccine formulation recommendations are made in the context of strains that are antigenically 'like' laboratory reference strains that are named according to a standard nomenclature for influenza viruses. For human isolates this nomenclature is based on type, the place of isolation, sequential number and year of isolation and for influenza A, the subtype of the HA and NA may also be included in brackets after the designation. An example of a human isolate is A/Sydney/5/97(H3N2), an influenza A(H3N2) virus that was the 5th sequential influenza A isolated in Sydney in the year 1997.

The WHO recommendations[2] are then translated into actual virus strains acceptable to regulatory authorities and vaccine manufacturers, by national and regional committees (such as the Australian Influenza Vaccine Committee).

Sentinel laboratory network

The New South Wales sentinel laboratory network collects influenza virology testing data from 6 major public laboratories, and influenza serology testing data from three. The number of laboratory requests for influenza laboratory tests was obtained weekly from New South Wales Influenza Surveillance Reports.

Absenteeism surveillance

A major nationwide employer, provided weekly absenteeism data in 2008. Absenteeism, defined as an absence recorded as 'sick-leave' for three or more consecutive days, was presented as a rate per 100 employees per week, on an average of 33,290 employees per reporting week.

Mortality

Death certificate data from the New South Wales Registry of Births, Deaths and Marriages provided an estimate of the number of deaths from pneu-

monia and influenza in New South Wales and compared the rate per 1,000 deaths with predicted seasonal mean plus a 95% confidence interval. These were obtained weekly from the New South Wales Influenza Surveillance Report.[3]

Deaths data compiled by the ABS from information provided by the state and territory Registrars of Births, Deaths and Marriages, and coded using the 10th revision of the *International Classification of Diseases and Related Health Problems* (ICD-10) were used to estimate levels of influenza deaths. In this report, deaths for 2008 with an underlying cause of influenza and pneumonia (ICD-10 J10–J18) are presented.[4]

Morbidity data

There was no effective measure of morbidity of disease readily available during the 2008 influenza season. Instead, morbidity was assessed through a number of indicators including:

- absenteeism surveillance;
- Paediatric Intensive Care Unit admissions to intensive care units (ICUs) and deaths data collected by the Australian Paediatrics Surveillance Unit (APSU);
- ED presentations for ILI in New South Wales and Western Australia; and
- ILI presentations to GP surveillance networks.

Hospital admissions for influenza and pneumonia were not available during the 2008 season.

Results

The 2008 influenza season began in mid-July, although there was a very gradual increase in notifications above non-seasonal levels from much earlier in the year. Between February and the start of the season in July, NNDSS notifications were above the 5-year mean. However all sentinel data sources were tracking below or similar to trends seen in previous years.

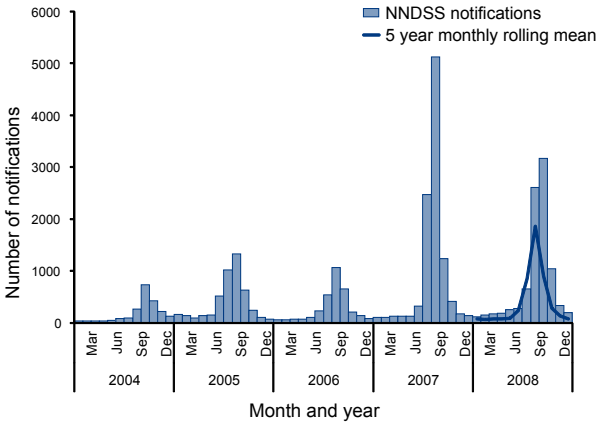
Laboratory confirmed cases

The first increases in notifications of laboratory-confirmed influenza in the 2008 season were registered in early July (week 28) when 105 cases were diagnosed. Notifications peaked in early September (week 36) and were almost back to inter-seasonal levels by the middle of November (week 64) (Figure 1). The total number of notifications for the year was 9,137, which was 1.9 times the 5-year mean.

Geographic spread

In 2008, 41% of laboratory-confirmed influenza notifications occurred in Queensland, 20% in New South Wales, 14% in Victoria, 11% in Western Australia, 5% in South Australia, 4% in Tasmania, 3% in the Australian Capital Territory and 2% in the Northern Territory (Figure 2). The number of notifications peaked at a similar time in most jurisdictions, (in weeks 35–36; weeks ending 29 August and 5 September respectively).

Figure 1: Laboratory-confirmed influenza notifications, 2004 to 2008, Australia, by month and year of diagnosis



Laboratory-confirmed influenza notification rates for 2008 varied across the country, ranging from 24 cases per 100,000 population in Victoria to 91 cases per 100,000 population in the Northern Territory. The crude annual notification rate of influenza infection for Australia was 43 cases per 100,000 population (Table 1).

The Map shows rates of laboratory-confirmed influenza in 2008 by Statistical Division* of residence. The highest rates of influenza occurred in Statistical Divisions that encompassed the central region of the Northern Territory and south-west region of Queensland.

Age-sex profile

Age-specific notification rates for laboratory-confirmed influenza reported to the NNDSS in 2008 are shown in Figure 3. The highest notification rates were seen in children aged 0–4 years, which were around 3.4 times higher than for other age groups (98 per 100,000 population compared with a total rate of 39 per 100,000 population for all notifi-

* A Statistical Division (SD) is an Australian Standard Geographic Classification defined area, which represents a relatively homogeneous region characterised by identifiable social and economic links within the region. They consist of one or more Statistical Subdivisions and cover, in aggregate, the whole of Australia without gaps or overlaps. They do not cross state or territory boundaries and are the largest statistical building blocks of states and territories. (Source: ABS)

Map: Notification rates of laboratory-confirmed influenza, Australia, 2007, by Statistical Division of residence

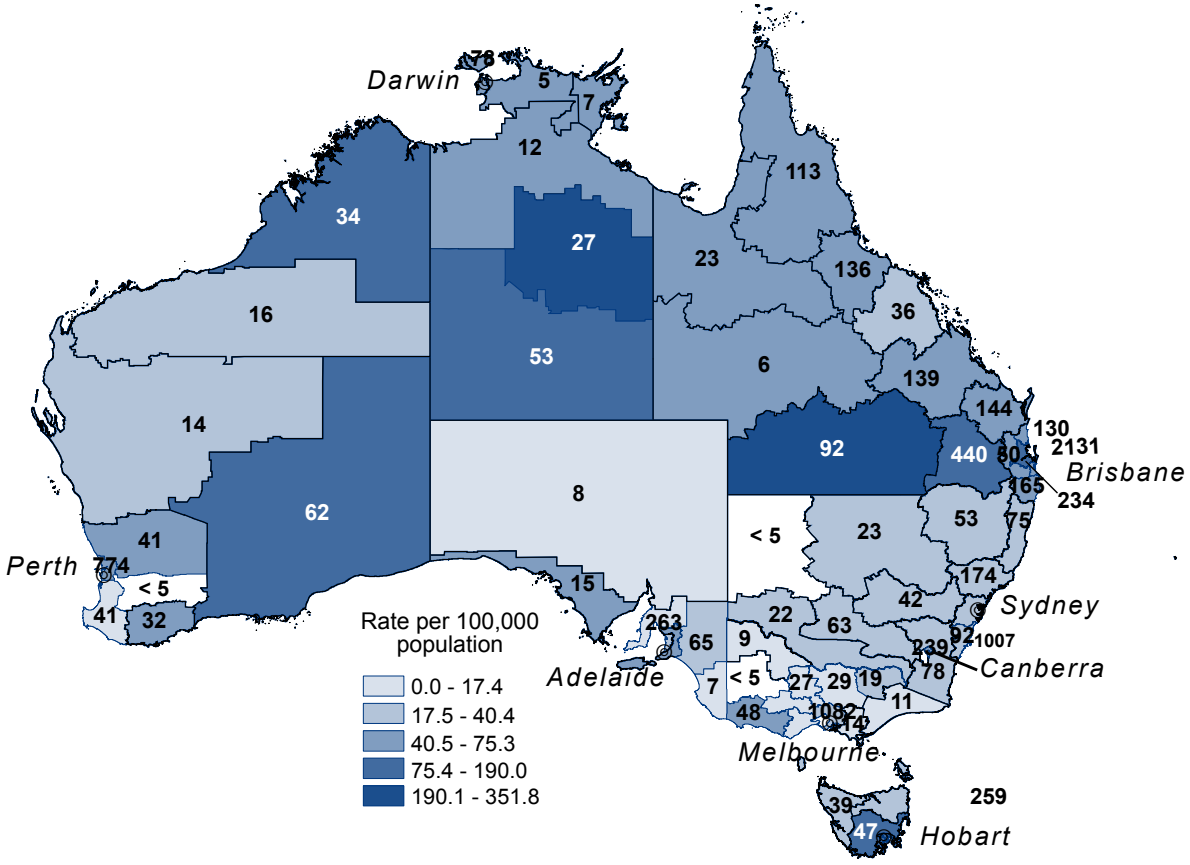


Figure 2. Laboratory-confirmed influenza notifications, June to December 2008, by state or territory and week of diagnosis

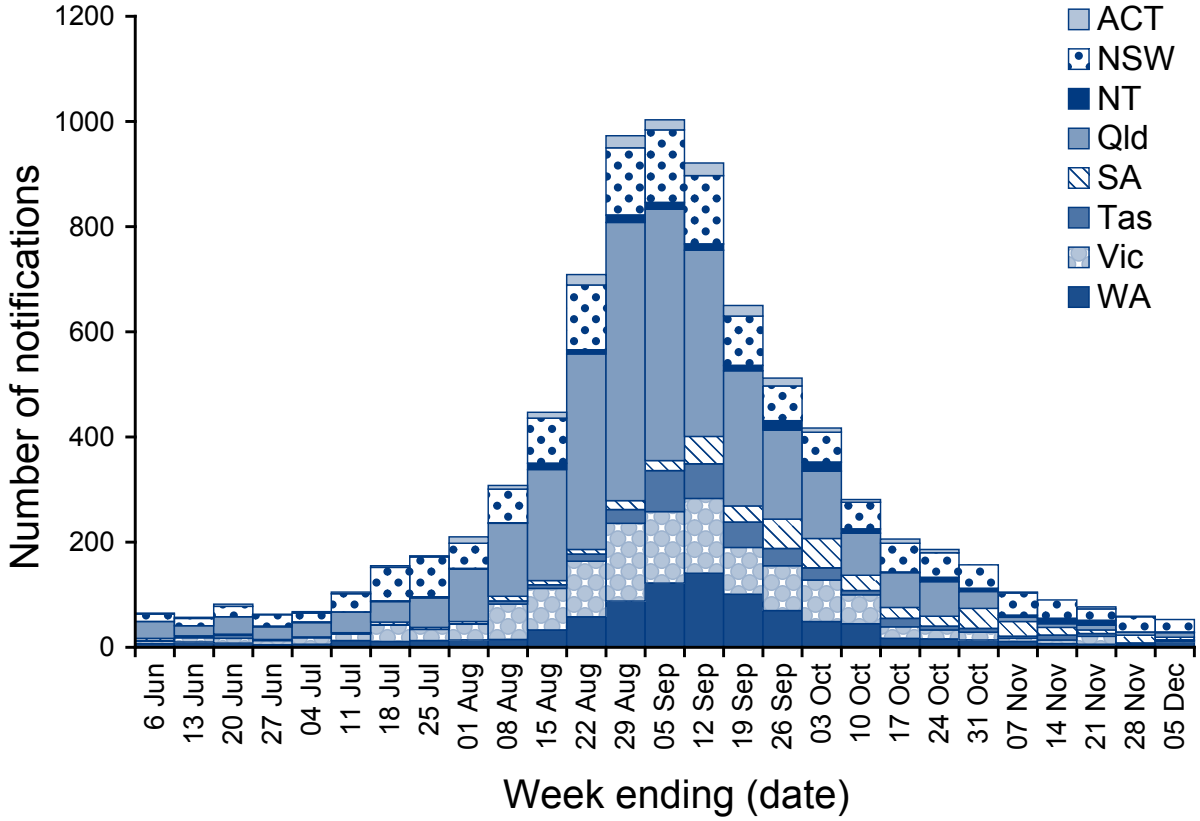


Table 1: Notifications and rates of laboratory-confirmed influenza, 2008, by state or territory and sex

ACT	244	3	71	99	145	58	83
NSW	1,814	20	26	863	944	25	27
NT	199	2	91	97	102	85	96
Qld	3703	41	86	1,718	1,985	80	92
SA	473	5	30	238	235	30	29
Tas	388	4	78	180	207	73	82
Vic	1,300	14	24	628	670	24	25
WA	1,016	11	47	521	495	47	46
Aus	9,137	100	43	4,344	4,783	41	44

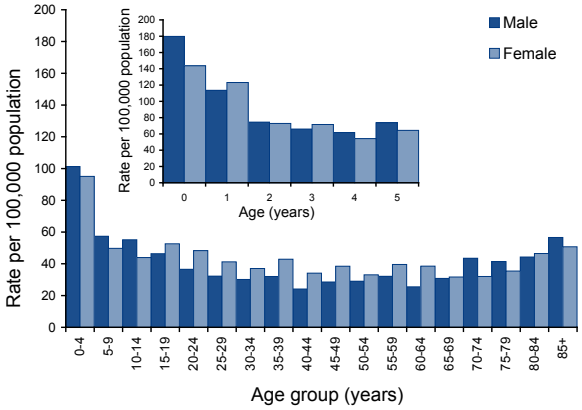
* Ten notifications of unknown sex were excluded from analysis.

cations). People aged 65 years or over are the target for influenza vaccination as they are at an increased risk of complications from influenza. Notification rates for people in this age group were 40 per 100,000 population for males and 38 per 100,000 population for females. This compares with 2007 where influenza notification rates in the 65 years or over age group were 43 per 100,000 population for both males and females.

Total notifications in 2008 were approximately equal for both males (47.5%) and females (52.5%). Notifications were slightly higher in females than in males for persons aged between 15 and 69 years, and 80–84 years. For children and the elderly, notifications for males exceeded those for females. The ratio of males to females in the population was approximately 1:1 for age groups up to 69 years. The ratio of males to females aged 70 years or older was 0.8:1. Although the number of notifications was higher in females aged over 70 years, there are fewer males in the population aged over 70 years, which accounts for the higher rate compared with females in this age group.

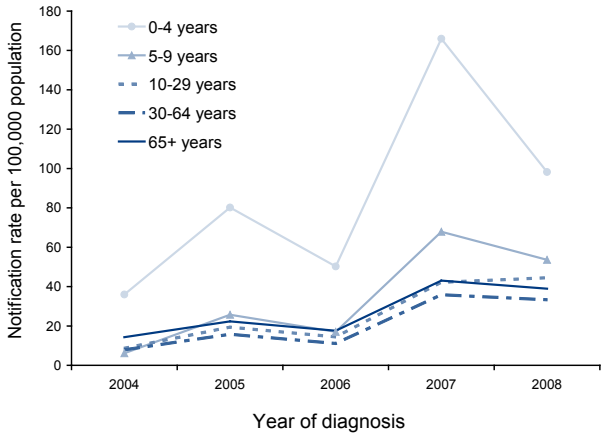
Figure 4 shows the notifications rates for key age groups for the years 2004 to 2008. Overall, notification rates were higher in 2008 for all age groups compared with the previous 4 years, with the exception of the 0–4 and 5–9 year age groups in 2007. Notification rates decreased dramatically in the 0–4 year age group, from 166 per 100,000 population in 2007 to 98 per 100,000 population in 2008. A decrease was also observed in the 5–9 year age group (68 notifications per 100,000 in 2007 to 54 notifications per 100,000 in 2008). Rates in all other age groups remained similar to those in 2007.

Figure 3: Notification rate of laboratory-confirmed influenza, Australia, 2008, by age group (insert – age) and sex*



* Notifications of unknown age (n= 4) or sex (n=10) were excluded from analysis.

Figure 4: Notification rate of laboratory-confirmed influenza reported to the National Notifiable Diseases Surveillance System, Australia, 2003 to 2008, by age group*

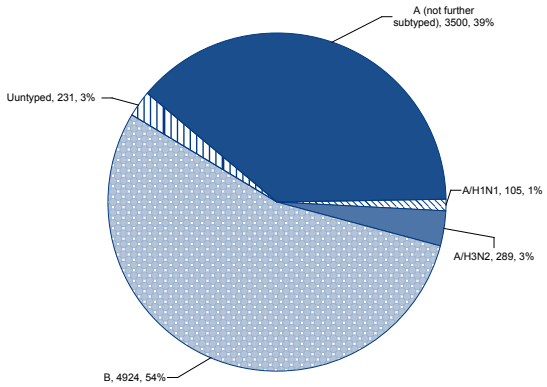


* Notifications of unknown age (n = 4) were excluded from analyis

Virus type and subtype

Analysis of NNDSS influenza typing data indicated a change in the proportion of circulating influenza subtypes from that seen in previous years. Of the 9,137 influenza cases notified to NNDSS in 2008, 98% included typing data. Influenza B was the predominant circulating type comprising 54% of isolates typed compared with 43% that were type A (type was unknown for 3% of influenza cases) (Figure 5). In 2007, 86% of typed isolates were influenza A and 9% were influenza B.

Figure 5: Number of influenza notifications reported to the National Notifiable Diseases Surveillance System, Australia, 2008, by type*



* Notifications of influenza type 'A&B' (n=86) and 'C' (n=2) were excluded from this analysis..

Prior to the influenza season, notifications were predominantly influenza A. However as the season commenced in mid-July, influenza B became more predominant (Figure 6). A predominantly influenza B season has not been recorded in Australia since influenza became nationally notifiable. Since 2004, the proportion of influenza B was 20%, 23%, and 26% in 2004–2006 (respectively), 9% in 2007 and 54% in 2008 (Figure 7).

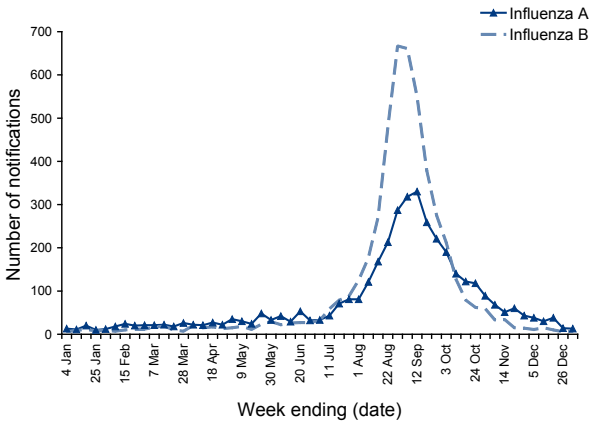
A breakdown of notifications by type and age indicated that compared with influenza A, the rate of influenza B was higher in younger age groups and lower in older age groups (Figure 8).

Virology

Antigenic characterisation

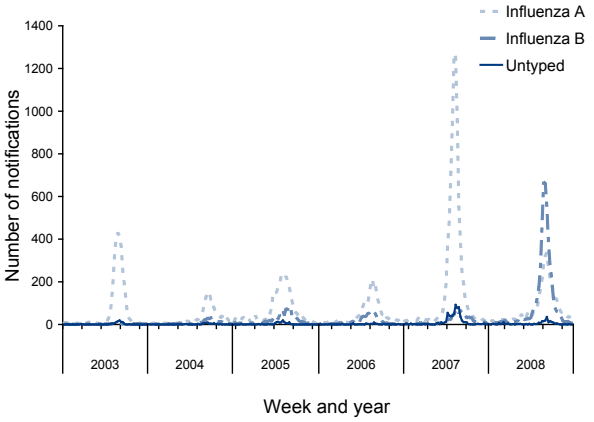
The WHO Collaborating Centre for Reference and Research on Influenza received 1,512 isolates or clinical specimens from Australian laboratories in 2008 that yielded 1,224 viable influenza viruses. All of the viable 2008 viruses were analysed antigenically using the haemagglutination inhibition (H1) assay, which identified 266 (21.7%) as A(H3N2) strains, 144 (11.8%) as A(H1N1) strains and 814 (66.5%) as influenza B strains. This was by far the highest proportion of influenza B viruses seen in the last 10 years with the previous highest proportion occurring in 2006 with 38.5% of the viruses typed as influenza B. Over this period, A(H3N2) viruses have predominated in 8 years, A(H1) in 1 year and now influenza B in 1 year (2008). The 2008 Australian A(H3N2) viruses were mostly antigenically similar to the vaccine strain A/Brisbane/10/2007 by HI assay, with few A/Wisconsin/67/2005 viruses detected. Antigenic analysis of the Australian 2008 A(H1) strains, showed that there was significant drift away from the 2008 Australian vaccine strain A/Solomon

Figure 6: Number of influenza notifications reported to the National Notifiable Diseases Surveillance System, Australia, 2008, by type and week of diagnosis*



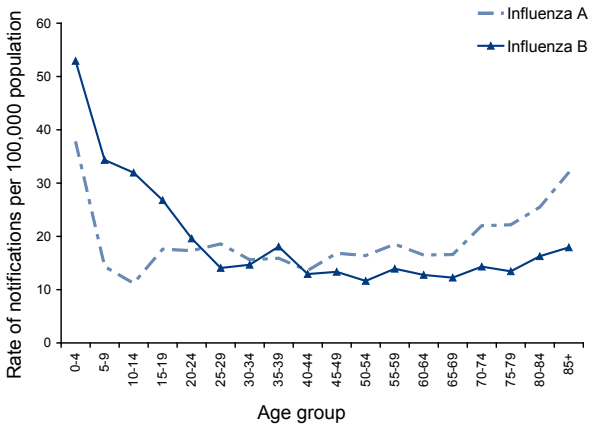
* 319 notifications of influenza were excluded from this analysis: type 'A&B' (n=86), 'C' (n=2) and 'Untyped' (n=231)

Figure 7: Number of influenza notifications reported to the National Notifiable Diseases Surveillance System, Australia, 2004 to 2008, by type and week of diagnosis



Islands/3/2006 to the A/Brisbane/59/2007-like viruses. A large number of influenza B viruses were isolated in 2008, with almost equal proportions of the 2 lineages being present; 50.7% (413) of viruses of the B/Victoria-lineage (B/Victoria/2/87) and 49.3% (401) of viruses of the B/Yamagata-lineage (B/Yamagata/16/88). The B/Victoria-lineage viruses reacted well with ferret sera raised to cell cultured recent B/Victoria-lineage like-viruses such as B/Victoria/304/2006 while the B/Yamagata-like viruses reacted well with antisera raised against the 2008 Australian vaccine strain B/Florida/4/2006 (Table 2).

Figure 8: Rate of influenza notifications reported to the National Notifiable Diseases Surveillance System, Australia, 2008, by type and age group*



* Notifications of 'unknown' age (n=4) and influenza types: 'A&B' (n=86), 'C' (n=2) and 'unknown' (n=231) were excluded from analysis.

Sequence analysis of the variable (HA1) region of the haemagglutinin (HA) gene was undertaken for 115 Australian 2008 viruses (33 A(H1), 29 A(H3) and 53 B) and for 111 neuraminidase genes, (67 H1, 16 H3, 28 B). The phylogenetic analysis of the 2008 (H3) virus HA1 sequences showed that most Australian A(H3) viruses were closely related to A/ Brisbane/10/2007-like viruses with the majority having a conserved amino acid change at position K173Q, although some viruses also fell into other closely related sub-groups (Figure 9).

When the HA1 genes from A(H1) viruses isolated in Australia in 2008 were compared phylogenetically, they fell almost exclusively into the group of viruses represented by A/Brisbane/59/2007 (Figure 10) and now

Figure 9: Evolutionary relationships between influenza A (H3) haemagglutinins (HAI region)

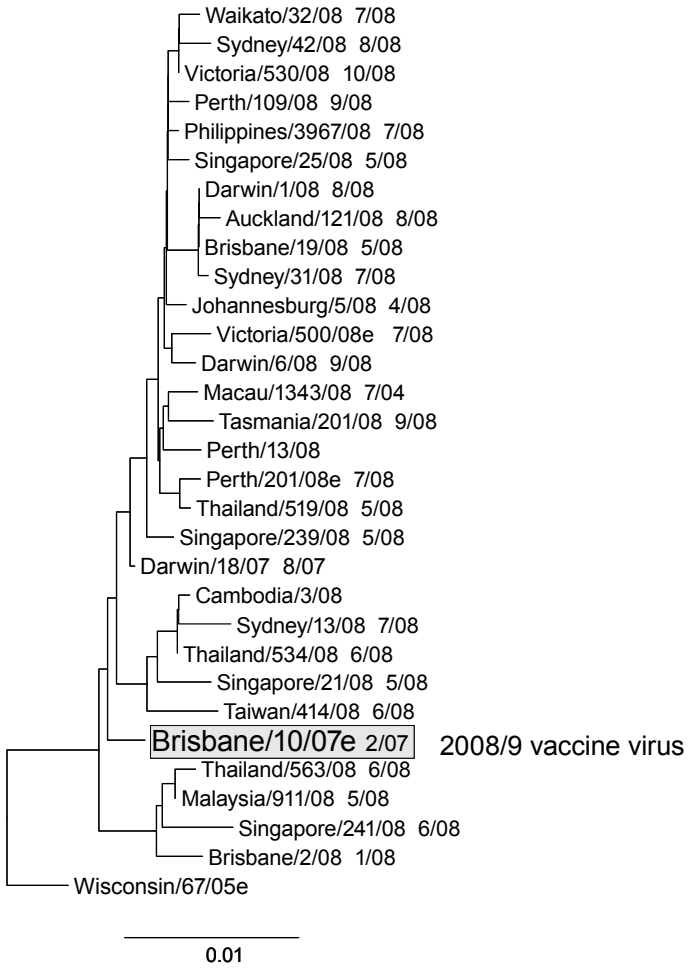
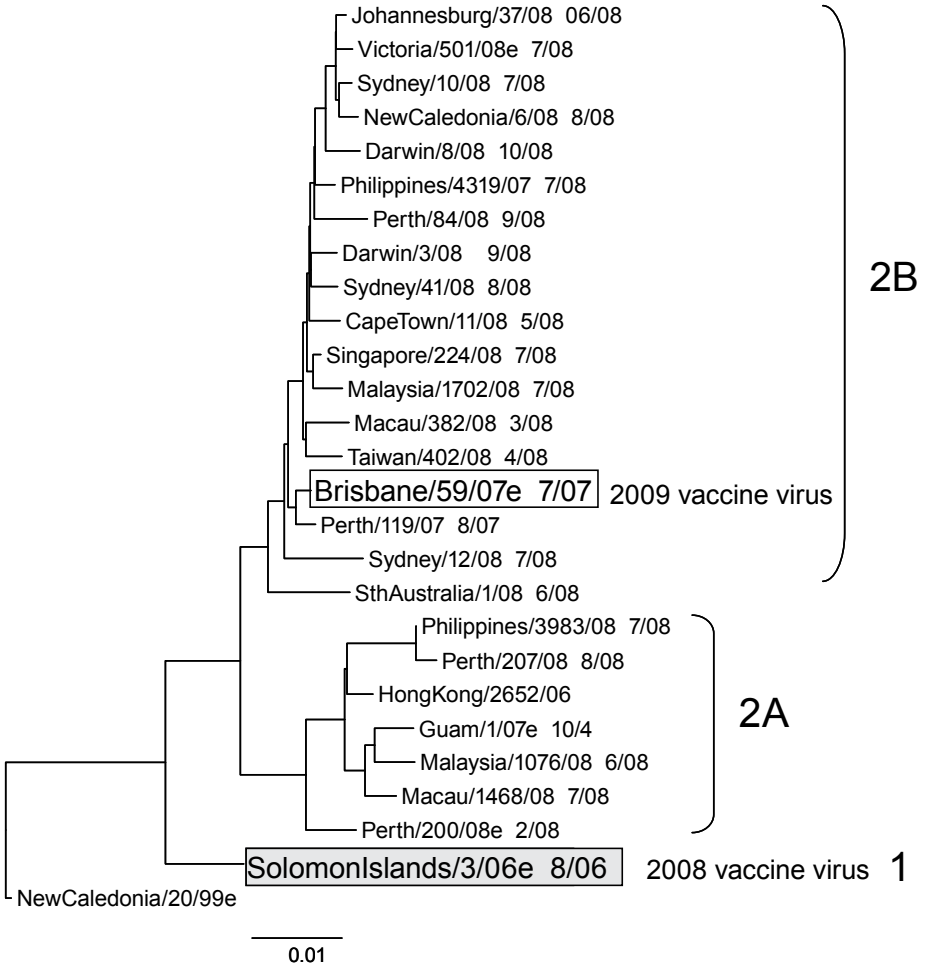


Table 2: Antigenic comparisons of the influenza B viruses, by the haemagglutination-inhibition assay

Viruses	Lineage	Ferret antisera Reciprocal haemagglutination–inhibition titre			
		B/Yamagata-lineage		B/Victoria-lineage	
		B/Florida/4/2006	B/Brisbane/3/2007	B/Malaysia/2506/2004	B/Brisbane/60/2008
Reference viruses					
B/Florida/4/2006	Yamagata	640	640	160	20
B/Brisbane/3/2007	Yamagata	320	640	80	20
B/Malaysia/2506/2004	Victoria	<20	<20	1280	160
B/Brisbane/60/2008	Victoria	<20	<20	640	320
Test viruses					
B/Victoria/227/2008	Victoria	<20	<20	80	320
B/Victoria/523/2008	Yamagata	160	160	<20	<20
B/Sydney/529/2008	Yamagata	160	320	20	<20
B/Perth/213/2008	Victoria	<20	<20	20	80
B/South Australia/180/2008	Victoria	–	–	20	160
B/South Australia/88/2008	Yamagata	320	1280	40	<20
B/Brisbane/134/2008	Yamagata	160	320	–	–
B/Canberra/100/2008	Victoria	–	–	40	160
B/Tasmania/205/2008	Victoria	–	–	40	320

Figure 10: Evolutionary relationships between influenza A (H1) haemagglutinins (HAI region)



distinguished as clade 2B viruses. Few viruses fell into other groups represented by A/Hong Kong/2652/2006 (clade 2C viruses), A/Solomon Islands/3/2006 (clade 2A viruses) or A/Brisbane/193/2004 (clade 1 viruses) (Figure 10).

The Australian 2008 influenza B viruses phylogenetically grouped into their respective lineages, either the B/Victoria or B/Yamagata lineage, with the B/Victoria lineage viruses showing a number of amino acid change from the reference/vaccine strain B/Malaysia/2506/2004 and grouping with the reference virus B/Brisbane/60/2008. The B/Yamagata-like viruses from Australia fell into 3 genetically distinct subgroups represented by B/Florida/4/2006 (Group 1) B/Brisbane/3/2007 (Group 2) and B/Bangladesh/3333/2007 (Group 3) in similar proportions (Figure 11).

Influenza-like illness consultations from sentinel general practitioner surveillance systems

Data from the ASPREN Sentinel GP Surveillance System showed that for 2008 there were 4,213 notifications for ILI. An average of 80 doctors reported to ASPREN each week (range 69–85), with an average of 7,453 consultations per week (range 3,573–8,464).

Overall, the rate of consultations for ILI was lower in 2008 than in 2006 and 2007 (Figure 12). Consultation rates were higher in May and June (weeks 18–25) compared with earlier in the year, decreased slightly in July and early August (weeks 26–32), and then increased sharply in mid-August (week 33). The rate peaked in early September (weeks 36–37), consistent with NNDSS notifications, at 34 ILI cases per 1,000 consultations, and returned to baseline levels by the end of October (week 43). Although ILI and NNDSS notifications

Figure 11: Evolutionary relationships between influenza B haemagglutinins (HAI region)

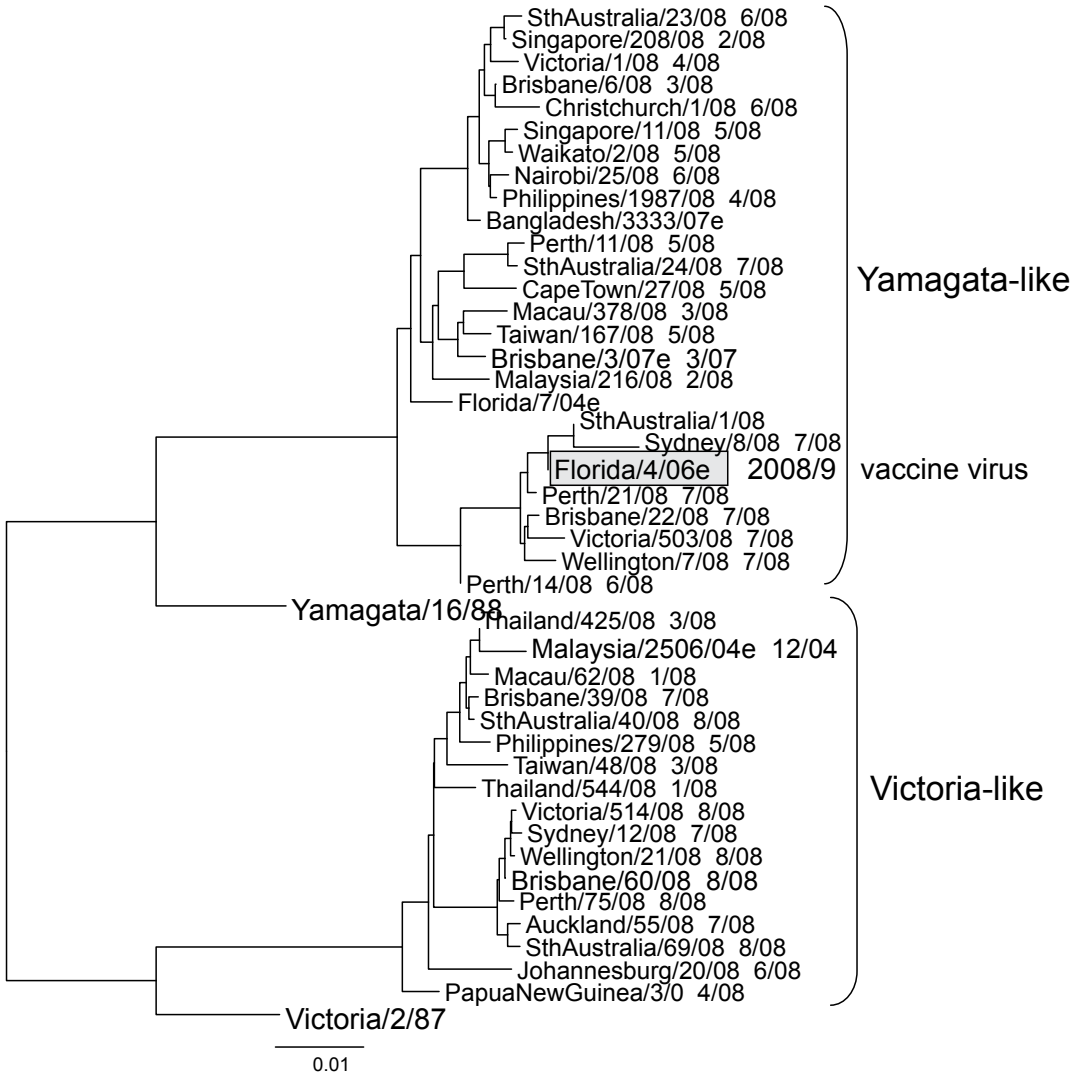
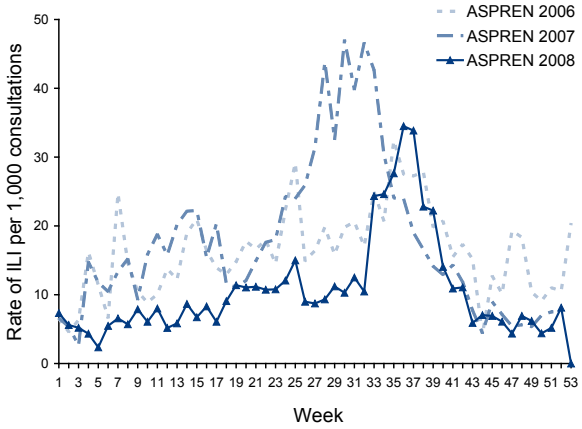


Figure 12: Consultation rates for influenza-like illness, ASPREN, 2006 to 2008, by week



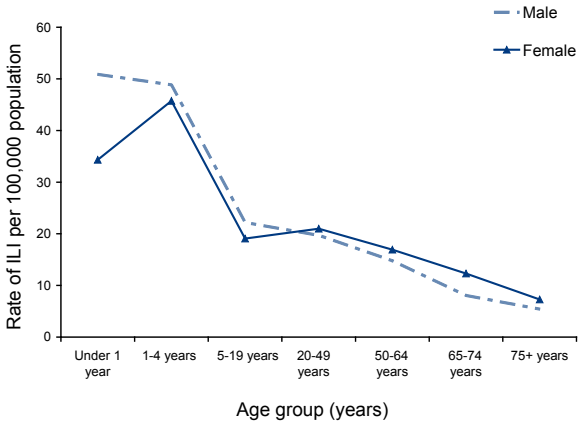
follow the same trends, during the 2008 season ILI notifications were more timely then NNDSS notifications and were used as an early alert and predictor of influenza in the community.

At a state level, ASPREN GPs in Western Australia and New South Wales had higher rates of ILI notifications compared with all the other states and territories.

The VIDRL GPSS followed a similar trend to ASPREN nationally, with more ILI presentations in 2008 than in 2006 but less than 2007, and similar timing for the start and end of the season. This trend was also seen in data from the Northern Territory Tropical Influenza Surveillance System.

A breakdown of ASPREN data by age and sex indicates that the highest rate of ILI presentations were recorded in children under 1 year of age and aged 1–4 years (Figure 13). This is consistent with NNDSS influenza notifications. Male children under 1 year of age had a higher number of presentations compared with female children of the same age. The rate of ILI presentations were similar for both males and females in all other age groups.

Figure 13: Consultation rates for influenza-like illness, ASPREN, 2008, by age group and sex



ASPREN data are not completely representative of the Australian population. In 2008, there were no GPs from the Northern Territory contributing data to ASPREN, and several states and territories had small numbers of GPs reporting. It is also difficult to compare across different years, as representativeness varies over time, due to changes in the number of reporting doctors.

No sampling of ILI patients for laboratory influenza testing occurred at a national level through the ASPREN scheme, although some state systems conducted random testing. Therefore the proportion of ILI presentations tested for influenza, and of those, the proportion positive for influenza could not be determined.

Influenza-like illness – emergency department surveillance (New South Wales and Western Australia only)

Presentations to New South Wales EDs for ILI began to rise in early July (week 27) and peaked at 5 presentations per 1,000 consultations in late August (week 35) (Figure 14). The increase in presentation rates reflected the rise in laboratory confirmed notifications of influenza to NNDSS. Presentation rates in 2008 were higher than in 2006 but did not exceed rates during the peak of the 2007 influenza season.

Presentations to emergency departments in Western Australia for ILI rose gradually from the beginning of the year, following trends seen in 2006 and 2007 (Figure 15). The number of presentations peaked in the week ending 14 September (week 37), which

Figure 14: Rate of influenza-like illness consultations from hospital emergency departments, New South Wales, April to September 2006 to 2008, by week of report

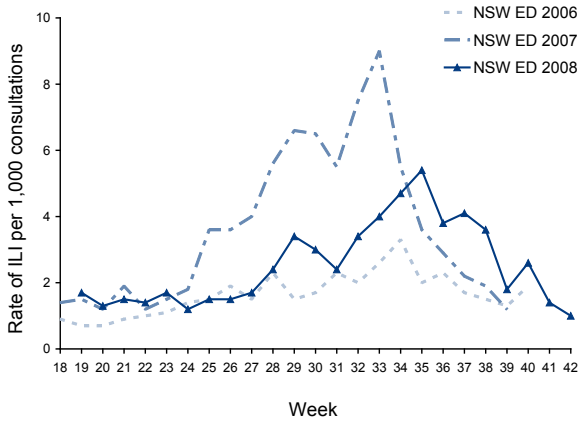


Figure 15. Rate of influenza-like illness consultations from hospital emergency departments, Western Australia, January to December, 2006 to 2008, by week of report

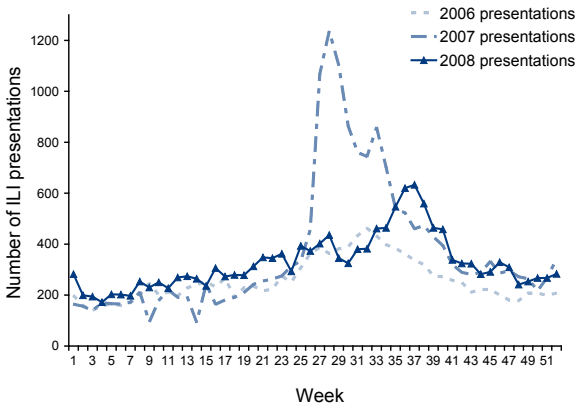
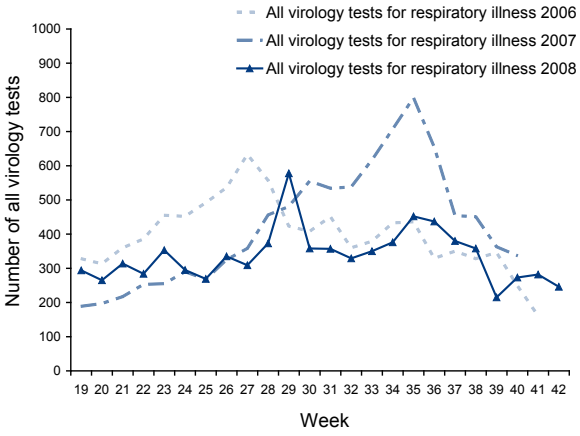


Figure 16: Total virology specimens tested and number positive for influenza, New South Wales, May to October 2006 to 2008



was consistent with the peak of Western Australia influenza notifications to NNDSS. ED presentations for ILI in 2008 were lower than in 2007.

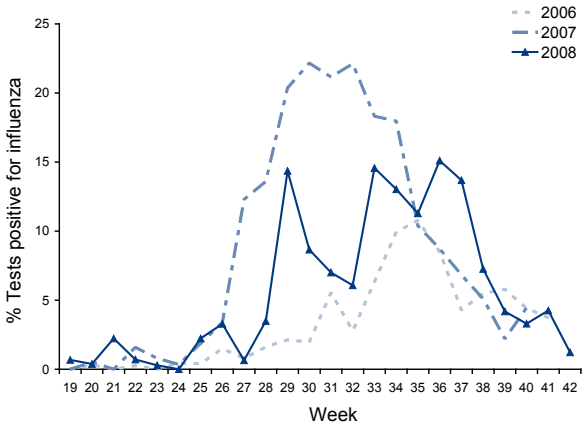
Sentinel ED surveillance data were timely, and a useful indicator of illness severity, but were only available from New South Wales and Western Australia. ED surveillance systems operate in other jurisdictions but these do not routinely report data to DoHA.

Laboratory surveillance

Data from New South Wales sentinel laboratory networks showed that the number of laboratory virology tests for respiratory illness (direct immunofluorescence, nucleic acid tests and viral culture) increased rapidly from baseline levels prior to early July 2008 (approximately 300 per week), to a peak of 578 tests per week in mid-July. This peak resulted from laboratory testing of influenza-like illness outbreaks in World Youth Day pilgrim groups. A second increase in testing during late August and early September were associated with the influenza season. The peak number of tests in 2008 did not exceed peaks in 2006 and 2007 (Figure 16).

The percentage of virology specimens testing positive peaked at approximately 15% in weeks 29, 33 and 36 (weeks ending 18 July, 15 August and 5 September respectively) (Figure 17). The peak in week 29 was again associated with World Youth Day pilgrims, and confirmed that these were influenza outbreaks. The peaks in weeks 33 and 36 were associated with the influenza season, and confirm the increase in the number of seasonal influenza cases. As the number of tests did not increase greatly during these weeks, it also indicates that increasing numbers were not an artefact of increased testing.

Figure 17: Percentage of virology specimens testing positive for influenza, New South Wales, May to October 2006 to 2008

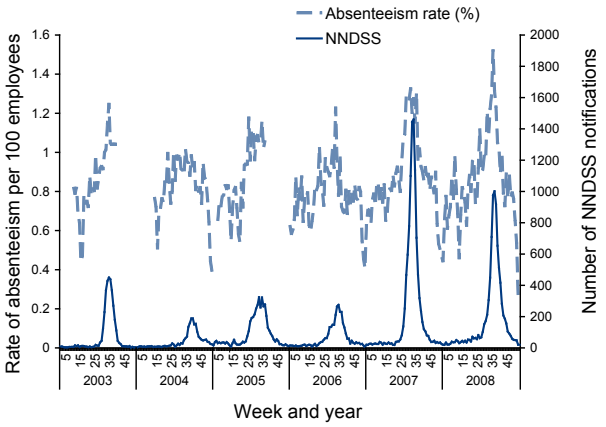


Absenteeism surveillance

Absenteeism surveillance can provide an early warning system of the severity of the influenza season as rates generally begin to increase several weeks before laboratory-confirmed notifications start increasing. Absenteeism rates are likely to be elevated by public health messages to stay home if unwell.

Absenteeism during the 2008 influenza season peaked in the week ending 3 September, which was consistent with the peak in laboratory-confirmed influenza notifications to NNDSS. Although laboratory-confirmed influenza notifications were lower in 2008 compared with 2007, absenteeism peaked higher in 2008, at 1.5% of employees (Figure 18).

Figure 18: National absenteeism (more than 3 consecutive days) rates and National Notifiable Diseases Surveillance System influenza notifications, 2003 to 2008, by week of report



Morbidity

Australian Paediatric Surveillance Unit surveillance

APSU reported that between 1 July and 17 October 2008, there were 43 cases of children aged 15 years or younger admitted to ICUs in Australia, following complications due to influenza infection. Of the admissions, 29 were influenza B, 12 were influenza A and two were unknown. Only 6 children were reported to have been vaccinated for influenza (27 reportedly unvaccinated; 10 unknown). At the time of admission, 12 cases were under 1 year of age, 14 were aged 1–4 years, and 15 were aged 5–15 years (2 ages unknown). The ages at admission ranged from 5 weeks to 14 years, with a median age of 3 years.

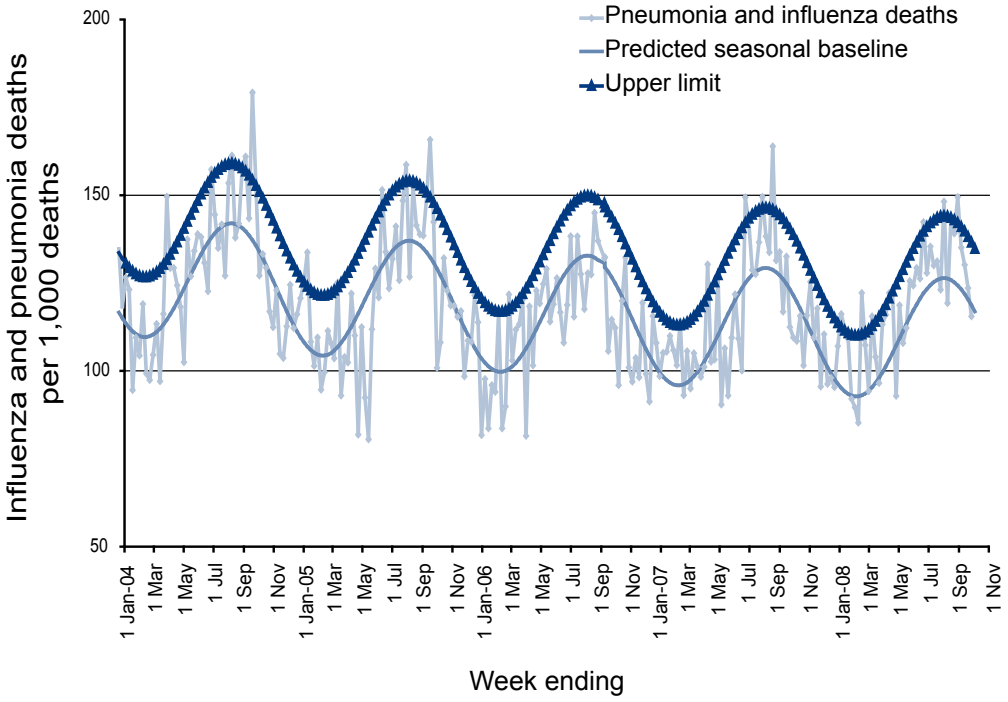
Mortality

Mortality from a primary influenza infection is rare and most of the deaths attributed to influenza occur from complications including pneumonia, obstructive airways disease and sudden cardiac deaths. These occur predominantly in identified risk groups such as those over 65 years or under 6 months of age; or those with chronic medical conditions.

Deaths from pneumonia and influenza – New South Wales

Mortality rates from influenza in New South Wales reported by the Registry of Births, Deaths and Marriages showed that rates of deaths from influenza and pneumonia peaked in early September at approximately 150 per 1,000 deaths (Figure 19). The combined pneumonia and influenza death rates were equal to or higher than the predicted seasonal baseline for the majority of the season. Death rates were equal to or higher than the upper 95% confidence interval of the predicted seasonal baseline for 4 weeks during the season.

Figure 19: Observed and predicted rate of influenza and pneumonia deaths as per New South Wales registered death certificates, 1 January 2004 to 3 October 2008



Australian Bureau of Statistics death data[4]

Influenza and pneumonia (ICD-10 codes J10–J18†) were noted as the underlying cause of death for 1,554 persons in 2008 (1.2% of all deaths). The rate of influenza and pneumonia deaths was 7 per 100,000 deaths. In 2008, there were 932 female and 622 male deaths noted as influenza and pneumonia being the underlying cause of death. The standardised death rate was higher in females, with 9 per 100,000 deaths, compared with males at 6 per 100,000 deaths.

International trends in influenza

Between February and September 2008, influenza activity reported by WHO in Africa, the Americas, Asia, Europe, Australia and New Zealand, was in general mild.

In the Northern Hemisphere, influenza activity following the 2007/08 influenza season began to decline in March in Europe and in April in both Asia and North America. It was reported that A(H1) predominated in many countries in Asia and Eastern Europe, while A(H3) predominated in the USA. Influenza B viruses co-circulated and outbreaks were reported in some countries.

In the Southern Hemisphere, influenza activity began in March, increasing in April, in South America, while in Africa activity started in May, increasing in July. Overall activity declined in August, except for Australia, Brazil and New Zealand. In South America, A(H1) and B viruses co-circulated, in Africa A(H1) predominated, and in Australia and New Zealand A(H3) and B viruses co-circulated.

Overall, influenza activity in New Zealand in 2008 was described as moderate compared with other influenza seasons between 1997 and 2007. In 2008, consultation rates for ILI in New Zealand started to increase at the start of May, and remained at a high level between the start of July (week 27) to the start of September (week 36). During this time there were 2 peaks, one in the middle of July (week 29) and another in the middle of August (week 33). ILI consultation rates returned to baseline levels by the middle of September (week 37). There were a total of 474 hospitalisations in 2008, which was more than in 2007 (347 people), and approximately equal to hospitalisations in 2006 (464 people).

The highest morbidity rates occurred in children aged under one year (120.1 per 100,000 population), followed by children aged 1–4 years (28.8 per 100,000 population) and adults aged over 65 years (15.5 per 100,000 population). These rates are consistent with morbidity seen in Australia during the 2008 season.

Of typed and subtyped viruses in 2008, the majority of viruses were characterised as influenza B (58.3%), with influenza A(H3) representing 41% and influenza A(H1) representing 0.8%.

Interestingly, influenza A(H3) was predominant at the start of the season (during June and July) however during August and September, influenza B was the most widely circulating strain in New Zealand. Prior to the start of the season, influenza B/Yamagata viruses (B/Florida/4/2006-like) predominated, however B/Victoria viruses (B/Malaysia/2506/2004) became more prevalent during the second half of the season and were predominant for the season overall (B/Yamagata 13.6%; B/Victoria 44.6% – of all influenza viruses). Since 1990, there have been 5 influenza seasons in New Zealand that have been predominantly type B: 1991 (92.3%), 1995 (68.8%), 1997 (53.5%), 2005 (87.0%), and 2008 (58.3%).

The full report on the 2008 influenza season in New Zealand is available on their web site.[6]

Outbreaks of A(H5N1) highly pathogenic avian influenza (HPAI) in poultry and wild birds still occurred in many parts of the world in 2008, with the continued exceptions of the Americas and Oceania. According to the official WHO figures, 44 H5N1 human infections occurred in 6 countries during 2008 resulting in 33 deaths. This was lower than cases and deaths in the previous 3 years (2005, 2006 and 2007). No highly pathogenic H5N1 infections were detected in humans or in birds in Australia in 2008. Details of avian influenza cases and deaths is available on the WHO avian influenza web site.[7]

Discussion

The severity and impact of influenza are difficult to measure due to the nature of the illness and limitations of surveillance systems. Influenza surveillance in Australia relies on a myriad of data sources and systems, varying in their ability to detect true cases of influenza. Ideally, the number of laboratory-confirmed notifications would include all cases, rather than just those that have been tested, and sentinel GP and ED surveillance systems would indicate the burden of disease on health systems

† The International Classification of Diseases (ICD) is the international standard diagnostic classification, and is used to classify diseases and other health problems recorded on many types of health and vital records, including death certificates and health records. ICD-10 (ICD version 10) codes J00-J99 relate to diseases of the respiratory system, and J10-J18 are specific for influenza and/or pneumonia.[5]

and the community. Hospitalisation and death data could also be improved to allow for true indicators of severity, morbidity and mortality related to influenza infection. It is possible that notifications in 2008 may have been affected by heightened media attention following the severity of the 2007 season, and it is not currently possible to measure the extent of this impact (if any). Influenza notifications following the 2007 season did not return to the same background levels as in previous years, and as such, notifications at the start of 2008 were slightly elevated in comparison. As all sentinel sources were indicating low levels of influenza-like illness in the community, it is possible that increased notifications early in 2008 were the result of increased testing and as such increased case finding.

Based on available data, the 2008 influenza season in Australia was considered moderate overall in comparison to previous seasons. Laboratory-confirmed influenza notifications were almost 2 times the 5-year mean, although this was less then notifications in 2007 (3 times the 5-year mean). There were no significant differences in laboratory-confirmed influenza notifications during the 2008 season compared with previous seasons in terms of geographic spread, with larger jurisdictions recording higher case numbers, and smaller jurisdictions having higher rates (due to smaller populations). Like previous years, the highest rates of laboratory-confirmed influenza occurred in children under 5 years of age, especially in those under 1 year of age. Although laboratory-confirmed influenza rates remained stable in ages over 10 years, the rate in children aged under 10 years decreased in comparison to 2007. This decrease was significant in children under 5 years of age, which may reflect higher rates of testing in this age group in 2007 following the heightened media attention.

In 2008 for the first time, sentinel GP surveillance system data were analysed by age and sex. It has been widely hypothesised that young children were under-represented in these data, as the vast majority of ILI presentations are in the 20–49 year age group. However, calculated rates indicate that the rate of ILI presentations to ASPREN GPs was highest in children under 5 years of age, especially male children under 1 year of age.

All sentinel data sources indicated that the 2008 season was moderate overall, consistent with laboratory-confirmed influenza data. ILI presentations to GPs and EDs, as well as influenza and pneumonia deaths, were lower in 2008 than in 2007, but higher than 2006.

Australia hosted World Youth Day (WYD) events in Sydney on 15–20 July 2008. Prior to WYD, influenza activity in Australia had been at baseline levels, with influenza notifications and outbreaks predominantly influenza A. Several influenza outbreaks (including a number due to influenza B) occurred in pilgrims who had travelled to Sydney for WYD events. An increase in influenza coincided with pilgrims travelling around Australia, but it cannot be concluded whether this was due to spread of the virus by pilgrims or normal seasonal spread.

The 2008 season was predominantly influenza B, which accounted for 54% of all laboratory-confirmed influenza notifications. Younger age groups had higher rates of influenza B infection than older age groups, who were more commonly infected with influenza A. The WYD outbreaks may have contributed to this trend, as influenza B may have been spread from pilgrim groups to similar age groups as they travelled around Australia or as Australian pilgrims returned home. To confirm that this change had been as a result of pilgrim travel would have required detailed case follow-up and contact tracing, which was not possible due to the high numbers of influenza during the 2008 season.

Of influenza viruses circulating during the 2008 season A(H3) viruses were mostly antigenically similar to the vaccine strain A/Brisbane/10/2007. A(H1) strains however, showed significant drift away from the vaccine strain A/Solomon Islands/3/2006 to the A/Brisbane/59/2007-like viruses. As the season was predominated by influenza B, a large number of influenza B viruses were isolated in 2008. While overall there were approximately equal proportions of viruses from the 2 influenza B lineages (B/Victoria and B/Yamagata), B/Yamagata viruses were predominant at the start of the season, and B/Victoria at the end of the season.

Based on data available during the 2008 Southern Hemisphere influenza season, at their technical meeting in September 2008, the WHO recommended the following influenza virus strains for inclusion in the 2009 Southern Hemisphere seasonal influenza vaccine:

- A/Brisbane/59/2007-like virus (H1N1);
- A/Brisbane/10/2007-like virus (H3N1); and
- B/Florida/4/2006-like virus.

The recommendation for the 2009 Southern Hemisphere vaccine had only one change compared with the 2008 Southern Hemisphere vaccine: a change to the A(H1) virus from A/Solomon Islands/3/2006-like virus to A/Brisbane/59/2007-like virus.

Although various data sources were used to characterise the 2008 Australian influenza season, they were all consistent in indicating that the season

occurred later than usually seen but was moderate overall, which is characteristic of a predominantly influenza B season.

Acknowledgements

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Peer-reviewed articles

Impact of faxed health alerts on the preparedness of general practitioners during communicable disease outbreaks

Alexander Rosewell, Mahomed Patel, Kerri Viney, Andrew Marich, Glenda L Lawrence

Abstract

The NSW Department of Health (NSW Health) faxed health alerts to general medical practitioners during measles outbreaks in March and May 2006. We conducted a retrospective cohort study of randomly selected general practitioners (GPs) (1 per medical practice) in New South Wales to investigate the effectiveness of faxing health alerts to GPs during a communicable disease outbreak. Fax transmission data allowed comparison of GPs sent and not sent the measles alert for self-reported awareness and practice actions aimed at the prevention and control of measles. A total of 328 GPs participated in the study. GPs who were sent the alert were more likely to be aware of the measles outbreak (RR 1.18, 95% CI 1.02, 1.38). When analysed by whether a fax had been received from either NSW Health or the Australian General Practice Network, GPs who reported receiving a faxed measles alert were more likely to be aware of the outbreak (RR 2.56, 95% CI 1.84, 3.56), to offer vaccination to susceptible staff (RR 6.46, 95% CI 2.49, 16.78), and be aware of other infection control recommendations. Respondents reported that the faxed alerts were useful with 65% reporting that the alerts had reminded them to consider measles in the differential diagnosis. This study shows that faxed health alerts were useful for preparing GPs to respond effectively to a communicable disease outbreak. The fax alert system could be improved by ensuring that all general practices in New South Wales are included in the faxstream database and that their contact details are updated regularly. *Commun Dis Intell* 2010;34(1):23–28.

Keywords: fax, health alert, measles, outbreak, communication, immunisation, public health, general practitioner, health department

Introduction

The importance of timely identification and response to significant public health events, including communicable disease outbreaks, has been demonstrated across a variety of epidemiological settings.[1,2] Rapid, mass communication between health departments and community-based clinicians is commonly regarded as a key element in an effective response to such events.[3,4] However, published evidence is sparse regarding the effectiveness of such communications methods in reaching the intended audience and clinicians taking the requested public health actions to identify cases and reduce disease transmission.

Traditionally, the NSW Department of Health (NSW Health) has collaborated with the local Australian General Practice Network (AGPN) to communicate rapidly with community general practitioners (GPs). However, not all GPs in New South Wales are members of the AGPN, nor are medical specialists and a range of other health professionals. The community-wide threat of pandemic influenza and a growing awareness of the need for rapid uniform communication with a range of health professionals during significant health events prompted NSW Health to seek a more comprehensive communication tool for providing up to date information to a more diverse audience.

In 2005, NSW Health investigated a range of options to address this need and procured a commercially available database (Database X). In addition to the contact information for 24,000 medical doctors in New South Wales and the Australian Capital Territory, Database X contains similar information for medical specialists, emergency departments and aged care facilities. Between late March and June 2006, there were 3 measles outbreaks (2 localised New South Wales outbreaks and 1 larger multi-state outbreak),[5,6] which provided the first opportunity to test the utility and effectiveness of the Database X faxstreaming system to New South Wales-based GPs, in combination with the AGPN faxstream process.

Since the 1990s, the incidence of measles in Australia has declined to a point where the disease is now uncommon.[7] As a result, many GPs have either never seen a person with measles or do not consider measles in their differential diagnoses.[8,9] During an outbreak of measles in this context, cases may

present to general practices and emergency departments several times during their infectious period before a correct diagnosis is made. This could result in a high risk of ongoing transmission in health care settings, particularly among unimmunised infants and susceptible adults.[10–12] Rapid, direct, mass communication with GPs and hospitals may minimise the risk of transmission in health care settings and result in better preparedness to control measles and other communicable diseases outbreaks.

Using contact details from Database X, NSW Health faxed measles health alerts to community general medical practices in March and again in May 2006. Information contained in the health alert included a brief description of the measles outbreak, the age groups most susceptible to measles infection and advice on actions required of GPs to help reduce ongoing transmission of measles in the community. These included consideration of measles in the differential diagnosis of patients with a febrile illness, a request to notify possible cases to the local public health unit, advice on collection of appropriate samples for laboratory tests, as well as information about offering measles, mumps, rubella (MMR) vaccine to all susceptible patients, their contacts and practice staff (i.e. those born after 1965 but not vaccinated with 2 doses of MMR), and appropriate infection control measures implemented when a patient with possible measles attended the practice. A record was kept of the practices sent faxes, whether fax transmission was successful, and practices not sent a fax as no fax number was recorded.

We aimed to evaluate the effectiveness of the faxed health alerts in achieving the objectives of improving measles control and prevention by comparing awareness and actions related to the recommendations contained in the health alert among general medical practices recorded as having been successfully sent one or more faxed alerts, and those not sent the alert (i.e. unsuccessful transmission or missing fax number). We also recognised that GPs may have received faxed information from other sources, including the AGPN, and therefore assessed the effectiveness of faxed health alerts by self-reported 'fax received' status as well as documented 'fax sent' status.

Methods

We conducted an historical cohort study of a random sample of general medical practices in New South Wales recorded in Database X. The unit of selection for both the NSW Health faxstream system and our study was the general practice, not individual GPs.

In sample size calculations, we estimated that 363 practices recorded as sent the faxed alert and 116 practices not sent the fax (i.e. a ratio of approximately 3:1) were required to detect differences with 80% power at a significance level of 0.05 in (i) awareness of the measles outbreak (assuming that 80% of practices sent the fax and 65% of those not sent the fax were aware of the outbreak), and (ii) offering MMR vaccine to susceptible practice staff (assuming that 15% of those sent and 5% of those not sent the fax would have done this). We over-sampled the 2 groups of practices assuming that up to 50% of the GPs selected for the study may not participate, based on previous studies of GPs in New South Wales. Random samples were selected from Database X of 725 practices sent the fax and 231 practices not sent the fax (Figure).

In August 2006, a self-administered questionnaire was mailed to 1 GP in each of the 956 randomly selected practices with a letter of invitation to participate in the study from the Chief Health Officer of New South Wales. A reminder letter and the questionnaire were mailed to all non-respondents after a period of 4 weeks. The group (i.e. fax sent and not sent) of each selected GP was identified from a coded sticker applied by research staff to the return-paid envelope. Data were collected under New South Wales public health legislation.

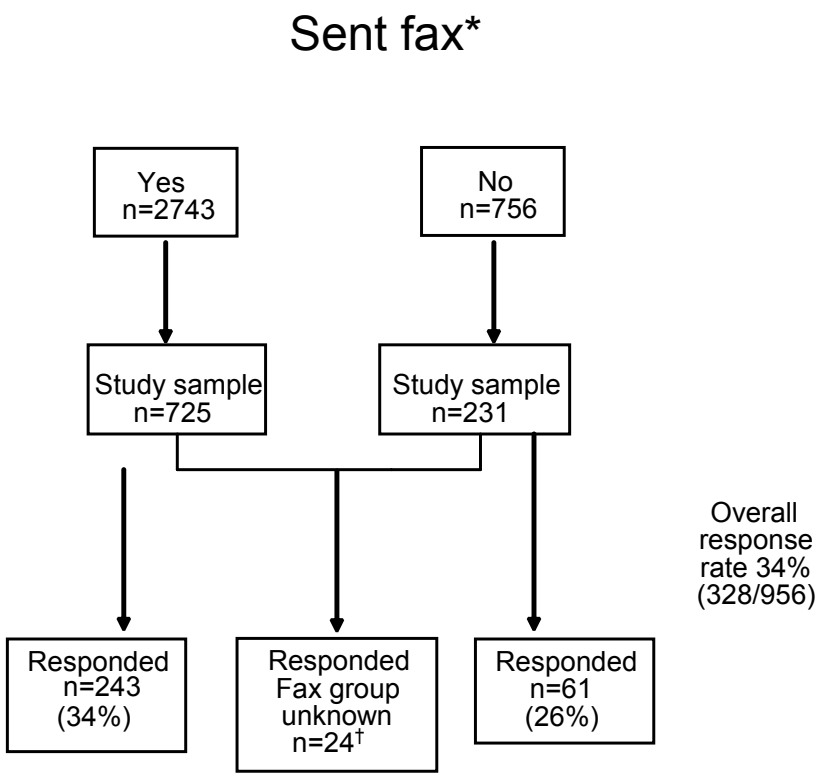
Information collected in the questionnaire included GP and practice demographics, information sources used during the measles outbreak including receipt of faxed measles health alerts from the AGPN, the usefulness of health alerts as well as awareness and implementation of NSW Health recommendations related to measles control.

All data cleaning, recoding and statistical analyses were performed using STATA Version 9 software (Stata Corp, College Station, TX, USA). The representativeness of survey respondents was assessed by comparing practice characteristics recorded in Database X for practices in the 'sent' and 'not sent' groups, from which the samples had been drawn. We calculated relative risks and 95% confidence intervals to compare proportions between groups. Chi-square tests were used to assess statistical significance. Two types of comparisons were conducted: (1) by 'fax sent' status, based on whether the practice was recorded as having been sent at least 1 alert by NSW Health, and (2) by 'fax received' status, based on whether the respondent reported receiving a faxed alert from NSW Health and/or the AGPN.

Results

The overall response rate was 34% after the second mail-out, with 33.5% (243/725) for the 'sent' group and 26.4% (61/231) for the 'not sent' group. Twenty-four surveys were excluded from 1 part of the analysis following loss of identifying stickers from envelopes during postage.

Figure: Recruitment of the study sample



* Based on records of NSW Department of Health.

† General practitioner identifying information was removed during postage.

Practice characteristics for survey respondents were found to be representative of all those sampled and the frames from which they were selected in terms of information contained in Database X (Table 1). Self-reported information from survey respondents in the 'sent' and 'not sent' fax groups showed that the two groups were similar in terms of urban/rural location, and self-reported characteristics including access to a fax machine, the proportion of GPs under 50 years of age, the proportion of female GPs and membership of the AGPN (Table 2). However, solo practitioners were less likely to have been sent a fax (Tables 1 and 2; RR 0.71, 95% CI 0.61, 0.82).

General practitioner knowledge and implementation of measles health alert information

GPs in practices faxed a health alert by NSW Health were more likely to report that they were aware of the measles outbreaks compared with GPs in practices not sent faxed alerts (Table 3; RR 1.18, 95% CI 1.02, 1.38). They were also more likely to report that susceptible staff in the practice had been offered MMR vaccine (RR 1.55; 95% CI 0.99, 2.45; $P < 0.05$). This was also true in practices without routine staff MMR vaccination policies ($P < 0.01$).

Associations were stronger when data were analysed by 'fax received' status (i.e. from NSW Health or the AGPN). For the outcome of being aware of the measles outbreak, the RR was 2.56 (95% CI 1.84, 3.56; Table 3). A higher proportion of GPs who reported that they had received a faxed health alert became aware of specific recommendations for measles control, including immunisation of susceptible staff (RR 6.46, 95% CI 2.49, 16.78; Table 3), isolation of patients with possible measles in the practice (RR 3.30, 95% CI 1.83, 5.97), and notification of suspected cases to a public health unit (RR 4.26, 95% CI 1.93, 9.41) (data not shown elsewhere).

GPs who reported they were a member of the local AGPN were more likely to be aware of the measles outbreak than those who were not part of the network (RR 1.20 $P < 0.05$; not shown elsewhere). The majority (92%) of GPs who reported receiving a faxed measles health alert from NSW Health and/or the AGPN found it useful, and 65% had considered measles in their differential diagnoses. GPs reported that the preferred method of receiving health alerts was by fax (87%) compared to email (24%). Some GPs preferred alerts to be sent by both fax and email (16%).

Table 1: Comparison of study respondents with the study sample and sampling frame for characteristics recorded on Database X, stratified by whether a fax was sent

	Sent a fax						Not sent a fax					
	Respondents n=243		Sample selected n=725		Sampling frame n=2,743		Respondents n=61		Sample selected n=231		Sampling frame n=756	
Variable	n	%	n	%	n	%	n	%	n	%	n	%
Location												
Urban	191	79	551	76	2,026	74	44	72	175	76	587	78
Rural	52	21	174	24	708	26	17	28	56	24	169	22
Unknown	0	0	0	0	9	0	0	0	0	0	0	0
Practice type												
Group*	39	16	116	16	416	15	3	5	18	8	70	9
Medical	126	52	354	49	1,328	48	14	23	71	31	153	20
Clinic	7	3	27	4	116	4	2	3	7	3	41	5
Solo	71	29	226	31	869	32	40	66	135	58	492	65
Other	0	0	2	0	14	1	2	3%	0	0	0	0

* Group practice is defined as a practice with multiple general practitioners in the same geographic location, not a medical practice or clinic

Table 2: Demographic characteristics reported by respondents, stratified by whether they were sent a fax

Characteristics*	Sent NSW Health fax			
	Yes n=243		No n=61	
	n	%	n	%
Gender				
Male	152	63	40	66
Female	87	36	17	28
Not reported	4	2	4	7
Age range				
30–49	108	44	20	33
50+	131	54	37	61
Not reported	4	2	4	7
AGPN member				
Yes	217	89	53	87
No	21	9	6	10
Not reported	5	2	2	3
Medical staff (full and part time)				
1	69	28†	39	64
2 or more	156	64	17	28
Not reported	18	7	5	8
Non-medical staff (full and part time)				
1	16	7	8	13
2 or more	167	69	30	49
Not reported	60	25	23	38
Modes of communication available				
Fax available‡	234	96	57	93
Email available‡	127	52	34	56
Not reported	9	4	4	7

* Not including 24 surveyed general practitioners of unknown 'fax sent' status.

† Difference in proportions was statistically significant compared to group not sent fax.

‡ Categories are not mutually exclusive.

Table 3: Self-reported preparedness of GPs during a measles outbreak, by whether a health alert was sent or received

		Sent NSW Health fax*				RR†	Received any fax				RR†
		Yes n=242		No n=61			Yes n=265		No n=58		
		n	%	n	%	95%CI	n	%	n	%	
Aware of measles outbreak	Yes	216	89	46	75	1.18	257	97	22	38	2.56
	No	26	11	15	25	(1.02,1.38)	8	3	36	62	(1.84,3.56)
		Sent NSW Health fax*				RR†	Received any fax				RR†
		Yes n=227		No n=54			Yes n=247		No n=56		
		n	%	n	%	95%CI	n	%	n	%	95%CI
Offered MMR to susceptible staff	Yes	98	43	15	28	1.55‡	114	46	4	7	6.46
	No	129	57	39	72	(0.99,2.45)	133	54	52	93	(2.49,16.78)

* Not including 24 surveyed general practitioners of unknown 'fax sent' status

† Relative risk (RR) (95% confidence interval)

‡ Chi square ($P < 0.05$)

Discussion

The study showed that faxed health alerts were associated with better preparedness among GPs to respond to the measles outbreak. These findings demonstrate the value of faxed health alerts to GPs in the context of measles outbreaks and, potentially, in other situations of public health importance such as an influenza pandemic, where rapid communication is required to provide GPs and other health practitioners with important information needed to manage the situation within the community.

Measles is now a rare disease in Australia, and this creates problems for effective disease surveillance and control. In the setting of low incidence, the positive predictive value of clinical signs of measles is low,[7,10] and consequently, clinicians may not consider measles in their differential diagnosis, nor seek laboratory confirmation of such cases nor institute prompt infection control measures. GPs who were recorded as having been sent a fax, and those who reported receiving a faxed measles alert at the medical practice, were more likely to be aware of the outbreak and be aware of specific infection control and notification recommendations compared with other GPs (i.e. not sent or not received a faxed health alert).

Transmission of measles in the health care setting to unvaccinated health care workers is well documented in Australia.[8,11,12] Offering MMR to susceptible staff was a recommendation applicable to all practices during the outbreak, and we considered it a key element of the public health response. It is particularly relevant that GPs who reported receiving an alert were more likely to offer MMR to susceptible staff, including those with no known policy for staff MMR immunisation.

Fewer solo-GPs were sent faxed alerts. Presumably, solo-GPs are less likely to receive visits from representatives who collect information for the commercial Database X and are therefore more likely to be excluded from the database, or have incomplete information, which is used by NSW Health for faxing health alerts directly to medical practitioners. While the ideal source of direct contact information for medical practitioners within New South Wales is likely to be medical registration data, because updates are frequent and performed by clinicians themselves, this information source cannot be made available to NSW Health until data sharing agreements have been established for these purposes.

The study had a number of limitations; the most important was a low response rate. Despite this limitation, which is commonly reported in surveys of Australian GPs,[13–15] we were able to demonstrate that respondent GPs were similar to the GPs in the database in terms of whether their practice was rural or urban and the type of practice (clinic, medical, grouped or solo) (see Table 1). A further limitation was that there may have been some misclassification error of outcome factors and whether a fax had been received due to recall error as GPs self-reported information several months after the faxed health alerts were sent.

In conclusion, the results of this study demonstrate the value of faxed health alerts for rapid communication with GPs during communicable disease outbreaks to promote public health practices needed for effective disease control and prevention. Although

sending health alerts by email, or newer web-based technologies, have obvious advantages in terms of timeliness, at the time this study was conducted faxing appeared to be the preferred method for GPs to receive health alerts from the NSW Health.

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An evaluation of the Australian National Serosurveillance Program

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Abstract

The Australian National Serosurveillance Program (ANSP) was established in 1997 to provide national estimates of population immunity to vaccine preventable diseases and inform immunisation policy in Australia. The 1st round tested opportunistically collected sera from pathology laboratories across Australia, a 2nd round was carried out in 2002, and a 3rd round of testing is currently ongoing using sera from 2007–08. This is the 1st systematic evaluation of the ANSP since its inception. Existing information and outputs from the ANSP were reviewed and used in conjunction with data collected from a survey of the program operators to evaluate the overall utility of the ANSP and the following system attributes; acceptability, stability, simplicity, flexibility, data quality, sensitivity, representativeness and timeliness. So far the ANSP has generated 26 peer-reviewed publications and provided useful data that have influenced and provided an evidence base for immunisation policy in Australia; for example informing mathematical models, which identified the need for the young adult measles-mumps-rubella immunisation campaign. However, difficulties have been encountered with obtaining enough samples for testing in the 3rd round currently being undertaken. This is a concern that has the potential to undermine the representativeness and stability of the system, and other methods of sample collection must be investigated. Serological surveillance is an important component of any comprehensive system for monitoring population immunity to vaccine preventable diseases and evaluating the effectiveness of immunisation programs. However, an effective ongoing program requires strong support to ensure it remains sustainable in an era when laboratory based population health research for the public good is becoming increasingly challenging. *Commun Dis Intell* 2010;34(1):29–36.

Keywords: evaluation, serological surveillance, vaccine preventable diseases

Introduction

Serological surveillance (serosurveillance) provides estimates of antibody levels against vaccine preventable diseases (VPDs) in the population and is an important surveillance component in conjunction with notification, hospitalisation, mortality and immunisation coverage data. The primary advantage of serosurveillance is that it provides an indicator of population immunity induced both by immunisation and natural infection. Therefore it is a useful tool for informing immunisation policy, can be used to monitor trends in population immunity before and after changes in immunisation programs, and provides a rich source of data for disease modelling.[1]

National serosurveillance programs are well established in many countries, with at least 3 distinct models of sample collection employed. England and Wales,[2] Belgium, Bulgaria, Hungary, Ireland, Israel, Lithuanua Malta, Romania and Slovenia request representative laboratories to submit residual samples collected for routine laboratory testing that would otherwise be discarded, which is referred to as residual or opportunistic sampling.[3] The 2nd model is specific population-based random serum collection such as that undertaken by the Netherlands[4] the Czech Republic, Latvia, Luxembourg, Slovakia, Spain and Sweden.[3] Thirdly, in the United States of America sera are collected along with a wide range of other information from participants in population-based, randomly selected National Health and Nutrition Examination Survey.[5] The advantages and disadvantages of each method of serum collection have been discussed extensively elsewhere.[1,2,4]

The Australian National Serosurveillance Program (ANSP) was established as a collaboration between the National Centre for Immunisation Research and Surveillance (NCIRS) and the Centre for Infectious Diseases and Microbiology (CIDM), the Institute of Clinical Pathology and Medical Research using the 1st model described above. In the 1st round, opportunistically collected sera from all 8 Australian jurisdictions were tested in 1997–99,[1] the 2nd round was carried out in 2002, and the 3rd round of testing is currently ongoing, using sera collected in 2007–08 (Table 1).

This paper reports the findings of a formal evaluation of the ANSP that was conducted in 2008 to describe the surveillance system, to assess its attributes and to determine the usefulness of the data it generates for informing immunisation policy in Australia.

Methods

Data sources used for the evaluation included existing information available at NCIRS regarding both the previous and current rounds of the ANSP,

discussions with the ANSP study co-ordinator at NCIRS and a survey of those involved in coordinating and overseeing the ANSP, both past and present. Existing information included a survey of participating laboratories conducted in March 2004 after the 2nd serosurvey regarding enabling factors and barriers to their involvement. All the relevant information and results of the survey were available at NCIRS. It was therefore decided that this information was sufficient to inform this aspect of the evaluation and repeating the survey of laboratories was not required.

The operator survey was sent to 10 people previously or presently involved with the ANSP, of which 9 (90%) replied. The participating laboratory questionnaire was sent to 67 personnel associated with 52 laboratories in 2004. Twenty-one laboratories (40%) completed and returned the questionnaire. Of these, 18 had contributed to the 1st serosurvey, 16 had again participated in the 2nd round and the remaining three had been invited but had not contributed to either.

The assessment of the ANSP usefulness and system attributes were adapted from the guidelines for evaluating public health surveillance systems produced by the US Centers for Disease Control and Prevention (CDC), which includes 11 attributes.[6] However, for the purpose of this paper only the 4 most relevant will be reported.

The usefulness and attributes of the ANSP were defined as follows:

- usefulness: the extent to which the ANSP system and data contribute to the control of vaccine preventable diseases in Australia;
- acceptability: the willingness and ability of contributing laboratories to participate in the ANSP;
- simplicity: the structure of the ANSP and the way it operates;
- representativeness: how representative the sample selected for inclusion in the serosurvey is of the Australian population;
- timeliness: the ability of the ANSP to produce results and reports in a timeframe that allows them to be used by stakeholders.

Results

System description

A detailed description of the ANSP has been given by Gidding[1] so only a brief outline will be included here. Information flow is summarised in the Figure and the antigens included in each round are listed in Table 1.

Ethics approval is obtained for each round of sample collection and participating laboratories may also seek their own individual approvals. Public and private sector diagnostic laboratories across all 8 Australian jurisdictions send residual serum

Table 1: Antigens included in each round of the Australian National Serosurveillance Program

	Serosurvey 1 1996–99	Serosurvey 2 2002	Serosurvey 3 2007–08
Laboratories participated/invited	45/52	37/50	27/49
Number of specimens collected	13,084	7,699	Collection ongoing
Antigens included			
Measles	✓	✓	✓
Mumps	✓		✓
Rubella	✓	✓	✓
Varicella	✓	✓	✓
Hepatitis A	✓		✓
Hepatitis B	✓	✓	✓
Hepatitis C	✓		
Diphtheria	✓		
Tetanus	✓		✓
Polio	✓		✓
Pertussis	✓	✓	✓
Meningococcal C		✓	✓
Cytomegalovirus		✓	
Helicobacter pylori		✓	

samples to CIDM. Exclusion criteria include infants less than one year of age, and subjects who are known to be immunosuppressed, HIV positive or have received blood transfusions in the previous 3 months. Laboratories are also requested to submit only 1 specimen of serum per person. Samples are tested for antibodies using immunoassays specific for the antigens of interest. Population immunity for each antigen is inferred using accepted immune correlates of protection. The results of the serosurveillance and resultant mathematical modelling or policy implications are reported to the relevant committees and working parties responsible for disease control and immunisation policy, and then published in peer-reviewed journals.

Usefulness

The CDC guidelines define a public health surveillance system as useful if it 'contributes to the prevention and control of adverse health-related events, including an improved understanding of the public health implications of such events'.[6] A total of 26 papers arising from the first 2 rounds of the ANSP have been published in peer reviewed journals, which have covered issues such as evaluation of immunisation campaigns, reporting of baseline levels of immunity, mathematical modelling of disease transmission dynamics and the impact of immunisation programs (Table 2).

Overall, the ANSP meets the definition given above, particularly through its ability to contribute both conceptual knowledge, through increased understanding and stimulation of research into prevention and control of VPDs; and instrumental knowledge, through evaluation of immunisation programs and policy recommendations.

Specific objectives for the ANSP have not been defined, however the stated purpose is 'to measure the age-specific prevalence, in Australia, of susceptibility or immunity to diseases that are, or will soon be, vaccine preventable' by providing valid data for the 5 key outcomes listed below. The extent to which these outcomes have been achieved is examined.

Outcome 1: To measure age group specific population immunity to vaccine preventable diseases in Australia

It is clear that the ANSP generates useful data for determining measures of age group specific population immunity to diseases that are, or could potentially become, vaccine preventable. This information is extremely valuable for informing immunisation policy when combined with data on vaccine coverage and disease notifications. Eleven of the research papers generated were produced specifically for this purpose as indicated in Table 2.

Figure: Flow chart representing the Australian National Serosurveillance Program

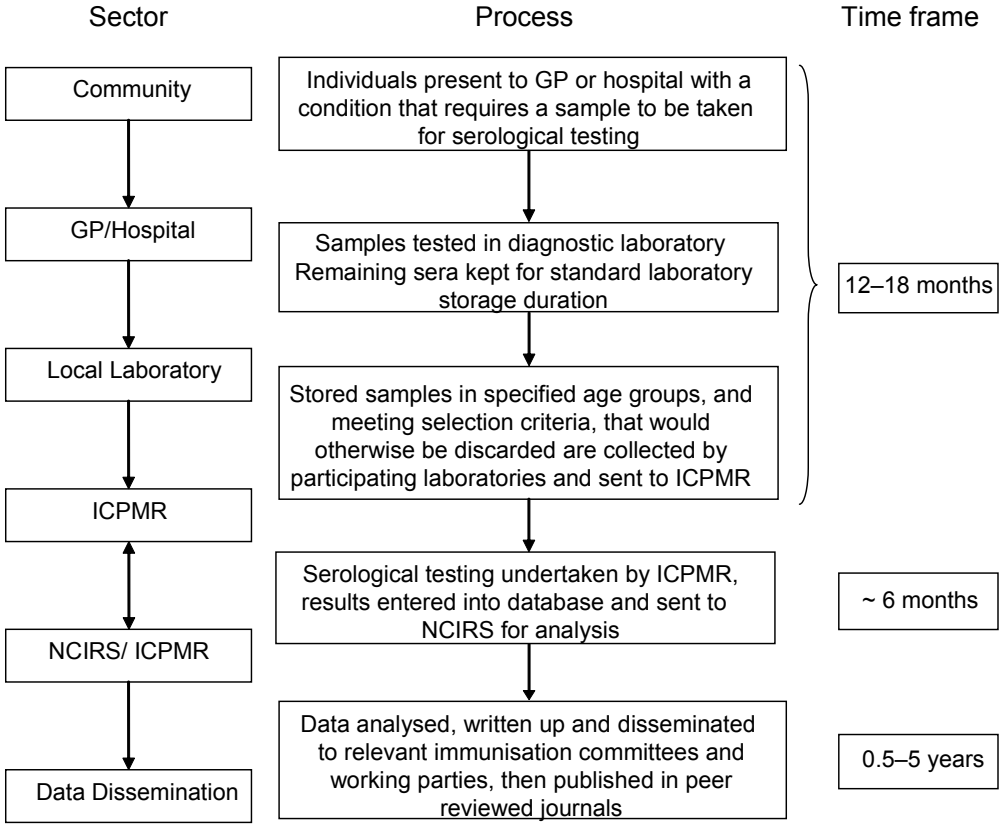

Table 2: The Australian National Serosurveillance Program has generated 26 publications and provided a valuable evidence base for immunisation policy in Australia

Study focus	Outcomes and/or policy recommendations
ROUND 1	
Discussion of serosurveillance	Outlines need for ongoing national serosurveillance in Australia[1]
Evaluation and/or discussion of vaccine program	Confirmation of measles control campaign (MCC) effectiveness and recommendation to continue serosurveillance in Australia using opportunistically collected sera[7]
Evaluation and/or discussion of vaccine program	Confirmation of MCC effectiveness[8]
Evaluation and/or discussion of vaccine program	Young adults should be encouraged to have a 2nd dose of measles-mumps-rubella (MMR) or serological confirmation of measles immunity[9]
Evaluation and/or discussion of vaccine program	Maintenance of high MMR coverage and collection of high quality surveillance data to detect and vaccinate non-immune females of child-bearing age[10]
Evaluation of laboratory testing procedures	Microimmune ELISA is a more appropriate assay than the Enzygnost ELISA for estimation of mumps seroprevalence[11]
Evaluation of sample collection methods	Opportunistically collected sera is a valid method serosurveillance as this method yielded similar seroprevalence estimates to a random cluster survey in Victoria[12]
Mathematical modelling	Sustained efforts are required to improve coverage with 2 doses of MMR and to ensure elimination of Indigenous measles transmission[13]
Mathematical modelling	Varicella vaccination should be aimed at children less than 5 years of age and further modelling using serosurvey data is warranted[14]
Population seroepidemiology	Ongoing need to improve MMR vaccine uptake in infants and recommendation of vaccination campaign targeting young adults[15]
Population seroepidemiology	Identification of young adult population group with low level of mumps immunity and recommendation to renew efforts to maximise MMR coverage[16]
Population seroepidemiology	Baseline population seroprevalence of varicella and mathematical modelling of disease transmission[17]
Population seroepidemiology	Any decision on national routine childhood hepatitis A vaccination requires a cost-benefit analysis before implementation[18]
Population seroepidemiology	People born in Asia are a high risk group for hepatitis B virus (HBV) infection in Australia and targeted vaccination of this group should be considered[19]
Population seroepidemiology	Higher evidence of past infection with HBV in the Northern Territory compared with Australian average for children aged ≤ 9 years[20]
Population seroepidemiology	Very low seroprevalence of immunity to hepatitis C virus in the over 50 years age group[21]
Population seroepidemiology	Additional efforts recommended to protect those aged over 50 years against diphtheria and tetanus, especially travellers[22]
Population seroepidemiology	Ongoing surveillance is required following the recent change back to inactivated polio vaccine[23]
Population seroepidemiology	Confirmation that changes in the scheduling of pertussis vaccination were necessary and recommendation for a sustained effort to ensure vaccination coverage remains above 90% for the benefit of herd immunity[24]
ROUND 2	
Evaluation and/or discussion of vaccine program	The young adult MMR campaign had no impact on measles immunity in Australia. To maintain elimination in the longer term, timeliness and coverage of childhood vaccination must improve and innovative strategies will be required to increase measles immunity among young adults[25]
Evaluation and/or discussion of vaccine program	The young adult MMR campaign had no impact on measles immunity in Victoria[26]
Evaluation and/or discussion of vaccine program	Varicella vaccination significantly increased immunity among children aged 3–5 years[27]
Evaluation and/or discussion of vaccine program	Demonstrated that the universal infant hepatitis B vaccination program was successful and that school-based programs for adolescents were effective[28]
Population seroepidemiology	Seroprevalence of antibody to meningococcus serogroup C was low before vaccine program introduction. Further serosurveys are required to evaluate vaccine impact[29]
Population seroepidemiology	High levels of cytomegalovirus exposure occur in the first few years of life therefore for a universal vaccination program to have maximal impact, the vaccine would need to be delivered to infants and have a long duration of protective efficacy[30]
Population seroepidemiology	Future *Helicobacter pylori* vaccines should be given in childhood as acquisition occurs from an early age[31]

Outcome 2: Identify groups in the population with low levels of protection to inform immunisation policy

The key issue of identifying groups in the population with low levels of protection has been covered in a number of the ANSP publications. For example, immunity to diphtheria and tetanus was demonstrated to be less than 60% for adults aged 50 years or over in the 1st serosurvey, and the authors recommended a booster dose among this age group.[22] A cohort of young adults that remained susceptible to measles following the Measles Control Campaign (MCC) in 1998 was also revealed in the 1st serosurvey, which led to the further provision of Commonwealth funding for the young adult measles, mumps and rubella immunisation program in 2001.[32] Following this, subsequent further residual measles susceptibility was identified using ANSP data.[12] A birth cohort, between 1978 and 1982, with a relatively high level of susceptibility to mumps has also been identified from serosurveillance data, further underlining the importance of maximising 2 dose MMR coverage.[16]

Outcome 3: Provide baseline measures of immunity to determine subsequent trends in future serosurveys

The 1st round of the serosurvey provided baseline estimates of immunity to 11 antigens, of which five were again included in the 2nd round along with 3 new antigens. The 3rd round included the 5 antigens from both previous serosurveys; four from the 1st round only and one from the 2nd round only (Table 1). Thus baseline estimates of age-specific susceptibility to 14 diseases that are or may soon be vaccine preventable have been published. Ten of these have been included in more than 1 round of the serosurvey, facilitating an examination of trends over time.

Outcome 4: Provide data for the evaluation of immunisation programs

The primary reason for determining the baseline level of population immunity is to facilitate the evaluation of immunisation programs, and a number of papers dealt with this issue as shown in Table 2. For example the 1st round of the ANSP was designed specifically to evaluate the MCC using the MMR vaccine and demonstrated significant increases in population immunity to all 3 antigens.[7]

Outcome 5: Provide data for mathematical modelling of vaccine preventable disease dynamics

Two papers developed mathematical models from ANSP data, to evaluate the impact of the MCC[13] subsequent to the young adult MMR immunisation program,[25] to determine the potential for another measles epidemic to occur in Australia, and to postulate what must be done to prevent it occurring. Recent evidence has indicated that measles control initiatives have been successful and endemic measles has been eliminated in Australia.[33] Baseline data have also been used to model epidemiological parameters associated with disease transmission dynamics such as the level of herd immunity required to prevent ongoing transmission of varicella.[14]

In conclusion, the ANSP is a valuable part of the comprehensive surveillance system of VPDs in Australia and has provided a broad range of useful, policy relevant data. It appears that the ANSP has largely met its stated aims but, as discussed below, there are some issues that need to be resolved in order for the ANSP to remain a useful and effective serosurveillance mechanism.

Evaluation of selected system attributes

Acceptability

The decreasing ability of laboratories to participate in the ANSP is reflected by the number contributing samples in each round, which has declined from 45 in the 1st round, to 37 in the 2nd round and 27 in the 3rd round. Some, but not all, of this decrease can be attributed to the fact that there have been significant changes in the business environment for laboratories over the last decade, which has resulted in mergers and centralisation of diagnostic services. Eight laboratories that contributed to the 1st serosurvey are no longer in existence; on the other hand 5 laboratories contributed to the 3rd round that did not participate initially. Thus 15 laboratories that still exist dropped out in later rounds, indicating that barriers to participation exist.

The laboratory survey in March 2004 identified several such barriers, including the need to acquire additional ethics approval at the laboratory level and competing research and other operational priorities. A financial contribution by NCIRS to assist with labour costs for specimen collection was identified as necessary by 10 laboratories, while two were unsure. The remainder reported financial reimbursement was not required as staff time was the major barrier and any financial reimbursement went to the laboratory general revenue, rather than the individual staff member responsible for sample collection. Thus there was no motivation for staff to work outside their normal duties to assist with sample collection. Only nine of the 21 laboratories that responded (43%) reported that a trained technician, employed by NCIRS, could be of assistance with sample collection.

On the basis of these results, payment per specimen contributed was offered to laboratories contributing to the 3rd round of the ANSP. However only 3 laboratories actually invoiced NCIRS and this incentive does not appear to have been effective. Sample collection in the 3rd round has been slower than anticipated and as a result the sample collection period was extended.

Simplicity

ANSP staff regard most components of the current system as relatively simple to operate. The centralised nature of the system greatly contributes to this as all samples can be tested quickly and easily without the need for complex inter-laboratory standardisation procedures. There is no specialised training required as all laboratory tests are part of routine practices at CIDM. The dataset is simple and small and seroprevalence estimates can be quickly generated upon the completion of laboratory testing. The majority (88%) of laboratories surveyed that participated in the 2nd serosuvey did not report any specific difficulties with sample collection. The two that did report difficulties indicated problems with using their laboratory database software to acquire the relevant information required for sample collection.

Representativeness

For the ANSP to generate national seroprevalence estimates, the samples tested must be representative of the Australian population. Age, gender and jurisdictional representativeness are built into the sample size calculations. However, the increasing difficulty to collect sufficient samples has the potential to compromise external study validity. There are a range of other variables of which the serosurvey should ideally be nationally representative (e.g. ethnicity, rural/remote locality etc.), but due to ethical and data availability constraints this is not possible.

Representativeness cannot be directly inferred by the number of contributing laboratories participating, but the decreasing trend is a concern. If a large laboratory does not contribute samples this may impact on the geographical representativeness of the samples contributed to the ANSP.

Finally, the opportunistic sampling of serum samples submitted to laboratories for diagnostic testing utilised by the ANSP also has implications for the ability to generalise the data.[1] Individuals who have serum samples taken for diagnostic testing are not necessarily representative of the entire Australian population and it is difficult to identify and control potential biases that may arise from this approach, as detailed risk factor information is not available.[2] A study was undertaken to compare immunity levels in Victorian school children estimated using ANSP opportunistic samples, by selecting results from Victorian subjects in the same age group, to a prospectively collected 3 stage random cluster sample. This demonstrated that similar estimates of immunity to measles, mumps, rubella, varicella and hepatitis B were generated by both sampling methodologies.[12] However the cost of sample collection and storage per antibody tested was over 7 times greater using the random cluster sampling compared with the ANSP opportunistic sampling. Random sampling is still the preferred methodology and would also overcome the difficulties encountered with obtaining samples from laboratories. However, this method also introduces potential biases, because it requires individual informed consent, the cost involved is prohibitive and it would require considerable dedicated funding to make it sustainable.

Timeliness

Serosurveillance is an inherently slow process (Figure 1). Serology testing results do not affect the clinical management of patients, so timeliness is not absolutely imperative. However, samples should still be collected, processed and the results made available to the relevant committees and working parties as rapidly as possible to ensure that ANSP produces data which are relevant and up to date.

Increasing automation within the laboratory is improving the speed at which samples can be tested. However, as discussed previously, the primary rate limiting step is sample collection. There is also a need to ensure sufficient capacity is available to analyse and publish the results. ANSP data are primarily disseminated through peer-reviewed publication, which is also an inherently protracted process. As shown in Table 1 the majority of papers arising from both the previous serosurveys were published 3 to 6 years after the completion of sample collection. More immediate results are reported to the relevant advisory committees and working parties as part of the formal vaccine impact evaluations that NCIRS is contracted to provide but results are not widely available until publication in the peer-reviewed literature.

Discussion

The first 2 rounds of the ANSP have generated useful data that have influenced and provided an evidence base for immunisation policy in Australia. However the key challenge for the ANSP lies in the increasing difficulties encountered with obtaining enough samples for testing. It is clear that the current method of sample collection is increasingly difficult to sustain, which has implications for all the system attributes discussed above.

Supplementary and alternative options for sample collection must be evaluated to ensure the ability to generalise the results to the Australian population and ongoing viability of the program. One option to increase engagement of laboratories and the acceptability of sample provision that has been considered is the establishment of jurisdictional-based serosurveillance systems. However this would greatly increase the complexity and cost of the system in order to standardise testing procedures and compromise the ability to generate nationally representative data.

It is important for the role and specific objectives of the ANSP to be clearly defined and communicated to all stakeholders. While peer reviewed publication is important, more immediate and regular feedback of results to participating laboratories may help to keep them engaged in the process and appreciate the importance of their contributions. Other approaches could include collaboration on resulting peer-reviewed publications, holding information dissemination forums, or the establishment of a formal consultation process, for example through the Public Health Laboratory Network, or creating a stakeholder reference group. Whilst all of these approaches are potentially useful, they are unlikely to address the fundamental problem of inadequate laboratory staff time available for sample collection.

Alternative options include collection of adult samples from blood bank donors, and request only paediatric samples from laboratories to reduce the collection workload. However as blood donors are restricted to healthy adults, such sample are not necessarily representative of the general population,[34] and would impact on the representativeness.[35,36] Furthermore individuals with certain diseases included in the ANSP (e.g. hepatitis B and C) are excluded from donating blood. The potential implications of this change in methodology would also need to be rigorously evaluated to ensure results would still be comparable with previous serosurveys, particularly for the in-house immunoassays.

Finally, there is also scope to strengthen the partnership between NCIRS, CIDM and the Commonwealth Department of Health and Aging by clearly defining the responsibilities of each organisation and establishing the ANSP as a cornerstone of VPD surveillance in Australia. An effective ongoing program requires strong support to ensure it remains sustainable in an era when laboratory-based population health research for the public good is becoming increasingly challenging.

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The impact of pre-departure screening and treatment on notifications of malaria in refugees in south-east Queensland

Megan K Young, Bradley J McCall, Karen Heel

Abstract

This study aimed to investigate changes in the notification rate of malaria in refugees over a period of national policy change on pre-departure screening. Notifying clinicians were interviewed to complete a standardised enhanced surveillance form. A decline in refugee malaria notifications occurred after implementation of a national policy to offer pre-departure malaria screening and treatment as necessary to refugees. Surveillance data support the benefit of offering pre-departure screening and treatment as necessary to refugees. *Commun Dis Intell* 2010;34(1):37–40.

Keywords: malaria, refugees, screening, south-east Queensland

Introduction

Around 3.2 billion people live at risk of malaria, with areas of 107 countries and territories at risk of transmission in 2004.[1] The disease causes around 1 million deaths each year, and average losses of economic growth of 1.3% annually in the most affected countries. Sub-Saharan Africa is the worst affected region, with 60% of cases and more than 80% of deaths.[1]

Malaria transmission is often heightened in refugee camps because of the lack of adequate shelter and mosquito protection, malnourishment in displaced populations, and difficulty accessing appropriate treatment.[2] Passenger manifests of refugees arriving in Australia confirm that many departed Africa from such camps, and Australian studies since the year 2001 have shown that between 5% and 10% of recently arrived refugees from Africa have parasitaemia.[3–6]

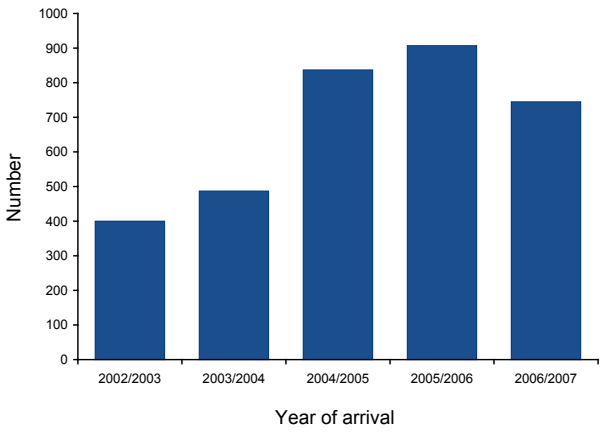
Queensland, similar to Western Australia and the Northern Territory[3,4] has resettled an increasing number of refugees from Africa since the turn of the century. Data for the area served by the Brisbane Southside Public Health Unit (BSPHU) in south-eastern Queensland (Figure 1) also demonstrate this increase in settlement numbers prior to the 2006/07 financial year.

In the decade prior to 2005, notifications of malaria to the BSPHU were limited in number and usually associated with overseas travellers returning to Australia. In 2005 there was a sudden increase in notifications to 116 for the year, from a previous 5 year average of 54.[7] This increase was noted in public health units around the country, and was widely noted to be associated with recently arrived refugees.

Application for entry to Australia as a refugee requires the applicant to undergo a visa medical examination, including age dependent HIV and tuberculosis screening.[8] Prior to 2005, the visa medical examination was the only health assessment protocol in place for those seeking entry to Australia via the Offshore Resettlement Program.

As national awareness developed that the increased malaria notifications were associated with recently arrived refugees, policy recommendations aimed at providing a broader health assessment for offshore refugees were developed. These initially (in the latter half of 2005) involved a pre-departure medical examination and treatment (unless contraindicated) with Fansidar® (sulfadoxine and pyrimethamine). However, a new policy to undertake rapid diagnostic testing (RDT) for malaria, and treatment (with artemether/lumefantrine combination) according to the result, prior to travel to Australia was implemented by the Commonwealth Department of Health and Ageing (DOHA), the (then) Commonwealth

Figure 1: Number of African humanitarian entrants settling in the Brisbane Southside Public Health Unit area, 2002 to 2007



Source: Statewide Multicultural Health Program, Queensland Health.

Department of Immigration and Multicultural Affairs (DIMA) and the Communicable Diseases Network Australia from March 2006.[9] The policy was implemented over the next 6 months.

Local enhanced surveillance was continued throughout 2006 and 2007 to monitor the expected changes in malaria notifications in response to implementation of these policies. These results are presented here.

Methods

During 2005, enhanced surveillance was commenced on all new notifications of malaria to the BSPHU. A one page questionnaire was completed by BSPHU staff by contacting the clinician who had requested the investigation for malaria on the case. Information was collected about the age, sex, refugee status, country of origin/country of first asylum, pre-departure treatment, and illness details for each case. A refugee was defined as a person who entered Australia via the Offshore Resettlement Program from any country. If the clinician was not aware of some of the details requested including whether the case had received pre-departure screening, this information was checked against the DIMA manifests that accompany offshore refugees. This surveillance continued throughout 2006 and 2007.

The information was entered into a Microsoft Excel database and periodically analysed using Excel and Epi Info ver 6 over the next 3 years. Chi-squared tests were used to assess the statistical significance of changes in proportions, while ANOVA was used to assess the statistical significance of changes in means.

Enhanced surveillance was conducted in accordance with Chapter 3 of the (Queensland) *Public Health Act 2005*. Electronic data were de-identified and password protected.

Results

The majority of refugees notified with malaria during 2005–2007 were young males who originated from Africa, and the vast majority were infected with *Plasmodium falciparum* (Table 1). The number and proportion of refugee notifications was greatest in 2005 and declined in the subsequent 2 years (Figure 2). This decrease was statistically significant ($P = 0.005$).

As the refugee notification numbers declined, the epidemiology of malaria notifications also changed, although not all observed changes were statistically significant. The proportion of notifications among refugee children aged less than 15 years was 66% in 2005, 74% in 2006 and 50% in 2007 ($P = 0.155$), while the average age of all malaria notifications increased from 20 years in 2005, to 22 years in 2006 and 28 years in 2007 ($P = 0.016$). A change in the country of origin/country of first asylum of the refugee cases also occurred. In 2005, the great majority of refugees with malaria came from camps in Liberia and Tanzania. In 2006, they mainly came from Tanzania, and in 2007, cases came from a variety of locations throughout Africa including Kenya, Liberia, Sudan, Tanzania and Uganda.

Figure 2: Number of notifications identified as refugees, by age group

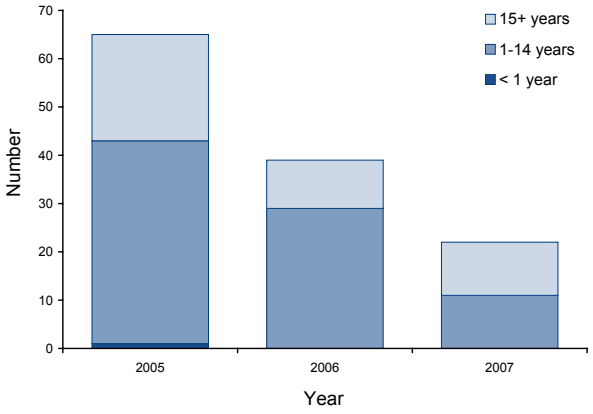


Table: Demographic characteristics of refugees notified with malaria, Brisbane Southside Public Health Unit, 2005 to 2007

Demographic	Range/per cent	Number
Age range	0–63 years	126
Age group		
< 15 years	66%	83
15+ years	34%	43
Gender		
Males	56%	71
Females	44%	55
Place of origin		
Africa	97%	123
Other	3%	3
***Plasmodium* spp**		
falciparum	89%	113
vivax	3%	3
ovale	2%	3
malariae	2%	2
Mixed infection	3%	4
Unknown	1%	1

* Numbers may not add to same total because of missing data; missing data have been excluded to calculate percentages.

The joint policy of the Commonwealth DIMA, DOHA, and the Communicable Diseases Network Australia dated March 2006, indicates locations with pre-departure screening available at that time included Kenya (including cases transiting from Uganda, Tanzania, Democratic Republic of the Congo, Burundi and Rwanda), Ethiopia, Ghana, Guinea, and Sierra Leone. The proportion of cases from countries evidently without pre-departure screening increased from 2006 to 2007 (8% to 41%; $P = 0.005$).

According to health manifest data, 31% of refugee notifications were screened for malaria prior to departure in 2006, with 77% screened in 2007. However, for 52% of notifications (69% in 2006 and 23% in 2007), health manifest data on pre-departure screening was incomplete.

The data on pre-departure malaria treatment of refugee notifications was also generally poor, with status unknown for 100% of cases in 2005 and an average of 40% of cases in 2006 and 2007. Where pre-departure treatment status was known, the proportion who were treated for malaria prior to departure decreased (17 of 23 cases [74%] in 2006 and 1 of 13 cases [8%] in 2007; $P < 0.001$).

Discussion

The decrease in malaria notifications to the BSPHU coincided with the implementation of the recommendation to offer offshore refugees screening (and treatment as appropriate) for malaria prior to travel to Australia. A causal association cannot be inferred, however the benefit of the introduction of pre-departure screening and treatment for malaria appears supported by this decrease in notifications. In addition to the decline in numbers, there was an increase in the proportion of refugee notifications from countries where pre-departure screening was not available across the study period. This also seems to support the benefit of pre-departure screening, although an increasing intake of refugees from such countries with a decrease in refugees from screening countries could have affected the results.

Other possible reasons for the decline in notifications include:

1. a decline in the number of refugees settling in the Public Health Unit's jurisdiction over the study period;
2. a decrease in screening for or detection of malaria once refugees arrived in Australia, and
3. changes in the incidence of malaria in the countries of asylum.

The first two of these reasons for the decline in notifications are unlikely. Firstly, data from the Statewide Multicultural Health Program show continued settlement of African refugees in south-east Queensland across the study period, with what appears to be only a small drop in total numbers of arrivals (Figure 1). Secondly, screening and investigation for malaria in the local area is likely to have increased rather than decreased. Prior to 2006, there were no co-ordinated screening services for refugees in the area of the BSPHU. February 2006 saw the commencement of a refugee health service with a focus on screening and immunisations in a local area where many offshore refugees were resettling.[10] Since 2006 the BSPHU has also worked to increase awareness of refugee health issues among other service providers.

However, these data are still subject to the same limitations as other notifiable conditions surveillance data. That is, that most notifications depend on the investigation practices of clinicians, which are closely associated with the health care seeking behaviours of the population. This may change over time, and in the refugee population particularly, may be influenced by a range of factors. Potential barriers to accessing health care that may fluctuate include a lack of access to culturally appropriate services, a past history of torture or trauma, and a distrust of government services. It cannot be determined how much influence, if any, these factors had on the health seeking behaviours of refugees over the study period, although the commencement of a local dedicated refugee health service would go some way to increasing access to culturally appropriate services over this period.

Changes in the incidence of malaria in different countries across Africa may explain at least some of the decline in notifications. It is noted that the countries of origin / countries of first asylum of cases changed across the study period, so different degrees of endemicity and / or transmission of malaria in these countries may have impacted on results.

When considering the data presented here in relation to the benefit of pre-departure screening for malaria, it should be noted that not all offshore refugees have access to RDT. There are inevitably some points of departure for Australia at which testing is not available. This will include some people who relocate under the Special Humanitarian Program, and may account for some of the missing data relating to screening and treatment. Further, treatment with artemether/lumefantrine is a 3 day course that is not observed by medical staff, so failure of therapy may result from non-compliance. Malaria upon arrival may also result from reinfection after treatment, or low antigen loads at the time of RDT causing a false negative result.

The change in policy from no pre-departure screening; to pre-departure medical exam and treatment with Fansidar® in 2005; to pre-departure medical exam, RDT for malaria and treatment with artemether/lumefantrine according to the result in 2006 would have affected the results in a number of ways. Firstly, the further decline in notifications noted in 2007 may be explained by the fact that RDT was phased in over 2006. Secondly, the increase in the proportion of notifications listed as having pre-departure screening from 2006 to 2007 may have resulted from the changing definition of pre-departure screening for malaria as RDT was phased in over 2006. Lastly, the decrease in the proportion of refugees who received pre-departure treatment for malaria from 2006 to 2007 may have been due to continued use of Fansidar® in 2006 while RDT and treatment with artemether/lumefantrine was gradually introduced. Artemether/lumefantrine is the recommended first line treatment for uncomplicated *falciparum* malaria.[11]

Other authors have reported on the benefits of pre-departure treatment of malaria[12] though it has been acknowledged that malaria still occurs in the refugee population after arrival in Australia.[13] To our knowledge, no other study has reported the results of malaria surveillance among refugee populations in Australia for the time period over which the national policy on pre-departure screening changed.

Our data support the continuation of pre-departure RDT and treatment as appropriate for malaria. However, as malaria cases will still occur despite pre-departure screening, post-arrival testing and treatment of refugees is still recommended.[14]

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A large point-source outbreak of *Salmonella* Typhimurium phage type 9 linked to a bakery in Sydney, March 2007

Trish Mannes, Leena Gupta, Adam Craig, Alexander Rosewell, Clancy Aimers McGuinness, Jennie Musto, Craig Shadbolt, Brian Biffin

Abstract

This report describes the investigation and public health response to a large point-source outbreak of salmonellosis in Sydney, Australia. The case-series investigation involved telephone interviews with 283 cases or their guardians and active surveillance through hospitals, general practitioners, laboratories and the public health network. In this outbreak 319 cases of gastroenteritis were identified, of which 221 cases (69%) presented to a hospital emergency department and 136 (43%) required hospital admission. This outbreak was unique in its scale and severity and the surge capacity of hospital emergency departments was stretched. It highlights that foodborne illness outbreaks can cause substantial preventable morbidity and resultant health service burden, requiring close attention to regulatory and non-regulatory interventions. *Commun Dis Intell* 2010;34(1):41–48.

Keywords: outbreak, foodborne illness, *Salmonella*, bakery

Introduction

Foodborne illness accounts for an estimated 32% of gastroenteritis illness in Australia, causing around 5.4 million episodes of illness, 18,000 hospitalisations and approximately 120 deaths annually.[1] *Salmonella* infection (mainly *Salmonella* Typhimurium) is predominantly a foodborne illness in Australia with annual seasonal peaks in the summer months.[2,3] In early 2007, *Salmonella* notifications in New South Wales increased to levels well above previous years[4] in part due to the outbreak reported in this paper.

On the evening of Monday 26 March 2007 a hospital emergency department (ED) in Sydney notified the Sydney South West Area Health Service (SSWAHS) Public Health Unit (PHU) of 5 people (three from the same family) with severe acute gastroenteritis who had presented to this ED reporting that they ate food from the same bakery, five to 24 hours prior to becoming ill. In the ensuing days this evolved to be one of the largest *Salmonella* outbreaks reported in New South Wales. The pathogen responsible was subsequently determined to be *Salmonella* Typhimurium phage type 9. This paper describes the public health investigation and response to the point-source outbreak.

Methods

Epidemiological investigation

Active case findings were conducted by requesting that other public health units in New South Wales, EDs and general practitioners (GP) notify the PHU of any possible cases. Laboratories and EDs close to the premises were contacted for updated possible case listings on a daily basis for 1 week.

Cases were also identified through routine laboratory notification of *Salmonella* species and *Salmonella* Typhimurium between 19 March and 10 April 2007. Case details were verified against an electronic clinical information system for probable cases who presented to facilities within the SSWAHS (n = 125). Where a person was unable to contacted directly, details were verified by discussion with the treating clinician or the notifier.

Details were collected on all food eaten in the 3 days preceding onset of illness for the first 20 cases notified to the PHU to confirm that food from the bakery was the only common exposure, hypothesising that this was the likely source of gastroenteritis illness. Other cases were interviewed with a standardised questionnaire to characterise exposure and obtain demographic, illness and exposure details. A case was defined as 'any person who had a consistent illness with symptoms of either diarrhoea and/or vomiting AND who ate food prepared at the bakery between 19 and 27 March 2007, prior to the onset of their illness'. Cases were confirmed by interview with the case or by discussions with treating clinicians.

All 5 symptomatic food handlers were interviewed. All symptomatic food handlers working at the bakery had stool cultures for *Salmonella* and arrangements were made for asymptomatic food handlers to be screened.

Six weeks after the commencement of the outbreak the SSWAHS PHU conducted a sub-investigation. Forty-five cases were randomly selected for follow-

up interview to obtain information on illness duration, illness severity and contact with health care facilities. The subset was randomly select using a random number generator matched with case identification numbers.

Data were entered into Epi Info 3.3.2 for Windows (CDC, Atlanta, GA, USA), and analysed using SAS System® version 8.02 and Microsoft® Excel 2000.

Environmental investigation

The NSW Food Authority was notified of the initial cluster of gastroenteritis cases by the PHU on the evening of Monday 26 March and inspected the bakery the following day. Food handling and cleaning practices were reviewed as part of the environmental investigation. Food items were sampled for microbial testing and environmental samples were taken from surfaces and equipment where food was prepared. A trace-back of all foods served at the premises was initiated, which included an inspection of an egg farm. The farm inspection involved a review of egg handling procedures and the collection of egg and environmental samples for microbial testing. Environmental samples included chicken faeces specimens, meat meal, stock feed and drag swabs collected from 4 laying sheds. Samples were tested by the Division of Analytical Laboratories.

Laboratory investigation

Salmonella isolates from stool and blood specimens, were cultured at laboratories throughout Sydney, and collated by the PHU. Serotyping of human *Salmonella* isolates and isolates from food and environmental sources collected at the food premises was performed by the Institute of Clinical Pathology and Medical Research and collated by the PHU. Multiple locus variable number of tandem repeats analysis (MLVA) was performed on these isolates.[5,6] These isolates were then tested by multiplex polymerase chain reaction-based reverse line blot (mPCR/RLB) phage-type (PT) prediction assay.

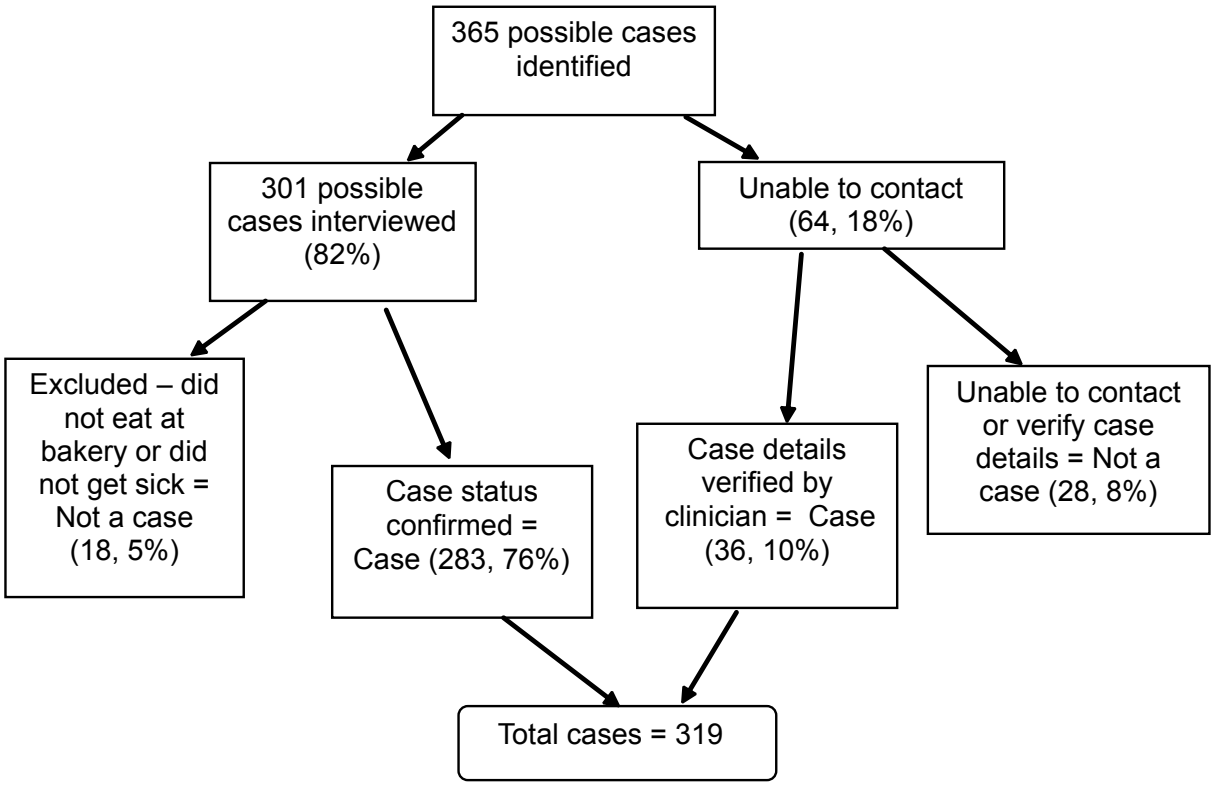
Results

Epidemiological investigation

The SSWAHS PHU was notified of 365 possible cases, from which 319 cases were identified. Of these, 283 were interviewed. The remaining 36 cases were confirmed by contacting the treating clinician (Figure 1). Five cases were food handlers who worked at the bakery and reported eating food from the bakery prior to becoming unwell. Onset dates for the food handler cases were between 23 and 27 March.

Demographic details are contained in Table 1. Cases were aged between one and 74 years

Figure 1: Case verification process



(median = 31 years) and most cases were male. Figure 2 contains the epidemic curve and the date of purchase of food from the bakery. The peak of the outbreak occurred on 25 and 26 March with 67% (189/283) of cases reporting disease onset on these 2 days. Incubation periods ranged from one to 118 hours (median 10 hours). The median reported incubation period decreased as the outbreak progressed (Figure 2); ranging from 104 hours for cases who purchased food on 22 March (n = 2) to 8 hours for those who purchased food on 27 March (n = 44).

Food was purchased by cases between Thursday 22 March and Tuesday 27 March (Figure 3). All but 7 cases purchased food over a 4-day period, with 40% (113/283) being purchased on a single day. Forty-four cases reported purchasing food on 27 March, after the outbreak was notified. Of these, 28 were admitted to hospital. All cases interviewed reported that they ate a chicken, pork or salad roll from the bakery prior to becoming unwell (Table 2). While most cases reported that they ate a whole roll prior to becoming unwell, 14 cases reported that they ate only a very small amount of the roll, as little as a single bite.

Attack rates could not be calculated, however, the owners of the food premises estimate that 320 pork rolls would be sold over a 5 day period (40 on weekdays and 100 weekend days). On 3 days at the peak of the outbreak (Sunday, Monday and Tuesday) the number of cases who purchased food exceeded the estimated number of rolls sold. The attack rate on these days therefore is likely to be close to 100%, although it may be much less on other days.

There were 8 food handlers at the bakery. Of these, five met the case definition and six had positive stool cultures for *Salmonella* Typhimurium phage type 9, with one initially being reported as asymptomatic. Later in the investigation this food handler admitted to having some stomach pain after consuming a small amount of a pork roll on 27 March.

Figure 2: Number of cases and incubation period (hours), *Salmonella* Typhimurium outbreak, Sydney, March 2007, by date of onset and date of purchase

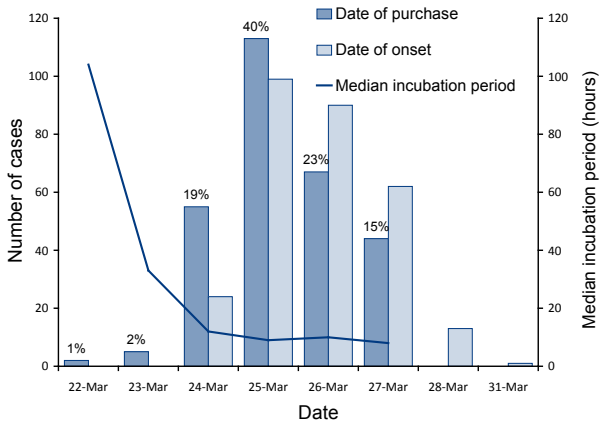


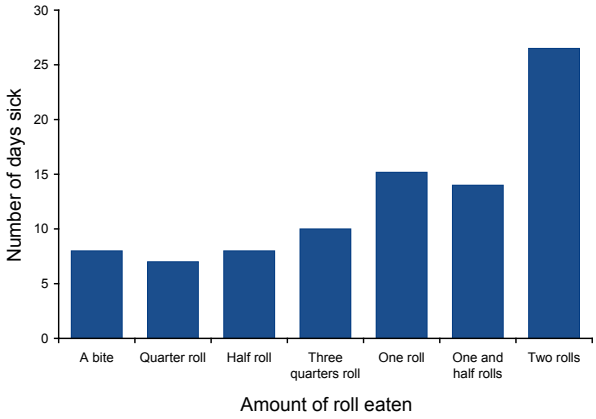
Table 1: Demographic and clinical characteristics for cases, *Salmonella* Typhimurium outbreak, Sydney, March 2007 (n = 283) and a subset of 45 cases interviewed a second time to determine illness severity and duration

	Full outbreak		Illness severity subset	
Sex	**n**	**%**	**n**	**%**
Male	171	60.4	21	46.7
Female	112	39.6	24	53.3
Age group				
0–9	10	3.5	2	4.4
10–19	62	21.9	11	24.4
20–29	57	20.1	10	22.2
30–39	55	19.4	9	20.0
40–49	48	17	6	13.3
50–59	28	9.9	4	8.8
60+	9	3.2	2	4.4
Unknown	14	4.9	1	2.2
Symptoms				
Diarrhoea	268	94.7		
Fever	235	83.0		
Vomiting	186	65.7		
Headache	185	65.4		

Table 2: Foods eaten by cases, *Salmonella* Typhimurium outbreak, Sydney, March 2007 (n = 283)

	Yes	No	Unknown	Percentage
Bread roll	280	1	2	98.9
Salad vegetables	263	10	10	92.9
Mayonnaise	149	82	52	52.7
Pate	138	86	59	48.8
Pork	149	108	26	52.7
Chicken	103	152	28	36.4
Ham	94	95	94	33.2
Salad only, no meat	14	269	0	04.9

Figure 3: Dose response curve: Amount of roll eaten, *Salmonella* Typhimurium outbreak subset of 45 cases, Sydney, March 2007, by number of days sick



During the outbreak, most cases (94%, 301/319) presented to a health care provider; 80 of these presented only to their GP and 69% (221/319 cases) presented to a hospital ED. A large number of the cases (62%, 136/221) who presented to EDs required hospital admission. Median duration of hospital admission was 24 hours. One case reported being admitted for 8 days. Of those cases who presented to a hospital within the SSWAHS boundary (n = 125), 65% of cases required hospital admission, with a median duration of 31 hours. Cases presented to 26 different hospitals across New South Wales. Seventy-six cases (24%) related to this outbreak presented to a single hospital ED over a 4-day period; 42 (13%) cases presented to a second hospital. These 2 hospitals are located within 5 kilometres of the bakery. Most presentations occurred on a single day.

Sub-investigation of illness severity

Of the 45 cases in the sub-analysis group (demographic characteristics are shown in Table 1), illness duration (defined as number of days before the case reported their health returned to normal) ranged from three to 45 days (median 14 days); at least 5 people reported still being unwell at the time of second interview. Eighteen per cent of this subset purchased food on 24 March, 40% on 25 March, and 20% on either 26 or 27 March. Thirty-nine of these cases (87%) reported taking time off work or school due to the illness; 20 cases reported taking less than a week off work or school, 14 between one and 2 weeks, one between two and 3 weeks and three greater than 3 weeks. Fifty-six per cent of this subset of cases indicated on questioning that they required intravenous fluid replacement at the time of the acute illness. Figure 3 displays a dose-response curve for this subset of cases, showing that duration of illness increased with the estimated quantity of the food eaten.

Laboratory findings

Salmonella Typhimurium was cultured on stool from blood from 173 cases (54%). Thirty-nine human isolates from cases were typed using MLVA and had MLVA type 01-03-20-04-06. These isolates exhibited a phage reaction pattern on mPCR/RLB that was consistent with *Salmonella* Typhimurium phage type 9, subsequently confirmed by phage typing. No other types of *Salmonella* were isolated from cases.

Environmental investigation

The bakery is a busy premises located near a train station in the inner west of Sydney. The bakery had previously been inspected by the NSW Food Authority in September 2005 during a food handling survey. The premises consisted of a shop fronted by a serving counter and a rear preparation area. The front counter was fitted with a glass pastry display unit and an adjoining takeaway food bar unit used to display ingredients for the preparation of pork and chicken rolls. The rolls contained any combination of ingredients including sliced hams, sliced pork, marinated cooked diced chicken, pate, raw egg mayonnaise, shredded carrot, coriander and cucumber.

The inspection and interviews with the owners of the premises revealed that there was a lack of effective sanitation of food handling equipment and surfaces. It was also noted that there was a refrigeration malfunction in the early hours of the morning of 25 March (after the outbreak had commenced). Inadequate refrigeration was also recorded for the raw egg mayonnaise in the display unit.

Salmonella Typhimurium phage type 9 was detected in 15 isolates from food and environmental samples taken at the source premises. *Salmonella* Typhimurium was isolated in the raw egg mayonnaise, ham, pork, chicken, pate and shell eggs and from swabs of the preparation bench, tongs, meat slicer, floor drain and display tray. All isolates from food and environmental samples were MLVA type 01-03-20-04-06, which is identical to that isolated from cases. A quantitative analysis of the raw egg mayonnaise sample yielded a count in excess of 1.1×10^7 colony forming units/mL.

The eggs used in the raw egg mayonnaise were traced to a farm in outer Sydney. The farm was inspected on 4 April 2007. The drag swab from a laying shed on the egg farm and from a meat meal sample were positive for *Salmonella* although subsequent serotyping and phage typing revealed that the egg farm isolates did not match the human and food isolates linked to the outbreak.

During the inspection of the premises on the morning of 27 March the NSW Food Authority removed the raw egg mayonnaise from sale. By 5 pm 27 March, a prohibition order was issued to the premises by the NSW Food Authority to prevent sale of chicken and pork rolls and associated ingredients. Upon receipt of analytical results a subsequent prohibition order was issued on 30 March preventing the business from operating.

Discussion

This describes one of the largest point source outbreaks of *Salmonella* in Australia for many years. Similar outbreaks linked to Vietnamese pork rolls in Victoria in 1997 resulted in 774 and 154 cases.[7] Other large outbreaks in recent years have related to commercially available products, including chicken meat[8] and fruit juice.[9] In the outbreak described in this report, routine surveillance through statutory reporting and active case finding identified 319 cases, about half of which were laboratory confirmed. This outbreak is notable for its scale, the severity of illness experienced by the cases and the degree of contamination at the point source.

A large proportion of cases required hospital admission, with many requiring prolonged stays. Hospital admission for salmonellosis generally only occurs in more severe cases, with most cases (estimated to be over 90%) being treated in the community.[1] In this outbreak, two-thirds of all identified cases presented to an emergency department at some point during their illness and half were admitted to hospital. In addition, the average duration of illness in this outbreak was long. Salmonellosis usually results in diarrhoea lasting 3–7 days,[3] whereas an average duration of diarrhoea of 14 days was observed in this outbreak, and many experienced illnesses of longer duration of up to 45 days. Over half of the cases in this sub-investigation reported requiring intravenous fluid replacement due to dehydration, further indicating that illness was severe.

It is estimated that the attack rate was close to 100%, which is consistent with or higher than similar outbreaks reported elsewhere.[10–13] The incubation period decreased as the outbreak progressed, indicating significant contamination at the point source, and likely bacterial growth in foods and spread to surfaces as the outbreak progressed. Inadequate temperature controls when storing foods, as was found in the environmental investigation, would have contributed to bacterial proliferation. The environmental investigation revealed widespread contamination throughout the premises; *Salmonella* was detected in most foods used in the preparation of the chicken and pork rolls, on the food slicer, preparation bench, serving tongs, display tray and floor drain. As a result, a specific source of contamination was not established. It is clear, however, that there were deficiencies in food handling and sanitation contributing to the proliferation of the organism and its spread throughout the premises after the initial contamination occurred. Similar deficiencies in handling and cross contamination were noted in 2 other outbreaks linked to similar premises in Victoria.[7]

The 2 most likely sources of the initial contamination are the eggs used for the raw egg mayonnaise or, less likely, an asymptomatic food handler. *Salmonella* was identified in the stool of a food handler with only mild symptoms of stomach pain after the outbreak had subsided, but it was not possible to confirm whether carriage commenced during or prior to the outbreak. Positive stool specimens were found in 2 asymptomatic food handlers working at a bakery associated with an outbreak of *Salmonella* Typhimurium phage type 9 in Victoria in 1997.[7] The significance of detection of *Salmonella* in this previous outbreak was also not established. Under the *NSW Food Act 2003* (with reference to the *Australia New Zealand Food Standards Code* covering health and hygiene requirements for food handlers) [14] food handlers with symptoms of foodborne illness, or carriage, are prohibited from handling food if there is a possibility they may contaminate it. NSW Health recommends that food handlers who have diarrhoea are excluded from work for 48 hours after

symptoms resolve. Neither the *NSW Food Act 2003* or the *Food Standards Code* allow for this and consideration should be given to strengthen, and clarify, the restriction of food handlers who are ill.

Salmonella Typhimurium phage type 9 was identified on the shells of one open tray of eggs kept on the premises. However, no *Salmonella* was found on eggs contained in 2 closed cartons also on the premises. Traceback to the farm identified other *Salmonella* serovars but not *Salmonella* Typhimurium phage type 9, however it is possible that *Salmonella* serovars may move in and out of poultry flocks with the introduction of contaminated feed, or other inputs and excretion of *Salmonella* may be intermittent.[11] Raw eggs are frequently implicated in large outbreaks of *Salmonella*[11] and in this outbreak it is believed that heavily contaminated mayonnaise, and subsequent cross contamination was the most the likely cause. Egg related outbreaks have increased in Australia in recent years. OzFoodNet data show that there have been 63 outbreaks in the last 3 years where egg or egg based dishes were suspected to be the source; 14% of all foodborne illness outbreaks in 2005 were considered to be related to eggs, compared with 23% in 2008.[15–20] While eggs are the most likely vector for the majority of these outbreaks, poor hygiene and food handling at retail level is also a major contributing factor and influences the size and severity of the incident.

There are several approaches to reduce the occurrence of such large outbreaks resulting from service of high risk foods, including raw egg based products. Starting with a raw product that has the lowest level of contamination possible is key. The reduction of pathogens on egg farms is a priority and many countries have developed schemes and/or regulations to reduce human pathogens such as *Salmonella* and *Campylobacter* in poultry.[21,22] In Australia, Food Standards Australia New Zealand (FSANZ) is developing a Primary Production and Processing Standard for Eggs and Egg Products.[23] This will impose requirements on producers and users of eggs at the primary production and retail level. The draft standard addresses specific food safety risks associated with cracked and dirty eggs and prohibits sale of unpasteurised egg pulp for retail or catering purposes. In New South Wales, the NSW Food Authority will be responsible for implementation and enforcement of the national standard. Additional cooperation between government regulators and industry is vital to ensure that food safety measures are of the highest standard to protect public health. Stronger, proactive engagement by government with industry, including the egg industry, will assist with better sharing of information and earlier identification of hazards in the food production chain.

The NSW Food Authority is working with all New South Wales local councils, who are responsible for routine inspection of retail businesses such as bakeries and cafés, to educate businesses on the risks of using products containing raw eggs. In 2005 the bakery at the source of this outbreak was presented with information about the risks of raw egg based foods and also advised to use a commercial, pasteurised mayonnaise product. This advice was ignored and a large fine was subsequently imposed by the courts as a result of prosecution action undertaken by the NSW Food Authority. This is a powerful deterrent for businesses to avoid using raw egg products where possible. Standard fact sheets and warning letters have been developed for distribution by local council officers to businesses where these are found to be using raw egg products.

The widespread contamination in the bakery indicates that food handling skills were not adequate. A national food handler survey undertaken by FSANZ in 2007 indicated that bakeries were less proficient in food handling activities compared with other businesses surveyed.[24] Protecting public health through food safety is primarily the responsibility of jurisdictional food regulators. The NSW Food Authority is responsible for regulating the food industry through the *NSW Food Act 2003*. This Act, through the *Food Standards Code*, requires food handlers to have adequate skills and knowledge in food safety and food hygiene if employed in food service. The owner of the business is responsible for ensuring staff have adequate skills and knowledge appropriate for their activities within the business. However, no formal training is currently required, even for the owner. In New South Wales this is being addressed through implementation of a Food Safety Supervisor Initiative. This will require every retail food business in New South Wales to have a dedicated food safety supervisor with responsibility for staff training. The scheme will be implemented in the middle of 2010.

Outbreaks, even in small premises, can result in high morbidity, high cost to the community and have a significant impact on health care provision. The scale and impact of this outbreak augurs for continued investment in risk assessment and food safety measures at all stages throughout the food chain and regulation of primary producers and food premises.

The most notable feature of this outbreak is its scale, the health and economic consequences for those affected and the resultant burden on the health system of a preventable foodborne illness. This outbreak is likely to have resulted in considerable costs to the healthcare system and the community. Given the number of people presenting to EDs and requiring admission to hospital, the cost to the hospitals involved would have been significant.

This outbreak was initially reported to local health authorities by an astute ED clinician. Early notification by clinicians requires the development of good relationships with their local public health authority. Continued co-operation between NSW Health and the NSW Food Authority will provide for early identification of outbreaks and timely public health action to protect public health. These authorities must seek to continually improve outbreak response procedures to reduce the impact on public health through, among other avenues, regular evaluation of outbreak response. Based on the estimated attack rate and the degree of environmental contamination, it is likely that the closure of the bakery prevented many more cases occurring. The co-operation and timely communication between clinicians at the coal face, public health officials and the regulators provided an opportunity for urgent and responsible public health action.

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Surveillance summaries

Supplementary report: surveillance of adverse events following immunisation among children aged less than seven years in Australia, 1 January to 30 June 2009

Deepika Mahajan, Rob Menzies, Ilnaz Roomiani, Glenda Lawrence

Introduction

This report summarises national passive surveillance data reported to the Therapeutic Goods Administration (TGA) to 31 August 2009 for adverse events following immunisation (AEFI) in children aged less than 7 years who received vaccines between 1 January and 30 June 2009. The report includes all vaccines administered to children in this age group with a focus on the vaccines included in the funded National Immunisation Program (NIP) schedule.[1]

The most recent change to vaccine funding and availability that impacts on the AEFI surveillance data in this report compared with the same period in 2008 is the changeover to the single hexavalent DTPa-IPV-HepB-Hib vaccine for all children at 2, 4 and 6 months of age,[2–4] due to an international shortage of some *Haemophilus influenzae* type b (Hib) (PedvaxHib® [monovalent] and Comvax® [Hib-HepB] vaccines.[5] In March 2008, Queensland, South Australia and Victoria changed from using 2 combination vaccines (quadrivalent DTPa-IPV and Hib-HepB) to the single hexavalent DTPa-IPV-HepB-Hib vaccine. In February 2009, Western Australia stopped using PedvaxHib® for Indigenous children so that all children received the single hexavalent DTPa-IPV-HepB-Hib vaccine. The Northern Territory continued using Comvax® until October 2009, when it also changed to the hexavalent vaccine. All other jurisdictions had already been using the hexavalent vaccine since November 2005.

The data reported here are provisional only. It is important to note that an AEFI is defined as a medical event that is temporally associated with immunisation but not necessarily causally associated with immunisation. Readers are referred to previous reports for a description of the national AEFI passive surveillance system,[6] methods used to analyse the data and information regarding limitations and interpretation of the data.[6–9] Often, several vaccines and reaction codes are listed in an AEFI record so the number of vaccines and reaction codes will exceed the total number of AEFI records. For the purpose of this report, an AEFI is defined as 'serious' if there is a code of life-threatening severity or an outcome code indicating recovery with sequelae, admission to hospital, prolongation of hospitalisation or death.

Average annual population-based AEFI reporting rates were calculated using mid-2008 population estimates. Reporting rates per 100,000 doses were calculated for vaccines on the NIP schedule for which reliable dosing data were available, for children aged from 2 months to < 7 years, using data from the Australian Childhood Immunisation Register (ACIR).

Results

There was a total of 305 AEFI records (annualised reporting rate of 32.0 per 100,000 population) for children aged < 7 years for vaccines administered in the first 6 months of 2009. This was an 18% decrease on the 374 records (39.2 per 100,000 population) for the corresponding period in 2008. Forty-four percent (n = 134) of the 305 AEFI records for the 2009 reporting period were for children aged < 1 year; 12% (n = 36) for those aged 1 to < 2 years; and 44% (n = 135) were for the 2 to < 7 year age group, similar to the age distribution in previous years.[7–10] The male to female ratio was 1.1:1, also similar to previous years.[7,9]

Of the 305 records, 24 listed one or more vaccines for which accurate dose information was not available. These were influenza (n = 10), 23-valent pneumococcal polysaccharide (n = 2), hepatitis A (n = 2), hepatitis B (n = 5), Inactivated poliomyelitis (IPV) (n = 1) and bacille Calmette-Guérin (n = 4) vaccines. AEFI reporting rates per 100,000 doses were calculated for the remainder of records (281) (Table). This was an overall AEFI reporting rate of 14.7 events per 100,000 doses recorded on the ACIR, lower than the rate for the corresponding period in 2008 (16.9 per 100,000 doses). Rates for all age groups

and reaction categories were lower in 2009 than for the same period in 2008 (Table). The age group with the largest reduction was children aged 2 to < 7 years (24%). Reporting rates for most vaccines were similar to, or lower than, those for the same period in 2008. There were substantial decreases in reported AEFI following receipt of diphtheria-tetanus-pertussis (DTP)-containing vaccines (12%), rotavirus (11%) and varicella (54%) vaccines. There were increases in reports following meningococcal C (20%), measles-mumps-rubella (7%) and pentavalent (DTPa-IPV-HepB) and Hib-HepB, although the total number of reports and doses administered for these latter 2 vaccines were small.

Twelve per cent (n = 37) of the 305 AEFI records had outcomes defined as 'serious' (i.e. recovery with sequelae, or hospitalisation, or life-threatening event or death), a rate of 1.8 per 100,000 doses, and lower than for the corresponding period in 2008 (2.3 per 100,000 doses). There was 1 report of life-threatening event and another 36 children were admitted to hospital. The only report of life-threatening event was apnoea, bradycardia and seizures in an infant born prematurely at 29 weeks, which followed the first vaccination with hexavalent DTPa-IPV-HepB-Hib, 7vPCV, and rotavirus vaccines at 38 weeks of age. The infant was receiving medication for apnoea of prematurity at the time of vaccination. Seizures and apnoea are listed in the product information for hexavalent DTPa-IPV-HepB-Hib and 7vPCV but not for rotavirus vaccine. Serious and other significant AEFIs reported included hypotonic-hyporesponsive episodes (HHE) (n = 17 of which 4 were associated with hospitalisation), seizures (n = 12), intussusception (n = 4) and anaphylaxis (n = 1). For the first 6 months of 2009, the overall reporting rate was the same as in 2008 for HHE in

Table: Reporting rates of adverse events following immunisation (AEFI) per 100,000 vaccine doses,* children aged less than 7 years, Therapeutic Goods Administration database, 1 January to 30 June 2009

	AEFI records‡ (n)	Vaccine doses* (n)	Reporting rate per 100,000 doses§		
			Jan–June 2009	Jan–June 2008	Jan–June 2007
Vaccine†					
DTPa-containing vaccines	212	533,490	39.7	45.1	29.2
DTPa-IPV	110	144,624	76.1	77.6	40.4
Pentavalent (DTPa-IPV-HepB)	3	6,450	46.5	11.2	42.0
Hexavalent (DTPa-IPV-HepB-Hib)	99	382,416	25.9	24.4	8.9
Haemophilus influenzae type b	23	129,697	17.7	17.4	18.0
Haemophilus influenzae type b-hepatitis B	3	3,586	83.7	40.6	24.9
Measles-mumps-rubella	98	277,992	35.3	33.1	17.8
Meningococcal C conjugate	26	138,678	18.7	15.6	9.2
Pneumococcal conjugate	106	390,026	27.2	28.4	17.9
Varicella	10	133,482	7.5	16.4	12.6
Rotavirus	102	306,024	33.3	37.2	50.9
Age group					
< 1 year	124	1,102,955	11.2	12.8	8.1
1 to < 2 years	35	491,792	7.1	7.4	5.2
2 to < 7 years	122	318,228	38.3	50.5	35.6
AEFI category†					
Total	281	1,912,975	14.7	16.9	11.9
'Certain' or 'probable' causality rating	43	1,912,975	2.2	5.1	4.2
'Serious' outcome	34	1,912,975	1.8	2.3	1.2

* Number of vaccine doses recorded on the Australian Childhood Immunisation Register (ACIR) and administered between 1 January and 30 June 2009.

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

‡ Number of AEFI records in which the vaccine was coded as 'suspected' of involvement in the reported adverse event and the vaccination was administered between 1 January and 30 June 2009. More than 1 vaccine may be coded as 'suspected' if several were administered at the same time.

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

total (1.5 per 100,000 doses) and HHE following DTPa-IPV-HepB-Hib (3.2 per 100,000 doses in 2009 versus 3.1 per 100,000 doses in 2008).

The most commonly reported reaction categories included injection site reaction (ISR) (n = 121; 40%), fever (n = 54; 18%), allergic reactions (n = 46; 15%), gastroenteritis following rotavirus vaccination (n = 37; 12%), rash (n = 34; 11%), screaming (n = 20; 7%) and seizure (n = 12; 4%), similar to the distribution in 2008.

Discussion

The total number of AEFI records and population-based reporting rates was 18% lower for the first 6 months of 2009 compared with the corresponding period in 2008. This reduction appears to be due to a combination of several factors. Firstly, approximately 4% fewer vaccine doses were administered in the 1st half of 2009 compared with 2008 due to the more widespread use of hexavalent vaccine for primary schedule doses across Australia. When expressed as a reported AEFI rate per 100,000 doses, the difference was less marked, being 12% lower in 2009 than in 2008. Secondly, reporting delay is likely to account for a level of under-estimation in the latest period (2009) similar to that seen in 2008 and 2007 of between 5% and 20%,[7,9] which would result in upwards revision for the final report. Thirdly, it is also likely that the decline in reports for infants is at least partly due to a stabilisation following the expected initial peak following the introduction of the rotavirus vaccine in 2007 (Figure). Unlike recent reports, there have been no known changes in surveillance systems expected to impact on this period.

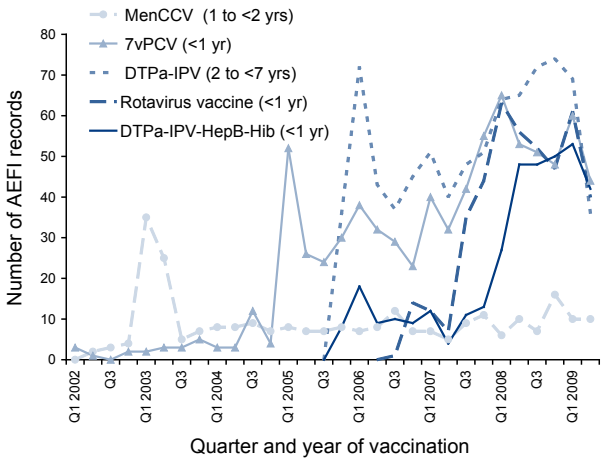
The increase in reporting rates for pentavalent DTPa and Hib-HepB should be interpreted with caution as the total number of doses administered and reported AEFI were low.

The episode of apnoea and reporting trends of HHE are deserving of further comment. Apnoea following immunisation in an infant born prematurely is a well recognised occurrence, which is usually self-limiting.[1] Trends in reporting HHE among children aged < 1 year are of particular interest. The reporting of HHE following receipt of hexavalent vaccine during the 1st half of 2009 (3.2 per 100,000 doses) was similar to that in 2008 (3.1 per 100,000 doses), after an increase from 0.5 per 100,000 doses in 2007 which has been attributed to changes in surveillance methods in Victoria.[11,12]

The observed reduction in the reporting rate for children aged 2 to < 7 years in 2009 compared with 2008 (38.3 and 50.5 per 100,000 doses, respectively) is primarily related to a reduction in ISR following acellular pertussis-containing vaccines. This AEFI is known to be very common among children receiving a 4th and 5th dose of acellular pertussis-containing vaccine.[6,8,13,14] The reporting rate of ISR in this age group declined in recent years (48 per 100,000 doses in 2007 compared with 78 per 100,000 doses in 2006), as was expected following the removal of the dose due at 18 months of age from the NIP schedule in September 2003. The reasons for the peak in 2008 and subsequent decrease are not entirely clear and may be at least partly due to changes in the propensity of general practitioners and others to notify adverse events and to reporting delays.

As discussed in previous reports, intussusception was associated with a previous rotavirus vaccine released only in the United States of America (USA) in 1998, and withdrawn from the market after 8 months.[15,16] All new generation rotavirus vaccines were closely monitored for intussusception during clinical trials and no association has been identified between the vaccines and intussusception.[17,18] Post-marketing surveillance for intussusception in association with vaccination continues internationally and, in Australia, through the Paediatric Active Enhanced Disease Surveillance pilot project. The 4 cases reported in the 1st half of 2009 is less than the number reported for the same period in 2008 (n = 11). The reporting rate in Australia in 2008 was found to be similar to that in the USA.[7]

Figure: Reports of adverse events following immunisation, Therapeutic Goods Administration database, 1 January 2002 to 30 June 2009, for vaccines recently introduced into the funded National Immunisation Program*



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

Conclusion

In the first half of 2009 the AEFI reporting rate per 100,000 doses was 12% lower than for the same period in 2008. This appears to be attributable to a stabilisation of reports after the initial peak at the commencement of the rotavirus vaccination program in 2007, fewer reports of injection site reactions following pertussis-containing vaccines and perhaps also reporting delay. The majority of AEFIs reported to the TGA were mild transient events and the data reported here are consistent with an overall high level of safety for vaccines included in the NIP schedule.

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Abbreviations of vaccine types

DTPa	diphtheria-tetanus-pertussis (acellular) – paediatric formulation
DTPa-IPV	combined diphtheria-tetanus-pertussis (acellular) and inactivated poliovirus (quadrivalent)
DTPa-IPV-HepB	combined diphtheria-tetanus-pertussis (acellular), inactivated poliovirus and hepatitis B (pentavalent)
DTPa-IPV-HepB-Hib	combined diphtheria-tetanus-pertussis (acellular), inactivated poliovirus, hepatitis B and *Haemophilus influenzae* type b vaccine (hexavalent)
HepB	hepatitis B
Hib	*Haemophilus influenzae* type b
Hib-HepB	combined *Haemophilus influenzae* type b and hepatitis B
IPV	inactivated poliovirus vaccine
7vPCV	7-valent pneumococcal conjugate vaccine

Short reports

An outbreak of Shiga toxin-producing *Escherichia coli* infection associated with a school camp

Bradley J McCall, Vicki G Slinko, Helen V Smith, Karen Heel, Terry H Culleton, Virgil R Kelk, Russell J Stafford

Abstract

In November 2008, a case of Shiga toxin-producing *Escherichia coli* (STEC) infection was reported to the Brisbane Southside Public Health Unit. The case had participated in a school camp. Subsequent investigations confirmed 5 other asymptomatic cases among camp attendees or visitors. Examination of the camp water supply identified that most water sources had high levels of *E. coli* and did not meet the *Australian Drinking Water Guidelines* with STEC isolated from 2 water sources. This outbreak highlights the emerging issue of asymptomatic carriage of STEC and the importance of thorough maintenance and attention to drinking water supplies in the rural and school camp setting. *Commun Dis Intell* 2010;34(1):54–56.

Keywords: *Escherichia coli*, foodborne illness, outbreak, school camp

Introduction

Shiga toxin-producing *Escherichia coli* (STEC), of which enterohaemorrhagic *Escherichia coli* (EHEC) are a sub-group, are an important cause of gastroenteritis with substantial associated mortality and morbidity. In up to 8% of cases the infection may progress to haemolytic uraemic syndrome (HUS) in children or thrombocytopenia purpura in adults.[1] Management is generally supportive with some evidence that antibiotics may prolong excretion of the organism or increase the risk of HUS.[2–4] Prevention of primary infection remains the best way to prevent the serious outcomes of STEC infection.

Large outbreaks of STEC infection are frequently reported from Europe, North America and other developed countries with contaminated food or water the most common source of transmission.[5–7] In contrast there have been few reported outbreaks of STEC in Australia.[8,9] Notifications of sporadic STEC cases have been increasing in Australia and Queensland since 2002.[10,11] This may be related to improved laboratory surveillance or an actual increase in disease incidence.[12]

In November 2008 the Brisbane Southside Public Health Unit received a notification of STEC infection in a teenage male. Case follow up identified a remote rural school camp as the likely source of infection. This paper describes an outbreak of STEC infection associated with the water supply at this camp.

Methods

Case investigation, contact tracing and collection of faecal samples was commenced in accordance with Queensland Health protocols.[13] The investigation included questionnaire and faecal sampling of household and school camp contacts of the index case.

A case was defined as:

- isolation of Shiga (-like) toxin-producing/vero toxin-producing *E. coli* from faeces: or
- detection of Shiga (-like) toxin or vero toxin from a clinical isolate of *E. coli*: or
- identification of the gene associated with the production of Shiga (-like) toxin or vero toxin in *E. coli* by nucleic acid testing on isolate or fresh faeces.

The school was contacted to provide information on other children attending the school camp. An environmental health investigation was undertaken at the site of the school camp. Water samples from rainwater tanks, bores and other potential environmental sources were collected and tested for evidence of STEC at Queensland Health Forensic and Scientific Services.

Students who attended the camp with the index case completed a questionnaire that included demographics and details of potential exposures at the camp, including water sources and facilities used, as well as animal and environmental exposures. Stool samples were requested from those who attended the school camp including school and site mainte-

nance staff. If other camp attendees were found to be positive for STEC, their household contacts were offered faecal sample testing for STEC.

Results

Various classes from the school had spent 4 weeks living in 4 dormitories (2 male, 2 female) at the camp throughout the school year. Twenty-five students, 3 teaching staff, a family of 4 residents and an unknown number of parents and family members on day visits were at the camp during the period 2–28 November 2008. Each dormitory contained its own cooking, shower and toilet facilities. No specific water source or other potential risk factors were identified among 20 students who completed the questionnaire.

A faecal sample from the index case was positive for Shiga-like toxin, had *stx1*, *stx2*, *eaeA* and *ehxA* genes detected and was serotyped as O26:H11. All faecal samples from household contacts of the index case were negative for STEC.

Thirteen other students provided faecal samples. Four students (who were asymptomatic) tested positive for a variety of STEC genes. These were: O112ab:H2 (2), O88 related:H25 (1), Ont:H7 (1), O174:H- (1), and O153:H21 (1). One of 15 household contacts of the 4 asymptomatic STEC cases tested positive for *stx2*. This was from a parent who had visited the camp for a Parents Day and consumed water from a container in one of the common areas prior to the introduction of bottled water measures.

STEC was isolated from 2 water samples collected from the camp. The sample from the case's dormitory had O2:H8 isolated and *stx1*, *stx2*, *ehxA* and *saa* genes detected. The 2nd positive sample had O91:H10 isolated and also had *stx2*, *ehxa* and *saa* genes detected. Ten of 13 samples from rainwater tanks and their outlets had unacceptably high levels of *E. coli* (up to 310 *E. coli* per 100 ml) and did not meet the microbiological requirements of the National Health and Medical Research Council *Australian Drinking Water Guidelines 2004*.[14] The bore water was found to be microbiologically safe. No Shiga toxin genes were detected in animal faecal samples collected at the camp.

Food preparation and general camp hygiene were described as satisfactory. Bore water was supplied to the toilets and showers and there were rainwater tanks that supplied the kitchen and various other amenities blocks. These tanks varied in size, construction materials and relative age. They were not fitted with 'first flush' devices (rainwater diverters), had not been cleaned out for many years and were not subject to any form of disinfection. There was no documented evidence of a routine maintenance, chemical or microbiological testing program for either the tank or bore water supply.

At the time of the environmental health investigation all camp attendees were advised to use only bottled water for drinking and cleaning teeth and that any other illness in children or staff should be reported. Apart from the case, all other attendees or residents remained well.

Discussion

This is the 1st reported Australian outbreak of STEC among children participating in a rural school camp. It is not possible to attribute the parent case with certainty, to the consumption of camp water or subsequent household contact. However, the variety of STEC serotypes identified in camp attendees, the detection of a variety of STEC isolates and genes in 2 water sources and the high level of *E. coli* contamination of water sources, lead us to postulate that the tank water supply at this camp was the most likely source of this outbreak.

Waterborne outbreaks of STEC have been well documented in both recreational and drinking water.[15] This outbreak demonstrates the potential risks associated with rainwater tanks where there is no active maintenance program in place and/or where there is no disinfection of the water prior to consumption. Not only were 2 drinking water sources contaminated with STEC, but most of the tank water sampled did not meet the standards for the current *Australian Drinking Water Guidelines*. This potential risk is amplified in the setting of a school camp.

As a result of this outbreak, Queensland Health required remedial action to be undertaken at the school camp to ensure the safety of the drinking water supply. The tank water reticulation system has been redesigned, ultraviolet sterilisation of the drinking water sources has been instituted and all the drinking water tanks have been cleaned and disinfected. Queensland Health is also reviewing its *Guidelines for Prevention and Management of Gastroenteritis Outbreaks in Camp Facilities* to place greater emphasis on the provision and maintenance of potable water supplies.

STEC infections have been notifiable in most jurisdictions since 2000. Routinely, clinical diagnostic laboratories only culture for *E. coli* O157. Therefore, STEC infections of other serotypes are likely to be under-reported. In a recent survey over 3 months at QHFSS laboratory, 4.7% of clinical diagnostic laboratory referred faecal samples that had evidence of blood but no other pathogen isolated were positive for non-0157 STEC infection (Helen Smith,

personal communication). This referral practice was an important factor in the identification of this outbreak.

In addition, this outbreak is notable for the high degree of asymptomatic carriage of STEC in those tested. Asymptomatic carriage has been previously described in children[16,17] and in rural populations.[18] It is currently not known how long asymptomatic carriage may occur prior to the onset of infection or if there are certain physiological triggers that can switch carriage to infection. While the clinical relevance of asymptomatic carriage of STEC is undetermined, the potential serious sequelae of STEC infection places further importance on measures that improve the detection of asymptomatic carriage of STEC and support disease control measures. This outbreak also demonstrates the importance of thorough maintenance and attention to drinking water supplies in the rural and school camp setting. As testing for STEC becomes more commonplace we expect that further similar outbreaks will be identified.

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Viraemic importations of dengue into north Queensland, 2009

Jeffrey N Hanna, Ann R Richards, Juliet V Esmonde, Steven Donohue, Jan L Humphreys, Alyssa T Pyke, Carmel T Taylor

Although dengue viruses are not endemic in north Queensland, the vector mosquito, *Aedes aegypti*, is present in urban settings in the region and on some Torres Strait islands. This means that north Queensland is prone to outbreaks of dengue, each one apparently initiated by a viraemic traveller recently arrived from a dengue-affected country abroad. For this reason, it is essential that viraemic importations of dengue from overseas into north Queensland are promptly recognised and notified so that appropriate mosquito-control measures can be rapidly implemented.

There were 28 laboratory-confirmed viraemic importations of dengue from overseas into north Queensland in 2009. (For public health purposes, in north Queensland the duration of viraemia is considered to be from 1 day before until 12 days after the onset of symptoms.[1]) This is the highest number of such importations recognised in any 1 year on record; previously the highest was 17 importations in 2008.[2] Four separate outbreaks of dengue occurred in north Queensland early in 2009, two of which had continued from 2008 into 2009. The increased awareness and testing surrounding those outbreaks may have increased the likelihood of detecting a newly imported case. Nevertheless, the recent increasing trend in importations[2] is consistent with the deteriorating dengue situation globally.[3]

Most (57%) of the infections were acquired in South East Asian countries (Table 1); these cases were acquired throughout the year. However, 32% were acquired in the South Pacific island nations (Samoa, Vanuatu, Fiji, Cook Islands and Tonga); all of these South Pacific cases were acquired in the first 4 months of the year. Again, there were relatively few (only 2) viraemic importations of dengue from Papua New Guinea.[2]

All 4 dengue virus serotypes were detected among the viraemic importations (Table 2). Four of the infecting viruses could not be serotyped because of serological cross-reactions. However one of these led to a serotype 1 outbreak (described below), and two were almost certainly serotype 4 as they were both acquired in South Pacific island nations.[4] The prominence of serotype 4 infections acquired in the South Pacific islands (8, but probably 10 cases) is striking. This is a reflection upon the widespread serotype 4 epidemic throughout the region since mid-2008,[4] and the burgeoning South Pacific island migrant population in north Queensland, several of whom acquired the infection while visiting their country-of-origin.

Table 1: Region/country where the viraemic importations of dengue were acquired

Region/country where the infections were acquired	Number of viraemic importations into north Queensland in 2009
South East Asia	16
South Pacific island nations	9
Papua New Guinea	2
Indian sub-continent	1

Table 2: Dengue virus serotypes detected among the viraemic importations

Dengue virus serotype	Number of viraemic importations into north Queensland in 2009
1	6
2	4
3	6
4	8
Not able to be serotyped	4

Two of the viraemic importations initiated outbreaks of dengue in north Queensland:

i. A 28-year-old woman became unwell a day after returning to Innisfail (~80 km south of Cairns) from Vanuatu. She saw a local medical practitioner the following day; dengue (subsequently identified as serotype 4) was notified 4 days after the consultation. She was viraemic in Innisfail for 6 days before mosquito-control measures were taken, and led to an outbreak with a total of 35 cases that lasted for 15 weeks.

ii. Local transmission of dengue was recognised in 2 suburbs of Townsville in late October to early November. The serotypes were soon identified

as type 1, and genotyping subsequently confirmed that serotype 1 viruses from the 2 suburbs were identical. During the outbreak investigation a 64-year-old man, who had become ill a day after returning from Timor-Leste in late September, was recognised. He saw a local medical practitioner 5 days after the onset; the practitioner diagnosed an influenza-like illness but did not consider dengue. The man had very strong links to cases in the 2 suburbs, and was subsequently shown to have had a secondary dengue infection, but his infecting serotype could not be identified. The outbreak consisted of a total of 10 cases and lasted 7 weeks (as of the end of 2009).

The median delay between the initial medical consultation in north Queensland and the notification of the cases to the local Public Health Unit was 3 days, with a range between 0 (when the notification occurred on the same day as the consultation) and 61 days (this importation was only recognised retrospectively during the investigation of an outbreak; see above). It is encouraging that the timeliness of the notifications has improved when compared with that documented in 31 importations into north Queensland over a decade ago (Table 3).[1] The improvement is probably mainly due to increased awareness among local medical practitioners, but the recent introduction of NS1 antigen testing (which is particularly useful early in the illness[5]) at the 2 main hospital laboratories in north Queensland may also have contributed.

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Table 3: The timeliness of the notifications of the viraemic importation in the 1990s, compared with that in 2009

	December 1994 to November 1998 (n=31)	2009 (n=28)
Median delay (days)	5.5	3
Notified on day of initial consultation	4 (13%)	6 (21%)
Notified within 48 hours of initial consultation	8 (26%)	11 (39%)
Notified within a week of initial notification	19 (61%)	24 (86%)

Quarterly reports

OzFoodNet quarterly report, 1 October to 31 December 2009

The OzFoodNet Working Group

Introduction

The Australian Government Department of Health and Ageing established OzFoodNet in 2000 to collaborate nationally to investigate foodborne disease. OzFoodNet conducts studies on the burden of illness, co-ordinates national investigations into outbreaks of foodborne disease, develops nationally standardised protocols and tools for surveillance, identifies foods or commodities that may cause human illness and trains people to investigate foodborne illness. This quarterly report documents investigation of outbreaks of gastrointestinal illness and clusters of disease potentially related to food, occurring in Australia from 1 October to 31 December 2009.

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

During the 4th quarter of 2009, OzFoodNet sites reported 622 outbreaks of enteric illness, including those transmitted by contaminated food. Outbreaks of gastroenteritis are often not reported to health agencies or the reports may be delayed, meaning that these figures under-represent the true burden of enteric illness. In total, these outbreaks affected 13,013 people, of whom 372 were hospitalised. There were 33 deaths reported during these outbreaks. The majority of outbreaks (87%, n=541) were due to person-to-person transmission (Table 1).

Table 1: Mode of transmission for outbreaks of gastrointestinal illness reported by OzFoodNet, 1 October to 31 December 2009

Transmission mode	Number of outbreaks	Percent of total
Foodborne and suspected foodborne	42	7
Person-to-person	541	87
Salmonella cluster	5	1
Suspected waterborne	2	<1
Unknown	32	5
Total	622	100

Foodborne and suspected foodborne disease outbreaks

There were 42 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as the primary mode of transmission (Table 2). These outbreaks affected 741 people and resulted in 48 hospitalisations. There were 2 reported deaths during these outbreaks. This compares with 31 outbreaks for the 4th quarter of 2008 [1] and 28 foodborne outbreaks for the 3rd quarter of 2009.[2] In addition to these newly reported outbreaks, in a continuing multi-jurisdictional outbreak of hepatitis A,[3] there were 193 cases of locally-acquired hepatitis A notified in Australia this quarter.* Included amongst these were 18 cases that are reported as 3 small foodborne outbreaks in single jurisdictions, which are included in outbreaks for this quarter.

Salmonella was the aetiological agent for 11 outbreaks during this quarter, with *S.* Typhimurium being the most common serotype (n=9). Of the remaining 31 outbreaks, 13 were due to norovirus, three to hepatitis A, two to *Clostridium perfringens*, two to *Escherichia coli*, and one each to *Campylobacter* and fish wax esters (or escolar). For 9 outbreaks, the aetiological agent was unknown or not specified.

Twenty outbreaks (48%) reported in this quarter were associated with food prepared in restaurants, four (10%) were associated with aged care facilities, four (10%) with private residences, three each (7%) were associated with commercial caterers and commercially manufactured food. Two outbreaks (5%) were associated with foods prepared in a military institution, two (5%) with foods prepared in a takeaway and two (5%) with camps. One outbreak (2%) was associated with a fair/festival or mobile service, and one (2%) with primary produce.

To investigate these outbreaks, sites conducted 14 cohort studies, 3 case-control studies, and col-

* This outbreak is considered to be a continuation of a previously reported outbreak and as such is not included in Table 2.

Table 2: Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 October to 31 December 2009 (n=42)

State	Month of outbreak	Setting prepared	Agent	Number affected	Hospitalised	Evidence	Responsible vehicles
NSW	Oct	Commercially manufactured	*S.* Typhimurium 170	4	0	M	Layered chocolate cake (no raw eggs used)
	Oct	Fair/festival/mobile service	Unknown	3	1	D	Unknown, possibly prawns or calamari
	Oct	Private residence	Unknown	8	0	D	Unknown
	Oct	Restaurant	Unknown	4	0	D	Unknown
	Oct	Restaurant	Unknown	4	0	D	Unknown, possibly salad items
	Nov	Commercially manufactured	Unknown	28	0	D	Unknown
	Nov	Private residence	*S.* Typhimurium 135	6	2	D	Unknown, probably tiramisu prepared with raw eggs
	Nov	Restaurant	*S.* Typhimurium	3	0	M	Cooked pork mince and leftover food (mix of tofu, rice, duck)
	Nov	Restaurant	Unknown	7	0	D	Unknown
	Dec	Restaurant	*S.* Singapore	3	0	M	Fried ice cream prepared with raw eggs, omelette
	Dec	Takeaway	Norovirus	30	0	D	Unknown
	Dec	Takeaway	*S.* Stanley	32	6	D	Unknown
NT	Dec	Restaurant	Unknown	999	0	D	Unknown
	Dec	Restaurant	Norovirus	999	0	D	Unknown
Qld	Oct	Restaurant	Norovirus	3	0	D	Unknown
	Oct	Restaurant	Norovirus	23	0	A	Chicken Caesar salad; roast chicken
	Dec	Restaurant	*C. perfringens*	2	0	D	Unknown
SA	Nov	Camp	*E. coli* O157	31	5	A	Potato salad
	Nov	Restaurant	Norovirus	21	0	A	Berry cheesecake
	Dec	Private residence	*S.* Typhimurium 44	16	2	A	Tiramisu
Tas	Nov	Commercial caterer	Norovirus	14	0	A	Green salad suspected
Vic	Oct	Commercial caterer	Unknown	41	0	D	Unknown
	Nov	Aged care facility	*C. perfringens*	4	0	D	Unknown
	Nov	Aged care facility	Unknown	6	0	D	Unknown
	Nov	Aged care facility	*S.* Typhimurium 170	22	5	D	Unknown
	Nov	Aged care facility	*S.* Typhimurium 170	20	2	D	Unknown
	Nov	Primary produce	Fish wax ester	27	0	D	Escolar/rudderfish
	Nov	Private residence	*S.* Typhimurium 3	6	3	D	Suspect eggs
	Nov	Restaurant	Norovirus	165	(blank)	D	Unknown
	Nov	Restaurant	Unknown	17	0	D	Unknown

Table 2: Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 October to 31 December 2009 (n=42), *continued*

State	Month of outbreak	Setting prepared	Agent	Number affected	Hospitalised	Evidence	Responsible vehicles
Vic, *cont*	Dec	Military institution	*Campylobacter*	5	Unknown	D	Unknown
	Dec	Military institution	Norovirus	18	1	D	Unknown
	Dec	Restaurant	Hepatitis A	3	1	D	Infectious food handler suspected
	Dec	Restaurant	Hepatitis A	6	2	D	Infectious food handler suspected
WA	Oct	Restaurant	*S.* Typhimurium 170	39	6	D	Raw egg mayonnaise
	Oct	Restaurant	*S.* Typhimurium 170	39	7	A	Scrambled eggs
	Nov	Commercial caterer	Norovirus	8	0	D	Unknown
	Nov	Commercially manufactured	Hepatitis A	9	5	M	Semi-dried tomatoes
	Dec	Camp	Norovirus	14	0	D	Unknown
	Dec	Restaurant	Norovirus	11	0	D	Unknown
	Dec	Restaurant	Norovirus	17	0	D	Unknown
	Dec	Restaurant	Norovirus	22	0	D	Unknown

* No foodborne outbreaks were reported by the Australian Capital Territory during the quarter.

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

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

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

The month of outbreak represents the month of onset of outbreak. The hepatitis A multi-jurisdictional outbreak was re-opened in November but some cases had dates of onset during this quarter.

lected descriptive case series data for 25 investigations. As evidence for the implicated food vehicle, investigators relied on microbiological evidence in 4 outbreaks and analytical epidemiological evidence in 6 outbreaks. Descriptive evidence only was obtained in 32 outbreaks.

The following jurisdictional summaries describe key outbreaks and public health actions that occurred in this quarter.

Australian Capital Territory

There were no outbreaks of foodborne disease investigated in the Australian Capital Territory during the 4th quarter.

New South Wales

New South Wales investigated 12 outbreaks of foodborne or suspected foodborne disease during the quarter. Three outbreaks were due to *S.* Typhimurium (phage types 170, 135 and pending), one to *S.* Singapore, one to *S.* Stanley, and one to norovirus. The aetiological agent in 6 outbreaks was unknown, and these outbreaks are not detailed here.

In October, 4 people amongst a group of six developed diarrhoea, fever, and severe abdominal cramps after consuming a layered chocolate cake, prepared with cream and ganache icing (no raw eggs were used). Two other people in the group who did not consume the cake remained well. One stool sample and leftover cake were positive for *S.* Typhimurium 170 (MLVA type 3-9-8-13-523).

In November, 3 people became ill after sharing a meal at a Chinese restaurant. An inspection of the premises identified inadequate disinfection and sanitation, insufficient cooking and insufficient reheating practices. Two stool samples and samples of cooked pork mince and left-over food (duck, tofu and rice) were positive for *S.* Typhimurium MLVA type 3-12-12-13-523 (phage type pending).

In December, it was reported that 30 people of a group of 40 became ill after attending a self-catered Christmas function held at a tennis club. Foods consumed at the function included pork, ham, beef and prawns purchased from a large supermarket, and Caesar, pasta, seafood and coleslaw salads from a takeaway outlet. One-third of stool samples collected were positive for norovirus. The local council inspected the premises and found inadequate storage of foods and inadequate hand washing facilities.

A cluster of 3 cases of *S.* Typhimurium 135 (MLVA 3-13-9-11-550) was identified through routine surveillance. Through interviews, it was revealed that cases had recently attended the same function, where 2 cases reported eating tiramisu prepared at home by one of the 10 to 12 people at the function. The tiramisu contained raw eggs. The 3rd case would not say whether she had consumed the tiramisu. One of the interviewees stated that 5–7 people at the function became ill. The mother of one of the cases did not attend the function but consumed some of the tiramisu and also became ill. The eggs used for making the tiramisu were bought at one of 2 major supermarkets, and the person preparing the tiramisu could not remember the brand.

The investigation of a cluster of 3 cases of *S.* Singapore was identified through routine surveillance. Interviews revealed that cases had common exposure to a variety of foods from a large buffet-style restaurant. *S.* Singapore was isolated from a sample of uncooked fried ice cream with raw egg used to bind the breadcrumb coating to the ice cream. Two of 3 cases had consumed the fried ice cream. The 3rd case reported consuming an omelette from the premises. The eggs were traced back to a farm previously implicated in an outbreak of *S.* Singapore.

In December, a sudden increase in the number of notifications of *S.* Stanley was investigated amongst people aged between 20 and 30 years of age, who were living or staying in Sydney's eastern suburbs. Of a total of 34 cases, 16 reported consuming a number of food products from a takeaway food premises in Bondi Beach, including salads, wraps and burgers. Seven people reported either eating commercially prepared food items from other venues at Bondi/Bondi Beach, or working or residing in that area and 11 either acquired their infection overseas, or were overseas travellers themselves with no contact details. Investigations at the takeaway premise linked to 16 cases, revealed that mayonnaise and sauces (ingredients common to most food items implicated by cases) were commercially prepared without raw egg. Stool specimens from 2 food handlers at the takeaway were positive for *S.* Stanley, although neither reported being symptomatic. MLVA typing identified an outbreak MLVA profile 2-15(14)-0-0-496, which was distinct to the profile from *S.* Stanley isolates from overseas travellers. The source of the outbreak remains unclear.

Northern Territory

Two outbreaks of foodborne illness were investigated during this quarter in the Northern Territory, both in December.

In the 1st outbreak, 13 cases of norovirus were investigated amongst people who had attended a corporate event, with buffet-style catering provided by a local restaurant. The outbreak was notified 12 days after the event, making investigations difficult. It is suspected that the outbreak was due to

norovirus, with food handler or patron contamination of the buffet-style foods likely to have been a contributing factor.

In the 2nd outbreak, 7 cases of gastrointestinal illness occurred amongst 3 separate groups after consuming a meal at a local restaurant. An environmental investigation identified several areas for improvement within the food business and the food business was voluntarily closed. *Escherichia coli* (4,000 CFU/g) and coliforms (77,000 CFU/g) were isolated from food samples. The illness was consistent with *Bacillus cereus* or *Staphylococcus aureus* intoxication, although all stool samples were negative for pathogens and toxins.

Queensland

Three outbreaks of foodborne or suspected foodborne illness were investigated during the quarter in Queensland.

In October, an outbreak of norovirus affected 23 of 60 guests at a wedding reception on the Sunshine Coast. Cases became ill between 8 and 73 hours (median 31 hours) following an evening meal. Two foods were associated with illness; Caesar salad (RR=1.5, 95% CI 1.12–2.02) and roast chicken (RR=1.5, 95% CI 1.04–2.06). Microbiological testing of several food samples supported the epidemiological findings with chicken and cos lettuce found to be of unsatisfactory bacterial quality. The outbreak was suspected to have been transmitted from an infected person to food-to-person transmission although there were no reported cases of illness among staff members prior to the reception.

In October, 3 people became ill with symptoms including diarrhoea, vomiting and fever after a meal at a Brisbane restaurant. No food vehicle was identified, but a number of food handlers had been recently ill with gastrointestinal symptoms. Norovirus was detected in faecal specimens from two of the 3 cases.

In December, 2 women became ill with diarrhoea and abdominal cramps approximately 10 hours after consuming a meal at a wedding, with *C. perfringens* (vegetative cell count 2.5 x 10^5 org/g) detected in 1 of 2 faecal specimens. There were no other reports of illness among the attendees. The meal consumed included curried rice and tofu curry. No leftover food samples were available for microbiological testing.

South Australia

Three outbreaks of foodborne or suspected foodborne illness were investigated in South Australia during the quarter.

In November, the Communicable Disease Control Branch investigated an outbreak of 31 cases of gastroenteritis in attendees at a 4 day church camp. Six of these were confirmed to have been infected with Shiga toxin-producing *E. coli* (STEC). Environmental investigation at the camp site found no environmental sources of STEC. No food from the camp was available for testing. A cohort study of 240 camp attendees found that eating potato or pasta salad at the camp increased the risk of illness. The only common ingredient in the 2 salads was parsley and the source of the parsley is unknown.

In November, the Communicable Disease Control Branch investigated an outbreak of gastroenteritis in diners at a hotel restaurant. Initial reports indicated that members of at least 2 unrelated groups of diners became ill with gastroenteritis approximately 24 hours after a meal at the restaurant. A cohort study was conducted to investigate the outbreak, and included 77 of 125 diners that could be contacted. Five people tested positive for norovirus, with a total of 25 people reporting illness after consumption of food at the restaurant. Illness was associated with consumption of berry cheesecake (RR 2.5, 95% CI 1.3–4.9). The restaurant was provided with education regarding the exclusion of ill food handlers and requested to address issues with refrigeration temperatures.

In December, an outbreak of gastroenteritis was reported amongst 16 of 27 attendees at a function held at a private residence. Six cases were confirmed to be infected with *S.* Typhimurium 44. A cohort study of all 27 attendees found a strong association between consumption of tiramisu and illness (95% CI 1.7–12.7). The tiramisu contained raw eggs sourced from the host's backyard chickens. Environmental investigation found the chicken coop was not regularly cleaned and eggs were not washed prior to use. Samples of chicken faeces tested positive for *S.* Typhimurium 44. No tiramisu was available for testing, however samples of leftover pavlova and cream (store-bought then dressed by the same person who made the tiramisu) tested positive for *S.* Typhimurium 44. The pavlova was not associated with illness (RR 1.1, 95% CI 0.6–2.1, *P*-value 0.701).

Tasmania

Tasmania investigated one foodborne outbreak during the quarter.

In November, 14 of 40 people reported gastrointestinal illness after attending a catered function. Norovirus was detected in the faecal specimen of 1 case. A cohort investigation involving 38 of the 40 people who had attended the function indicated point source transmission. Analysis revealed a green

salad to be the most likely vehicle of transmission with those who consumed the salad being at least twice as likely to develop symptoms as those who did not consume the salad. A possible explanation is that the food handler directly contaminated the salad during preparation prior to consumption. While the food handler was not symptomatic when the salad was prepared she had been caring for her partner and infant daughter, both of whom were ill with vomiting and diarrhoea in the days preceding the function

Victoria

Victoria investigated 13 foodborne or suspected foodborne outbreaks during the quarter.

In October, an unidentified outbreak caused symptoms of vomiting and diarrhoea amongst a group of people after they attended a wedding. Sixty-five guests were interviewed and 41 people suffered vomiting and/or diarrhoea with a median incubation period of 40 hours after consumption of food at the reception. A cohort study was conducted but no foods were significantly associated with illness. The pattern of illness suggested a viral aetiology and the high attack rate suggested that food was the vehicle of transmission.

In November, 2 separate outbreaks of *S.* Typhimurium 170 were investigated in 2 separate aged care facilities. The 1st affected 22 of the 71 residents at the facility with onsets occurring over a 2 week period. The 2nd affected 20 of the 60 residents with onsets occurring over a 13 day period. Investigators were unable to identify a food source for either outbreak despite intensive investigations, however, in both instances the spread of onsets of illness over a 2 week period were suggestive of ongoing low dose contamination of foods and/or kitchen equipment.

An outbreak affecting 4 residents of a small aged care facility was investigated in November. Cases' symptoms were consistent with *C. perfringens* and faecal specimens for two of the cases were positive for *C. perfringens* enterotoxin. Three cases became ill on the same day, with a 4th case becoming symptomatic 2 days later. Investigators were unable to identify a food vehicle for this outbreak.

An outbreak of gastrointestinal illness was reported amongst a group of 25 people who shared a meal at a private residence in November. The meal consisted of chicken and rice, a stir fried noodle and prawn dish, a garden salad and a bread pudding. The prawn and noodle dish had raw eggs added to it at the end, and it is suspected that the eggs were inadequately cooked. Six guests submitted faecal specimens, all of which were positive for *S.* Typhimurium 3.

In November, 6 residents from a 30-bed aged care facility became ill with diarrhoea within 12 hours of each other, and symptoms resolved within 24 hours. Lamb chops and roast beef were 2 foods that all cases had in common. No faecal specimens were collected but *C. perfringens* was suspected.

Within 24 hours after attending a function in November, 28 people suffered yellow, oily diarrhoea and nausea after consuming a meal of 'butterfish'. Investigators confirmed that the fish was escolar, which has been previously associated with these symptoms.

In November, an outbreak of vomiting and diarrhoea occurred in approximately 25 people of a group of 106 people who attended a function at a restaurant. Two food handlers reported gastrointestinal illness prior to the event, one of whom returned to work within 48 hours after symptoms resolved. The aetiology of this outbreak was unable to be confirmed but it is suspected to have been viral transmitted via contaminated food.

In November, an outbreak of gastroenteritis was reported amongst a large group of 260 people who attended a function at a restaurant with a set menu. The reported attack rate was approximately 60%. One case had norovirus detected in a faecal specimen and 2 secondary cases were reported amongst family members who did not attend the function. Gastrointestinal illness was also reported in a 2nd group of 24 people who dined at the same restaurant complex the following evening, with nine of the 19 who were interviewed reporting symptoms. While data from interviews were suggestive that a seafood entrée may have been the food vehicle, investigators were unable to make any firm conclusions. There was no reported illness amongst staff.

Two separate outbreaks of hepatitis A associated with 2 separate restaurants were investigated this quarter. In the outbreak at the 1st restaurant, 3 cases developed symptoms within 4 days of each other and are likely to have acquired their illness through consumption of semi-dried tomatoes. One of these cases was a food handler at the restaurant. A further 6 cases who had eaten at this restaurant during their incubation period were subsequently identified. These cases had onsets between three and 5 weeks later. The source may have been food contaminated by the infected food handler, foods cross-contaminated by contaminated semi-dried tomatoes or direct consumption of contaminated semi-dried tomatoes. There was also a case in the 2nd outbreak who was a food handler at a restaurant to which 4 cases were subsequently linked. These 4 cases had onsets approximately 3–5 weeks after the food handler. Again, it cannot be certain

what the source for these cases may have been as there are various possibilities (see also the section on multi-jurisdictional outbreak investigations).

Two separate outbreaks were reported at the same military barracks in December. The 1st was an outbreak of *Campylobacter*, with 5 confirmed cases having onsets over an 8 day period. Only 1 case was interviewed and the source of illness could not be identified. The 2nd outbreak occurred approximately 2 weeks later and was confirmed as norovirus. The outbreak affected 18 military personnel and all had an onset of gastroenteritis on the same day. Only staff that ate at the same mess became unwell and those that went home remained well. It is suspected that this was a foodborne norovirus outbreak.

Western Australia

Western Australia investigated 8 foodborne and suspected foodborne disease outbreaks during the quarter.

An outbreak of *S.* Typhimurium 170 (pulsed field gel electrophoresis [PFGE] type 0011) amongst patrons of a hamburger takeaway restaurant was investigated in October. Follow-up of complaints received and interviews with notified cases of infection revealed that 39 cases with gastroenteritis had eaten food from a hamburger takeaway, and 21 were laboratory-confirmed. Cases had eaten a variety of burgers and 10 had also eaten aioli with hot chips. The aioli and a mayonnaise used in the burgers were made using raw egg. Food samples and swabs including mayonnaise (not the same batch as eaten) and eggs were negative for *Salmonella*. Eggs used in the restaurant were traced to a Western Australia egg producer, but eggs and drag swabs obtained from the farm were negative for *Salmonella*. The restaurant began using pasteurised egg in aioli and mayonnaise in response to this outbreak.

In October, a 2nd outbreak of *S.* Typhimurium 170 (PFGE type 0011) was investigated amongst patrons of a metropolitan restaurant, with 39 cases of gastroenteritis. Of these 39 cases, 27 were confirmed to have been infected with the outbreak strain. A case control study conducted to investigate the outbreak found an association between illness and eating scrambled eggs. Investigations revealed that raw eggs were routinely added to the scrambled eggs before serving. An aioli served at the restaurant and eaten by a small number of cases was also prepared with raw eggs. Food samples including aioli (from a different batch to that eaten by cases) and eggs, and swabs taken from the restaurant were negative for *Salmonella*. Trace back revealed that eggs used in the restaurant were sourced from the same Western Australian producer as those used by the hamburger takeaway restaurant involved in an outbreak in the same month (as described above). The restaurant stopped adding raw eggs to the scrambled eggs prior to serving and changed to a different egg supplier in response to this outbreak.

In November, eight of 11 people became ill with gastroenteritis after attending a private lunch function catered by 2 food businesses. Two cases were diagnosed with norovirus. There were no reports of illness among the cases prior to the lunch. Food served at the function included sandwiches, foccacia, meatballs and roast chicken. No ill food handlers were reported. One of the food businesses had supplied food to another group of people on the same day, and people from this group also developed gastroenteritis. A cohort study was unable to identify a food vehicle, but the outbreak was considered foodborne because an offsite food business prepared the meals and illness occurred amongst 2 separate groups making person-to-person spread unlikely.

From October to December 2009, eight of 9 cases of hepatitis A in Western Australia were epidemiologically linked a brand of semi-dried tomatoes that was previously shown to be contaminated with hepatitis A virus. These cases reported either eating a particular brand of semi-dried tomatoes, or eating at one of 2 food outlets at a large entertainment complex where this same brand of semi-dried tomatoes was served in a variety of dishes. Six of these cases were confirmed to have been infected with the genotype (1B) that has been reported amongst cases in the concurrent multi-jurisdictional outbreak (see also the section on multi-jurisdictional outbreaks).

In December, 3 separate norovirus outbreaks were investigated amongst people who dined at 3 different restaurants. In the first, 22 people became ill with gastrointestinal symptoms following meals at a Thai restaurant. The 2nd was amongst eight from a group of 22 people who reported gastrointestinal illness following a lunch from a set menu at a café with 3 staff also reporting illness, but after the group had dined there. The 3rd outbreak affected 18 people from 4 separate groups who had buffet meals from the same restaurant. In each of these 3 outbreaks, investigators were unable to identify a specific food, but foodborne transmission was suspected in the absence of evidence of person-to-person transmission.

In December, 12 of 25 people camping in dormitory style accommodation reported gastroenteritis. There was no reported illness amongst staff responsible for catering in the days prior to the group's stay, but 2 staff members (not food handlers) did report illness at the same time as the campers. Investigators were unable to identify a specific food

vehicle, but foodborne transmission was suspected in the absence of evidence of person-to-person transmission.

Multi-jurisdictional outbreak investigations

Hepatitis A

In late June 2009, Victoria reported a marked increase in locally-acquired cases of hepatitis A. There were also a small number of cases in other jurisdictions. The number of cases in Victoria and other jurisdictions continued to increase and the multi-jurisdictional outbreak investigation was re-opened on 2 November. This was a second wave of cases following an earlier multi-jurisdictional outbreak associated with semi-dried tomatoes in May 2009.[†,2] Genotyping of isolates in Victoria showed that the outbreak strain from the 2nd wave of cases was identical to that in the 1st wave. All jurisdictions were asked to follow-up locally-acquired cases and request genotyping on all isolates to determine whether they matched the outbreak strain.

During the quarter, there were 193 locally-acquired cases of hepatitis A in Australia. These cases were reported from Victoria (150), New South Wales (12), Queensland (10), Western Australia (10) South Australia (7), Tasmania (3) and the Australian Capital Territory (1). Of these locally-acquired cases, 52% (100/193) recalled eating semi-dried tomatoes during the period when they were likely to have been exposed. The 2nd wave of the outbreak appears to have peaked in October, with the number of new cases reported decreasing since mid-November 2009. A univariate analysis of data from a case-control study conducted in Victoria to investigate the 2nd wave of the outbreak, showed a significant association between consumption of semi-dried tomatoes and illness with hepatitis A (OR=10.32; 95% CI 4.7–22.7).

Trace back in Victoria of semi-dried tomatoes consumed by cases has revealed a complicated supply chain with multiple suppliers to multiple brands and imported product may be mixed or re-packaged for sale. However, trace back investigations were suggestive of a link to imported frozen tomatoes, from which the final product was manufactured locally. The manufacturer of 1 brand of semi-dried tomatoes that is made from imported product, conducted a voluntary trade-level recall of the product on 30 October 2009, following detection of hepatitis A genomic material in the undressed product. Evidence linking this product to cases was not conclusive for all cases.

† This outbreak is considered a continuation of an earlier outbreak it is not included in Table 2.

The Chief Health Officer of Victoria issued emergency orders mandating pasteurisation or chlorine washing of tomatoes used in the manufacture of semi-dried tomatoes, and measures to improve traceability of all ingredients used in the product. These emergency orders expired in January 2010. Victoria, New South Wales, Western Australia and Tasmania advised consumers to avoid eating loose semi-dried tomatoes unless thoroughly cooked.

Waterborne and suspected waterborne outbreaks

OzFoodNet epidemiologists investigated 2 probable waterborne outbreaks during the quarter. The following jurisdictional summaries describe the key features of these outbreaks.

In December, the Northern Territory investigated an outbreak of gastroenteritis amongst a group of workers at a remote mine site. One case was hospitalised. Stool culture isolated *S.* Reading. Investigations revealed that the possible source was the water supply that was used for drinking and cooking. *E. coli* (30 CFU/g in spring) and faecal coliforms (40 CFU/g at spring and 80 CFU/g in pipe) were detected in the water. Camp management sourced bottled water for drinking and cooking and there were no further reports of illness.

In November, an outbreak of gastroenteritis at a rural school was investigated in Victoria. Absentee records showed that 135 students (out of 250) were absent over a 3 week period with the majority (100 students) becoming ill over a 1 week period. Water testing carried out by the school revealed that the private drinking water supply (underground concrete rainwater tank) was not suitable for consumption due to high levels of *E. coli*, but investigations were unable to determine how the water became contaminated. Symptoms appeared to be consistent with a viral illness. The staff drank bottled filtered water during this period and did not appear to be affected in this outbreak.

Cluster investigations

Five cluster investigations were conducted during the quarter; all were clusters of *Salmonella* infection with cases being linked in place and time, but the source of infection and mode of transmission remain unknown. Clusters were due to *S.* Heidelberg, *S.* Stanley, *S.* Typhimurium U302 and two clusters of *S.* Virchow phage type 8.

In New South Wales, a cluster of cluster of 7 cases (6 were children under 9 years of age and 5 lived in the same suburb) of *S.* Heidelberg phage type 1 were identified through routine surveillance of laboratory notifications. No common exposures such

as to animals (e.g. through a petting zoo) could be identified and while interviews revealed a number of foods commonly consumed by cases, the source of this outbreak remains unknown.

In Queensland, a cluster of 6 cases of *S.* Stanley were investigated; 1 case reported overseas travel prior to illness (Thailand), another case had travelled to Sydney and was subsequently linked to the concurrent investigation of *S.* Stanley cases being conducted there (*see Foodborne disease outbreaks*).

A cluster of 13 cases of *S.* Virchow phage type 8 was investigated in South Australia. Hypothesis generating interviews were completed for 9 cases, all of whom reported consuming chicken. Trace back revealed that chicken consumed by eight of 9 cases was sourced from 1 poultry supplier. Routine testing of poultry from this company did not identify *S.* Virchow. Victoria also investigated a cluster of *S.* Virchow 8, with no source identified.

Comments

There were a higher number of foodborne outbreaks (n=42) during the 4th quarter of 2009 than in the previous quarter (n=28) and this was also an increase when compared with the same quarter in 2008 (n=31). This was due to the increased number of foodborne outbreaks of *Salmonella* (n=11) and norovirus (n=13) investigated. There were also several *Salmonella* clusters that were potentially foodborne for which investigators were unable to determine the source.

Fourteen outbreaks of foodborne disease were linked to imported foods between 2001 and 2007.[4] The multi-jurisdictional outbreak of hepatitis A associated with semi-dried tomatoes demonstrates that the safety of foods containing imported ingredients is of continuing concern. Trace back of semi-dried tomatoes consumed by cases in Victoria revealed a complicated supply chain with multiple suppliers to multiple brands and imported product that may be mixed or re-packaged for sale. Improvements to the documentation of the supply and distribution chain of tomatoes and other components used for the production of semi-dried tomatoes in Australia would assist in trace back during outbreaks of this kind in the future. Australia notified the World Health Organization of this outbreak in accordance with reporting requirements under the *International Health Regulations* (2005)[4] due to the suspected link with imported ingredients. OzFoodNet has continued to communicate with the international public health community about developments. Since the end of the quarter, OzFoodNet has been in communication with countries in Europe where potentially linked outbreaks of hepatitis A are occurring. Outcomes of investigations in Australia are helping to inform investigations in those countries.

A limitation of the outbreak data provided by OzFoodNet sites for this report is the potential for variation in categorisation of the features of outbreaks depending on circumstances and investigator interpretation. Changes in the incidence of foodborne outbreaks should be interpreted with caution due to the small numbers each quarter.

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Communicable diseases surveillance

Tables

National Notifiable Diseases Surveillance System

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 43,344 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 October to 31 December 2009 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1: Reporting of notifiable diseases by jurisdiction

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

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

Disease	Data received from:
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza (laboratory confirmed)*	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease (invasive)	All jurisdictions
Poliomyelitis	All jurisdictions
Rubella	All jurisdictions
Rubella - congenital	All jurisdictions
Tetanus	All jurisdictions
Varicella zoster (chickenpox)	All jurisdictions except New South Wales
Varicella zoster (shingles)	All jurisdictions except New South Wales
Varicella zoster (unspecified)	All jurisdictions except New South Wales
Vectorborne diseases	
Arbovirus infection (NEC)†	All jurisdictions
Barmah Forest virus infection	All jurisdictions
Dengue virus infection	All jurisdictions
Japanese encephalitis virus infection	All jurisdictions
Kunjin virus infection	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus infection	All jurisdictions
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssavirus (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Tularaemia	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

* Notifiable in South Australia as of 1 May 2008.

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

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities in the period 1 October to 31 December 2009, by date of diagnosis*

Disease	State or territory								Total 4th quarter 2009†	Total 3rd quarter 2009	Total 4th quarter 2008	Last 5 years mean 4th quarter	Ratio‡	Year to date 2009	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	1	0.2	0.0	0	0.6
Hepatitis B (newly acquired)	3	12	3	12	1	2	20	0	53	48	45	62.2	0.9	197	272.2
Hepatitis B (unspecified)	20	803	38	246	99	21	508	197	1,932	2,040	1,418	1,510.6	1.3	7,878	6,289.8
Hepatitis C (newly acquired)	2	11	1	NN	13	4	51	0	82	69	100	89.0	0.9	297	370.8
Hepatitis C (unspecified)	38	1,261	45	649	122	65	521	302	3,003	3,270	2,491	2,831.4	1.1	12,695	11,800.2
Hepatitis D	0	1	0	2	0	0	3	0	6	5	6	5.8	1.0	32	33.4
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	1	1.2
Campylobacteriosis§	94	NN	29	1,059	440	174	1,699	697	4,192	3,819	4,082	4,543.8	0.9	15,851	16,008.2
Cryptosporidiosis	5	110	28	40	14	29	90	27	343	259	423	589.0	0.6	4,599	2,577.2
Haemolytic uraemic syndrome	0	1	1	0	1	0	1	0	4	0	11	8.2	0.5	12	20.0
Hepatitis A	2	29	0	17	7	5	167	13	240	86	41	52.6	4.6	562	272.4
Hepatitis E	0	1	0	0	0	0	0	0	1	8	9	5.2	0.2	35	28.8
Listeriosis	1	5	0	2	0	1	9	3	21	22	8	14.0	1.5	91	59.8
STEC, VTEC‖	0	6	1	25	21	0	3	2	58	23	36	27.0	2.1	160	82.8
Salmonellosis	53	724	132	504	168	40	460	326	2,407	1,501	2,054	2,149.6	1.1	9,526	8,455.4
Shigellosis	3	25	20	29	11	0	12	19	119	124	199	155.8	0.8	629	643.4
Typhoid	0	16	0	2	1	0	14	2	35	25	19	15.6	2.2	116	79.0
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	1	4	1.8	0.0	4	3.8
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0

Table 2: Notifications of diseases received by state and territory health authorities in the period 1 October to 31 December 2009, by date of diagnosis,* *continued*

Disease	State or territory								Total 4th quarter 2009†	Total 3rd quarter 2009	Total 4th quarter 2008	Last 5 years mean 4th quarter	Ratio‡	Year to date 2009	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection¶	226	3,716	470	4,052	850	352	3,378	2,149	15,193	15,124	13,966	11,548.4	1.3	62,677	47,062.6
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	1.6	0.0	1	6.8
Gonococcal infection	6	496	349	381	61	1	343	278	1,915	1,699	1,807	1,856.2	1.0	8,094	7,828.6
Syphilis (all)	4	236	24	119	5	5	204	43	640	736	704	661.0	1.0	2,840	2,704.0
Syphilis <2 years duration	0	96	5	42	5	3	83	18	252	331	275	241.6	1.0	1,259	975.4
Syphilis >2 years or unspecified duration	4	140	19	77	NDP	2	121	25	388	405	429	419.4	0.9	1,581	1,728.6
Syphilis - congenital	0	0	1	0	0	0	0	0	1	1	2	2.4	0.4	4	11.2
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Haemophilus influenzae type b	0	0	1	0	0	0	1	0	2	2	5	5.0	0.4	20	19.2
Influenza (laboratory confirmed)	8	0	24	275	177	11	70	135	700	34,824	1,521	767.0	0.9	47,718	5,897.8
Measles	0	7	0	0	0	0	1	1	9	8	1	6.2	1.5	104	51.4
Mumps	0	9	2	10	2	0	0	6	29	34	35	90.4	0.3	162	297.6
Pertussis	77	1,815	25	1,710	1,888	100	1,095	205	6,915	6,454	6,777	3,145.4	2.2	29,514	10,032.4
Pneumococcal disease (invasive)	7	97	19	63	33	2	80	26	327	625	353	349.6	0.9	1,564	1,734.8
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Rubella	0	0	0	1	0	0	0	1	2	7	9	7.6	0.3	25	38.2
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.8
Tetanus	0	0	0	0	0	0	0	0	0	0	0	1.0	0.0	3	3.4
Varicella zoster (chickenpox)	1	NN	17	4	124	8	22	68	244	328	768	500.0	0.5	1,238	1,672.0
Varicella zoster (shingles)	7	NN	26	2	263	27	225	151	701	609	756	387.5	1.8	2,828	1,241.8
Varicella zoster (unspecified)	20	NN	2	1,001	77	25	431	214	1,770	1,680	1,381	873.5	2.0	6,693	3,097.8
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	0	3	0	0	0	0	3	5	10	5.6	0.5	27	33.4
Barmah Forest virus infection	0	75	19	163	8	1	4	23	293	271	414	346.8	0.8	1,493	1,672.6
Dengue virus infection	6	17	5	42	1	0	9	30	110	87	193	74.0	1.5	1,401	322.8
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.4
Kunjin virus infection	0	0	0	0	0	0	0	0	0	0	0	0.6	0.0	2	2.4
Malaria	1	12	2	35	7	1	16	21	95	151	127	141.4	0.7	533	646.2
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	4	1.2
Ross River virus infection	0	128	71	358	94	4	16	107	778	833	914	728.2	1.1	4,777	4,425.2

Table 2: Notifications of diseases received by state and territory health authorities in the period 1 October to 31 December 2009, by date of diagnosis,* *continued*

Disease	State or territory								Total 4th quarter 2009†	Total 3rd quarter 2009	Total 4th quarter 2008	Last 5 years mean 4th quarter	Ratio‡	Year to date 2009	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.4
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Brucellosis	0	1	0	7	0	0	0	0	8	6	12	13.6	0.6	31	43.0
Leptospirosis	0	1	0	6	0	0	7	0	14	16	22	22.8	0.6	145	134.0
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	0	0	0	0	0	10	1	11	18	17	33.6	0.3	60	152.2
Q fever	0	26	0	27	7	0	5	0	65	66	88	98.8	0.7	308	406.6
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Other bacterial infections															
Legionellosis	1	19	0	7	14	0	14	8	63	74	70	84.0	0.8	305	313.0
Leprosy	0	0	0	2	0	0	0	0	2	0	3	1.8	1.1	3	9.4
Meningococcal infection**	0	20	1	19	3	0	14	6	63	86	63	78.2	0.8	259	340.8
Tuberculosis	13	162	5	73	13	2	159	35	462	340	351	331.4	1.4	1,325	1,142.2
Total	598	9,844	1,361	11,283	4,525	887	9,663	5,183	43,344	75,744	41,794	34,612.0	1.3	228,226	139,573.3

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

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

‡ Ratio = ratio of current quarter total to the mean of last 5 years for the same quarter. Note: Ratios for syphilis <2 years; syphilis >2 years or unspecified duration are based on 5 years of data. Ratios for varicella zoster (chickenpox), varicella zoster (shingles) and varicella zoster (unspecified) are based on 2 years of data

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

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

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

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

NN Not notifiable.

NEC Not elsewhere classified.

NDP No data provided.

Table 3: Notification rates of diseases, 1 October to 31 December 2009, by state or territory. (Annualised rate per 100,000 population)

Disease*	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)	3.4	0.7	5.3	1.1	0.2	1.6	1.5	0.0	1.0
Hepatitis B (unspecified)	22.8	45.2	67.6	22.3	24.4	16.7	37.4	35.2	35.3
Hepatitis C (newly acquired)	2.3	0.6	1.8	NN	3.2	3.2	3.8	0.0	1.5
Hepatitis C (unspecified)	43.3	71.0	80.1	58.9	30.1	51.7	38.4	54.0	54.9
Hepatitis D	0.0	0.1	0.0	0.2	0.0	0.0	0.2	0.0	0.1
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis†	107.1	NN	51.6	96.1	108.5	138.5	125.2	124.6	76.7
Cryptosporidiosis	5.7	6.2	49.8	3.6	3.5	23.1	6.6	4.8	6.3
Haemolytic uraemic syndrome	0.0	0.1	1.8	0.0	0.2	0.0	0.1	0.0	0.1
Hepatitis A	2.3	1.6	0.0	1.5	1.7	4.0	12.3	2.3	4.4
Hepatitis E	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Listeriosis	1.1	0.3	0.0	0.2	0.0	0.8	0.7	0.5	0.4
STEC, VTEC‡	0.0	0.3	1.8	2.3	5.2	0.0	0.2	0.4	1.1
Salmonellosis	60.4	40.8	234.8	45.7	41.4	31.8	33.9	58.3	44.0
Shigellosis	3.4	1.4	35.6	2.6	2.7	0.0	0.9	3.4	2.2
Typhoid	0.0	0.9	0.0	0.2	0.2	0.0	1.0	0.4	0.6
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§	257.4	209.4	836.1	367.8	209.5	280.1	248.9	384.3	277.8
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	6.8	27.9	620.9	34.6	15.0	0.8	25.3	49.7	35.0
Syphilis (all)	4.6	13.3	42.7	10.8	1.2	4.0	15.0	7.7	11.7
Syphilis <2 years duration	0.0	5.4	8.9	3.8	1.2	2.4	6.1	3.2	4.6
Syphilis >2 years or unspecified duration	4.6	7.9	33.8	7.0	NDP	1.6	8.9	4.5	7.1
Syphilis - congenital	0.0	0.0	1.8	0.0	0.0	0.0	0.0	0.0	0.0
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.0	1.8	0.0	0.0	0.0	0.1	0.0	0.0
Influenza (laboratory confirmed)	9.1	0.0	42.7	25.0	43.6	8.8	5.2	24.1	12.8
Measles	0.0	0.4	0.0	0.0	0.0	0.0	0.1	0.2	0.2
Mumps	0.0	0.5	3.6	0.9	0.5	0.0	0.0	1.1	0.5
Pertussis	87.7	102.3	44.5	155.2	465.4	79.6	80.7	36.7	126.5
Pneumococcal disease (invasive)	8.0	5.5	33.8	5.7	8.1	1.6	5.9	4.6	6.0

Table 3: Notification rates of diseases, 1 October to 31 December 2009, by state or territory. (Annualised rate per 100,000 population), *continued*

Disease*	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vaccine preventable diseases, continued									
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.2	0.0
Rubella - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Varicella zoster (chickenpox)	1.1	NN	30.2	0.4	30.6	6.4	1.6	12.2	4.5
Varicella zoster (shingles)	8.0	NN	46.3	0.2	64.8	21.5	16.6	27.0	12.8
Varicella zoster (unspecified)	22.8	NN	3.6	90.9	19.0	19.9	31.8	38.3	32.4
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	0.0	4.2	33.8	14.8	2.0	0.8	0.3	4.1	5.4
Dengue virus infection	6.8	1.0	8.9	3.8	0.2	0.0	0.7	5.4	2.0
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	1.1	0.7	3.6	3.2	1.7	0.8	1.2	3.8	1.7
Murray Valley encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	0.0	7.2	126.3	32.5	23.2	3.2	1.2	19.1	14.2
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	0.6	0.0	0.0	0.0	0.0	0.1
Leptospirosis	0.0	0.1	0.0	0.5	0.0	0.0	0.5	0.0	0.3
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.0	0.0	0.0	0.0	0.0	0.7	0.2	0.2
Q fever	0.0	1.5	0.0	2.5	1.7	0.0	0.4	0.0	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	1.1	1.1	0.0	0.6	3.5	0.0	1.0	1.4	1.2
Leprosy	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0
Meningococcal infection‖	0.0	1.1	1.8	1.7	0.7	0.0	1.0	1.1	1.2
Tuberculosis	14.8	9.1	8.9	6.6	3.2	1.6	11.7	6.3	8.4

* Rates are subject to retrospective revision.

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

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

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

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

NN Not notifiable.

NEC Not elsewhere classified.

NDP No data provided.

Additional reports

Australian Sentinel Practice Research Network

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

The network consists of general practitioners who report presentations on a number of defined medical conditions each week. ASPREN was established in 1991 to provide a rapid monitoring scheme for infectious diseases that can alert public health officials of epidemics in their early stages as well as play a role in the evaluation of public health campaigns and research of conditions commonly seen in general practice. Electronic data collection was established in 2006 and currently, further development of ASPREN is in progress to create an automatic reporting system.

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published. In 2009, four conditions are being monitored. They include influenza-like (ILI) 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 *2010;34(1):82–83.*

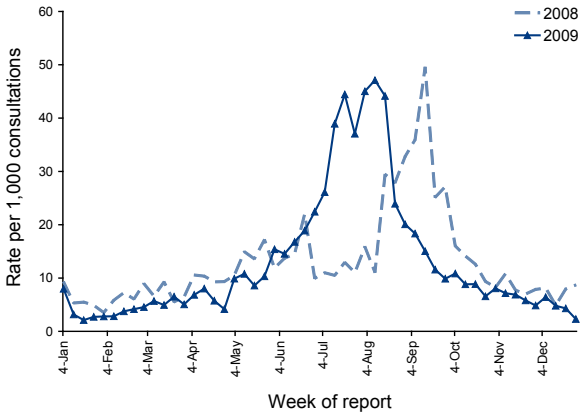
Data on influenza-like illness, gastroenteritis, chickenpox and shingles from 1 October to 31 December 2009 compared with 2008, are shown as the rate per 1,000 consultations in Figures 1, 2, 3 and 4, respectively.

Reporting period 1 October to 31 December 2009

Sentinel practices contributing to ASPREN were located in all jurisdictions other than the Northern Territory. A total of 84 general practitioners contributed data to ASPREN in the 4th quarter of 2009. Each week an average of 71 general practitioners provided information to ASPREN at an average of 7,207 (range 5,182–7,789) consultations per week and an average of 118 (range 57–138) notifications per week.

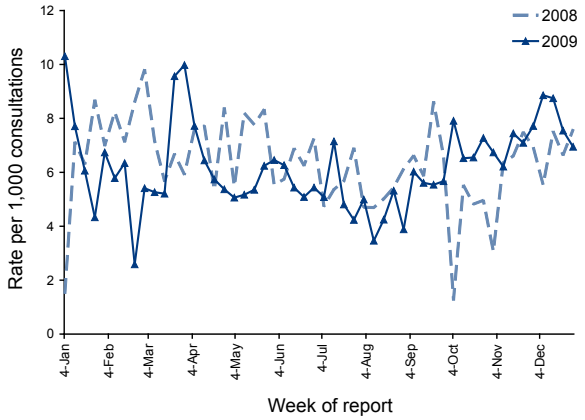
ILI rates reported from 1 October to 31 December 2009 were 2–9 cases per 1,000 consultations. The reported rates in October, November and December 2009 were lower (7–11 cases per 1,000 consultations, 5–8 cases per 1,000 consultations and 2–6 cases per 1,000 consultations respectively) compared with the same reporting period in 2008 (9–14 cases per 1,000 consultations, 7–11 cases per 1,000 consultations and 5–9 cases per 1,000 consultations respectively) (Figure 1).

Figure 1: Consultation rates for influenza-like illness, ASPREN, 1 January 2008 to 31 December 2009, by week of report



During this reporting period, consultation rates for gastroenteritis ranged from 6 to 9 cases per 1,000 (Figure 2). This was slightly higher compared with the same reporting period in 2008 (3 to 8 cases per 1,000 consultations).

Figure 2: Consultation rates for gastroenteritis, ASPREN, 1 January 2008 to 31 December 2009, by week of report



Varicella infections were reported at a similar rate for the 4th quarter of 2009 compared with the same

period in 2008. From 1 October to 31 December 2009, recorded rates for chickenpox were between 0.1 and 0.8 cases per 1,000 consultations (Figure 3).

In the 4th quarter of 2009, reported rates for shingles were between 0.4 and 1.8 cases per 1,000 consultations (Figure 4), similar to the same reporting period in 2008.

Figure 3: Consultation rates for chickenpox, ASPREN, 1 January 2008 to 31 December 2009, by week of report

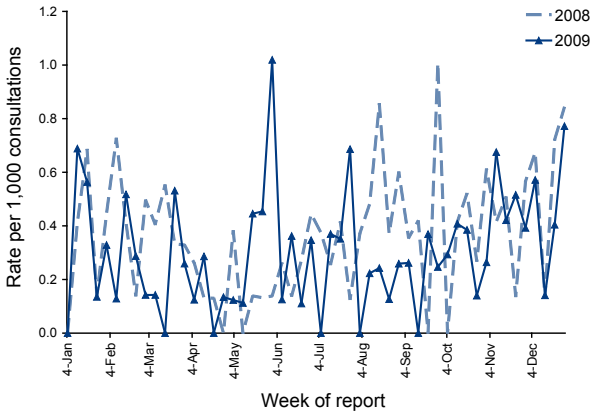
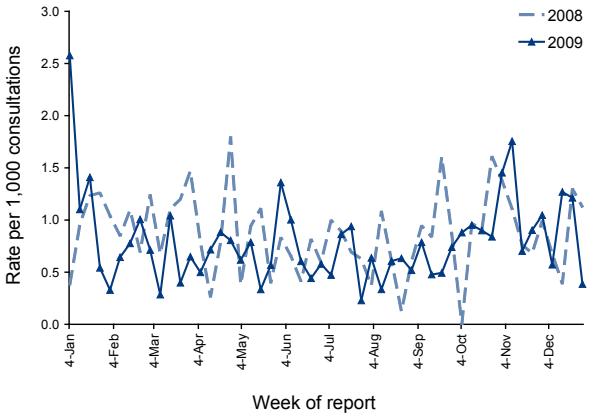


Figure 4: Consultation rates for shingles, ASPREN, 1 January 2008 to 31 December 2009, by week of report



Australian childhood immunisation coverage

The Australian Childhood Immunisation Register (ACIR) commenced operation on 1 January 1996 and is now an important component of the Immunise Australia Program. *It is administered and operated by Medicare Australia (formerly the Health Insurance Commission). The Register was established by transferring data on all children under the age of 7 years enrolled with Medicare to the ACIR.*

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

The data show the percentage of children fully immunised at 12 months of age for the cohort born between 1 July and 30 September 2008, at 24 months of age for the cohort born between 1 July and 30 September 2007, and at 5 years of age for the cohort born between 1 July and 30 September 2004 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 PRP-OMP containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other *Haemophilus influenzae* type b (Hib) vaccine, and 2 or 3 doses of Comvax hepatitis B vaccine or 3 doses of all other hepatitis B vaccines. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of 3 or 4 doses of a DTP-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP containing *Haemophilus influenzae* type b (Hib) vaccine or 4 doses of any other *Haemophilus influenzae* type b (Hib) vaccine, 3 or 4 doses of Comvax hepatitis B vaccine or 4 doses of all other hepatitis B vaccines, and 1 dose of a measles, mumps and rubella-containing (MMR) vaccine. 'Fully immunised' at 5 years of age is defined as a child having a record on the ACIR of 4 or 5 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

Immunisation coverage for children 'fully immunised' at 12 months of age for Australia decreased slightly by 0.4 of a percentage point to 91.6% (Table 1). However, there were important changes in coverage in all jurisdictions for both *Haemophilus influenzae* type b and hepatitis B vaccines. Coverage for *Haemophilus influenzae* type b vaccine fell 2.3 to 3.9 percentage points across all jurisdictions, whilst coverage for hepatitis B vaccine fell 2.4 to 6.8 percentage points across all jurisdictions. The biggest decrease in hepatitis B coverage was experienced in the Northern Territory (6.8 percentage points). These decreases are likely to be entirely due to the changes in the coverage calculation algorithms introduced as at 31 December 2009. The changes tightened the rules, outlined above, regarding *Haemophilus influenzae* type b and hepatitis B vaccines for 12-month-olds to lead to more accurate measures of *Haemophilus influenzae* type b and hepatitis B vaccine coverage in Australia.

Immunisation coverage for children 'fully immunised' at 24 months of age for Australia decreased importantly by 1.7 percentage points to 91.0 (Table 2). As with coverage at 12 months of age, there were also important changes in coverage in all jurisdictions for both *Haemophilus influenzae* type b and hepatitis B vaccines, but the decreases were smaller in magnitude. Coverage for *Haemophilus influenzae* type b vaccine fell 0.6 to 2.5 percentage points across all jurisdictions, whilst coverage for hepatitis B vaccine fell 1.5 to 3.2 percentage points across all jurisdictions. As with coverage at 12 months of age, the biggest decrease in hepatitis B coverage was experienced in the Northern Territory (3.2 percentage points). These decreases are also likely to be entirely due to the changes in the coverage calculation algorithms introduced as at 31 December 2009. The changes tightened the rules, outlined above, regarding *Haemophilus influenzae* type b and hepatitis B vaccines for 24-month-olds

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

Total number of children	1,258	25,091	974	15,896	5,083	1,678	18,341	7,767	76,088
Diphtheria, tetanus, pertussis (%)	94.0	92.7	89.8	92.2	92.0	92.8	92.8	90.1	92.3
Poliomyelitis (%)	93.9	92.6	89.7	92.2	92.0	92.7	92.8	90.1	92.3
Haemophilus influenzae type b (%)	93.6	92.5	92.8	92.1	91.6	92.7	92.3	90.0	92.1
Hepatitis B (%)	93.2	92.3	89.7	91.9	91.5	92.5	92.0	89.7	91.8
Fully immunised (%)	93.2	92.1	88.0	91.8	91.3	92.5	91.9	89.3	91.6
Change in fully immunised since last quarter (%)	-1.2	-0.0	-3.9	-0.1	-0.3	-0.4	-0.7	-1.1	-0.4

Table 2. Percentage of children immunised at 2 years of age, results by disease and state or territory for the birth cohort 1 July to 30 September 2007; assessment date 31 December 2009*

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,226	25,067	911	16,062	5,066	1,701	18,408	7,755	76,196
Diphtheria, tetanus, pertussis (%)	95.4	94.4	94.3	94.3	95.3	94.8	95.5	94.3	94.7
Poliomyelitis (%)	95.3	94.4	94.3	94.3	95.3	94.8	95.4	94.3	94.7
Haemophilus influenzae type b (%)	95.6	94.6	92.2	91.8	91.4	95.1	93.9	92.8	93.4
Measles, mumps, rubella (%)	94.5	93.3	94.2	93.3	94.3	94.4	94.4	93.6	93.7
Hepatitis B (%)	95.1	93.9	93.6	92.8	93.8	94.4	93.9	93.8	93.7
Fully immunised (%)	93.2	91.7	90.7	89.6	89.5	92.8	91.7	90.0	91.0
Change in fully immunised since last quarter (%)	-1.2	-0.7	-3.2	-2.5	-3.2	-1.9	-2.0	-1.8	-1.7

* The 12 months age data for this cohort were published in *Commun Dis Intell* 2009;33(1):75.

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

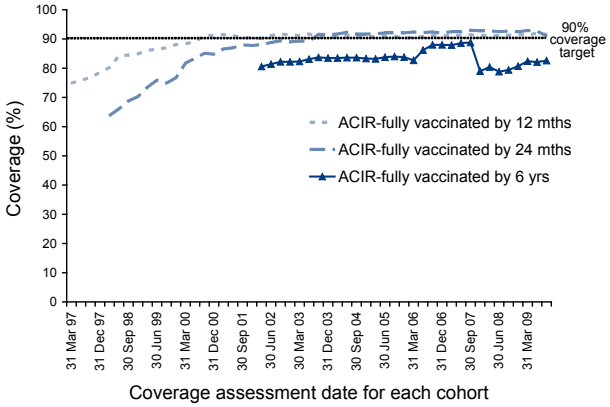
Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,117	23,003	817	14,487	4,524	1,571	16,857	7,029	69,405
Diphtheria, tetanus, pertussis (%)	86.2	81.4	82.3	83.1	81.2	87.4	86.8	82.1	83.3
Poliomyelitis (%)	85.9	81.3	82.1	83.0	81.2	87.3	86.8	82.0	83.3
Measles, mumps, rubella (%)	85.9	81.2	82.0	83.0	80.9	87.1	86.5	81.5	83.1
Fully immunised (%)	85.5	80.8	81.2	82.3	80.6	86.2	86.2	80.9	82.6
Change in fully immunised since last quarter (%)	-1.5	-0.6	+1.9	-1.0	+2.2	+1.8	+1.9	+1.7	+0.5

to lead to more accurate measures of *Haemophilus influenzae* type b and hepatitis B vaccine coverage in Australia.

Immunisation coverage for children 'fully immunised' at 5 years of age for Australia increased marginally by 0.5 of a percentage point to sit currently at 82.6% (Table 3). However, 'fully immunised' coverage increased 1.2 to 1.7 percentage points in 5 jurisdictions (the Northern Territory, South Australia, Tasmania, Victoria and Western Australia) and is now above 80% in all jurisdictions. These same 5 jurisdictions also experienced similar increases in coverage for all individual vaccines due at 5 years of age.

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 (5 years from March 2008), although coverage for vaccines due at 4 years decreases significantly due to the change in assessment age from 6 to 5 years. It should also be noted that, currently, coverage for the vaccines added to the NIP since 2003 (varicella at 18 months, meningococcal C conjugate at 12 months and pneumococcal conjugate at 2,4, and 6 months) are not included in the 12 or 24 months coverage data respectively.

Figure 5: Trends in vaccination coverage, Australia, 1997 to 30 September 2009, by age cohorts



Gonococcal surveillance

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

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various states and territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5 per cent or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatment.[1] *Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as TRNG. Tetracyclines are however, not a recommended therapy for gonorrhoea in Australia. Comparability of data is achieved by means of a standardised system of testing and a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. For more information see* Commun Dis Intell *2010;34(1):81–82.*

Reporting period 1 July to 30 September 2009

The AGSP laboratories received a total of 713 gonococcal isolates of which 705 remained viable for susceptibility testing. This was about 4.5% less than the 746 gonococci reported for the same period in

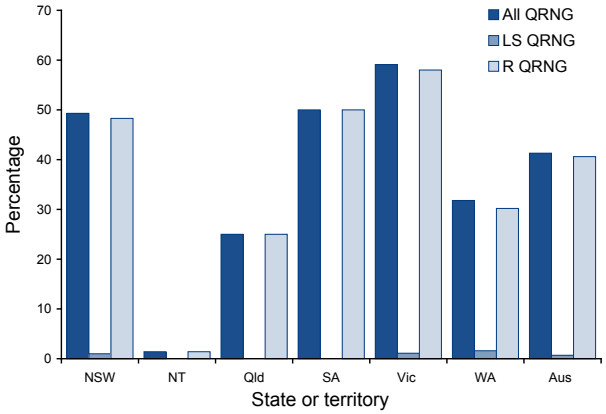
2008. About 29% of this total was from New South Wales, 27% from Victoria, 16% from Queensland, 10% each from Western Australia and the Northern Territory, 5% from South Australia, 2% from the Australian Capital Territory and 1% from Tasmania.

Penicillins

Of the 705 isolates examined 225 (36.2%) were penicillin resistant by one or more mechanisms, 102 (14.5%) were penicillinase producing *N. gonorrhoeae* (PPNG). This was a substantial increase from the 82 (11%) PPNG in the same quarter in 2008. One hundred and fifty-three (21.7%) isolates were resistant by chromosomal mechanisms (CMRP), which was a large decrease from the 206 (28%) seen in the same quarter in 2008. The proportion of all strains resistant to the penicillins by any mechanism ranged from 4.3 % in the Northern Territory to 57.9% in South Australia. High rates of penicillin resistance were also found in Victoria (55%), New South Wales (41%), Western Australia (22.2%) and Queensland (16.3%). Five of 12 gonococci isolated in the Australian Capital Territory and two of the 8 strains from Tasmania were penicillin resistant.

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

Figure 6: Categorisation of gonococci isolated in Australia, 1 January to 30 September 2009, by penicillin susceptibility and region



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

In Victoria, most of the penicillin resistance was with CMRP (67, 34.7%) with 39 PPNG (20.2%) while in New South Wales there were 32 PPNG (15.6%) but 52 CMRP (25.4%). In South Australia PPNG represented 15.8% and CMRP 42.1% of all isolates tested. In Western Australia PPNG were 12.7% and CMRP 9.5%; in Queensland PPNG were 11.2% and CMRP 5.1% of isolates tested. PPNG were present in Northern Territory (3 isolates), but there were no CMRP. Five CMRP isolates, but no PPNG, were present in the Australian Capital Territory. There was 1 CMRP and 1 PPNG from Tasmania. All the penicillin resistant strains in the Northern Territory were from Darwin.

Ceftriaxone

Seventeen isolates with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected, eight from Victoria, two from Queensland and one each from New South Wales, South Australia and the Northern Territory. It is emphasised that no treatment failures have been documented locally when a 250 mg, or currently a 500 mg, IM dose of ceftriaxone has been used.

Spectinomycin

All isolates susceptible to this injectable agent.

Quinolone antibiotics

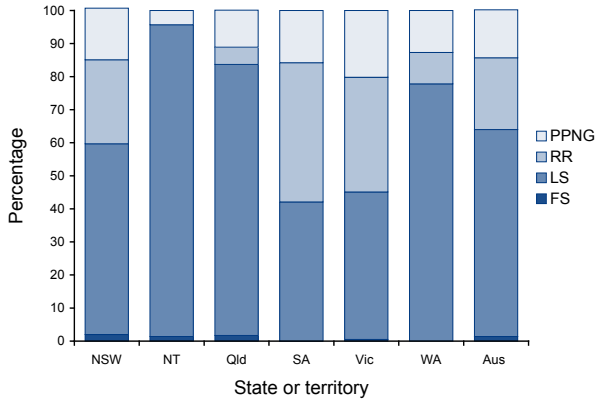
Nationally, the 291 quinolone resistant *N. gonorrhoeae* (QRNG) detected in this quarter represented 41.3% of all isolates tested. This was a decrease from the 368 (50.6%) QRNG recorded in the 3rd quarter of 2008 and the 321 QRNG (50.5) seen in 2007. The majority of QRNG (286 of 291, 98.2%) had higher-level resistance to ciprofloxacin of 1 mg/L or more. QRNG are defined as those isolates with a MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06–0.5 mg/L) or resistant (MIC ≥ 1 mg/L) groups.

QRNG were detected in all states and territories and the highest proportion of QRNG was found in Victoria where there were 114 QRNG (59.1% of isolates) (Figure 7). New South Wales had 101 QRNG (49.3%) with 19 QRNG (50%) present in South Australia, 20 QRNG (31.8%) in Western Australia and 29 (25%) in Queensland. There were 5 QRNG detected in the Australian Capital Territory, two in Tasmania and one in the Northern Territory.

High level tetracycline resistance

The number (145) and proportion (20.6%) of high level tetracycline resistance (TRNG) detected was slightly higher than that recorded in this quarter in 2008 (128, 17.6%). TRNG were found in all states and territories except for Tasmania and

Figure 7: The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 January to 30 September 2009, by jurisdiction



LS QRNG Ciprofloxacin MICs 0.06–0.5 mg/L.

R QRNG Ciprofloxacin MICs ≥1 mg/L.

the Australian Capital Territory and represented between 13.8% (Queensland) and 36.5% (Western Australia) of all isolates tested.

Reference

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

Administration

Surveillance systems reported in *CDI*, 2010

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

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

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

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

Surveillance has been defined by the World Health Organization as the 'continuing scrutiny of all aspects of the occurrence and spread of disease that are pertinent to effective control.' It is characterised by 'methods distinguished by their practicability, uniformity, and frequently by their rapidity, rather than complete accuracy.'[1] Although some surveillance schemes aim for complete case ascertainment, others include only a proportion of all cases of the conditions under surveillance, and these samples are subject to systematic and other biases. Results generated from surveillance schemes must be interpreted with caution, particularly when comparing results between schemes, between different geographical areas or jurisdictions and over time. Surveillance data may also differ from data on communicable diseases gathered in other settings.

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

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

National Notifiable Diseases Surveillance System

National compilations of notifiable diseases have been published intermittently in a number of publications since 1917.[2] The National Notifiable Diseases Surveillance System (NNDSS) was established in 1990 under the auspices of the Communicable Diseases Network Australia (CDNA).

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

The NNDSS core dataset includes data fields for a unique record reference number; notifying state or territory; disease code; age; sex; Indigenous status;

postcode of residence; date of onset of the disease; death, date of report to the state or territory health department and outbreak reference (to identify cases linked to an outbreak). Where relevant, information on the species, serogroups/subtypes and phage types of organisms isolated, and on the vaccination status of the case is collected. Data quality is monitored by DoHA and the National Surveillance Committee (NSC) and there is a continual process of improving the national consistency of communicable disease surveillance.

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

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

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

Australian Childhood Immunisation Register

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

From the data finally entered onto the ACIR, Medicare Australia provides regular quarterly coverage reports at the national and state level. Coverage for these reports is calculated using the cohort method described in *Commun Dis Intell* 1998;22:36–37. With this method, a cohort of children is defined by date of birth in 3-month groups. This birth cohort has the immunisation status of its members assessed at the 3 key milestones of 12 months, 24 months and 5 years of age. Analysis of coverage is undertaken 3 months after the due date for completion of each milestone, so that time is available for processing notifications and the impact on coverage estimates of delayed notification to the ACIR is minimised. Only children enrolled with Medicare are included in order to minimise inaccuracies in coverage estimates due to duplicate records.

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

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

Australian Gonococcal Surveillance Programme

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

and intermittent surveys of azithromycin resistance are conducted. Comparability of data is achieved by means of a standardised system of MIC testing and a program-specific quality assurance process.

Australian Meningococcal Surveillance Programme

The reference laboratories of the Australian Meningococcal Surveillance Programme report data of laboratory-confirmed cases confirmed either by culture or by non-culture techniques. Culture-positive cases where a *Neisseria meningitidis* is grown from a normally sterile site or skin, and non-culture based diagnoses, derived from results of nucleic acid amplification assays and serological techniques are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions.

Data are reported annually and quarterly in *CDI*. Data in the quarterly reports are restricted to a description of the number of cases per jurisdiction, and serogroup where known. A full analysis of laboratory-confirmed cases of IMD, including phenotyping and antibiotic susceptibility data are published annually (*Commun Dis Intell* 2009;33(1):1–9).

Australian Paediatric Surveillance Unit

The Australian Paediatric Surveillance Unit (APSU) is an active surveillance mechanism for prospective, national identification and study of children (< 15 years) with uncommon conditions of childhood, including rare infectious and vaccine preventable diseases, genetic disorders, child mental health problems, and rare injuries. Each month the APSU sends an e-mail or paper report card to approximately 1,300 paediatricians and other child health clinicians. Clinicians are asked to indicate whether or not they have seen a child newly diagnosed with any of the listed conditions listed. Clinicians reporting cases are asked to provide details about demographics, diagnosis, treatments and short-term outcomes. All negative and positive reports are logged into a database and the report card return rate has been maintained at over 90% over the last 16 years.

Communicable diseases currently under surveillance include: acute flaccid paralysis (to identify potential cases of poliovirus infection); congenital cytomegalovirus infection; congenital rubella; perinatal exposure to HIV and HIV infection, neonatal herpes simplex virus infection; neonatal varicella, congenital varicella, severe complications of varicella, intussusception and its causes (e.g. rotavirus infection), and acute rheumatic fever (group A *Streptococcus* infection). After demonstrating feasibility in 2007, APSU has also conducted surveillance for severe complications of influenza during the influenza season. In 2009 APSU contributed to the national surveillance effort during the Influenza H1N1 09 pandemic.

APSU is a unit of the Royal Australasian College of Physicians, and its activities are supported by the Department of Health and Ageing; Sydney Medical School, The University of Sydney; NHMRC Enabling Grant No: 402784, Practitioner Fellowship No: 457084, E. Elliott, and H1N1 Grant no: 633028; the Creswick Foundation Fellowship (Y Zurynski), and Kids Research Institute at the Children's Hospital at Westmead.

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Australian National Creutzfeldt-Jakob Disease Registry

The surveillance for CJD in Australia is conducted through the Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR). CJD has been scheduled as a notifiable disease in all Australian states and territories. The ANCJDR is under contract to the Commonwealth to identify and investigate all suspect cases of transmissible spongiform encephalopathies (TSE) in Australia. An annual update is published in *CDI (Commun Dis Intell* 2009;33(2):188–191).

Australian Sentinel Practice Research Network

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

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

There are currently 96 general practitioners participating in the network from all jurisdictions other than the Northern Territory. Sixty-eight per cent of

these are in metropolitan areas, 26% in rural and 14% in remote areas of Australia. Approximately 6,000 consultations are recorded each week.

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

Influenza-like illness – record once only per patient

Must have the following: fever, cough and fatigue

Gastroenteritis – record once only per patient

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

Chickenpox – record once only per patient

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

Shingles – record once only per patient

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

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

HIV and AIDS surveillance

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

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

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

Each year from 1997, the NCHECR has published the *HIV/AIDS, Viral Hepatitis and Sexually Transmissible Infections in Australia Annual Surveillance Report*. The annual surveillance report, available through www.nchecr.unsw.edu.au, provides a comprehensive analysis and interpretation of surveillance data on HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia. The report *Bloodborne viral and sexually transmitted infections in Aboriginal and Torres Strait Islander people: Surveillance and Evaluation Report* has been published from 2007, as an accompanying document to the *Annual Surveillance Report*. The *Surveillance and Evaluation Report* provides detailed analysis and interpretation of the occurrence of these infections in Aboriginal and Torres Strait Islander communities in Australia.

Laboratory Virology and Serology Reporting Scheme

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

LabVISE data should be interpreted with caution. The number and type of reports received are subject to a number of biases. These include the number of participating laboratories, which has varied over time. The locations of participating laboratories also create bias, as some jurisdictions are better represented than others. Also changes in diagnostic practices, particularly the introduction of new testing methodologies, may affect laboratory reports. The ability of laboratory tests to distinguish acute from chronic or past infection must also be considered in interpretation of the data. Although changes in incidence cannot be determined with precision from this data, general trends can be observed, for example with respect to seasonality and the age-sex distribution of patients. (Review in *Commun Dis Intell* 2002;26(4):323–374).

National Influenza Surveillance Scheme

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

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

The results of each of the schemes are published together fortnightly throughout the influenza season (May to October) on the department's web site as the Australian Influenza Report.

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

OzFoodNet: enhanced foodborne disease surveillance

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

OzFoodNet reports quarterly on investigations of gastroenteritis outbreaks and clusters of disease potentially related to food. Annual reports have been produced and published in *CDI* (*Commun Dis Intell* 2009;33(4):389–413) since 2001. Data are reported from all Australian jurisdictions.

Sentinel Chicken Surveillance Programme

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

These viruses are enzootic in parts of the north-east Kimberley region of Western Australia and the Top End of the Northern Territory but are epizootic in other areas of the Kimberley, Pilbara, Gascoyne Murchison and Mid-west regions of Western Australia, in north Queensland and in Central Australia. MVEV is also responsible for occasional epidemics of encephalitis in eastern Australia. Since 1974, a number of sentinel chicken flocks have been established in Australia to provide an early warning of increased MVEV activity. These programs are supported by individual state health departments. Each state has a contingency plan that will be implemented if one or more chickens in a flock seroconverts to MVEV.

Currently, flocks are maintained in the north of Western Australia, the Northern Territory, New South Wales and in Victoria. The flocks in Western Australia and the Northern Territory are tested all year round but those in New South Wales and Victoria are tested only in the summer months, during the main MVEV risk season. Results are posted on the National Arbovirus Surveillance Website by state representatives. A yearly summary is presented in *CDI* (*Commun Dis Intell* 2009;33(2):155–169).

References

1. Last JM. A dictionary of epidemiology. New York: Oxford University Press, 1988.
2. Hall R. Notifiable diseases surveillance, 1917 to 1991. *Commun Dis Intell* 1993;226–236. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-pubs-annlrpt-oz_dis19_91.htm Accessed March 2010.

COMMUNICABLE DISEASES INTELLIGENCE INSTRUCTIONS FOR AUTHORS

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

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

The objectives of *CDI* are:

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

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

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

Manuscripts for submission

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

- a list of all authors;
- confirmation that the manuscript content (in part or in full) has not been submitted or published elsewhere; and
- whether the manuscript is being submitted as an article, short report, surveillance summary, outbreak report or case report.
- In addition, manuscripts should include a title page that should contain the following information:
- title (e.g. Prof, Dr, Ms, Miss, Mrs, Mr), full name including middle initial, position held, and institution at the time the article was produced, of each author;
- name of corresponding author, including current postal address, telephone, facsimile and email; and
- word count of the main text and of the abstract.

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

Authorship

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

Types of manuscript

Original Articles

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

Short reports

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

Surveillance summaries

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

Outbreak reports

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

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

Case reports

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

Letters to the Editor

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

Document preparation

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

In addition:

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

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

Tables

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

Figures and illustrations

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

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

Charts

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

Illustrations

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

Photographs

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

Maps

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

References

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

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

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

Ethics committee approvals and patients' rights to privacy

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

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

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

Review process

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

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

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

Copyright

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

Submission of manuscripts

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

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