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Annual reports

Annual report: Surveillance of adverse events following immunisation in Australia, 2007

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Abstract

This report summarises Australian passive surveillance data for adverse events following immunisation (AEFI) reported to the Therapeutic Goods Administration for 2007, and describes reporting trends over the 8-year period 2000 to 2007. There were 1,538 AEFI records for vaccines administered in 2007. This is an annual AEFI reporting rate of 7.3 per 100,000 population, the highest since 2003 and an 85% increase compared with 2006 (835 AEFI records; 4.0 records per 100,000 population). The increase was almost entirely due to reports following the commencement of the national 3-dose human papillomavirus (HPV) vaccine program for females aged 12 to 26 years in April 2007 (n=705 reports) and the national infant rotavirus vaccine program in July 2007 (n=72 reports). AEFI reporting rates in 2007 were 2.3 per 100,000 administered doses of influenza vaccine for adults aged ≥18 years, 18.6 per 100,000 administered doses of pneumococcal polysaccharide vaccine for those aged ≥65 years and 12.7 per 100,000 administered doses of scheduled vaccines for children aged <7 years. The majority of the 1,538 AEFI reports for 2007 described non-serious events while 9% (n=141) were classified as serious. Two deaths temporally associated with immunisation were reported; there was no evidence to suggest a causal association. The most significant AEFI reported following HPV vaccine were anaphylaxis (n=11) and convulsion (n=18), mostly associated with syncope. The most commonly reported reactions were allergic reaction, injection site reaction, headache and nausea. The data confirm that, despite the low rate of AEFI reporting in Australia, the passive surveillance system is sufficiently robust to detect safety signals which are expected following changes in the immunisation program, allowing these to be investigated further. *Commun Dis Intell* 2008;32:371–387.

Keywords: AEFI, adverse events, vaccines, surveillance, immunisation, vaccine safety

Introduction

This report summarises national passive surveillance data for adverse events following immunisation (AEFI) reported to the Therapeutic Goods Administration (TGA) to 31 March 2008. The report focuses on AEFI reported for vaccines administered during 2007 and trends in AEFI reporting for the 8-year period 2000 to 2007.

The aim of passive post-licensure AEFI surveillance is to monitor vaccine and immunisation program safety and to detect population-specific, rare, late-onset or unexpected adverse events that may not be identified in pre-licensure vaccine trials.[1,2] An 'adverse event following immunisation' is defined as any serious or unexpected adverse event that occurs *after* a vaccine has been given, which may be related to the vaccine itself or to its handling or administration. An AEFI can be *coincidentally* associated with the *timing* of immunisation without necessarily being caused by the vaccine or the immunisation process.

In Australia, AEFI are notified to the TGA by state and territory health departments, health professionals, vaccine manufacturers and members of the public.[3,4] All reports are assessed using internationally consistent criteria[5] and entered into the Australian Adverse Drug Reactions System (ADRS) database. All reports for vaccines and complementary medicines, plus all serious reports for drugs, are forwarded to the Adverse Drug Reactions Advisory Committee (ADRAC) for review at regular meetings. ADRAC is an expert committee of the TGA composed of independent medical experts who have expertise in areas of importance to the evaluation of medicine safety.

Passive AEFI surveillance data have been collated in the ADRS database since 2000 and used to monitor trends, detect signals and generate hypotheses. Reports summarising national AEFI surveillance data have been published regularly since 2003.[6–14] Several important changes to vaccine funding and availability occurred in 2007 that impact on the AEFI surveillance data presented in this report.

- A national human papillomavirus (HPV) immunisation program commenced in April 2007 for all girls aged 12 to 18 years, and was extended to the 19 to 26 year age group in July 2007.[15] The program is delivered through a secondary school immunisation program and general practice for those not vaccinated in a school program. In 2007, the funded program delivered only the quadrivalent vaccine (Gardasil®); the bivalent vaccine (Cervarix®) became available on the private market only during 2007. Both vaccines are given as a 3-dose course.
- Rotavirus (RotaTeq® and Rotarix®) vaccine was added to the National Immunisation Program (NIP) for all infants in Australia on 1 July 2007.[15] From August 2006, the vaccine was publicly funded for infants resident in the Northern Territory, and was available on the private market for other infants. Infants receive either a 2-dose schedule (Rotarix®) at 2 and 4 months of age, or a 3-dose schedule (RotaTeq®) at 2, 4 and 6 months of age.

Previous changes to the NIP schedule in 2003 and 2005[3,4,15] also impact on the interpretation of trend data: (i) on 1 January 2003, the meningococcal C conjugate vaccine (MenCCV) immunisation program commenced when the vaccine was introduced into the NIP schedule at 12 months of age with a catch-up program for all those born between 1984 and 2001;[15] (ii) in September 2003, the 4th dose of DTPa vaccine, given at 18 months of age, was removed from the immunisation schedule;[3] (iii) in January 2005, funded national pneumococcal immunisation programs commenced for infants at 2, 4 and 6 months of age (7-valent conjugate vaccine; 7vPCV), and for adults aged ≥65 years (23-valent polysaccharide vaccine; 23vPPV);[15] (iv) in November 2005, varicella vaccine was added to the NIP schedule as a single dose due at 18 months (for children born on or after 1 May 2004) or at 12–13 years of age if they have no evidence of either vaccination or varicella infection; and (v) in November 2005, inactivated poliovirus vaccine (IPV) replaced oral poliovirus vaccine (OPV) for all age groups. All IPV-containing combination vaccines include diphtheria-tetanus-acellular pertussis (DTPa) antigens (i.e. quadrivalent vaccines) and some also include hepatitis B (HepB) and/or *Haemophilus influenzae* type b (Hib) antigens (i.e. pentavalent and hexavalent vaccines). The specific combination vaccines administered at 2, 4, and 6 months of age vary between states and territories but all provide DTPa-IPV quadrivalent vaccine at 4 years of age.[4]

Methods

Adverse events following immunisation data

De-identified information was released to the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) for all drug and vaccine adverse event notifications received by the TGA to 31 March 2008. Readers are referred to previous AEFI surveillance reports for a description of the surveillance system and methods used to evaluate reports to the TGA.[6,7]

AEFI records* contained in the ADRS database were eligible for inclusion in the analysis if a vaccine was recorded as 'suspected' of involvement in the reported adverse event and *either*

(a) the vaccination and onset occurred between 1 January 2000 and 31 December 2007; *or*

(b) for records where the vaccination date was not recorded, the date of onset of symptoms or signs occurred between 1 January 2000 and 31 December 2007.

Definitions of outcomes and reactions

AEFI were defined as 'serious' or 'non-serious' based on information recorded in the ADRS database and criteria similar to those used by the World Health Organization[5] and the US Vaccine Adverse Events Reporting System.[16] In this report, an AEFI is defined as 'serious' if the record indicated that the person had recovered with sequelae, been admitted to a hospital, experienced a life-threatening event, or died.

The causality ratings of 'certain', 'probable' and 'possible' are assigned to individual AEFI records by the TGA and reviewed by ADRAC. They describe the likelihood that a suspected vaccine or vaccines was/were associated with the reported reaction at the level of the individual vaccine recipient. Factors that are considered in assigning causality ratings include the timing (minutes, hours etc) and the spatial correlation (for injection site reactions) of symptoms and signs in relation to vaccination, and whether one or more vaccines were administered.[6] Because children in particular receive several vaccines at the same time, all administered vaccines are usually listed as 'suspected' of involvement in a systemic adverse event as it is usually not possible to attribute the AEFI to a single vaccine.

Typically, each AEFI record listed several symptoms, signs and diagnoses that had been re-coded by TGA staff from the reporter's description into

* The term 'AEFI record' is used throughout this report because a single AEFI notification can generate more than 1 record in the ADRS database. This usually occurs if a notification describes an injection site reaction plus symptoms and signs of a systemic adverse event. Two records will appear in the database: one containing information relevant to the injection site reaction and the other for the systemic adverse event.

standardised terms using the Medical Dictionary for Regulatory Activities (MedDRA®).[17] AEFI reports of suspected anaphylaxis and hypotonic-hyporesponsive episodes (HHE) were reviewed by ADRAC using the Brighton Collaboration case definitions.[18,19] If an AEFI report met any level of the Brighton Collaboration case definition it was coded accordingly.

To simplify data analysis, we grouped MedDRA® coding terms to create a set of reaction categories. Firstly, reaction categories were created that were analogous to the AEFI listed and defined in *The Australian Immunisation Handbook* (8th edition).[3] Additional categories were created for MedDRA® coding terms that were listed in more than 1% of AEFI records (e.g. headache, irritability, cough). Reaction terms listed in less than 1% of records were grouped into broader categories based on the organ system where the reaction was manifested (e.g. gastrointestinal, neurological).

Data analysis

All data analyses were performed using SAS software version 9.1.3.[20] The distribution of AEFI records was analysed by age, gender and jurisdiction. Average annual population-based reporting rates were calculated for each state and territory and by age group using population estimates obtained from the Australian Bureau of Statistics.

The frequency and age distribution of AEFI outcomes, reaction categories and vaccines listed as 'suspected' of involvement in the reported adverse event were assessed. For each vaccine, the age distribution of vaccinees notified with AEFI was calculated, as well as the proportion of AEFI records where (i) the vaccine was the only suspected vaccine or drug, (ii) the AEFI record was assigned a 'certain' or 'probable' causality rating, and (iii) the AEFI was defined as 'serious'.

AEFI reporting rates per 100,000 administered doses were estimated for influenza vaccine for adults aged ≥18 years, for 23vPPV for adults aged ≥65 years, and for 10 vaccines funded through the NIP for children aged <7 years. These were DTPa-IPV, DTPa-IPV-HepB, DTPa-IPV-HepB-Hib, Hib, Hib-HepB, measles-mumps-rubella (MMR), MenCCV, 7vPCV, varicella and rotavirus vaccines. The 2007 AEFI reporting rates were compared with those for 2006 and 2005.

Denominator data to estimate influenza and 23vPPV AEFI reporting rates were obtained from the biennial national adult coverage survey conducted in 2006 (unpublished) for adults aged ≥65 years and 18 to 64 years (influenza only). The number of administered doses of each of the 10 childhood vaccines was calculated from the Australian Childhood Immunisation Register (ACIR), a national population-based register of approximately 99% of children aged <7 years.[21]

Dose-based AEFI reporting rates could not be calculated for other vaccines and age groups as reliable denominator data for the number of vaccine doses distributed or administered were not available.

Notes on interpretation

Caution is required when interpreting the AEFI data presented in this report. Due to reporting delays and late onset of some AEFI, the data are considered preliminary, particularly for the 4th quarter of 2007. Data published in previous reports for 2000–2006[6–14] differ to that presented in this report for the same period because the data have been updated to include AEFI notified to the TGA during 2007 for vaccines administered in previous years.

The information collated in the ADRS database is intended primarily for signal detection and hypothesis generation. While AEFI reporting rates can be estimated using appropriate denominators, such as the number of vaccine doses administered, they cannot be interpreted as incidence rates due to under-reporting and biased reporting of suspected AEFI, and the variable quality and completeness of information provided in individual AEFI notifications.[6–14,22] In addition, AEFI that were assessed as mild by the health care provider may not be reported to the passive surveillance system, which could impact the comprehensiveness of the report. *The Australian Immunisation Handbook* indicates that immunisation providers need not report common reactions to the TGA.[3,4]

It is important to note that this report is based on vaccine and reaction term information collated in the ADRS database and not on comprehensive clinical notes. Individual database records list symptoms, signs and diagnoses that were used to define a set of reaction categories based on the case definitions provided in the 8th edition of *The Australian Immunisation Handbook*.[3] These reaction categories are similar, but not identical, to the AEFI case definitions.

The reported symptoms, signs and diagnoses in each AEFI record in the ADRS database are temporally associated with vaccination but are not necessarily causally associated with a vaccine or vaccines. The causality ratings assigned to individual AEFI records describe the likelihood that a suspected vaccine or vaccines was/were associated with the reported reaction at the level of the individual vaccine recipient.

Results

Summary of data

There was a total of 1,538 AEFI records in the ADRS database where the date of vaccination (or onset of an adverse event, if vaccination date was not reported) occurred in 2007. Approximately 2% of AEFI notifications resulted in more than 1AEFI record in the database, usually an injection site reaction (ISR) and a systemic reaction.

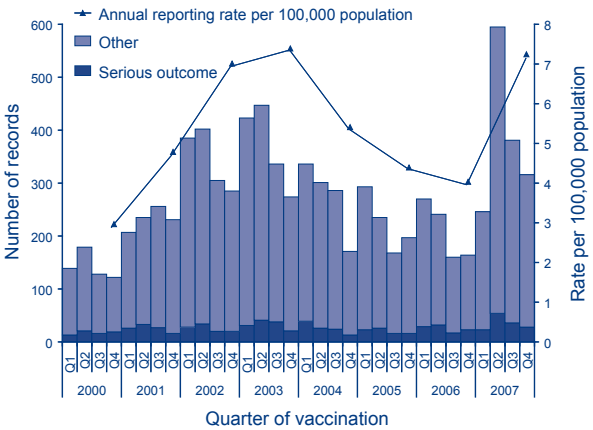
The number of AEFI records for vaccines administered in 2007 is almost twice the 835 AEFI records for vaccines administered in 2006. The increase is largely due to AEFI notifications related to HPV vaccination (n=705) following the commencement of the national school-based HPV immunisation program in April 2007, and to the commencement of the national rotavirus vaccine program in July 2007 (n=72).

One hundred and forty-one (9%) of the 1,538 AEFI records were defined as 'serious' (i.e. recovery with sequelae, requiring hospitalisation, experiencing a life-threatening event or death). A total of 511 (33%) AEFI records were assigned causality ratings of 'certain' (n=391, 25%) or 'probable' (n=120, 8%).

Reporting trends

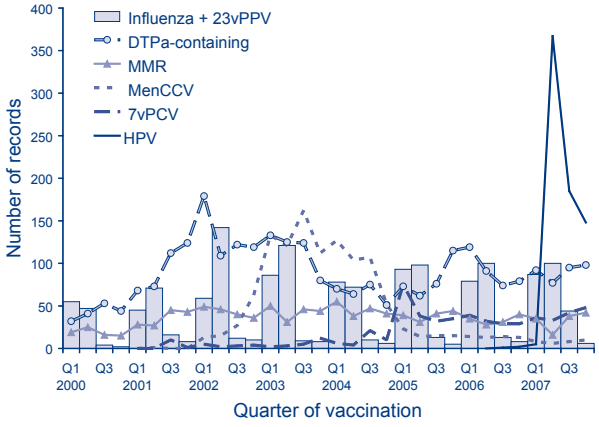
The AEFI reporting rate for 2007 was 7.3 per 100,000 population, compared with 4.0 per 100,000 population in 2006 (Figure 1). This is the second highest reporting rate for the period 2000 to 2007, and is similar to the peak in 2003 that coincided with the national MenCCV catch-up immunisation program for the 1 to 19 year age group. The trends in AEFI notifications shown in Figure 1 are reflected in the trends in vaccines frequently suspected of involvement in reported AEFI (Figure 2), and in the types of reactions frequently reported (Figure 3). Many of these changes correspond in time with changes in the funded NIP schedule. The most recent changes were the commencement of the national school-based HPV immunisation program in April 2007 (which was followed by the highest quarterly peak in AEFI reporting, shown in Figures 1 and 2), and a peak following the commencement of the national infant rotavirus vaccination program in July 2007. Previously, AEFI reporting for MenCCV and 7vPCV increased when the national routine and catch-up programs first commenced in January 2003 (MenCCV) and January 2005 (7vPCV), then stabilised over time (Figure 2). AEFI reports for DTPa-containing vaccines declined following the removal of the 4th dose from the immunisation schedule in the third quarter of 2003, and increased again following the introduction of DTPa and IPV-containing multivalent vaccines in the 4th quarter of 2005.

Figure 1. Adverse events following immunisation, ADRS database, 2000 to 2007, by quarter of vaccination



For reports where the date of vaccination was not recorded, the date of onset was used as a proxy for vaccination date.

Figure 2. Frequently suspected vaccines, adverse events following immunisation, ADRS database, 2000 to 2007, by quarter of vaccination

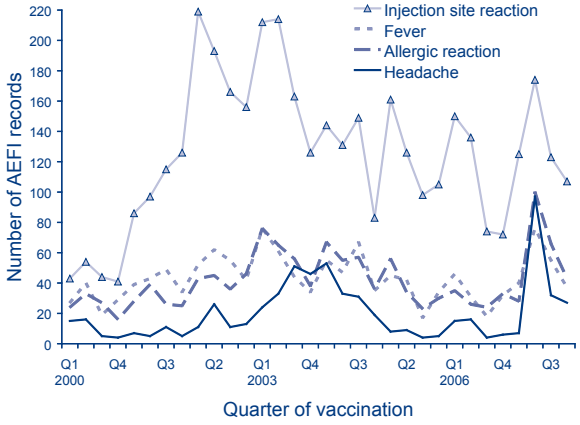


See appendix for abbreviations of vaccine names. DTPa-containing vaccines include DTPa, and the combination vaccines DTPa-HepB, DTPa-IPV, DTPa-IPV-HepB and DTPa-IPV-HepB-Hib.

The usual seasonal pattern of AEFI reporting, with peaks in the 1st half of the year, was less apparent in 2007 following the commencement of the national HPV program in the second quarter of the year, where 3 doses were delivered over a period of several months (Figure 1). The seasonal peaks generally correspond to the months when more vaccinations are administered in Australia, particularly among 4- and 5-year-old children receiving MMR and DTPa-containing vaccines prior to commencing

school in February and older Australians receiving 23vPPV and influenza vaccine during the autumn months (March to June) (Figure 2).

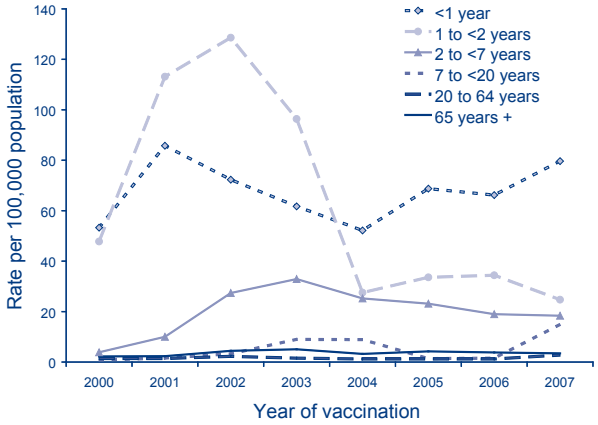
Figure 3. Selected frequently reported adverse events following immunisation, ADRS database, 2000 to 2007, by quarter of vaccination



Age distribution

In 2007, the highest population-based AEFI reporting rate occurred in infants <1 year of age, the age group that received the highest number of vaccines (Figure 4). Compared with 2006, AEFI reporting rates increased among the <1 year age group (from 66.2 to 79.6 per 100,000 population), the 7 to 19 year age group (1.5 to 14.8 per 100,000) and the 20 to 64 year age group (1.2 to 2.8 per 100,000). Rates were stable or declined slightly for other age groups. The changes over time reflect the introduction or removal of scheduled vaccines for specific age groups.

Figure 4. Reporting rates of adverse events following immunisation per 100,000 population, ADRS database, 2000 to 2007, by age group and year of vaccination



Geographical distribution

As in previous annual reports,[6,7,9,11,13] AEFI reporting patterns varied between states and territories for vaccines received during 2007 (Table 1). The Northern Territory, South Australia and the Australian Capital Territory had the highest reporting rates (20.9, 19.8 and 18.2 per 100,000 population, respectively) while Tasmania and New South Wales had the lowest rates (4.7 and 4.5 per 100,000 population, respectively). AEFI reporting rates increased in all jurisdictions in 2007, largely related to the commencement of the school-based HPV program. An increase in the reporting rate for Victoria (from 3.7 per 100,000 in 2006 to 6.7 in 2007) also followed the implementation of a new AEFI reporting and evaluation system in that state in April 2007.[23]

Outcomes

Sixty per cent of reported AEFI in 2007 were defined as 'non-serious' while 9% were defined as 'serious' (Table 2), similar to the proportions observed in previous years. Fewer 'serious' AEFI were assigned certain or probable causality ratings compared with 'non-serious' AEFI (20% versus 35%) (Table 2). Vaccines listed as 'suspected' of involvement in reported AEFI and with outcomes defined as 'serious' are shown in Table 3.

Two deaths were recorded as temporally associated with receipt of vaccines. One was a 16-month-old child who had received influenza and varicella vaccines 3 days prior to death. The child had Down Syndrome and a pre-existing cardiac condition; autopsy was inconclusive. The other reported death was a 55-year-old who died 1 day after receiving influenza vaccine. Further information was requested by the TGA and has not been provided by the reporter.

Vaccines

Thirty-one vaccines were recorded as 'suspected' of involvement in the adverse events described in the 1,538 AEFI records for vaccines received in 2007 (Table 3). The percentage of records where only 1 vaccine was suspected of involvement in the adverse event differed by vaccine, as did the percentage assigned causality ratings of 'certain' or 'probable', and with outcomes defined as 'serious'. This is to be expected as vaccines are routinely co-administered at specific ages in the immunisation schedule.

HPV vaccine was the most frequently reported vaccine (705 records; 46%) (Table 3). Vaccines containing diphtheria, tetanus and acellular pertussis antigens (including combination vaccines and dTpa) were suspected in 391 (25%) records (Table 3) with DTPa-IPV the most frequently suspected vaccine in

Table 1. Adverse events following immunisation (AEFI), ADRS database, 1 January to 31 December 2007, by jurisdiction

Jurisdiction	AEFI records		Annual reporting rate per 100,000 population*			
	n	%	Overall	'Certain' or 'probable' causality rating†	'Serious' outcome‡	Aged <7 years
Australian Capital Territory	62	4	18.2	4.1	0.6	64.8
New South Wales	313	20	4.5	1.7	0.5	9.8
Northern Territory	45	3	20.9	12.6	2.3	120.1
Queensland	228	15	5.5	1.8	0.4	14.0
South Australia	313	20	19.8	5.3	0.9	91.2
Tasmania	23	1	4.7	1.4	0.2	16.2
Victoria	347	23	6.7	2.4	0.6	39.4
Western Australia	129	8	6.1	2.2	0.7	21.2
Other§	78	5	na	na	na	na
Total	1,538	100	7.3	2.4	0.7	28.3

* Average annual rates per 100,000 population calculated using mid-2007 population estimates (Australian Bureau of Statistics).

† See previous report[6] for criteria used to assign causality ratings.

‡ AEFI records defined as 'serious' (i.e. recovery with sequelae, hospitalisation, life-threatening or death – see Table 2).

§ Records where the jurisdiction in which the AEFI occurred was not reported or was unclear. AEFI records in this category were notified by pharmaceutical companies (n=67), members of the public (8), general practitioners (2) and from a hospital (1).

Table 2. Outcomes of adverse events following immunisation (AEFI), ADRS database, 2007

Outcome	AEFI records		'Certain' or 'probable' causality rating†		Age group‡			
					<7 years		≥7 years	
	n	%*	n	%§	n	%§	n	%§
Non-serious	920	60	321	35	318	35	591	64
Not recovered at time of report	328	21	119	36	95	29	229	70
Not known (missing data)	149	10	43	29	50	34	93	62
Serious:	141	9	28	20	63	45	78	55
recovered with sequelae	(3)		(2)		(0)		(3)	
hospital treatment – admission	(125)		(24)		(59)		(66)	
life-threatening event	(11)		(2)		(3)		(8)	
death (maybe drug)	(2)		(0)		(1)		(1)	
Total	1,538	100	511	33	526	34	991	64

* Percentages relate to the total number of AEFI records (n=1538).

† Causality ratings were assigned to AEFI records using criteria described previously.[6]

‡ AEFI records where both age and date of birth were not recorded are not shown (21 missing).

§ Percentages relate to the number of AEFI records with the specific outcome, e.g. of 920 AEFI records with a 'non-serious' outcome, 35% had causality ratings of 'certain' or 'probable' and 35% were for children aged <7 years.

this group (288 records; 19%). Influenza vaccine and 23vPPV were among the more common vaccines listed as suspected of involvement in reported AEFI, particularly where only 1 vaccine was listed as suspected (Table 3). The relative frequency of reports for specific vaccines relates both to the number of doses administered and the types of AEFI reported for each vaccine.

Reports related to MMR vaccine remained relatively stable over time (Figure 2), while there have been peaks in AEFI reporting for vaccines recently introduced into the routine childhood immunisation schedule, followed by a reduction and stabilisation in reporting over time (Figure 5). This pattern has been particularly evident with the introduction of the scheduled MenCCV dose at 12 months of age

Table 3. Vaccine types listed as 'suspected' in records of adverse events following immunisation (AEFI), ADRS database, 2007

Suspected vaccine type*	AEFI records	One suspected vaccine or drug only†		'Certain' or 'probable' causality rating‡		'Serious' outcome§		Age group‖			
								<7 years		≥7 years	
	n	n	%¶	n	%¶	n	%¶	n	%¶	n	%¶
HPV**	705	674	96	203	29	43	6	0	–	689	98
DTPa-IPV	288	128	44	113	39	24	8	287	100	1	0
7vPCV	159	7	4	9	6	26	16	158	99	1	1
Influenza	150	111	74	45	30	21	14	30	20	116	77
MMR	131	27	21	16	12	15	11	118	90	13	10
23vPPV	118	87	74	73	62	10	8	4	3	112	95
Hib-Hepatitis B	118	118	100	4	3	14	12	118	100	0	–
Rotavirus††	90	26	29	5	6	19	21	90	100	0	–
Hepatitis B	53	22	42	7	13	5	9	7	13	46	87
Varicella	44	32	73	8	18	6	14	28	64	16	36
DTPa-IPV-HepB-Hib	39	2	5	2	5	9	23	39	100	0	–
MenCCV	32	4	13	3	9	1	3	30	94	2	6
dTpa	29	18	62	10	34	2	7	0	–	29	100
DTPa	27	7	26	5	19	2	7	27	100	0	–
Hib	17	1	6	1	6	2	12	17	100	0	–
dT	15	9	60	7	47	2	13	0	–	15	100
Hepatitis A	13	4	31	2	15	2	15	5	38	8	62
Hepatitis A + B	9	7	78	2	22	3	33	0	–	9	100
DTPa-IPV-HepB	8	2	25	2	25	2	25	8	100	0	–
Yellow fever	8	3	38	0	–	3	38	0	–	8	100
Men4PV	5	2	40	1	20	2	40	0	–	5	100
BCG	4	3	75	1	25	1	25	4	100	0	–
IPV	4	0	–	0	–	2	50	1	25	3	75
Q fever	4	4	100	1	25	0	–	0	–	4	100
Hepatitis A-Typhoid	3	2	67	1	33	0	–	0	–	3	100
Rabies	3	0	–	0	–	2	67	1	33	2	67
Typhoid	3	1	33	1	33	1	33	1	33	2	67
dTpa-IPV	1	0	–	0	–	1	100	0	–	1	100
Cholera	1	0	–	0	–	1	100	0	–	1	100
Japanese encephalitis	1	0	–	0	–	1	100	1	100	0	–
Tetanus	1	1	100	0	–	0	–	0	–	1	100
Total‡‡	1,538	579	38	515	33	141	9	526	34	991	64

* See appendix for abbreviations of vaccine names.

† AEFI records where only 1 vaccine was suspected of involvement in a reported adverse event.

‡ Causality ratings were assigned to AEFI records using criteria described previously.[6]

§ 'Serious' outcomes are defined in the Methods section (see also Table 2).

‖ AEFI records are not shown if both age and date of birth were not reported.

¶ Percentages are calculated for the number of AEFI records where the vaccine was suspected of involvement in the AEFI, e.g. HPV was 'suspected' in 705 AEFI records; this was the only suspected vaccine in 96% of the 705 AEFI records, 29% had 'certain' or 'probable' causality ratings, 6% were defined as 'serious' and 98% were for those aged ≥7 years.

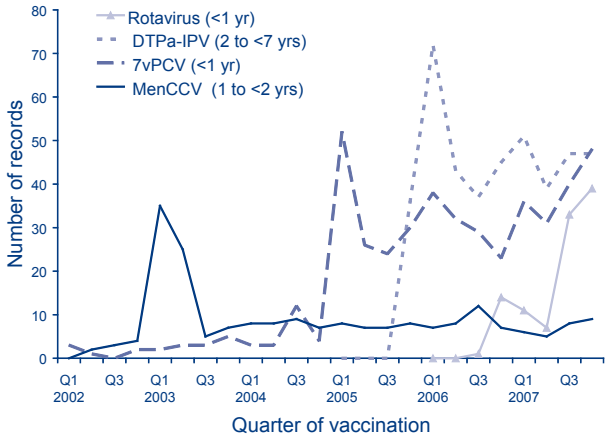
** Human papillomavirus vaccine was added to the National Immunisation Program schedule on 1 April 2007.[15]

†† Rotavirus vaccine was added to the National Immunisation Program schedule on 1 July 2007.[15]

‡‡ Total number of AEFI records analysed, not the total in each column as categories are not mutually exclusive and an AEFI record may list more than 1 vaccine.

in January 2003, 7vPCV at 2, 4, and 6 months of age in January 2005, and the DTPa-IPV containing vaccines at 2, 4, 6 months and 4 years of age in November 2005. The most recent peak evident is shown for rotavirus vaccine from the second quarter of 2007. Smaller peaks in 7vPCV AEFI reporting coincide with the later introduction of the infant DTPa-IPV and rotavirus programs (Figure 5), presumably related to the simultaneous administration of the 3 vaccines at 2, 4 and 6 months of age.

Figure 5. Reports of adverse events following immunisation, ADRS database, 2002 to 2007, for vaccines recently introduced into the funded National Immunisation Program,* by quarter of vaccination



* Meningococcal C conjugate vaccine (MenCCV) was introduced into the National Immunisation Program on 1 January 2003, 7-valent pneumococcal conjugate vaccine (7vPCV) on 1 January 2005, both DTPa-IPV and combination vaccines on 1 November 2005, and rotavirus vaccine on 1 July 2007.

Reactions

The distribution and frequency of reactions listed in AEFI records for 2007 are shown in Tables 4 and 5. In Table 4, only the reaction categories analogous to those listed in *The Australian Immunisation Handbook*[3] are shown. In Table 5, other reaction categories are listed in descending order of frequency.

The most frequently reported adverse events were ISR (34% of 1,538 AEFI records) followed by allergic reaction (17%), fever (14%), rash (11%), headache (11%) and malaise (10%) (Tables 4 and 5). ISR was the most commonly reported individual adverse event following receipt of 23vPPV (78%; 92/118), MMR (60%; 71/118), DTPa-containing vaccines (52%; 202/391), and influenza vaccine (37%; 56/150), administered alone or in combination with other vaccines. Twenty per cent (143/705) of HPV vaccine-related AEFI records listed ISR.

More severe AEFI included reports of anaphylactic reaction (n=13), HHE (n=37), thrombocytopenia (n=2), encephalitis (n=2), convulsion (n=35), Guillain-Barré syndrome (GBS; n=7) and death (n=2; described previously in this report). The 7 records coded as GBS included 2 reports in adolescent girls following HPV vaccine, and 5 reports following influenza vaccine in adults aged 23 to 72 years.

Ten of the 13 reports of anaphylaxis in 2007 occurred in women following receipt of HPV vaccine; two had also received dTpa.[24] There were a total of 35 reports of convulsion, including syncopal and febrile convulsions. Twelve were for children aged <7 years. The most commonly suspected vaccines were HPV (n=18), 7vPCV (n=6) and MMR (n=6).

The majority (34/37) of HHE were notified by Victoria (22), South Australia (6) and Queensland (6). DTPa-containing vaccines were listed as suspected in 30 reports, with DTPa-IPV suspected in 27 reports. 7vPCV (n=31) and Hib-HepB (n=26) were also commonly suspected vaccines in HHE reports.

Reactions shown in Table 5 include headache, malaise, nausea, dizziness and reduced sensation (paraesthesia). The most commonly reported categories for grouped reactions involved the gastrointestinal, neurological and musculoskeletal organ systems.

The trends in the most frequently reported types of reactions changed over time (Figure 3). Reports of allergic reaction, fever and rash were less variable compared with reports of ISR. Reports of headache peaked in 2003 and again in 2007, coinciding with the national school-based MenCCV immunisation program in 2003 and the HPV program in 2007. Much of the variation in reporting of ISR relates to specific changes in the immunisation schedules for vaccines that are known to have higher rates of ISR, including DTPa-containing vaccines, MenCCV, 23vPCV and HPV vaccine.[6–14,25,26]

Dose-based reporting rates

Influenza vaccine and adults aged ≥18 years

In 2007, influenza vaccine was suspected of involvement in 109 AEFI records for people aged ≥18 years. The AEFI reporting rate was 2.3 per 100,000 administered doses, similar to the rate in 2005 and 2006 (Table 6). As seen in previous years, both the overall and serious AEFI reporting rates were higher for vaccinees aged 18 to 64 years than among older vaccinees.

Table 4. Reaction categories of interest* mentioned in records of adverse events following immunisation (AEFI), ADRS database, 2007

Reaction category*	AEFI records	Only reaction reported†		Certain/probable causality rating‡		Age group§			
						<7 years		≥7 years	
	n	n	%‖	n	%‖	n	%‖	n	%‖
Injection site reaction	529	293	55	366	69	235	44	288	54
Allergic reaction¶	269	46	17	48	18	68	25	198	74
Fever	208	7	3	25	12	92	44	115	55
Rash	164	52	32	23	14	71	43	91	55
Abnormal crying	52	1	2	1	2	47	90	5	10
HHE**	37	18	49	2	5	37	100	0	–
Convulsions	35	8	23	4	11	12	34	22	63
Arthralgia	25	1	4	4	16	0	–	25	100
Lymphadenopathy/itis††	21	6	29	2	10	5	24	16	76
Anaphylactic reaction	13	0	–	10	77	2	15	11	85
Arthritis	7	3	43	0	–	1	14	6	86
Guillain-Barré syndrome	7	5	71	0	–	0	–	7	100
Abscess	4	3	75	2	50	3	75	1	25
Death	2	2	100	0	–	1	50	1	50
Encephalitis	2	1	50	0	–	1	50	1	50
Orchitis	2	0	–	0	–	1	50	1	50
Parotitis	2	0	–	0	–	0	–	2	100
Thrombocytopenia	2	1	50	0	–	1	50	1	50
Meningitis	1	0	–	0	–	0	–	1	100
Sepsis	1	0	–	0	–	0	–	1	100
Acute flaccid paralysis	0	–		–		–		–	
Brachial neuritis	0	–		–		–		–	
Encephalopathy	0	–		–		–		–	
Osteitis	0	–		–		–		–	
Osteomyelitis	0	–		–		–		–	
SSPE‡‡	0	–		–		–		–	
Toxic shock syndrome	0			–		–		–	
Total§§	1,538	579	38	515	33	526	34	991	64

* Reaction categories were created for the AEFI of interest listed and defined in *The Australian Immunisation Handbook,* (8th edition, p 22–23 and 271–275)[3] as described in Methods section.

† AEFI records where only 1 reaction was reported.

‡ Causality ratings were assigned to AEFI records using criteria described previously.[6]

§ Not shown if neither age nor date of birth were recorded.

‖ Percentages relate to the number of AEFI records in which the specific reaction term was listed, e.g. of 529 AEFI records listing injection site reaction, 55% listed only 1 type of reaction while 69% had a causality rating of 'certain' or 'probable' and 44% were for children aged <7 years.

¶ Allergic reaction includes skin and/or gastrointestinal (e.g. diarrhoea, vomiting) symptoms and signs.[3]

†† Includes lymphadenitis following Bacille Calmette-Guèrin vaccination and the more general term of 'lymphadenopathy'.

** Hypotonic-hyporesponsive episode.

‡‡ Subacute sclerosing panencephalitis.

§§ Total number of AEFI records analysed, not the total in each column as categories are not mutually exclusive and an AEFI record may list more than 1 reaction term.

Table 5. 'Other'* reaction terms listed in records of adverse events following immunisation (AEFI), ADRS database, 2007

Reaction term*	AEFI records	Only reaction reported†		Certain/probable causality rating‡		Age group§			
						<7 years		≥7 years	
	n	n	%‖	n	%‖	n	%‖	n	%‖
Headache	163	15	9	29	18	3	2	153	94
Malaise	159	1	1	21	13	28	18	129	81
Nausea	149	2	1	34	23	3	2	142	95
Dizziness	125	2	2	36	29	0	–	122	98
Reduced sensation	71	9	13	19	27	0	–	70	99
Syncope	69	18	26	19	28	0	–	68	99
Pain	62	2	3	9	15	3	5	59	95
Resp. rate/rhythm change	62	3	5	14	23	24	39	38	61
Myalgia	42	0	–	3	7	2	5	40	95
Oedema	42	4	10	17	40	13	31	29	69
Irritability	39	0	–	0	–	36	92	3	8
Pallor	39	2	5	10	26	17	44	22	56
Gastrointestinal – RVV	37	9	24	3	8	37	100	0	–
Weakness	36	0	–	7	19	1	3	35	97
Heart rate/rhythm change	31	1	3	6	19	13	42	18	58
Increased sweating	29	0	–	5	17	4	14	25	86
Somnolence	29	1	3	4	14	11	38	17	59
Anorexia	23	0	–	3	13	11	48	11	48
Flushing	21	0	–	7	33	1	5	18	86
Visual disturbance	21	1	5	4	19	0	–	20	95
Abdominal pain	20	0	–	3	15	2	10	18	90
Erythema	20	1	5	4	20	7	35	12	60
Tremor	16	1	6	6	38	1	6	15	94
Genital/menstrual – HPV	15	7	47	0	–	0	–	15	100
Other	721	51	12	73	17	104	25	311	74
gastrointestinal	61	9	15	9	15	21	34	40	66
neurological	59	3	5	13	22	10	17	49	83
musculoskeletal	46	2	4	12	26	4	9	42	91
psychological	46	3	7	6	13	11	24	33	72
respiratory	44	6	14	10	23	13	30	28	64
general non-specific	37	1	3	3	8	4	11	32	86
eye or ear	35	0	–	5	14	6	17	28	80
cardiovascular	33	2	6	13	39	6	18	27	82
skin	33	5	15	6	18	6	18	27	82
infection	23	7	30	23	100	13	57	10	43
metabolic/endocrine	16	0	–	16	100	7	44	9	56
renal/urogenital	14	1	7	3	21	3	21	11	79
haematological	12	2	17	1	8	1	8	11	92
miscellaneous	4	2	50	0	–	0	–	4	100
pregnancy/congenital	1	1	100	0	–	0	–	1	100

* Reaction terms not listed in *The Australian Immunisation Handbook*[3] but included in AEFI records in the ADRAC database. The top part of the table shows reaction terms included in 1% or more of AEFI records; the bottom part of the table shows reaction terms grouped by organ system that were included in less than 1% of AEFI records.

Please see Table 4 for the description of other footnotes.

The most frequently reported adverse events were ISR, allergic reaction, fever, and malaise (1.1, 0.5, 0.5 and 0.4 per 100,000 doses, respectively). Reporting rates for each of these reactions were higher in the 18 to 64 year age group. There were 5 reports of GBS following influenza vaccination in 2007 giving a reporting rate of 0.11 per 100,000 doses. This is higher than in recent years, when only 1 or 2 reports were received annually,[11,13] but well within the expected reporting rates.

Pneumococcal vaccine and adults aged ≥65 years

There were 82 AEFI reports for older adults where 23vPPV was listed as suspected of involvement in the reported adverse event, with 6 reports coded as serious and 64 reports of ISR. Using the 2006 estimate of the number of doses of 23vPPV administered to people aged ≥65 years (n=429,500), the AEFI reporting rate was 18.6 per 100,000 doses, with 1.4 serious and 14.9 ISR reports per 100,000 doses. This is similar to the reporting rates estimated for 2006.[13]

Scheduled vaccines for children aged <7 years

There was a total of 526 AEFI records for vaccines administered in 2007 for children aged <7 years. Of these, 470 records listed as suspected one of the 10 vaccines for which ACIR data could be used to estimate AEFI reporting rates per 100,000 administered doses (Table 7). Vaccines for which reliable denominator data were not available included Bacille Calmette-Guèrin (n=4), influenza (n=30), 23vPPV (n=4), hepatitis A (n=5) and hepatitis B (n=7) (Table 3). Eighteen reports for rotavirus vaccine administered prior to the commencement of the national program on 1 July 2007 were also excluded from the assessment due to lower reliability of denominator data recorded on the ACIR for this time period.

The overall reporting rate for the 10 NIP vaccines was 12.7 per 100,000 administered doses, while the reporting rate for serious AEFI was 1.5 per 100,000 doses (Table 7). Reporting rates were similar to, or lower than, those in 2006 for most vaccine types including MenCCV, MMR and DTPa-containing vaccines (Table 7). The apparent increase in the reporting rates for Hib-HepB and 7vPCV vaccines may be related to reporting of AEFI for rotavirus vaccine as the vaccines are all given at 2 and 4 months of age.[15]

Reporting rates for the different DTPa-IPV combination vaccines varied by vaccine type and age group. The reporting rate for pentavalent vaccine is likely to be inaccurate due to the small number of reports and some under-reporting to the ACIR of doses administered. AEFI reports following quadrivalent DTPa-IPV include both children aged <1 year who were scheduled to receive the vaccine at 2, 4, and 6 months of age (reporting rate of 24.6 per 100,000 doses) and the 2 to <7 year age group (reporting rate of 73 per 100,000 doses). The reporting rate of ISR following DTPa-IPV in this older age group was 63 per 100,000 doses compared with 70 per 100,000 doses in 2006 and 76–80 per 100,000 doses of DTPa vaccine over the 2002–2005 period.

The AEFI reporting rate for children aged <1 year was higher for quadrivalent DTPa-IPV compared with the hexavalent DTPa-IPV-HepB-Hib vaccine (24.6 vs 10.3 reports per 100,000 administered doses) (Table 7). Reporting rates among infants for most reaction categories were approximately 2 to 3 times

Table 6. Reporting rate of adverse events following immunisation (AEFI) per 100,000 doses of influenza vaccine,* 18 years and over, ADRS database, 2007

AEFI category†	Age group	AEFI records‡ (n)	Vaccine doses* (n)	Rate per 100,000 doses§		
				2007	2006	2005
Overall	≥18 years	109	4,746,900	2.3	1.9	2.1
	18 to 64 years	79	2,626,400	3.0	2.5	2.8
	≥65 years	30	2,120,500	1.4	1.1	1.2
Serious	≥18 years	14	4,746,900	0.29	0.19	0.37
	18 to 64 years	11	2,626,400	0.42	0.27	0.49
	≥65 years	3	2,120,500	0.14	0.09	0.27

* Number of administered doses of influenza vaccine estimated from the 2006 national survey (unpublished).

† AEFI category includes all records, and those defined as 'serious' where influenza vaccine was suspected of involvement in the reported adverse event. The definition of a 'serious' outcome is shown in the Methods section.

‡ Number of AEFI records in which influenza vaccine was 'suspected' and the vaccination was administered in 2007.

§ The estimated reporting rate of adverse events per 100,000 administered doses of influenza vaccine.

higher for DTPa-IPV, except for HHE which was 12-fold higher for DTPa-IPV (6.6 per 100,000 doses; 95% CI 4.3–9.6) compared with DTPa-IPV-HepB-Hib (0.5 per 100,000 doses; 95% CI 0.1–2.0). The differing reporting rates and surveillance practices by jurisdiction (Table 1) need to be borne in mind as higher reporting jurisdictions (South Australia and Victoria) use quadrivalent DTPa-IPV for this age group.

New National Immunisation Program schedule vaccines

Rotavirus vaccine

There were a total of 90 AEFI records for 2007 where a rotavirus vaccine was listed as a suspected vaccine (Table 3). Of these, 72 were for the period following the commencement of the national program in July 2007 (reporting rate of 33.2 per 100,000 doses; Table 7). As expected, the majority (71%) of the 90 rotavirus vaccine AEFI reports also listed other vaccines as suspected of involvement in the reported adverse event, as most infants now receive rotavirus vaccine at the same time as other scheduled vaccines at 2, 4 and 6 months of age. Six per cent of the 90 rotavirus AEFI records had a certain or probable causality rating and 21% described events that met the definition of 'serious'.

The most commonly reported AEFI were vomiting/diarrhoea (n=37; 41%), abnormal crying (n=19; 21%) and other gastrointestinal events (n=16; 18%)

Table 7. Reporting rates of adverse events following immunisation (AEFI) per 100,000 vaccine doses,* children aged less than 7 years, ADRS database, 2007

	AEFI records‡ (n)	Vaccine doses* (n)	Reporting rate per 100,000 doses§ 2007	2006	2005
Vaccine†					
DTPa-containing vaccines	334	1,064,713	31.4	32.3	34.8
DTPa-IPV	287	669,451	42.9	43.0	–
Pentavalent (DTPa-IPV-HepB)	8	17,862	44.8	37.4	–
Hexavalent (DTPa-IPV-HepB-Hib)	39	377,400	10.3	12.9	–
Haemophilus influenzae type b	17	111,389	15.3	22.1	17.8
Haemophilus influenzae type b-hepatitis B	118	422,838	27.9	24.8	18.9
Measles-mumps-rubella	118	527,082	22.4	24.4	29.0
Meningococcal C conjugate	30	282,527	10.6	18.4	17.7
Pneumococcal conjugate	158	825,018	19.2	15.8	15.1
Rotavirus vaccine¶	72	219,791	33.2	–	–
Varicella	28	251,766	11.1	18.5	–
Age group					
<1 year	195	1,790,663	9.0	8.6	6.6
1 to <2 years	56	990,723	5.9	9.3	7.7
2 to <7 years	219	488,695	38.3	39.5	32.0
AEFI category†					
Total	470	3,702,124	12.7	13.9	12.0
'Certain' or 'probable' causality rating	150	3,702,124	4.1	5.4	5.3
'Serious' outcome	53	3,702,124	1.48	1.35	0.76

* Number of vaccine doses recorded on the Australian Childhood Immunisation Register (ACIR) and administered between 1 January and 31 December 2007.

† Records where at least one of the 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.6 A 'serious' outcome is defined as recovery with sequelae, hospitalisation, life-threatening event or death.[6]

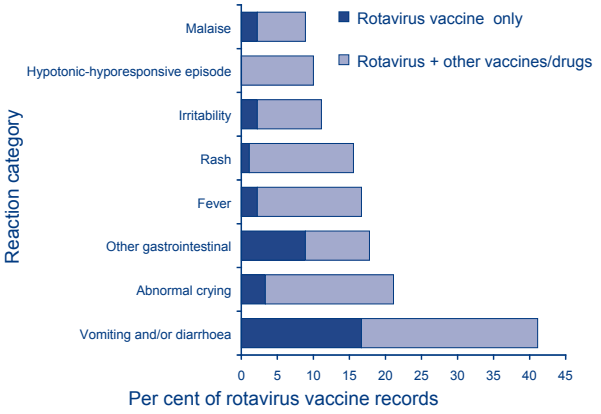
‡ Number of AEFI records in which the vaccine was coded as 'suspected' of involvement in the reported adverse event and the vaccination was administered between 1 January and 31 December 2007. More than 1 vaccine may be coded as 'suspected' if several were administered at the same time.

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

¶ Rotavirus vaccine AEFI reporting rate estimated for July–December 2007 only, the period where the vaccine was included in the funded National Immunisation Program schedule.

(Figure 6). Other gastrointestinal events included 3 reports of intussusception (reporting rate of 1.4 per 100,000 administered doses). The intervals between receipt of rotavirus vaccine and onset for the 3 cases of intussusception were 6, 16 and 31 days.

Figure 6. Most frequently reported adverse events following rotavirus immunisation,* ADRS database, 2007, by number of vaccines suspected of involvement in the reported adverse event



* Per cent of 90 adverse events following immunisation (AEFI) records where rotavirus vaccine was listed as suspected of involvement in the reported AEFI.

Human papillomavirus vaccine

A total of 705 AEFI reports were received for 2007 where HPV vaccine was the suspected vaccine. The age range was 11 to 31 years with a median of 16 years. HPV vaccine was the only suspected vaccine in 674 (96%) records, 203 (29%) had causality ratings of 'certain' or 'probable' and 43 (6%) were defined as 'serious' (Table 3). No deaths were reported.

The most frequently reported categories of reactions associated with HPV administration are shown in Figure 7. They included non-anaphylactic allergic reactions (23%; n=161), ISR (20%), headache (19%), nausea (16%), dizziness (14%) and malaise (13%).

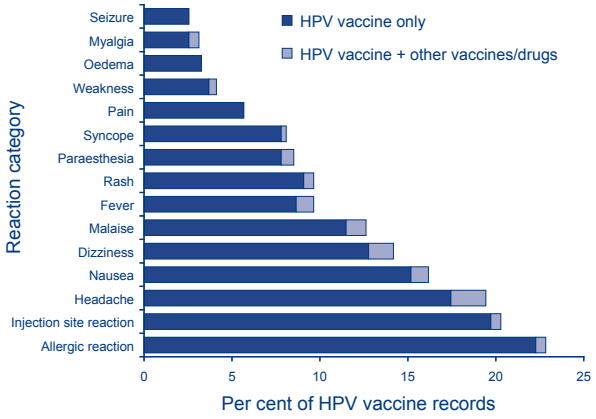
There were a total of 11 reports of anaphylactic reaction (in 2 cases, dTpa had also been administered)[24] and 18 reports of convulsion, mainly associated with syncope. The 2 cases initially reported as GBS were subsequently assigned alternate or uncertain diagnostic labels and, in 1 case, *Mycoplasma* infection was identified as the probable antecedent.

More recent information about AEFI and HPV vaccine reported up to June 2008 can be obtained from the TGA website.[27]

Discussion

As in previous years, the majority of AEFI reported to the TGA in 2007 were mild, transient and well-recognised vaccine side-effects. There was a large increase in the number of AEFI reports received for 2007 compared with recent years, mainly related to the commencement of the national HPV vaccine immunisation program in April 2007 for women aged 12 to 26 years. Other factors that may have contributed to the increase in reporting include the commencement of the national rotavirus immunisation program on 1 July 2007 and enhanced AEFI surveillance in the state of Victoria from April 2007.[23] Although the number of AEFI reports increased substantially, the proportion defined as serious remained stable at around 9%.

Figure 7. Most frequently reported adverse events following HPV immunisation,* ADRS database, 2007, by number of vaccines suspected of involvement in the reported adverse event



* Per cent of 705 AEFI records where human papillomavirus (HPV) vaccine was listed as suspected of involvement in the reported AEFI

There was a higher than expected number of anaphylactic reactions following HPV vaccine detected in New South Wales.[24,27] An expert multidisciplinary panel was convened by NSW Health to investigate all reports of anaphylaxis and severe allergic reaction following HPV vaccine. The panel found that the rate of anaphylaxis in New South Wales was significantly higher for the school-delivered HPV vaccination program compared with the 2003 school-delivered MenCCV program.[24] However, the overall rate was low, and all cases were managed appropriately without serious sequelae.[24,27,28] The results of the study were shared nationally and internationally. It

is recommended that vaccine recipients be observed for 15 minutes following administration of HPV vaccine[4,15,24,28] and that any symptoms and/or signs that may suggest anaphylaxis are clearly documented to allow an accurate assessment of the AEFI report using Brighton Collaboration case definitions.[18]

The majority of the 705 AEFI reports for HPV vaccine during 2007 were mild vaccine side-effects that had been identified in pre-licensure clinical trials.[25,26] These included mainly injection site reactions and milder allergic reactions. A range of non-specific symptoms were also reported, including headache, nausea, dizziness, malaise and weakness. (Table 5; Figure 7).[27,29] These symptoms have previously been reported to the TGA for secondary school students following receipt of MenCCV as part of the national catch-up program in 2003 and 2004.[9,11] This constellation of symptoms, which are likely to be due to a conversion reaction, are known to be associated with the event of vaccination rather than any specific vaccine.[4] They are more commonly reported in settings such as schools where many people are being vaccinated at the same time and can lead to a mass psychogenic response.[29,30] Immunisation providers of mass campaigns in this age group need to be aware of this response and attempt to put measures in place to prevent these events from occurring.[29]

The rotavirus vaccines used in Australia (RotaTeq® and Rotarix®) underwent extensive pre-licensure clinical trials which involved over 140,000 infants in developed and developing countries.[4,31,32] The major reason for these larger than usual clinical trials related to an apparent association between intussusception within 21 days of receipt of a previously licensed rotavirus vaccine, RotaShield, which was licensed in the United States of America (USA) in 1998 and withdrawn soon afterwards.[33,34] In the pre-licensure clinical trials for both RotaTeq® and Rotarix®, there was no difference in the rate of intussusception among vaccine recipients and the placebo group, while vaccine recipients were found to have an increased risk of up to 3% for gastrointestinal symptoms, predominately diarrhoea and vomiting, within 1 week of vaccination.[31,32] The most commonly reported AEFI to the TGA following rotavirus vaccine was gastrointestinal symptoms, predominantly diarrhoea and vomiting (41%). Post-licensure analysis of USA passive and active AEFI surveillance data for RotaTeq® indicated no association with intussusception.[35] The overall passive reporting rate of intussusception in Australia of 1.4 per 100,000 administered doses is similar to the rate estimated for the US passive surveillance system of 1.3 per 100,000 administered doses (calculated from data presented in the published paper of 160 reports and administration of 75% of 9.1 million distributed doses).[35]

After excluding reports for HPV and rotavirus vaccines, the number and patterns of AEFI reported to the TGA in 2007 was generally similar to that seen in 2006,[13] both for the older age groups receiving influenza vaccine and 23vPPV, and among children aged <7 years for scheduled vaccines. The only substantive changes identified for children were a further reduction in reported ISRs among 4- and 5-year-old children receiving DTPa-containing vaccines, and a significantly higher reporting rate of HHE among infants receiving DTPa-IPV compared with DTPa-IPV-HepB-Hib vaccines.

Children born after 1 April 2002 were due to receive their 4th dose of acellular pertussis-containing vaccines at 4 years of age following the removal of the dose due at 18 months of age from the immunisation schedule in September 2003.[3] The rate of ISR following acellular pertussis-containing vaccines in the 2 to <7 year age group has declined from a consistent reporting rate of approximately 80 per 100,000 doses during the 4 years 2002–2005[11] to 70 per 100,000 in 2006 and 63 per 100,000 in 2007. This suggests that the removal of the dose due at 18 months of age is having an impact on extensive limb swelling following receipt of a 4th versus 5th scheduled dose of vaccine prior to school entry.[36,37] However, other surveillance and schedule-related factors may also be impacting on the observed reporting trends, including the change to DTPa-IPV quadrivalent vaccine in November 2005, increased reporting and awareness that usually follows the introduction of new vaccines, and commencement of enhanced AEFI surveillance in Victoria in April 2007.

The significantly higher reporting rate of HHE in children aged <1 year following DTPa-IPV versus DTPa-IPV-HepB-Hib is difficult to interpret due to confounding related to changes in surveillance practices, the higher overall AEFI reporting rates from the South Australia and Victoria, and in the application of the Brighton Collaboration case definition for HHE by the TGA.[19] There were 2 reports of HHE following hexavalent DTPa-IPV-HepB-Hib in both 2006 and 2007 (from New South Wales and Western Australia) compared with 13 reports following DTPa-IPV in 2006 and 27 in 2007. The increase in 2007 was predominantly from Victoria and Queensland (16 Victoria, 6 South Australia and 5 Queensland in 2007 vs 8 Victoria, 4 South Australia and 1 Queensland in 2006). While most other jurisdictions use the hexavalent DTPa-IPV-HepB-Hib vaccine at 2, 4 and 6 months of age, these 3 states use quadrivalent DTPa-IPV vaccine at 2, 4 and 6 months plus Hib-HepB vaccine at 2 and 4 months. In 2007, Queensland changed the formulation of DTPa-IPV vaccine to the same as that used in Victoria and South Australia. Taken

together, the available information suggests that the higher HHE reporting rate following quadrivalent DTPa-IPV vaccine and Hib-HepB might be related to differences in surveillance practices (including enhanced clinical referral and assessment processes in Victoria from April 2007), as well as a true difference in HHE rates. Differences in reporting of HHE by vaccine type will continue to be monitored. A recent study in The Netherlands identified reporting rates of HHE through enhanced passive surveillance mechanisms to be up to 10 times higher than that identified from TGA data.[38]

Conclusion

The benefits of immunisation in reducing morbidity and mortality due to vaccine preventable diseases outweigh the risks of immunisation-related adverse reactions in Australia. Disease notification data show the impact of immunisation on reducing the number of cases of many severe infections,[39,40] including significant impacts on the incidence of both invasive meningococcal disease and invasive pneumococcal disease following the introduction of these national immunisation programs in 2003 and 2005.

While under-reporting is a known disadvantage of passive surveillance systems,[1,2,16,22] the Australian national AEFI passive surveillance system is sufficiently sensitive to detect expected changes in AEFI reporting associated with changes in immunisation programs, such as higher apparent reporting of anaphylaxis following receipt of HPV vaccine. Processes are in place to investigate signals and monitor trends in AEFI reporting.[24,27] The regular analysis and publication of national AEFI surveillance data collated in the ADRAC database remains an important aspect of Australia's immunisation programs. The next report will present AEFI data for children <7 years of age for vaccines administered in the first 6 months of 2008.

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Abbreviations of vaccine types

7vPCV	7-valent pneumococcal conjugate vaccine
23vPPV	23-valent pneumococcal polysaccharide vaccine
BCG	Bacille Calmette-Guèrin (i.e. tuberculosis)
dT	diphtheria-tetanus – adolescent and adult formulation
DTPa	diphtheria-tetanus-pertussis (acellular) – paediatric formulation
dTpa	diphtheria-tetanus-pertussis (acellular) – adolescent and adult formulation
dTpa-IPV	combined dTpa and inactivated poliovirus
DTPa-HepB	combined diphtheria-tetanus-pertussis (acellular) and hepatitis B
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
HPV	human papillomavirus
IPV	inactivated poliovirus vaccine
Men4PV	meningococcal polysaccharide tetravalent vaccine
MenCCV	meningococcal C conjugate vaccine
MMR	measles-mumps-rubella
RRV	rotavirus vaccine

Trachoma surveillance annual report, 2007 A report by the National Trachoma Surveillance and Reporting Unit

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Abstract

Trachoma screening was conducted in 2007 in trachoma-endemic regions and communities in the Northern Territory, South Australia and Western Australia. Aboriginal children aged 1 to 9 years were examined using the World Health Organization grading criteria. Screening in the Northern Territory was conducted by the primary health staff from the Healthy School Age Kids program, the Australian Government Emergency Intervention and Aboriginal Community Controlled Health Services with 60 of the 117 communities screened in 5 regions (1,703 children). In South Australia, the Eye Health and Chronic Disease Specialist Support Program and a team of eye specialists screened eight out of 91 communities in areas serviced by 5 Aboriginal Controlled Health Services (128 children). In Western Australia, population health unit and primary health care staff screened 62 out of 167 communities in 4 regions (1,666 children). Active trachoma prevalence rates varied between the regions with reported prevalence ranging from 5%–26% in the Northern Territory, 0%–21% in South Australia and 4%–22% in Western Australia. Comparisons of 2006 and 2007 regional active trachoma prevalence showed no consistent pattern in changes. Only a small amount of data were reported for the surgery and environmental improvement components of the World Health Organization recommended trachoma control activities of surgery (for trichiasis), antibiotic treatment (with azithromycin), facial cleanliness and environmental improvement. Reporting for the antibiotic treatment and facial cleanliness components has improved since 2006; however, many gaps still exist. A method to monitor bacterial resistance to azithromycin has been implemented. Baseline data collected by pathology services found similar results to national data collected by the Advisory Group on Antibiotic Resistance. *Commun Dis Intell* 2008;32:388–399.

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

Introduction

Trachoma is the most common infectious cause of blindness worldwide.[1] It is caused by specific strains of the bacteria *Chlamydia trachomatis* that in time leads to scarring of the eyelid, inturned eyelashes (trichiasis) and blindness.[2] Trachoma occurs predominantly in developing countries where living conditions are crowded and hygiene is poor.[3] Australia is the only developed country where trachoma still exists.[2]

In its resolve to eliminate blinding trachoma by 2020, the World Health Organization (WHO) recommends the adoption of a 4 component strategy: surgery (for trichiasis), antibiotic treatment (with azithromycin), facial cleanliness and environmental improvement (SAFE).[4] Based on the SAFE strategy, the Communicable Diseases Network Australia (CDNA) in 2006 developed the *Guidelines for the Public Health Management of Trachoma in Australia*.[2]

In 2006 the Australian Government awarded the tender to establish the National Trachoma Surveillance and Reporting Unit (NTSRU) to the Centre for Eye Research Australia (CERA). The NTSRU is responsible for providing high quality national information on trachoma prevalence based on data received from state and territory jurisdictions.

Screening was conducted at remote Aboriginal communities during 2007 in trachoma-endemic regions in the Northern Territory, South Australia and Western Australia. Data from communities and regions were reported to the NTSRU. This current report compares 2007 data with results from the screening in 2006. It comments on the jurisdictions' implementation of the CDNA guidelines 'minimum best-practice approach' and makes recommendations regarding future reporting.[2]

Methods

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

Trachoma at ≥10% is considered to be endemic hence the use of this threshold.[2]

A detailed account of the methods used has been documented in the 2007 surveillance report.[6]

In brief, in 2007, screening was conducted once in regions of the Northern Territory and Western Australia, and twice in three of the 5 Aboriginal Community Controlled Health Services (ACCHS) in South Australia. Data were reported for active trachoma prevalence, antibiotic treatment of children and household and community contacts, facial cleanliness, trachomatous trichiasis (TT), surgery for trichiasis, and trachoma control activities.

A method to assess the bacterial resistance to azithromycin has been implemented and baseline data have been collected (Annex: Antibiotic resistance).

Northern Territory

Most of the screening for trachoma was conducted between March and October 2007 by the Healthy School Age Kids (HSAK) program in the 5 regions where active trachoma is believed to be present (Map 1). Primary health care staff from the Maternal, Child and Youth Health program of the Department of Health and Families conducted screening in partnership with community health centres and the ACCHS.

In July 2007, the Australian Government Emergency Intervention (AGEI) conducted Child Health Checks in the Northern Territory. A decision was made by the AGEI clinical advisory panel that trachoma screening was only to be conducted where members of the intervention teams had appropriate skills and training to do so. Communities that were visited by the AGEI were not revisited by the HSAK program and this contributed to the smaller number of communities and children that were screened in 2007.

South Australia

Screening for trachoma was conducted twice in 2007, from February to July and again from July to December. Two ACCHS were visited only once in 2007. Data for a 6th ACCHS were reported in 2006 but were not reported in 2007 due to another program providing services in this area. Screening was undertaken by the project coordinator of the Eye Health and Chronic Disease Specialist Support Program and a team of ophthalmologists and optometrists in areas serviced by 5 ACCHS (Map 2). Data for 2 ACCHS were reported together in 2006. Similarly data for some communities were combined or pooled in 2006. In 2007 data for all ACCHS and communities were reported separately making comparisons difficult.

Some Aboriginal children who were identified for screening were seen in schools, while others were brought to the clinics by family members, Aboriginal health workers and other clinic staff.

Western Australia

Screening for trachoma was conducted between August and September 2007 in 4 population health regions where active trachoma is believed to be present (Map 3). Population health units collected data in partnership with primary health care staff from state government and ACCHS.

In 2007, 6 communities from the Goldfields region reported as 3 pairs; results for trachoma prevalence, clean faces and treatment counted each pair as 1 community.

Data analysis and reporting

A community was defined as an area which has a school. The denominator for the number of communities within each region or area serviced by an ACCHS was derived from school lists from each state and territory department of education.[7–9] For South Australia, schools in areas serviced by the Nganampa, Oak Valley and Tullawon ACCHS were grouped together by the NTSRU to match the reporting of school district categories used by the Department of Education. Key representatives from each state and territory nominated those communities that were believed not to have trachoma, those that had been screened, and those that may have trachoma and so should have been screened but had not.

Community coverage was calculated using the number of communities that were screened as a proportion of those that were identified by each state or territory to 'possibly have trachoma'. Communities reported as 'believed not to have trachoma' and those that reported zero prevalence in both 2006 and 2007 were not included in this calculation.

Australian Bureau of Statistics (ABS) 2006 Census data regarding the number of Aboriginal people residing in a region or enrolled in pre– and primary schools, were used to calculate 2007 high and low series population growth projections.[10,11]

Screening coverage was calculated using the number of children who were examined for trachoma as a proportion of those who were reported to be currently in the community/school by the population health units. Where the reported number of children in the

community was not provided (Northern Territory and South Australia), the ABS school enrolment 2007 projections were used. The screening coverage for Oak Valley and Tullawon was combined for 2007 data because data for these ACCHS were reported together in 2006.

The prevalence includes active trachoma detected by trachoma screening programs and in some instances detected through other sources such as clinics and other health checks. Thus, the reported prevalence may not truly reflect the population prevalence. Regional prevalence figures of active trachoma are reported on maps of each state and territory (Maps 1–3). In South Australia the prevalence of active trachoma is based on the first round of screening.

Chi square tests were used to measure and compare prevalences/proportions of active trachoma for communities that examined 10 or more children in both 2006 and 2007. Where numbers were less than five in any cell, a Fishers exact test was used. Statistical comparisons for the Pilbara region could not be made because in 2006 follicular trachoma was not graded according to the WHO grading system. Comparisons between each state and territory need to be interpreted with caution because of the variation in data collection and reporting.

Results

National perspective

Community coverage between 2006 and 2007 varied between each state and territory with higher coverage in Western Australia and consistently low coverage in South Australia (Tables 1 and 2).

A comparison between 2006 and 2007 regional prevalence data found a statistically significant decrease in prevalence in 4 regions and a statistically significant increase in 1 region (Table 2). Many communities from each state or territory still reported active trachoma prevalence ≥10% (Table 3).

Data were reported for 103 of 165 communities for both 2006 and 2007. Data from 39 communities were reported in 2006 only and 23 in 2007 only. In 2006, data for some communities were combined, leaving 34 communities from which data were reported in 2006 only, of which 19 (56%) had an active trachoma prevalence ≥10%.

Of the 27,171 Aboriginal people aged 30 years or over residing in these jurisdictions, only 987 (4%) were examined for trichiasis, of which 17 (2%) were found to have trichiasis.

Information on the implementation of SAFE trachoma control activities was not reported for any communities in South Australia. Data on activities were reported for few communities from the Northern Territory and Western Australia; however the distribution of antibiotics was reported for most communities in Western Australia (Table 4).

Northern Territory

Of the 117 communities in the 5 trachoma-endemic regions, 92 (79%) were identified as possibly having trachoma, of which 47 (50%) were screened in 2007 (Table 1). Data were reported from the 47 communities and an additional 13 that were screened but were identified as believed not to have trachoma (Table 1 and Map 1).

Table 1. Screening in communities believed not to have trachoma and those that possibly have trachoma, 2007, by state or territory

	Number of communities			Total
	Northern Territory	South Australia	Western Australia	
Believed not to have trachoma				
Screened	13	0	2	15
Not screened	12	0	97	109
Subtotal	25	0	99	124
Possibly have trachoma				
Screened with no trachoma found	16	2	19	37
Screened with trachoma found	31	6	37	74
Reported screened but no data received	4	0	4	8
Not screened	41	83	8	132
Subtotal	92	91	68	251
Total*	117	91	167	375

* Based on the number of schools provided by the Department of Education in the Northern Territory, South Australia and Western Australia.

Table 2. Community coverage, screening coverage and active trachoma prevalence of Aboriginal children aged 1 to 9 years, 2006 and 2007, by state and territory, region and Aboriginal Community Controlled Health Service

State and territory and region	2006 data						2007 data					
	Community coverage	%	Screening coverage	%	Active trachoma	%	Community coverage	%	Screening coverage	%	Active trachoma	%
Northern Territory												
Alice Springs Remote	23/31	74	530/1,382	38	94	18	15/31	48	231/1,402	16	46	20
Barkly	4/7	57	105/437	24	22	21	4/7	57	68/443	15	18	26
Darwin Rural*	15/25	60	522/1,407	37	84	16	11/25	44	377/1,427	26	25	7
East Arnhem†	7/8	88	879/1,187	73	22	3	7/8	88	465/1,204	39	23	5
Katherine*	11/22	50	218/1,344	16	65	30	10/22	45	562/1,363	41	104	19
Subtotal	60/93	65	2,254/5,757	39	287	13	47/93	51	1,703/5,839	29	216	13
South Australia (Screening 1)												
Ceduna/Koonibba	1/26	4	18/131	14	1	6	1/26	4	16/134	12	1	6
Nganampa	10/11	91	27/255	11	5	19	6/11	55	76/260	29	10	13
Oak Valley & Tullawon	‡		28/NA		7	25	‡		34 /NA		7	21
Pika Wiya	5/29	17	51/77	66	6	12	0/29	0	0/79	0	NS	
Umoona Tjutagku	1/25	4	6/49	12	1	17	1/25	4	2/50	4	0	0
Subtotal	17/91	19	130/512		20	15	8/91	9	128/523		18	14
Western Australia												
Goldfields*	6/14	43	231/873	26	43	19	10/14	71	227/1,047	22	8	4
Kimberley*	30/33	91	1,048/1,586	66	192	18	27/33	82	1,006/1,584	64	164	16
Midwest	6/6	100	167/981	17	32	19	5/6	83	127/201	63	28	22
Pilbara	9/15	60	273/935	29	146§	53	14/15	93	306/545	56	50§	16
Subtotal	51/68	75	1,719/4,375	39	413	24	56/68	82	1,666/3,377	49	250	15
Australia												
Total	128/252	51	4,103/10,644		720	18	111/252	44	3,497/9,739		484	14

NA Not available.

NS Not screened.

* p<0.05, † p<0.01 = statistical significance between 2006 and 2007 active trachoma prevalence

‡ Communities in areas serviced by these Aboriginal Community Controlled Health Services were reported with communities from the Nganampa Aboriginal Community Controlled Health Service.

§ Change in grading.

Of the 5,839 children reported by the ABS to be enrolled in schools, 1,703 (29%) were examined for trachoma and 216 of these had active trachoma (prevalence = 13%, 95% CI, 11%–15%) (Table 2). Twenty-nine of the 60 communities screened (48%) had no children with active trachoma; of those with active trachoma, 20 (33%) had a prevalence ≥10% (Table 3).

Data for facial cleanliness were reported for some communities (Table 5), and the use of resources or programs to promote clean faces was reported for few communities (Table 4). Four of the 31 communities (13%) that required treatment complied with the CDNA antibiotic treatment guidelines (Table 6).

Data on trichiasis were reported for the Katherine region only, but no cases were found. However, a community-based survey of trachoma was conducted in 5 communities in this region by an independent team from CERA and the Fred Hollows Foundation. Six people were found to have trichiasis and an additional person was reported to have undergone surgery.[12]

Table 3. Community prevalence of active trachoma in Aboriginal children aged 1 to 9 years, 2006 and 2007, by state or territory

Community prevalence	Number and percentage of communities where active trachoma data were reported						Total	
	Northern Territory		South Australia		Western Australia			
	n	%	n	%	n	%	n	%
2006 data								
0%	30	42	0		5	9	35	26
1 to <5%	7	10	0		3	6	10	8
5 to <10%	7	10	2	25	8	15	17	13
≥10%	28	39	6	75	37	70	71	53
Total	72	100	8	100	53	100	133	100
2007 data								
0%	29	48	2	25	20	36	51	41
1 to <5%	7	12	0		0		7	6
5 to <10%	4	7	2	25	5	9	11	9
≥10%	20	33	4	50	30	55	54	44
Total	60	100	8	100	55	100	123	100

Table 4. Number of communities where SAFE trachoma control activities were reported, 2007, by state or territory

SAFE trachoma control activities	Number and percentage of communities						Total N=124	
	Northern Territory N=60		South Australia N=8		Western Australia* N=56			
	n	%	n	%	n	%	n	%
Surgery	–		–		5	9	5	4
Antibiotics	7	12	–		44	78	51	41
Facial cleanliness resources	1	2	–		24	43	25	20
Facial cleanliness programs	5	8	–		21	38	26	21
Environmental improvement	1	2	–		6	11	7	6
Other	4	7	–		8	14	12	10

* Includes the paired communities from the Goldfields.

N Number of communities that reported trachoma screening data, including the community that provided treatment data only.

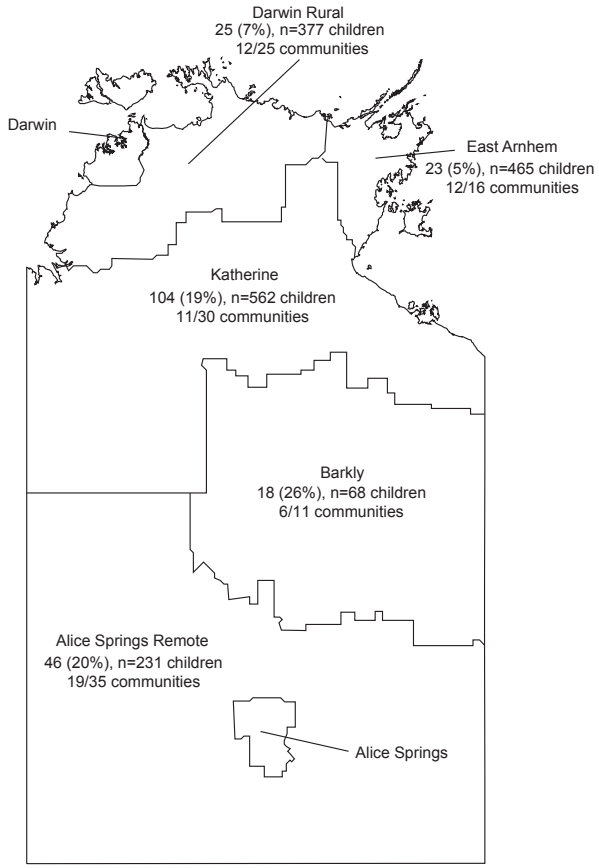
– Data not reported.

Of the 20 communities where sufficient children were examined to compare 2006 and 2007 trachoma data, prevalence was found to have increased significantly ($p<0.05$) in 6 communities and decreased significantly in four.

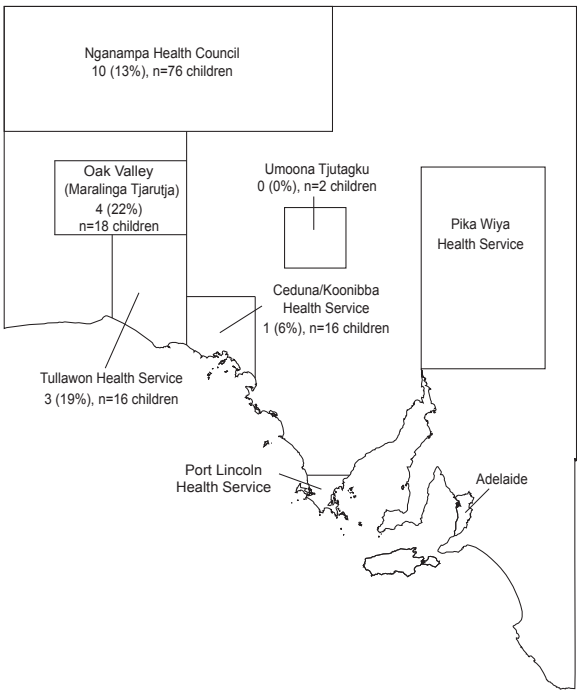
South Australia

Of the 91 communities in the 5 ACCHS, all were considered as possibly having trachoma, of which eight (9%) were screened in 2007 and reported data (Table 1 and Map 2). Data from 6 communities in

Map 1. Number of Aboriginal children with active trachoma (prevalence) aged 1 to 9 years, number examined and communities where trachoma data were reported, Northern Territory, 2007, by region



Map 2. Number of Aboriginal children with active trachoma (prevalence) aged 1 to 9 years and number examined, South Australia, 2007, by Aboriginal Community Controlled Health Service



Nganampa, Oak Valley and Tullawon = 6/11 communities

Ceduna/Koonibba = 1/26 communities (denominator also includes communities in Port Lincoln)

Umoona Tjutagku = 1/25 communities

Pika Wiya = 0/29 communities

Table 5. Number of resident Aboriginal children aged 1 to 9 years, those enrolled in schools, and communities and children examined for facial cleanliness, Northern Territory, 2007, by region

	Alice Springs remote	Barkly	Darwin rural	East Arnhem	Katherine	Total
Regional population (ABS)						
Resident children*	1,792	652	2,116	1,889	1,964	8,413
Children enrolled in schools†	1,402	443	1,427	1,204	1,363	5,839
Facial cleanliness						
Communities screened	13	6	9	4	2	34
Children examined	135	53	94	59	35	376
Prevalence of clean faces	49%	98%	91%	97%	100%	79%

* Projected 2007 population data for the whole region based on the Australian Bureau of Statistics 1.4% low series population growth rate in the Northern Territory.

Table 6. Number of communities with active trachoma and compliance with treatment according to Communicable Diseases Network Australia (CDNA) guidelines, Northern Territory, 2007, by region

Region	Number and percentage of communities					
	With active trachoma	%	Treated	%	Treated according to CDNA guidelines	%
Alice Springs Remote	9/19	47	5/9	56	3/9	33
Barkly	2/6	33	1/2	50	1/2	50
Darwin Rural	7/12	58	0/7	0	0/7	0
East Arnhem	5/12	42	1/5	20	0/5	0
Katherine	8/11	67	2/8	25	0/8	0
Total	31/60	52	9/31	29	4/31	13

areas serviced by 3 ACCHS (Nganampa, Tullawon and Umoona Tjutagku) were reported from the second round of screening.

Of the 444 children reported by the ABS to be enrolled in schools from the ACCHS areas where screening was conducted, 128 (29%) were examined for trachoma during the 1st screening and 18 of these had active trachoma (prevalence = 14%, 95% CI, 8%–20%) (Table 2). Fifty-nine children (13%) were examined during the second screening with nine having active trachoma (prevalence=15%, 95% CI, 6%–24%). From the 1st screening, two of the 8 communities screened had no children with active trachoma. Of those with active trachoma, four (50%) had a prevalence ≥10% (Table 3). During the second screening two of the 6 communities had no children with active trachoma. Of those with active trachoma four (75%) had a prevalence ≥10%.

Data for facial cleanliness were reported for all communities (Table 7), but the use of resources or programs to promote clean faces was not reported for any communities (Table 4). Although all of the children who were found to have active trachoma were treated within 2 weeks of examination, no household or community contacts were treated in 2007, clearly not complying with the CDNA treatment guidelines.

Adults were examined for trichiasis when they were at the ACCHS clinics for a diabetes examination. Data were reported for 11 communities during the 1st screening and 10 during the second. Data were reported for trichiasis but not for trachoma screening for some communities. Overall, 329 Aboriginal people were examined for trichiasis during the 1st screening, and 277 during the second; no cases of trichiasis were reported.

No significant changes were found in the 3 communities where sufficient children were examined to compare 2006 and 2007 trachoma data.

Western Australia

Of the 167 communities in the 4 trachoma-endemic regions, 68 (41%) were identified as possibly having trachoma, of which 56 (82%) were screened in 2007 (Table 1). Data were reported for the 56 communities and an additional two that were screened but were identified as believed not to have trachoma (Table 1 and Map 3). Data for treatment but not for screening were reported for 1 community.

Map 3. Number of Aboriginal children with active trachoma (prevalence) aged 1 to 9 years, number examined and communities where trachoma data were reported, Western Australia, 2007, by region

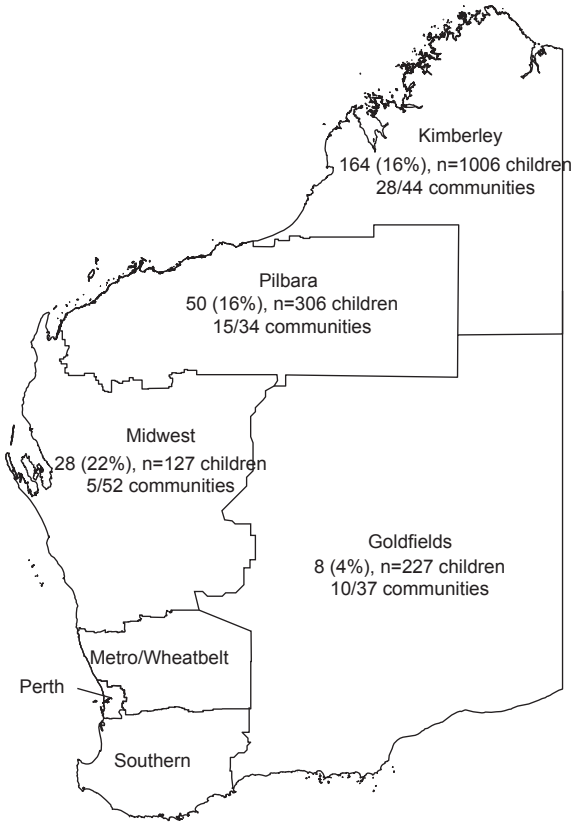


Table 7. Number of resident Aboriginal children aged 1 to 9 years, those enrolled in schools, and communities and children examined for facial cleanliness (Screening 1 and 2), South Australia, 2007, by Aboriginal Community Controlled Health Service

	Ceduna/ Koonibba	Nganampa	Oak Valley (Maralinga Tjarutja)	Pika Wiya	Tullawon	Umoona Tjutagku	Total
Regional population (ABS)							
Resident children*	165	349	NA	75	NA	76	665
Children enrolled in schools†	134	260	NA	79	NA	50	523
Facial cleanliness (Screening 1)							
Communities screened	1	4	1	0	1	1	8
Children examined	16	76	18	0	16	2	128
Prevalence of clean faces	100%	76%	100%		100%	100%	86%
Facial cleanliness (Screening 2)							
Communities screened	0	4	0	0	1	1	6
Children examined	0	34	0	0	23	2	59
Prevalence of clean faces		71%			100%	100%	83%

NA There were no data available from the Australian Bureau of Statistics for these locations because they had a very low population count.

* Projected 2007 population data for the whole region based on the Australian Bureau of Statistics 1.9% low series population growth rate in South Australia.

† Projected 2007 Australian Bureau of Statistics enrolment data for the whole region for pre– and primary school children based on the Australian Bureau of Statistics 1.9% low series population growth rate in South Australia.

In communities where screening was conducted, 1,666 (49%) of the 3,377 children believed to be attending school at the time of trachoma screening were examined for trachoma. Of these, 250 had active trachoma (prevalence = 15%, 95% CI, 13%–17%) (Table 2). Twenty of the 55 communities screened (36%) had no children with active trachoma. Of those with active trachoma 30 (55%) had a prevalence ≥10% (Table 3).

Data for facial cleanliness were reported for most communities (Table 8), and the use of resources and programs to promote clean faces was reported for many communities (Table 4). Eight of the 35 communities (23%) that required treatment complied with the CDNA treatment guidelines (Table 9).

Data on trichiasis were reported for the Goldfields region only. Adults were examined during an annual influenza vaccination program and no cases of trichiasis were found.

Table 8. Number of resident Aboriginal children aged 1 to 9 years, those enrolled in schools, and communities and children examined for facial cleanliness, Western Australia, 2007, by region

	Goldfields	Kimberley	Midwest	Pilbara	Total
Regional population					
Resident children*	1,163	2,824	1,218	1,178	6,383
Children enrolled in schools†	889	2,213	999	952	5,053
Facial cleanliness					
Communities screened	3	28	5	15	51
Children examined	104	1,006	127	306	1,543
Prevalence of clean faces	96%	81%	87%	78%	82%

* Projected 2007 population data for the whole region based on the Australian Bureau of Statistics 1.8% low series population growth rate in Western Australia.

† Projected 2007 Australian Bureau of Statistics enrolment data for the whole region for pre– and primary school children based on the Australian Bureau of Statistics 1.8% low series population growth rate in Western Australia.

Table 9. Number of communities with active trachoma and compliance to treatment according to the Communicable Diseases Network Australia guidelines, Western Australia, 2007, by region

Region	Number and percentage of communities					
	With active trachoma	%	Treated	%	Treated according to CDNA guidelines	%
Goldfields	3/7	43	3/3	100	1/3	33
Kimberley	19/28	68	17/19	89	4/19	21
Midwest	5/5	100	4/5	80	1/5	20
Pilbara	8/15	53	8/8	100	2/8	25
Total	35/55	64	32/35	91	8/35	23

CDNA Communicable Diseases Network Australia.

Of the 33 communities where sufficient children were examined to compare 2006 and 2007 trachoma data, prevalence was found to have increased significantly ($p<0.05$) in 1 community and decreased significantly in 3. For the Kimberley region, it was difficult to determine if there was a significant change due to missing data for the number of children examined in 2006. Although 2007 rates appeared to decrease in the Pilbara region this is almost certainly due to a change in the trachoma grading criterion used for screening in this region in 2007.

Discussion

Of the 375 communities in trachoma-endemic regions of Australia, 251 were identified as possibly having trachoma. Of these, 111 (44%) were screened in 2007. Screening was not conducted or not reported for the majority of communities (56%). A concerted effort to delineate which communities have trachoma and which do not is required before confident estimates can be made of the extent of trachoma in Australia.

Direct comparisons cannot be made between each state and territory because methods used in screening programs varied. For example, although in the Northern Territory 60 communities were screened, many of these communities had data for fewer than 10 children. Similarly, in South Australia, few communities were visited and, in those that were, few children were seen.

The screening coverage of children could not be calculated accurately as the number of children enrolled in school within a given region was not always provided. The coverage rate was 23% of the ABS estimate of the number of children resident in the area, or 31% of the ABS estimate of the number of children enrolled in schools.

Overall, of the 72 communities that were reported as having active trachoma, 47 (65%) were reported as giving antibiotic treatment. However very few (17%) complied fully with the CDNA guidelines. The distribution of antibiotics was lowest in the Northern Territory, however it is unclear whether this was due to a reporting issue or distribution issue or both. The data show a clear lapse in best practice adherence to the national guidelines by each state and territory.

Poor facial hygiene is an important risk factor for trachoma and the promotion of facial cleanliness is a key component of the SAFE strategy. Reporting of facial cleanliness data has improved since 2006. Regional means range between 45% and 100% of children having clean faces. However, the 2007 data still have many gaps. In the Northern Territory, data for only 34 out of 60 communities (57%) were reported to the NTSRU as it was considered a sensitive issue by some. Moreover, resources and programs for promoting facial cleanliness have not been reported for many communities. Such programs are important in order to integrate behavioural change regarding hygiene.

Only South Australia reported the systematic screening for trichiasis while the Northern Territory and Western Australia each provided data for 1 region only. Although seen relatively infrequently in communities, age specific prevalence rates of 5% to 10% are reported for some Aboriginal communities.[3,12] The routine screening and reporting of trichiasis in endemic areas needs to be strengthened. This is starting to occur for 2008 data collection, with more regions examining adults for trichiasis during an annual influenza vaccination program.

Of the 103 communities where data for trachoma were reported in both 2006 and 2007, 55 (53%) had examined sufficient children (≥10 examined) to make comparisons. Where comparison was possible, no consistent changes in prevalence were found as there were both increases and decreases.

It is apparent that the 4 components of the SAFE strategy trachoma control measures are not being implemented formally or comprehensively.

Each state and territory should identify all communities that are in need of screening for trachoma and aim to examine all children aged 1 to 9 years in these communities. The monitoring of trachoma can be successful only if meaningful and consistent data are collected with high rates of screening coverage (80+%) of all communities at risk of trachoma. Similarly, the lack of data regarding trichiasis and surgery for trichiasis provides an incomplete picture of what is happening at the end stages of this disease. This information is required before one could claim the elimination of blinding trachoma.

With collaboration and cooperation from each state and territory the NTSRU hopes to build a sustainable and effective monitoring system by which the elimination of trachoma can be documented.

Annex

Antibiotic resistance

Although *Chlamydia* remains sensitive to azithromycin, some studies have shown antibiotic resistance developing in other bacteria following community-based azithromycin treatment.[13,14] For these reasons, CDNA recommended that some monitoring of azithromycin resistance in other bacteria be conducted. The organism usually monitored for this purpose is *Streptococcus pneumoniae*. Resistance to azithromycin can be predicted by testing resistance to erythromycin and this is the recommended method.[15]

Data sources

The NTSRU contacted 3 pathology services to monitor macrolide resistance from specimens collected from Indigenous people:

- the Institute of Medical Veterinary Science (IMVS), South Australia;
- the Northern Territory Government Pathology Service (NTGPS); and
- the Western Diagnostics Pathology Service (WDPS), Northern Territory.

Following the IMVS requirements, the NTSRU obtained consent from 4 services that collected specimens from Indigenous people in South Australia and Central Australia: Ngaanyatjarra Health Service, Nganampa Health Council, Pika Wiya Health Service and the Royal Flying Doctors Service (South Australia). The NTGPS reported specimens collected from outpatients or those in the emergency room of the Alice Springs hospital.

Information on Indigenous status was only reported from the NTGPS as it is not routinely collected by the other 2 pathology services. IMVS and WDPS collected data for specimens from those regions or health services that serve predominantly Aboriginal people.

Sampling framework

The participating laboratories and health services reported erythromycin resistance (defined as both intermediate and high level resistance) for any invasive and non-invasive *S. pneumoniae* isolates collected from all specimen sites within the specified 3 month period (1 July to 30 September). Western Diagnostics laboratories collected data from 1 October to 31 December in 2007.

Data on patients' age, gender, region of residence, and specimen source were reported by each pathology service when available. Isolates were de-identified for personal and community data therefore regional information is reported in the tables.

Data analysis

Each participating laboratory performed antimicrobial susceptibility tests according to their routine standardised methodology (calibrated dichotomous sensitivity test, Clinical and Laboratory Standards Institute, agar dilution or minimum inhibitory concentration testing methods are identified in other sources).[15,16]

Results

Overall, 17 of 62 isolates (27%) were reported to be resistant or have intermediate resistance (Table 10). The numbers were too small to explore any regional variation in susceptibility rates.

Discussion

In a 3 month period only a small number of specimens were able to be identified as being from Aboriginal people or communities, however, a 6 month period will be used for 2008.

As part of the NTSRU monitoring of treatment of Aboriginal people with azithromycin in endemic areas, few data were reported in 2006 and the timing of administration of antibiotics was not specified as this was not a requirement of the 2006 report. No data were reported from the Northern Territory but 36 were reported to be treated in South Australia and 305 were reported to be treated in Western Australia. Reporting of treatment in 2007, when the antibiotic resistance data were collected, revealed that 328 people were reported to be treated in the Northern Territory from March to October, 18 in

Table 10. Erythromycin susceptibility of *Streptococcus pneumoniae* isolates, 2007, by pathology service

Pathology service/ region	Number and percentage of isolates						Total	%
	Resistant	%	Intermediate	%	Susceptible	%		
Institute of Medical Veterinary Science								
Nganampa	5	50	0		5	50	10	100
Ngaanyatjarra	0		0		2	100	2	100
Pika Wiya	0		0		1	100	1	100
Subtotal	5	38	0		8	62	13	100
Northern Territory Government Pathology Service								
Alice Springs	1	17	1	17	4	66	6	100
Alice Springs remote	3	27	0		8	73	1	100
Barkly	0		0		2	100	2	100
Darwin	0		0		1	100	1	100
Nganampa	0		1	50	1	50	2	100
Subtotal	4	18	2	9	16	73	22	100
Western Diagnostics Pathology Service								
Alice Springs	0		0		1	100	1	100
Alice Springs remote	1	33	0		2	67	3	100
Darwin	1	11	0		8	89	9	100
Darwin rural	2	29	0		5	71	7	100
East Arnhem	1	33	0		2	67	3	100
Katherine	1	25	0		3	75	4	100
Subtotal	6	22	0		21	78	27	100
Total	15	24	2	3	45	73	62	100

South Australia from February to July and 11 from July to December, and 1,675 in Western Australia between August and September.

The 2005 AGAR *S. pneumoniae* Survey reported antibiotic resistance to erythromycin in invasive and non-invasive isolates from 20 institutions around Australia. Laboratories collected up to 100 consecutive significant isolates starting from 1 January 2005.[17] South Australia reported 20.9% resistance in 392 isolates (12.3% in the 73 invasive strains and 22.9% in the 319 non-invasive strains). Western Australia reported 16.2% resistance in 296 isolates (11.1% in the 54 invasive strains and 17.4% in the 242 non-invasive strains). No data were reported for the Northern Territory. The 27% resistance (95% CI, 16%–39%) that was found in this study is comparable to the 22.7% resistance (95% CI, 20%–25%) reported by the AGAR survey.

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Data collection

The organisations that collected and/or reported data were:

Northern Territory

Aboriginal Community Controlled Health Services staff

Australian Government Emergency Intervention

Centre for Disease Control, Northern Territory Department of Health and Community Services

Healthy School Age Kids program: Top End and Central Australia

South Australia

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

Country Health South Australia

Ceduna/ Koonibba Health Service

Nganampa Health Council

Oak Valley (Maralinga Tjarutja) Health Service

Tullawon Health Service

Umoona Tjutagku Health Service

Western Australia

Aboriginal Community Controlled Health Services staff

Communicable Diseases Control Directorate, Western Australian Department of Health

Goldfields Population Health Unit

Kimberley Population Health Unit

Midwest Population Health Unit

Pilbara Regions Population Health Unit

Trachoma reference group

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

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

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

Communicable Disease Control Directorate, Western Australian Department of Health

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

National Aboriginal Community Controlled Health Organisation

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

Surveillance Branch, Office of Health Protection, Australian Government Department of Health and Ageing

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Monitoring the incidence and causes of diseases potentially transmitted by food in Australia: Annual report of the OzFoodNet Network, 2007

The OzFoodNet Working Group

Abstract

In 2007, OzFoodNet sites reported 27,332 notifications of 8 diseases or conditions that are commonly transmitted by food. The most frequently notified infections were *Campylobacter* (16,984 notifications) and *Salmonella* (9,484 notifications). Public health authorities provided complete serotype and phage type information on 96% of all *Salmonella* infections in 2007. The most common *Salmonella* serotype notified in Australia during 2007 was *Salmonella* Typhimurium, and the most common phage type was *Salmonella* Typhimurium 135. During 2007, OzFoodNet sites reported 1,882 outbreaks of gastrointestinal illness; the majority of these were spread person to person but included those transmitted by contaminated food. In total, these outbreaks affected 37,474 people and resulted in 1,034 people being admitted to hospital. During these outbreaks there were 114 deaths reported. Food was suspected or confirmed as the mode of transmission for 149 of these outbreaks, which affected 2,290 persons, hospitalised 266 persons and 5 deaths were reported during these outbreaks. For these foodborne outbreaks, *S.* Typhimurium was the most common aetiological agent and restaurants were the most common setting where foods were prepared. Twenty-four of these foodborne outbreaks were related to the consumption of eggs; the majority (n=22) of these outbreaks were due to various phage types of *S.* Typhimurium. This report summarises the incidence of disease potentially transmitted by food in Australia and details outbreaks associated with various food vehicles in 2007. These data assist agencies to identify emerging disease, develop food safety policies, and prevent foodborne illness. *Commun Dis Intell* 2008;32:400–424.

Keywords: foodborne disease, surveillance, disease outbreak

Introduction

Foodborne diseases are important globally because of their high incidence, their cost to society, and their potential to manifest as outbreaks.[1] In Australia, it has been estimated that there are 5.4 million cases of foodborne disease annually, costing an estimated $1.2 billion dollars per year.[2] Health departments conduct surveillance for foodborne diseases and diseases potentially transmitted by food to monitor trends in illness, detect outbreaks, and inform prevention efforts.[3] Surveillance data collected by health departments are an under estimate of the true burden of disease. Only a proportion of cases are reported to health departments and this is dependent on several factors including: health care seeking behaviour, stool testing practices differing between general practitioners, a patient submitting a stool specimen, the laboratory testing for the pathogen, and the result of testing being reported to public health authorities. Additionally, some pathogens are not notifiable to jurisdictional health departments.[4,5]

OzFoodNet, Australia's enhanced foodborne disease surveillance system, was established by the Australian Government Department of Health and Ageing in 2000 to improve national surveillance of gastrointestinal and foodborne illness.[6] OzFoodNet monitors the incidence of diseases caused by pathogens commonly transmitted by food through population-based, passive and enhanced surveillance for notifiable enteric diseases and for outbreaks of gastroenteritis and enteric diseases. This report summarises surveillance data for 2007 and compares them with data from previous years. The network includes collaborators from the National Centre for Epidemiology and Population Health at the Australian National University, the Public Health Laboratory Network, Food Standards Australia New Zealand, and the Department of Agriculture Fisheries and Forestry. OzFoodNet is a member of the Communicable Diseases Network Australia (CDNA), which is Australia's peak body for communicable disease control.[7]

Surveillance data are used primarily to monitor trends in the incidence of disease and to detect outbreaks. Surveillance data provide a historical perspective to assess changes in incidence and as such assist in the identification of outbreaks and clusters of disease. Long-term trends identified in surveillance data can also provide an indication of the success or otherwise of public health interventions.[8]

Methods

Population under surveillance

In 2007, the network covered the whole of the Australian population, which was estimated to be 21,015,042 persons.[9] All states and territories in Australia (New South Wales, Victoria, Queensland, South Australia, Western Australia, Tasmania, the Northern Territory, and the Australian Capital Territory) participated in OzFoodNet in 2007.

Data sources

Notified infections

All Australian states and territories have public health legislation that require doctors and/or pathology laboratories to notify cases of infectious diseases that are important to public health. In September 2007, the *National Health Security Act 2007 (National Health Security Act 2007, No. 174, 2007)* received royal assent. This Act provides a legislative basis for, and authorises the exchange of, health information, including personal information between Australian jurisdictions and the Commonwealth. The Act provides for the establishment of a National Notifiable Diseases List which specifies the diseases about which personal information can be provided. The National Health Security Agreement, 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. De-identified data on these diseases are provided via the Australian Government Department of Health and Ageing's National Notifiable Diseases Surveillance System (NNDSS). OzFoodNet aggregated and analysed data from NNDSS and enhanced surveillance data from OzFoodNet sites on the following 8 diseases or conditions, a proportion of which may be transmitted by food:

- non-typhoidal *Salmonella* infections;
- *Campylobacter* infections (except in New South Wales);
- *Listeria* infections;
- *Shigella* infections;
- *Salmonella* Typhi infection;
- Shiga toxin-producing *Escherichia coli* (STEC) infections;
- haemolytic uraemic syndrome (HUS); and
- botulism.

This report used a NNDSS dataset extracted in September 2008 and was analysed by the date of diagnosis to estimate disease activity within the reporting period 1 January to 31 December 2007. The diagnosis date is the onset date or where this was not known, the earliest date of specimen date, notification date or notification received date was used. Crude and category-specific rates of disease were calculated using the estimated resident populations for each state or territory as at June 2007. Age standardised rates were calculated only for those diseases with more than 100 cases overall using the direct method and the estimated 2007 Australian population as the reference population.[10]

Gastrointestinal and foodborne disease outbreaks

OzFoodNet collected summary information on gastrointestinal and foodborne disease outbreaks that occurred in Australia during 2007. An outbreak of foodborne disease is defined as two or more people with a particular infection or illness associated with a common food or meal. A cluster is defined as an increase in infections that are epidemiologically related in time, place or person where investigators were unable to implicate a vehicle or determine a mode of transmission.

For foodborne and suspected foodborne outbreaks, the summary information collected on each outbreak included the setting where the outbreak occurred, where the food was prepared, the month the outbreak occurred, the aetiological agent, the number of persons exposed and affected (including hospitalisations and deaths), the type of investigation conducted, the level of evidence obtained, and the food vehicle responsible for the outbreak. To summarise the data, outbreaks were categorised by aetiological agents, food vehicles and settings where the implicated food was prepared. Data on outbreaks due to waterborne transmission and data from clusters investigated by jurisdictional health departments were also summarised. The number of outbreaks and documented causes reported here may vary from summaries previously published by individual jurisdictions as these can take time to finalise.

Results

Rates of notified infections

In 2007, OzFoodNet sites reported 27,332 notifications of 8 diseases or conditions that are commonly transmitted by food (Table 1). This represents a 13% increase over the mean of 24,155 notifications per year for the previous 5 years (2002 to 2006).

Salmonella *infections*

In 2007, OzFoodNet sites reported 9,484 cases of *Salmonella* infection, a rate of 45 cases per 100,000 population. The 2007 rate was a 15% increase over the mean of the previous 5 years (Table 1). Notification rates ranged from 32 cases per 100,000 population in the Australian Capital Territory to 244 cases per 100,000 population in the Northern Territory, which

Table 1. Number of notified cases, crude rate and 5-year mean (2002–2006) rate per 100,000 population of diseases commonly transmitted by food, Australia, 2007, by disease and state or territory

Disease or aetiological agent		State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Salmonella	Notified cases, 2007	110	2,555	524	2,371	854	225	1,856	989	9,484
	Crude rate, 2007	32.4	37.1	243.8	56.7	53.9	45.6	35.7	47.0	45.1
	Mean rate, 2002–2006	30.1	30.6	187.2	66.6	34.3	38.3	26.2	36.2	39.0
Campylobacter	Notified cases, 2007	418	*	289	4,438	2,675	712	6,352	2,100	16,984
	Rate, 2007	123.0	*	134.5	106.1	168.9	144.3	122.0	99.7	120.2
	Mean rate, 2002–2006	119.7	*	120.4	103.7	152.1	132.7	115.4	105.4	115.5
Listeria	Notified cases, 2007	0	22	0	7	7	2	10	2	50
	Rate, 2007	0.0	0.3	0.0	0.3	0.4	0.4	0.1	0.1	0.2
	Mean rate, 2002–2006	0.4	0.4	0.1	0.2	0.2	0.3	0.3	0.5	0.3
Typhoid	Notified cases, 2007	0	34	3	6	5	3	30	9	90
	Rate, 2007	0.0	0.5	1.4	0.1	0.3	0.6	0.6	0.4	0.4
	Mean rate, 2002–2006	0.1	0.4	0.3	0.2	0.2	0.1	0.2	0.4	0.3
Shigella	Notified cases, 2007	0	71	173	88	62	3	96	104	597
	Rate, 2007	0.0	1.0	80.5	2.1	3.9	0.6	1.8	4.9	2.8
	Mean rate, 2002–2006	0.9	1.3	66.9	2.0	2.6	0.7	1.5	6.4	2.7
Shiga toxin-producing *E. coli*	Notified cases, 2007	1	23	3	24	41	0	13	2	107
	Rate, 2007	0.3	0.3	1.4	0.6	2.6	0.0	0.3	0.1	0.5
	Mean rate, 2002–2006	0.0	0.1	0.2	0.2	2.3	0.1	0.1	0.2	0.3
Haemolytic uraemic syndrome	Notified cases, 2007	1	13	0	1	1	0	3	0	19
	Rate, 2007	0.29	0.19	0.00	0.02	0.06	0.00	0.06	0.00	0.09
	Mean rate, 2002–2006	0.00	0.13	0.03	0.03	0.10	0.08	0.05	0.03	0.08
Botulism	Notified cases, 2007	0	0	0	0	0	0	1	0	1
	Rate, 2007	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	Mean rate, 2002–2006	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

* *Campylobacter* is not a notifiable disease in New South Wales.

usually has the highest rate of salmonellosis. For all jurisdictions, age-standardised rates were not different from crude rates. The exception was the Northern Territory where the age-standardised rate of salmonellosis was 222 cases per 100,000 population. Half (50%) of *Salmonella* notifications were in males. The highest age-specific rate of *Salmonella* infection was 202 cases per 100,000 population in children aged 0–4 years (Figure 1).

In 2007, the most commonly notified *Salmonella* serotype was *S.* Typhimurium. The most commonly notified phage type was *S.* Typhimurium 135, with 722 notifications in 2007 (Table 2). *S.* Typhimurium 9 was the second most common phage type notified in Australia in 2007; a large outbreak of *S.* Typhimurium 9 affecting more than 300 people associated with Vietnamese pork rolls in New South Wales in March where *S.* Typhimurium 9 isolated in more than 170 cases contributed to the large number of notifications of this phage type. Western Australia ceased routine phage typing of *S.* Typhimurium, *S.* Enteritidis and *S.* Virchow in July 2007; the top 5 *Salmonella* infections by serotype only are presented for Western Australia. (Table 3).

Figure 1. Notification rate of salmonellosis, Australia, 2007, by age group and sex

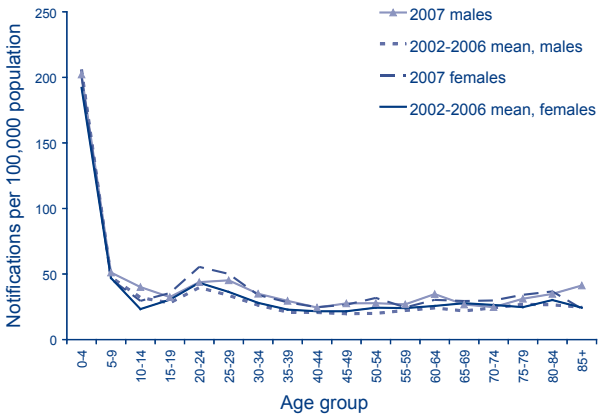


Table 2. Numbers, rates and proportions of the top 5 *Salmonella* infections, Australia (excluding Western Australia), 2006 to 2007, by OzFoodNet site*

OzFoodNet site	*Salmonella* sero/phage type	2007 n	2007 rate†	Proportion (%)‡	2006 n	2006 rate	2007/2006 ratio§
Australian Capital Territory	Muenchen	8	2.4	7.3	0	0.0	–
	Typhimurium 135	8	2.4	7.3	12	3.6	0.7
	Typhimurium U290	7	2.1	6.4	4	1.2	1.7
	Typhimurium 44	6	1.8	5.5	6	1.8	1.0
	Typhimurium 9	6	1.8	5.5	7	2.1	0.8
New South Wales	Typhimurium 9	362	5.3	14.2	82	1.2	4.5
	Typhimurium 135	231	3.4	9.0	206	3.0	1.1
	Typhimurium 170/108	137	2.0	5.4	221	3.2	0.6
	Birkenhead	105	1.5	4.1	109	1.6	1.0
	Typhimurium 44	86	1.3	3.4	42	0.6	2.1
Northern Territory	Oslo	48	22.3	9.2	2	1.0	23.2
	Ball	38	17.7	7.3	31	14.9	1.2
	Saintpaul	32	14.9	6.1	31	14.9	1.0
	Chester	20	9.3	3.8	17	8.2	1.1
	Subsp I Ser 16:L,V:–	20	9.3	3.8	13	6.3	1.5
	Infantis	20	9.3	3.8	15	7.2	1.3
	Anatum	17	7.9	3.2	10	4.8	1.6
Queensland	Saintpaul	219	5.2	9.2	271	6.6	0.8
	Virchow 8	183	4.4	7.7	212	5.1	0.9
	Typhimurium 135	155	3.7	6.5	171	4.2	0.9
	Aberdeen	121	2.9	5.1	136	3.3	0.9
	Birkenhead	116	2.8	4.9	152	3.7	0.8
South Australia	Typhimurium 9	122	7.7	14.3	58	3.6	2.1
	Typhimurium 29	75	4.7	8.8	6	0.4	12.6
	Typhimurium 135	64	4.0	7.5	79	5.0	0.8
	Typhimurium 44	51	3.2	6.0	17	1.1	3.0
	Typhimurium 170/108	44	2.8	5.2	58	3.6	0.8
Tasmania	Mississippi	118	23.9	52.4	67	13.4	1.8
	Typhimurium 135	43	8.7	19.1	39	7.8	1.1
	Typhimurium 170/108	5	1.0	2.2	14	2.8	0.4
	Subsp I Ser Rough :B:1,5	4	0.8	1.8	0	0.0	–
	Typhimurium 9	4	0.8	1.8	14	2.8	0.3
Victoria	Typhimurium 44	276	5.3	14.9	146	2.8	1.9
	Typhimurium 135	211	4.1	11.4	146	2.8	1.4
	Typhimurium 9	138	2.7	7.4	111	2.1	1.2
	Typhimurium 170/108	112	2.2	6.0	106	2.0	1.1
	Stanley	44	0.9	2.4	26	0.5	1.7
Australia (excluding Western Australia)	Typhimurium 135	722	3.4	8.5	673	3.6	1.0
	Typhimurium 9	674	3.2	7.9	335	1.8	1.8
	Typhimurium 44	460	2.2	5.4	245	1.3	1.7
	Typhimurium 170/108	337	1.6	4.0	470	2.5	0.7
	Saintpaul	329	1.6	3.9	511	2.7	0.6

* Where there were multiple 5th ranking *Salmonella* types all data have been shown; Western Australia data not included due to incomplete phage typing in 2007.

† Rate per 100,000 population.

‡ Proportion of total *Salmonella* notified for this jurisdiction in 2007.

§ Ratio of the rate in 2007 compared to the rate in 2006.

The highest specific rates for a single serotype other than *S.* Typhimurium were for *S.* Mississippi (24 cases per 100,000 population) in Tasmania and *S.* Oslo (22 cases per 100,000 population), *S.* Ball (18 cases per 100,000 population), and *S.* Saintpaul (15 cases per 100,000 population) in the Northern Territory.

Salmonella Enteritidis

Salmonella Enteritidis is a globally important *Salmonella* serotype that can infect the internal contents of eggs, but is not endemic in Australian egg layer flocks. To monitor the emergence of this strain in Australia, OzFoodNet conducts enhanced surveillance, including travel history, to detect outbreaks of locally-acquired *S.* Enteritidis. The majority of cases in Australia are associated with overseas travel.

During 2007, OzFoodNet sites reported 396 cases of *S.* Enteritidis infection (Table 4). Of those cases where travel status was reported, 92% (297/322) had travelled overseas and cases often reported visiting several countries. A travel history could not be obtained for 19% (74/396) of cases, compared to 24% (72/305) of cases in 2006 and 11% (44/387) of cases in 2005.

We compared the incidence of salmonellosis in returned travellers with the number of travellers to that region using customs data derived from incoming passenger cards. The field 'country where you spent the most time abroad' was used as the numerator. Of the cases that were known to have been acquired overseas, 66% (195/297) reported travel to South East Asia. This compares with only 29% of returning travellers coming from South East Asia in 2006.[11] The most common country of acquisition for overseas acquired cases was Indonesia, with 33% (98/297) of cases reporting travel there, while comprising only 2.2% of travel undertaken in 2006. Thailand was the second most common country of acquisition with 15% (44/297) of all notifications that were known to have been acquired overseas, followed by Singapore with 7% (20/297) and Malaysia with 6% (18/297). The most common infecting phage types amongst overseas-acquired cases were 6a (21%) and 1b (12%) (Table 5).

Table 3. Numbers, rates, and proportions of top 5 *Salmonella* infections, 2006 to 2007, Western Australia

OzFoodNet site	*Salmonella* serotype	2007 n	2007 rate*	Proportion (%)†	2006 n	2006 rate	2007/2006 ratio‡
Western Australia	Typhimurium	391	18.6	39.5	212	10.2	1.8
	Enteritidis	104	4.9	10.5	69	3.3	1.5
	Saintpaul	48	2.3	4.9	59	2.8	0.8
	Virchow	36	1.7	3.6	12	0.6	3.0
	Chester	26	1.2	2.6	27	1.3	1.0

* Rate per 100,000 population (of Western Australia)

† Proportion of total *Salmonella* notified for this jurisdiction in 2007.

‡ Ratio of the rate in 2007 compared to the rate in 2006.

Table 4. Number of *Salmonella* Enteritidis infections, Australia, 2007, by travel history and state or territory

State/territory	History of overseas travel			
	Yes	No	Unknown	Total
Australian Capital Territory	2	0	0	2
New South Wales	68	6	27	101
Northern Territory	8	1	1	10
Queensland	31	5	44	80
South Australia	22	1	1	24
Tasmania	3	0	0	3
Victoria	62	7	0	69
Western Australia	102	5	0	107
Total	298	25	73	396

Table 5. Number and percentage of each phage type for of overseas-acquired cases of *Salmonella* Enteritidis

Phage type	Number of cases	Percentage
6a	62	20.9
1b	37	12.5
1	24	8.1
4	17	5.7
Reaction does not conform (RDNC)	15	5.1
21	14	4.7
26	10	3.4
21var1	9	3.0
8	6	2.0
21c	6	2.0
Other phage types	33	11.1
No phage type was provided	64	22.5
Total	297	100

All states and territories except Tasmania and the Australian Capital Territory reported locally-acquired *S.* Enteritidis cases in 2007; 25 *S.* Enteritidis cases (6.3%) in 2007 were known to have acquired the infection locally, compared with an average of 48 cases per year between 2003 and 2006. The travel status of 44 cases from Queensland was unknown due to incomplete follow-up. A number of different phage types of *S.* Enteritidis were reported in locally-acquired cases, and no one phage type predominated, compared with 2003 to 2006, where *S.* Enteritidis 26 predominated. The number of locally acquired *S.* Enteritidis 26 in 2007 (n = 3) was lower than in previous years, where 15 to 34 cases of this phage type were reported per year between 2003 and 2006. This is likely due to incomplete follow-up of cases for travel history from Queensland during 2007 as the majority of locally-acquired *S.* Enteritidis cases (and particularly *S.* Enteritidis 26) are reported from Queensland each year.[12,13] Of the 44 *S.* Enteritidis cases from Queensland in 2007 where travel status was unknown, 22 (50%) were infected with *S.* Enteritidis 26.

Completeness of Salmonella serotyping and phage typing

Overall, 96% of *Salmonella* notifications on state and territory notification databases contained information about serotype and/or phage type. In Australia, 6 serotypes are routinely phage typed – Bovismorbificans, Enteritidis, Hadar, Heidelberg, Typhimurium and Virchow. Phage typing was greater than 90% complete for serotypes Typhimurium and Bovismorbificans (Table 6). All states and territories (with the exception of Western Australia which ceased routine phage typing in June 2007) had greater than 97% complete serotype and phage type information for all *Salmonella* notifications during 2007.

Campylobacter *infections*

In 2007, OzFoodNet sites (excluding New South Wales) reported 16,984 cases of *Campylobacter* infection; a rate of 120 cases per 100,000 population (Table 1). The lowest and highest rates of *Campylobacter* notification were in Western Australia (100 cases per 100,000 population) and in South Australia (169 cases per 100,000 population), respectively. Age-standardised rates were not different from crude rates for all jurisdictions except the Northern Territory (age standardised rate 131 cases per 100,000 population) and the Australian Capital Territory (age standardised rate 125 cases per 100,000 population). Fifty-five per cent of notified cases were male. The highest age-specific rate of notifications was in the 0–4 year age group for both males and females (239 cases per 100,000 population and 169 cases per 100,000 population, respectively) with additional peaks in the 20–29 year age group for both males and females and the 70–85+ year age group for males (Figure 2).

Table 6. Proportion of *Salmonella* infections for 6 serotypes notified to state and territory health departments with phage type information available, Australia, 2002 to 2007

Salmonella serotype	2002 %	2003 %	2004 %	2005 %	2006 %	2007 %	2007* %
S. Bovismorbificans	98.1	97.3	96.1	96.6	98.3	98.5	98.2
S. Enteritidis	96.5	98.3	95.8	97.6	98.3	79.2	96.1
S. Hadar	90.7	97.1	90.0	91.7	100.0	88.9	90.0
S. Heidelberg	93.3	96.4	94.7	97.6	98.4	86.5	86.3
S. Typhimurium	98.5	99.0	99.2	99.1	98.7	93.6	98.9
S. Virchow	97.6	98.3	97.1	96.8	98.6	89.7	94.9

* 2007 data excluding Western Australia have incomplete phage typing from June 2007.

Figure 2. Notification rate of campylobacteriosis, Australia, 2002 to 2007, by age group and sex

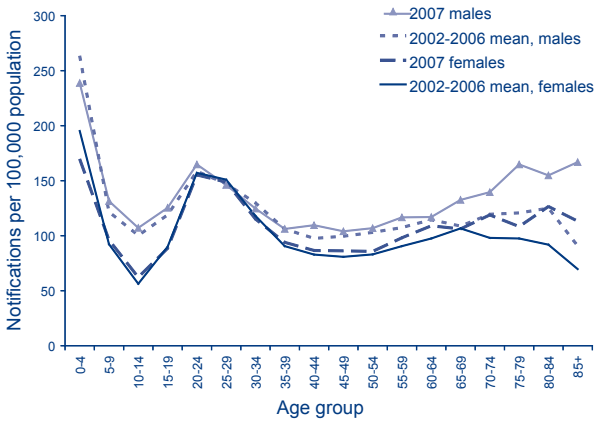
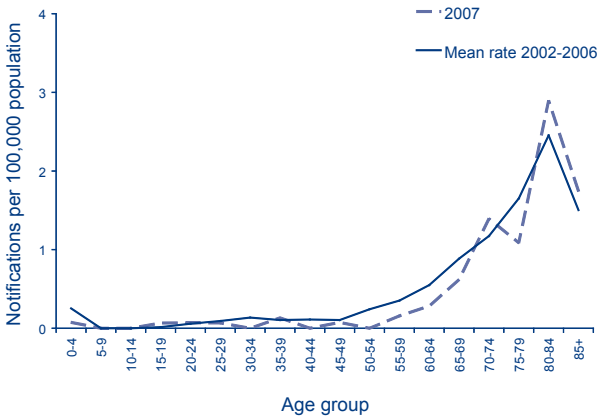


Figure 3. Notification rate of listeriosis, Australia, 2002 to 2007, by age group

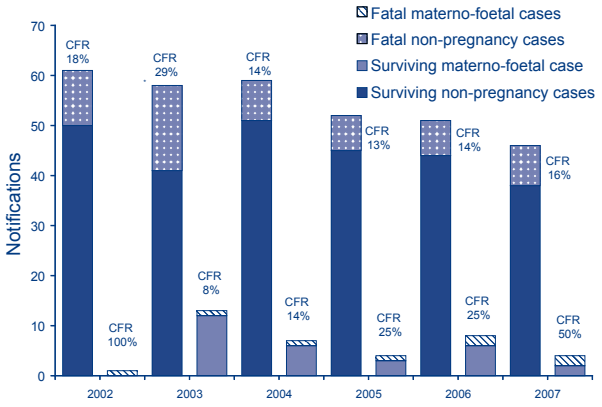


Listeria *infections*

OzFoodNet sites reported 50 cases of *Listeria monocytogenes* infection in 2007, a crude rate of 0.2 cases per 100,000 population. The 2007 notification rate was similar to the 5-year historical mean (0.3 cases per 100,000 population) (Table 1).

Seventy-six per cent (38/50) of notifications were in people aged 60 years or more. The highest age-specific notification rate was in 80–84 year-olds, with a notification rate of 2.9 cases per 100,000 population (Figure 3). Forty-eight per cent (22/46) of the non-pregnancy related cases, and 52% of all cases, were female. Only four of the 50 listeriosis cases notified in 2007 were pregnancy related, which is similar to previous years. Half (2/4) of pregnancy-related cases and 16% (8/38) of the non-pregnancy associated cases in 2007 were fatal (Figure 4).

Figure 4. Notifications of listeriosis showing non-pregnancy related cases and deaths, and materno-foetal infections and deaths, Australia, 2002 to 2007



* The case-fatality rate (CFR) is the percentage of cases that were fatal.

Shigella *infections*

In 2007, OzFoodNet sites reported 597 cases of shigellosis, with a crude rate of 2.8 cases per 100,000 population compared with a mean of 2.7 per 100,000 population between 2002 and 2006 (Table 1). As in previous years, the highest notification rate was in the Northern Territory (age standardised rate 79.8 cases per 100,000 population) (Table 1). The highest age-specific notification rates were amongst males and females aged 0–4 years, with age-specific rates of 11.6 and 11.7 notifications per 100,000 population. Females from 20 to 35 have higher age-specific rates than males of the same age. (Figure 5).

The most common biotypes in 2007 were *Shigella sonnei* biotype a (21%) and *Shigella sonnei* biotype g (16%). In 2007, these 2 biotypes increased in number

Figure 5. Notification rate of shigellosis, Australia, 2002 to 2007, by age group and sex

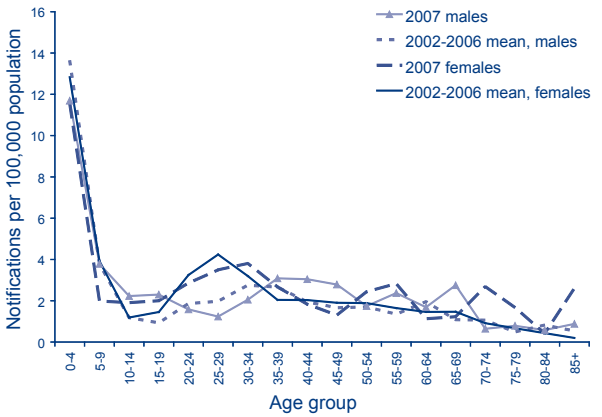


Table 7. Number, percentage and ratio of the top 5 *Shigella* infections, Australia, 2006 to 2007

	2007 n	Proportion* %	2006 n	Proportion %	2007/2006 ratio†
Shigella sonnei biotype a	128	21	80	15	1.6
Shigella sonnei biotype g	98	16	76	14	1.3
Shigella flexneri 4a mannitol negative	69	12	94	17	0.7
Shigella flexneri 2a	64	11	54	10	1.2
Shigella flexneri 4	47	8	83	15	0.6

* Proportion of total *Shigella* notified in 2007.

† Ratio of the number of reported cases in 2007 compared to the number reported in 2006.

and proportion of notified cases compared to 2006 (Table 7) In 2006 the most common biotype was *Shigella flexneri* 4a mannitol negative.[12]

Salmonella *Typhi infections*

OzFoodNet sites reported 90 cases of typhoid (*Salmonella* Typhi) infection during 2007; a crude rate of 0.4 cases per 100,000 population (Table 1) compared with a mean of 0.3 cases per 100,000 for the previous 5 years. Overseas travel is a significant risk factor for typhoid infection in Australia; in 2007, 92% (83/90) of cases reported overseas travel (Table 8).

More than half of all overseas-acquired cases reporting overseas travel had travelled to India (51%, 42/83). Bangladesh was the second most frequently reported country or region with 13% (11/83) of overseas-acquired cases reporting travel there. The predominant phage types isolated from cases returning from travel to India were E1 (19 cases) and E9 (9 cases), similarly in cases returning from travel to Bangladesh, the most common infecting phage type was E9 (4 cases) (Table 9).

The highest rates of typhoid notification were in people aged 20–24, with 0.8 cases per 100,000 and 25–29 with 1.1 cases per 100,000 population, compared with the overall notification rate of 0.4 per 100,000 population, which is likely to be due to high rates of overseas travel in this age-group.

Shiga toxin-producing Escherichia coli infections

In 2007, OzFoodNet sites reported 107 cases of Shiga toxin-producing *Escherichia coli* infections (STEC), a crude rate of 0.5 notifications per 100,000 population and an increase of 65% compared with an annual mean of 0.3 notifications per 100,000 population per year between 2002 and 2006 (Figure 6, Table 1).

South Australia reported 38% (41/107) of the STEC notifications, followed by Queensland (22%, 24/107), New South Wales (22%, 23/107), Victoria (12%, 13/107), the Northern Territory (2.8%, 3/107), Western Australia (2%, 2/107) and the Australian Capital Territory (1%, 1/107). The highest notification rates were in South Australia (2.6 cases per 100,000) and the Northern Territory (1.4 case per 100,000 population) (Table 1).

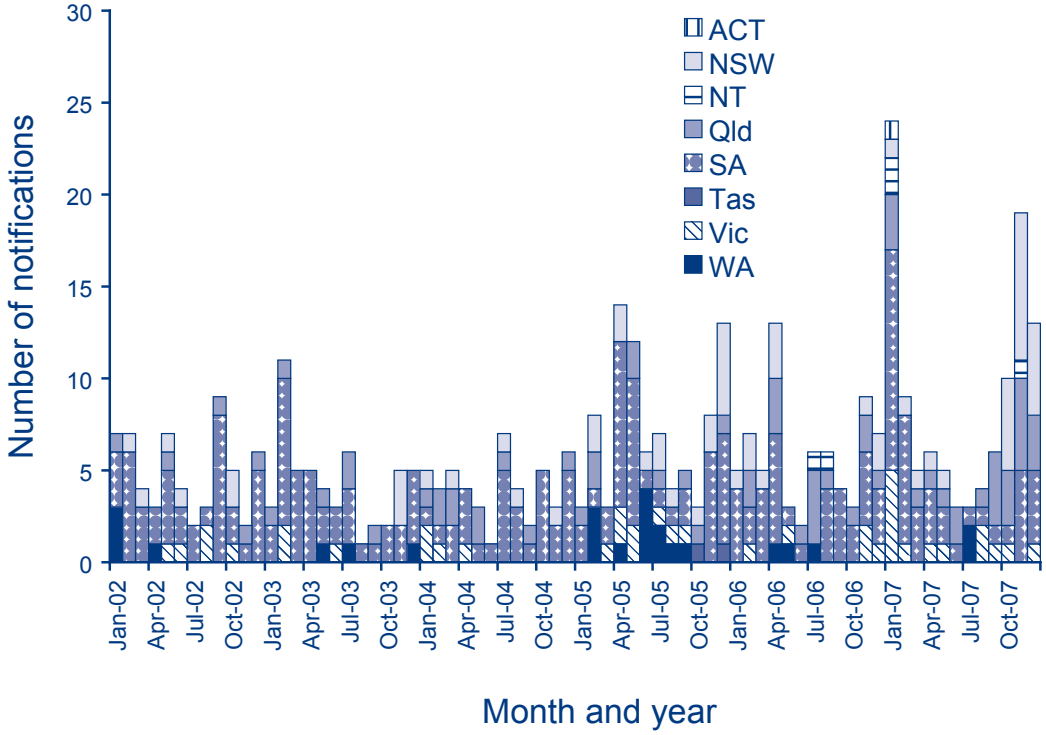
Table 8. Travel status for notified cases of typhoid, Australia, 2007

State or territory	History of overseas travel			
	Yes	No	Unknown	Total
Australian Capital Territory	0	0	0	0
New South Wales	32	1	1	34
Northern Territory	2	1	0	3
Queensland	4	1	1	6
South Australia	5	0	0	5
Tasmania	3	0	0	3
Victoria	30	0	0	30
Western Australia	7	2	0	9
Total	83	5	2	90

Table 9. *Salmonella* Typhi phage types isolated from cases (n=90), Australia, 2007, by place of acquisition

Place of acquisition	Number of cases	Phage types isolated from cases (n)
Africa (country not specified)	1	Degraded/ untypeable (1)
Bangladesh	11	E9 (4), untypeable/degraded (2), 59 (1), C2 (1), D2 (1), E1 (1), unknown (1),
Egypt	1	40 (1)
El Salvador	2	59 (1), untypeable/degraded (1)
India	41	E1 (19), E9 (8), untypeable/degraded (4), unknown (2), E1 (1), 28 (1), A (1), D1 (1), D2 (1), E2 (1), E3 (1), O variant (1)
India + Thailand	1	E9 (1)
Indonesia	9	E2 (4), D2 (2), untypeable/degraded (2), A degraded (1)
Liberia	2	D1 (2)
Morocco	1	C1 (1)
Nepal	2	Untypeable/degraded (2)
Pakistan	7	E1 (2), E9 (2), untypeable/degraded (2), D1 (1)
Papua New Guinea	2	D2 (2)
Samoa	2	E9 (2)
Thailand	1	Untypeable/degraded (1)
Unknown	2	E1 (2)
No overseas travel reported	5	E1 (3), D1 (1)

Figure 6. Number of notifications of Shiga toxin-producing *Escherichia coli*, 2002 to 2007, by date of diagnosis and state or territory, Australia



The highest age specific notification rate was amongst children aged 0–4 years (1.5 cases per 100,000 population), with peaks in older ages as well, with 1.0 cases per 100,000 amongst the 60–65 year age group and 0.8 notifications per 100,000 amongst the 70–74 year age group. During 2007, O157 was the most common serotype of STEC identified, with 38% (41/107) of cases due to this serotype, which was similar to previous years. The next most common serotypes reported were *E. coli* O111 and O26 (8 and

7 notifications respectively). In 36% (38/107) of cases, the organism either was not isolated or typed, or no information was provided.

Haemolytic uraemic syndrome

During 2007, OzFoodNet sites reported 19 cases of haemolytic uraemic syndrome (HUS); a rate of 0.09 cases per 100,000 population (Table 1) compared with a mean of 0.08 cases per 100,000 between 2002 and 2006. The majority of these were reported from New South Wales (13 cases). The median age of notifications was 6 years, with a range of 1–44 years. Similar to previous years, the highest notification rate was in children aged 0–4 years, with eight of the 19 notifications in this age group (0.6 notifications per 100,000 population).

In 2007, HUS notifications showed a seasonal pattern, similar to previous years, tending to increase during the warmer months, with 11 of 19 cases diagnosed in either January, November or December 2007 (Figure 7). There was little information available on the aetiological agents of HUS cases, with *E. coli* (unknown serotype) isolated from only 1 case. One case was known to have been due to infection with bacteria other than *E. coli* (*S. pneumoniae).*

Botulism

There was one case of botulism notified in 2007. In January, the Victorian Department of Human Services (DHS) was notified of a case of suspected botulism in a 25 year old male. The notifying clinician gave a history of onset of dizziness, lethargy, blurred vision and respiratory distress followed by a rapid decline which included respiratory failure requiring intubation and ventilation in ICU. A provisional diagnosis of stroke or multiple sclerosis was made but initial investigations were negative. The day following notification to DHS, the case became completely paralysed. A faecal enema specimen was forwarded to the University of Melbourne, Microbiological Diagnostic Unit for confirmation of the diagnosis. *Clostridium botulinum* toxin was detected in the faecal specimen, which was later identified as A2. An extensive investigation of a possible food source was conducted by DHS. At the time of the case notification, extensive case finding was conducted and no other cases were identified.

Gastrointestinal and foodborne disease outbreaks

During 2007, OzFoodNet sites reported 1,882 outbreaks of gastroenteritis, including foodborne disease, which affected 37,474 people. Associated with these outbreaks, 1,034 people were hospitalised and there were 114 deaths (Table 10). This compared with 1,544 and 624 outbreaks reported across Australia in 2006 and 2005, respectively. The number of outbreaks reported in 2007 was the largest since national surveillance began in 2001.

Outbreaks spread person-to-person

In 2007, 83% (1,556/1,882) of all gastroenteritis outbreaks were spread from person to person. There were

Figure 7. Number of notifications of haemolytic uraemic syndrome, Australia, 2002 to 2007, by state or territory and date of diagnosis

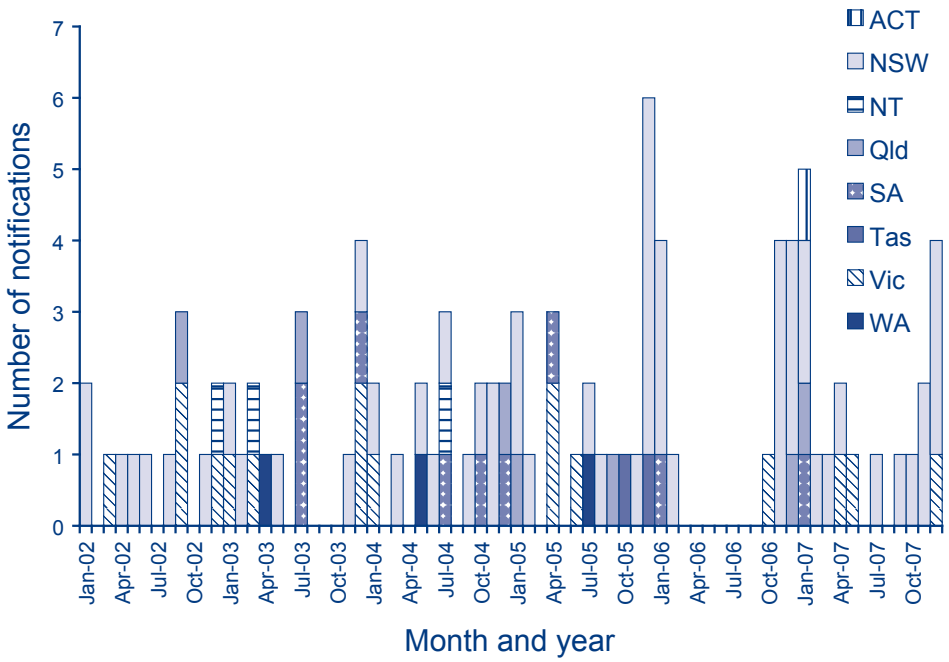


Table 10. Outbreaks of gastroenteritis including foodborne disease reported to state and territory health departments, Australia, 2007

Mode of transmission	Number of outbreaks	Number affected	Hospitalised	Fatalities
Foodborne	149	2,290	266	5
Person-to-person	1,556	32,988	652	108
Unknown mode (*Salmonella* cluster)	54	565	47	0
Unknown mode (other pathogen)	18	682	47	0
Unknown mode (unknown aetiology)	105	949	22	1
Total	1,882	37,474	1,034	114

32,988 people affected as part of these outbreaks and 108 deaths. Person-to-person outbreaks were most common in aged care homes, with 60% (939/1,556) of outbreaks occurring in this setting, followed by 19% (288/1,556) and 13% (208/1,556) in hospitals and child care centres respectively. Approximately 51% (789/1,556) of outbreaks spread from person to person were caused by norovirus, which was followed by 3% of outbreaks caused by rotavirus. Forty-three per cent (679/1,556) of person-to-person outbreaks were of unknown aetiology. Spring was the peak season for outbreaks of person-to-person transmission, with 46% (709/1,556) of outbreaks reported in the months of September to November 2007.

Outbreaks with unknown mode of transmission

There were 177 outbreaks where the mode of transmission was not determined, affecting a total of 2,196 people. There were 54 clusters of *Salmonella* and 18 clusters due to other pathogens that were clustered in time, place or person, where investigators were unable to develop an adequate hypothesis for the source of illness. There were 105 outbreaks where investigators were unable to determine the mode of transmission and the aetiology.

Foodborne outbreaks

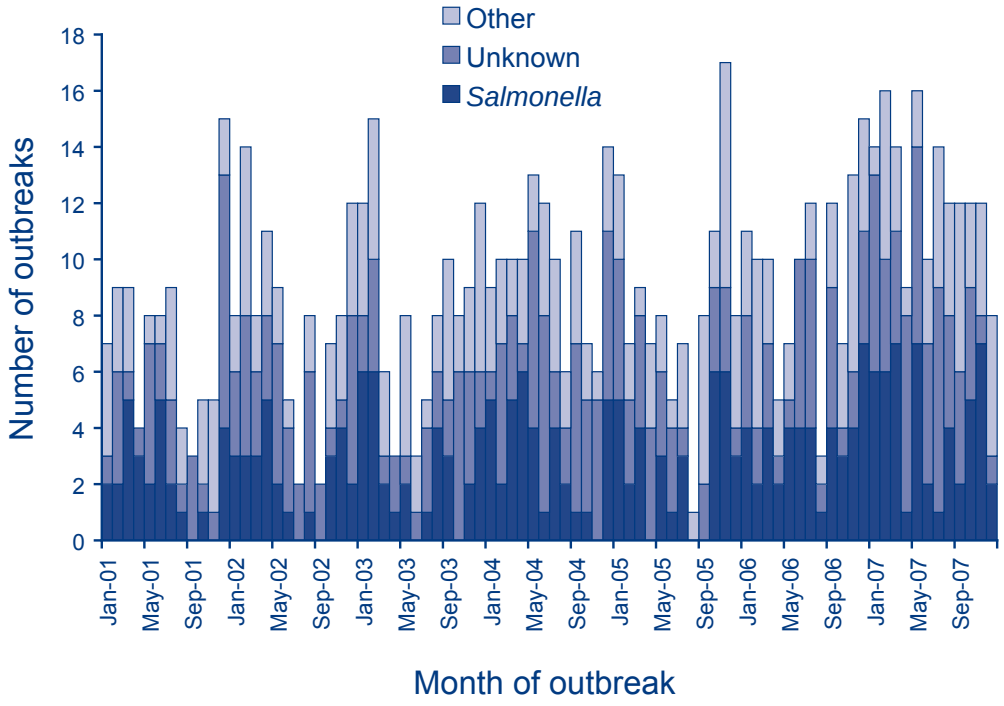
In 2007, there were 149 outbreaks of foodborne disease affecting 2,290 people, which resulted in 266 people being hospitalised. There were 5 deaths reported during these outbreaks (Appendix). This compares to 115 and 102 foodborne outbreaks in 2006 and 2005, respectively.

The overall rate of reported foodborne disease outbreaks for Australia was 7.1 per million population in 2007 (Table 11). The highest rates of reporting were from the Northern Territory (23.3 per million population) and Tasmania (10.1 per million population), although these represent a small number of outbreaks. Outbreaks in the Northern Territory were smaller in size, with a mean of 5.2 persons affected per outbreak, when compared to the mean of 15.4 persons for all outbreaks. The rates of outbreak reporting for the 3 most populous states of Queensland, New South Wales, and Victoria, were similar (7.7, 7.7, and 6.9 per million population, respectively). Outbreaks were more common in warmer months, with *Salmonella* outbreaks being most common in summer, as in previous years (Figure 8).

Table 11. Outbreaks of foodborne disease in Australia, 2007, by OzFoodNet site

State or territory	Number of outbreaks	Number affected	Mean size (persons)	Hospitalised	Outbreaks per million population
ACT	3	46	15.3	0	8.8
NSW	53	829	15.6	187	7.7
NT	5	26	5.2	3	23.3
Qld	32	406	12.7	19	7.7
SA	6	115	19.2	0	3.8
Tas	5	55	11	2	10.1
Vic	36	642	17.8	39	6.9
WA	9	171	19	16	4.3
Total	149	2,290	15.4	266	7.1

Figure 8. Outbreaks of foodborne disease reported to state and territory health departments (n=760), Australia, 2001 to 2007, by month of outbreak



Aetiological agents

The most common agent responsible for foodborne disease outbreaks was *Salmonella*, which caused 34% (50/149) of outbreaks (Table 12). *S.* Typhimurium was responsible for 78% (39/50) of foodborne *Salmonella* outbreaks.

Seventy-one per cent (15/21) of the toxin-mediated outbreaks in 2007 were related to fish toxins. As in previous years, outbreaks of ciguatera fish poisoning (8 outbreaks) and histamine poisoning (7 outbreaks) were small with a mean of three and 2 persons affected, respectively. There were 3 outbreaks of *Clostridium perfringens* and 2 outbreaks of *Bacillus cereus* intoxication. There was 1 small outbreak of lupin intoxication from flour affecting 2 people.

In 2007, there were 16 foodborne outbreaks due to norovirus affecting 520 people, compared with 11 foodborne outbreaks due to norovirus affecting 369 people in 2006. There were also 4 small out-

Table 12. Aetiological agents responsible for foodborne disease outbreaks, number of outbreaks and persons affected, Australia, 2007

Agent category	Number of outbreaks	Number affected	Mean size (persons)	Hospitalised
S. Typhimurium	39	914	23	225
Norovirus	16	520	33	6
S. other	11	125	11	15
Ciguatoxin	8	24	3	1
Histamine poisoning	7	17	2	4
Bacterial toxin	5	78	16	0
Campylobacter	4	20	5	1
Plant toxins	1	2	2	2
Shigella	1	55	55	3
Cyclospora	1	8	1	0
Unknown	56	527	9	9
Total	149	2,290	15	266

Table 13. Categories of food vehicles implicated in foodborne disease outbreaks, Australia, 2007

Vehicle category	Number of outbreaks	Number affected	Mean size (persons)	Hospitalised
Fish	17	75	4	7
Mixed foods	13	550	42	151
Egg-containing dish	11	129	12	15
Dessert	9	124	14	23
Meat & meat products	7	46	7	3
Fresh produce	7	186	27	13
Poultry	5	41	8	1
Water	4	85	21	3
Beverage	3	16	5	2
Seafood	3	42	14	0
Dips	2	77	39	10
Egg-based sauce/dressing	2	31	16	9
Pasta	2	34	17	0
Sushi	2	35	18	5
Cheese	1	10	10	0
Sandwich	1	6	6	0
Unknown	60	803	13	24
Total	149	2,290	15	266

breaks of *Campylobacter* affecting 20 people, 1 outbreak of *Shigella sonnei* biotype a (55 persons), and 1 outbreak of cyclosporiasis (8 persons).

Thirty-eight per cent (56/149) of foodborne outbreaks in 2007 were of unknown aetiology, compared with 31% for the previous year. These outbreaks of unknown aetiology affected 527 people.

Food vehicles

There was a wide variety of foods implicated in outbreaks of foodborne disease during 2007 (Table 13), although investigators were unable to identify a specific food vehicle in 40% (60/149) of outbreaks.

There were 24 outbreaks associated with eggs in 2007; this was 16% (24/149) of all foodborne outbreaks and included all outbreaks that investigators considered were egg-associated, including 11 due to egg-containing dishes, seven due to desserts, two due to egg based sauces/dressings, two due to mixed foods, and one each due to beverages and sushi (Table 14). These outbreaks affected a total of 629 people and hospitalised 195 people.

Contaminated fish was the most common food vehicle and was responsible for 11% (17/149) of foodborne outbreaks. Of the 8 outbreaks of ciguatoxin, Queensland reported seven and the Northern Territory reported one. The types of fish implicated in these outbreaks included: mackerel (4 outbreaks), coral trout (3 outbreaks) and 'mother-in-law fish' (1 outbreak). Histamine poisoning outbreaks from consumption of tuna were reported in New South Wales (2 outbreaks), Queensland (2 outbreaks), the Northern Territory (1 outbreak), and Victoria (1 outbreak). Victoria also reported an outbreak of suspected histamine poisoning following consumption of Mahi Mahi. Contaminated fish also caused an outbreak of *Bacillus cereus* following consumption of Gelfite fish balls, and an outbreak of unknown aetiology following tuna consumption.

Thirteen outbreaks were associated with mixed foods, which include multiple ingredients or buffet meals where a wide variety of foods and dishes were served. Consumption of poultry was responsible for 5 outbreaks and meat other than poultry for 7 outbreaks. There were 7 outbreaks associated with fresh produce, such as fresh fruits and vegetables. These food vehicles included: salads (3 outbreaks) and single outbreaks associated with watermelon, baby corn, fruit salad and lupin flour.

Settings where food was prepared

The most common settings where food was prepared in outbreaks was in restaurants (38%, 57/149), and private residences (11%, 17/149; Table 15). Foods prepared at a takeaway or by commercial caterers were responsible for 15 and 12 outbreaks, respectively. Foods that were contaminated in primary production environments ('primary produce'), such as fish contaminated with ciguatera toxin and fresh fruits and vegetables contaminated with *Salmonella*,

Table 14. Outbreaks of foodborne illness associated with eggs, Australia, 2007 (n=24)

State	Month of outbreak	Setting prepared	Agent responsible	Number affected	Evidence	Responsible vehicles	Comments
NSW	March	Bakery	*Salmonella* Typhimurium 9	319	M	Pork/chicken and salad rolls (with raw egg mayonnaise)	Vietnamese pork rolls; *S.* Tm 9 isolated from multiple foods at bakery including raw egg mayonnaise, ham, cooked chicken, pate, roast pork, shell eggs, and from environmental swabs. Traceback of shell eggs and chicken livers did not identify source of contamination.
	October	Bakery	*Salmonella* Typhimurium	27	D	Cheese or cream cake	Suspected raw egg in cake
	June	Bakery	Unknown	15	D	Raw egg mayonnaise, suspected	
	May	Restaurant	*Salmonella* Typhimurium 9	12	M	Fried ice cream	*S.* Tm 9 found in pre-prepared ice cream
	November	Private residence	*Salmonella* Typhimurium 9	3	M	Egg nog or undercooked chicken	
	November	Private residence	*Salmonella* Typhimurium 9	11	D	Raw eggs	Same MLVA* pattern as outbreak implicating Vietnamese pork rolls in March
Qld	January	Not applicable	*Salmonella* Typhimurium 197	25	M	Egg-based dish, suspected	25 community cases with same MLVA profile as environmental swabs positive for *S.* Tm 197 from egg farm A
	January	Restaurant	*Salmonella* Typhimurium 197	3	M	Egg-based dish, suspected	Restaurant supplied eggs from egg farm A and cases had matching MLVA to environmental swabs from egg farm A
	February	Restaurant	*Salmonella* Typhimurium 197	12	M	Egg-based dish, suspected	Restaurant supplied eggs from egg farm A and cases had matching MLVA to environmental swabs from egg farm A
	February	Restaurant	*Salmonella* Typhimurium 197	6	M	Egg-based dish, suspected	Restaurant supplied eggs from egg farm A and cases had matching MLVA to environmental swabs from egg farm A
	February	Restaurant	*Salmonella* Typhimurium 197	2	M	Egg-based dish, suspected	Restaurant supplied eggs from egg farm A and cases had matching MLVA to environmental swabs from egg farm A
	March	Restaurant	*Salmonella* Typhimurium U302	18	D	Egg-based dish, suspected	Raw eggs used in foods served at restaurant. Proprietor advised not to use raw eggs.
	May	Bakery	*Salmonella* Typhimurium 135a	7	M	Cheesecake	Bakery used raw eggs; instructed to stop
Tas	March	Other	*Salmonella* Typhimurium 135a	20	D	Eggs, suspected	Cases consumed salad rolls from bakery (n=18) and eggs (n=2) from same farm that provided eggs to bakery; cracked and dirty eggs being sold

Table 14. Outbreaks of foodborne illness associated with eggs, Australia, 2007, *continued*

State	Month of outbreak	Setting prepared	Agent responsible	Number affected	Evidence	Responsible vehicles	Comments
Vic	January	Private residence	*Salmonella* Typhimurium 44	4	M	Milkshake	Raw egg milkshake; blender used was positive for *S.* Tm 44
	January	Private residence	*Salmonella* Typhimurium 44	11	A	Trifle	Made with raw egg
	January	Private residence	*Salmonella* Typhimurium 44	10	A	Tiramisu	Made with raw egg, accounted for 90% of cases
	January	Restaurant	*Salmonella* Typhimurium 44	15	D	Caesar salad dressing	Made with raw egg
	May	Private residence	*Salmonella* Typhimurium 9	8	M	Chocolate mousse	Made with raw egg, mousse was positive for *S.* Tm 9
	May	Private residence	*Salmonella* Typhimurium 9	3	D	Chocolate mousse, suspected	Made with raw eggs
	June	Bakery	*Salmonella* Typhimurium 44	45	M	Pork rolls	Vietnamese pork rolls, leftover rolls and pate positive for *S.* Tm 44, raw egg mayonnaise used on all rolls.
	October	Restaurant	*Salmonella* Typhimurium 44	16	M	Chicken foccacia with raw egg aioli	Aioli made with raw egg, blender used to make aioli positive for *S.* Tm 44
	December	Restaurant	*Salmonella* Typhimurium 44	14	M	Risottini, undercooked	Risottini positive for *S.* Tm 44
WA	September	Takeaway	*Salmonella* Virchow 45	23	D	Sushi and Katsudon (served with raw egg mayonnaise)	Implicated premises made and used a raw egg mayonnaise; all food samples negative for *Salmonella*

D Descriptive evidence implicating the vehicle.

A Analytical epidemiological association between illness and vehicle.

M Microbiological confirmation of aetiology in vehicle and cases.

* Multilocus Variable-number tandem repeat Analysis (MLVA).

Table 15. Food preparation settings implicated in disease outbreaks, Australia, 2007

Setting prepared	Number of outbreaks	Proportion of all outbreaks (%)	Number affected (persons)
Restaurant	57	38	714
Private residence	17	11	134
Takeaway	15	10	152
Commercial caterer	12	8	285
Aged care facility	10	7	107
Primary produce	9	6	79
Institution – other	6	4	108
Bakery	5	3	413
Camp	4	3	85
Other	4	3	84
Unknown	3	2	94
Commercial manufactured food	3	2	17
Cruise/airline	1	1	8
Hospital	1	1	4
National franchised fast food restaurant	1	1	4
Grocery store/delicatessen	1	1	2
Total	149	100	2,290

accounted for another 9 outbreaks. Ten outbreaks were associated with food prepared in aged care facilities, while food prepared in other institutions was responsible for a further 6 outbreaks. There were 5 outbreaks associated with foods prepared at bakeries.

Investigative methods and levels of evidence

States and territories investigated 29 outbreaks using retrospective cohort studies and 4 outbreaks using case control studies. In 83 outbreaks, case series information was collected during the investigation. To attribute the cause of the outbreak to a specific food vehicle, investigators obtained analytical evidence from epidemiological studies in 19 outbreaks. Microbiological evidence of contaminated food was found in 20 outbreaks, with a further 2 outbreak investigations obtaining both microbiological and analytical evidence. Investigators obtained analytical and/or microbiological evidence for 46% (23/50) of *Salmonella* outbreaks, which was similar to the proportion in 2006 (41%). Seventy-two per cent (108/149) of outbreaks relied on descriptive evidence to implicate a food or foodborne transmission.

Significant outbreaks

There were 11 outbreaks affecting 40 or more persons in 2007. Five of these outbreaks were due to *Salmonella*, five were due to norovirus and one was due to *Shigella sonnei* biotype a. In total, these significant outbreaks affected 909 people, with a median of 55 persons affected per outbreak (range 45–319 people), and 163 people hospitalised.

New South Wales reported the largest of these outbreaks, which was an explosive outbreak of *S.* Typhimurium 9 in March 2007 due to Vietnamese pork rolls, which affected over 300 people, 170 of whom were laboratory-confirmed *S.* Typhimurium 9 cases. This outbreak was the largest single foodborne outbreak reported in New South Wales in several years. Victoria also reported a large outbreak of *S.* Typhimurium 44 associated with Vietnamese pork rolls in June 2007, which affected at least 45 people.

Queensland reported a large outbreak of shigellosis affecting 55 people, which was due to contaminated baby corn imported from Thailand. This outbreak was linked concurrently with a large outbreak in Denmark, to the same product from Thailand. An international investigation traced the source of the baby corn that both countries had received to the same packing shed in Thailand.

In addition, Queensland investigated 5 outbreaks of *S.* Typhimurium 197 associated with a single egg farm, which affected at least 48 people in total. These outbreaks were all detected by routine molecular subtyping following isolation of *S.* Typhimurium 197 with a specific multilocus

variable-number tandem repeat analysis (MLVA) pattern from environmental samples taken at the egg farm that matched the MLVA pattern of human cases of *S.* Typhimurium 197. The egg farm conducted a voluntary withdrawal of eggs in February 2007. However, eggs from the farm were still being sold in the community from several outlets in Brisbane after this date. After cases continued to be notified, a state-wide consumer level recall was initiated in early March 2007. The recall included a media release, which provided information to the public on the nature of the *Salmonella* outbreak. No further cases of *S.* Typhimurium PT 197 infection associated with the implicated eggs were detected in 2 months of surveillance following these interventions. The farm was prosecuted and pleaded guilty to selling cracked and dirty eggs to a retailer. A conviction was recorded and the farm was fined $1,500. The outbreak investigation identified problems with tracing eggs back to the farm due to the lack of labelling or stamping of the eggs.

Discussion

This report summarises the rates of gastrointestinal diseases commonly transmitted by foods in Australia. While notification rates in Australia have remained stable in recent years, the incidence of *Salmonella* and *Campylobacter* infections in particular, are high compared with other developed countries.[14] In contrast to *Salmonella, Campylobacter* infections in Australia are predominately sporadic, that is, they do not occur as part of a recognised outbreak and may be related to the lack of a robust typing scheme for *Campylobacter*.[15] In 2007, there were 4 outbreaks of campylobacteriosis, which affected 20 people compared with 16,984 notifications in 2007. It is also of note that in 2007 there was an increase in the rate of *Campylobacter* notifications in males aged over 65 compared with the mean notification rate in this age group for the last 5 years. Risk factors for *Campylobacter* infection in Australia are well-characterised[16–18] and it is estimated that in a typical year 50,500 (95% credible interval 10,000–105,500) cases of *Campylobacter* infection in persons over 5 years of age could be directly attributed each year to the consumption of chicken in Australia.[19] A primary piece of work for the OzFoodNet network in the coming year is to summarise existing epidemiological and microbiological data on *Campylobacter* infections to identify areas to target further research and prevention efforts.

In 2007 more than 96% of *Salmonella* notifications contained complete information about serotype and phage type. In Australia, serotyping is conducted by public health reference laboratories in Queensland, New South Wales, Victoria, South Australia, and Western Australia. The smaller jurisdictions, Tasmania, the Australian Capital Territory, and the Northern Territory, forward their *Salmonella* isolates to the Microbiological Diagnostic Unit at the University of Melbourne in Victoria and/or the Australian Salmonella Reference Centre at the Institute of Medical and Veterinary Sciences in South Australia, where phage typing is also performed. In particular, *S.* Typhimurium, the most common serotype in Australia, is routinely phage typed by state reference laboratories to provide epidemiologically relevant discrimination within the serotype to assist in outbreak detection and investigation. In 2007, 3 jurisdictions, Queensland, New South Wales, and Western Australia, introduced alternative subtyping methods to complement existing routine subtyping. In Queensland and New South Wales, MLVA is routinely done in addition to routine phage typing of *S.* Typhimurium. While no MLVA data are presented in this annual report, MLVA patterns are increasingly being used by New South Wales and Queensland to identify and investigate outbreaks of *S.* Typhimurium. In July 2007, Western Australia began to routinely type *S.* Typhimurium isolates with pulsed field gel electrophoresis (PFGE), which has been used by that state since June 2004 and is conducted in accordance to international PulseNet protocols (http://www.cdc.gov/pulsenet/protocols.htm). However, since Western Australia is the only jurisdiction in Australia to routinely use PFGE for *Salmonella* subtyping, OzFoodNet's ability to detect multi-state clusters, particularly of *S.* Typhimurium, that include Western Australia is hampered. Australia continues to work towards a harmonised approach to subtyping of *Salmonella* so that timely identification and investigation of *Salmonella* clusters can occur.

South Australia had the highest notification rate of STEC in 2007, as in previous years. South Australian pathology laboratories refer all stool specimens with macroscopic blood to a reference laboratory for screening using polymerase chain reaction tests to identify the presence of STEC toxin genes. This screening process results in higher ascertainment of cases in South Australia compared with other states and territories.[20]

In 2007, OzFoodNet sites reported 1,882 outbreaks of gastroenteritis, the largest number recorded since surveillance began in 2001. The majority of these outbreaks were due to person-to-person transmission and reflect the large burden of these outbreaks in institutions around Australia. In addition, outbreaks of gastroenteritis are easier to identify in institutions, such as aged care homes and hospitals, and are more likely to be reported from these settings. Just over half of the person-to-person outbreaks in 2007 were due to norovirus, which is highly infectious. A strain which had been circulating in recent years (strain 2006b), was the main cause of the spike in reported outbreaks in spring 2007. OzFoodNet epidemiologists contributed to the creation of the *Guidelines for the Public Health Management of Gastroenteritis*

Outbreaks Due to Norovirus or Suspected Viral Agents in Australia, targeted at aged care facilities, which will be circulated to states and territories under the auspices of CDNA.

In 2007, the rate of foodborne outbreaks in Australia was 7.1 outbreaks per million population and the mean size of these outbreaks was 15 people. This compares with an estimated rate of 18 foodborne outbreaks per million people in New Zealand,[21] and an estimated rate of 4.1 foodborne outbreaks per million people in the United States of America.[22]

Eggs continue to be the most commonly identified food vehicle in foodborne disease outbreaks in 2007. In 2007, there were 24 outbreaks associated with eggs, compared with 16 outbreaks reported in 2006. These outbreaks were due to a variety of dishes and food items containing raw or undercooked eggs. As highlighted in Table 13 and the Appendix, the assignment of outbreak vehicles into summary vehicle categories is a complex process, into which investigator experience, epidemiologic context and concurrent events contribute. In 2007, the risks associated with using raw egg products in foods are illustrated by the 2 large outbreaks associated with the consumption of Vietnamese pork rolls (made with a homemade raw egg mayonnaise) in New South Wales and Victoria. In August 2007 Federal, State and Territory governments met with industry at the National Egg Food Safety Summit to discuss how to address egg-associated illness. The increased number of egg-related outbreaks across Australia in 2007 highlights the need for a national primary production and processing standard for eggs and egg products. Currently, the Food Standards Code does not prohibit cracked and dirty eggs being sold to non-retail food businesses, provided eggs undergo adequate heat treatment. This standard does not address the risk of cross-contamination occurring in the kitchen of non-retail businesses such as restaurants and catering firms, when staff handle soiled eggs.

Fresh produce was associated with 7 outbreaks in 2007, an increase from the 4 produce-associated outbreaks reported in 2006. The outbreak associated with baby corn was linked to an international outbreak of shigellosis that affected Denmark and Australia and was due to baby corn imported from Thailand. This outbreak was identified in Australia by comparing antibiotic resistance patterns with those published from the Danish outbreak in the monthly publication *Eurosurveillance*.[23,24] After identification of the cluster in Australia, international communications through the World Health Organization (WHO) International Food Safety Authorities Network and under the auspices of the International Health Regulations (2005), enabled trace back of the baby corn to the same packing shed in Thailand.[25]

In 2007, OzFoodNet continued its international collaboration with the WHO Global Salmonella Surveillance (WHO Global Salm-Surv) Network (http://www.who.int/salmsurv/en/), a capacity building network focussed on foodborne disease epidemiology and microbiology. WHO Global Salm-Surv is part of WHO's endeavours to strengthen the capacities of its member states in the surveillance and control of major foodborne diseases and to contribute to the global effort of containment of antimicrobial resistance in foodborne pathogens. OzFoodNet has been a steering committee member of WHO Global Salm-Surv since 2004 and in 2007 OzFoodNet epidemiologists attended regional training courses in Thailand and Papua New Guinea as epidemiology trainers.

It is important to recognise some of the limitations of the data used in this report. Some of the most common enteric pathogens are not notifiable, particularly norovirus and *Clostridium perfringens*. These organisms may be notified as the cause of outbreaks, but not as individual cases of disease. A limitation of the outbreak data provided by OzFoodNet sites for this report is the potential for variation in categorising features of outbreaks depending on investigator interpretation and circumstances. States and territories are working towards harmonising surveillance and outbreak data to address some of these issues.

Foodborne disease surveillance provides information to assist in not only immediate public health action and the prevention of these diseases, but also contributes to the assessment of food safety policies and campaigns. A national program of surveillance for foodborne diseases and outbreak investigation has many benefits including identifying foods that cause human illness. Ongoing efforts to strengthen the quality of these data will ensure continued use by agencies to develop food safety policy and prevent foodborne illness.

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Appendix. Foodborne outbreak summary for OzFoodNet sites, Australia, 2007 (n=149)

State	Month of outbreak	Setting prepared	Aetiology	Number affected	Hospitalised	Fatalities	Evidence	Vehicle	Vehicle category
ACT	April	Restaurant	Unknown	29	0	0	A	Unknown	Unknown
	May	Restaurant	Unknown	8	0	0	D	Unknown	Unknown
	October	Restaurant	Unknown	9	0	0	D	Unknown	Unknown
NSW	January	Unknown	Norovirus	58	0	0	D	Unknown	Unknown
	January	Restaurant	Unknown	9	0	0	D	Chicken stirfry or beef massaman	Mixed foods
	January	Institution – other	Unknown	7	0	0	D	Unknown	Unknown
	January	National franchised fast food restaurant	Unknown	4	1	0	D	Deep fried chicken, suspected	Poultry
	January	Restaurant	Unknown	3	0	0	D	Unknown	Unknown
	February	Commercial caterer	Bacillus cereus	32	0	0	AM	Boiled gefilte fish (fish balls)	Fish
	February	Restaurant	*Salmonella* Typhimurium 12	6	1	0	D	Unknown	Beverage
	February	Commercially manufactured	Unknown	6	0	0	D	Flavoured drink	Dips
	February	Takeaway	Clostridium perfringens	6	0	0	M	Hommus	Unknown
	February	Restaurant	Unknown	5	0	0	D	Unknown	Unknown
	February	Restaurant	Unknown	4	0	0	D	Seafood platter	Seafood
	February	Private residence	Histamine	3	2	0	M	Tuna kebab steaks	Fish
	February	Restaurant	Unknown	3	0	0	D	Butter chicken or boiled rice, suspected	Mixed foods
	February	Restaurant	Histamine	2	2	0	D	Tuna steak	Fish
	March	Bakery	*Salmonella* Typhimurium 9	319	136	0	M	Pork/chicken and salad rolls (with raw egg mayonnaise)	Mixed foods
	March	Takeaway	*Salmonella* Typhimurium U302	71	10	0	M	Hommus and tabouli	Dips
	March	Restaurant	Unknown	5	0	0	D	Banquet style meal	Mixed foods
	April	Commercially manufactured	Unknown	9	3	0	A	Commercially prepared cake, suspected	Dessert
	April	Restaurant	Unknown	7	0	0	D	Fried rice, suspected	Mixed foods
	April	Takeaway	Unknown	5	0	0	D	Hot dogs	Meat & meat products
	May	Restaurant	Unknown	14	0	0	D	Raw capsicum, onions, fresh herbs, chicken and/or beef	Mixed foods

Appendix. Foodborne outbreak summary for OzFoodNet sites, Australia, 2007 (n=149), *continued*

State	Month of outbreak	Setting prepared	Aetiology	Number affected	Hospitalised	Fatalities	Evidence	Vehicle	Vehicle category
NSW, *cont'd*	May	Restaurant	*Salmonella* Typhimurium 9	12	4	0	M	Fried ice cream	Dessert
	May	Takeaway	Unknown	6	0	0	D	Fresh fruit juices, suspected	Beverage
	May	Restaurant	Unknown	6	0	0	D	Unknown	Unknown
	June	Bakery	Unknown	15	0	0	D	Raw egg mayonnaise, suspected	Egg-containing dish
	June	Takeaway	Unknown	2	2	0	D	Grilled tuna	Fish
	June	Takeaway	Unknown	2	0	0	D	Unknown	Unknown
	June	Takeaway	Unknown	2	0	0	D	Unknown	Unknown
	July	Restaurant	Unknown	6	0	0	D	Sandwich	Sandwich
	July	Restaurant	Unknown	5	1	0	D	Unknown	Unknown
	July	Restaurant	Unknown	5	0	0	D	Unknown	Unknown
	July	Restaurant	Unknown	5	0	0	D	Unknown	Unknown
	July	Restaurant	Unknown	3	0	0	D	Unknown	Unknown
	July	Restaurant	Unknown	3	0	0	D	Unknown	Unknown
	July	Restaurant	Unknown	3	0	0	D	Unknown	Unknown
	July	Restaurant	Unknown	3	0	0	D	Unknown	Unknown
	August	Aged care facility	Unknown	9	0	0	D	Unknown	Unknown
	August	Takeaway	Unknown	4	0	0	D	Beef or lamb kebab, suspected	Meat & meat products
	August	Takeaway	Unknown	2	0	0	D	Fried rice or Singapore noodles	Mixed foods
	September	Restaurant	Unknown	19	0	0	D	Oysters	Seafood
	September	Commercial caterer	Unknown	15	1	0	D	Unknown	Unknown
	September	Aged care facility	Unknown	6	0	0	D	Unknown	Unknown
	September	Takeaway	Unknown	3	0	0	D	Unknown	Unknown
	October	Bakery	*Salmonella* Typhimurium	27	10	0	D	Cheese or cream cake	Dessert
	October	Private residence	Unknown	7	0	0	D	Watermelon	Fresh produce
	October	Takeaway	Unknown	4	0	0	D	Chicken kebab	Poultry
	November	Private residence	*Salmonella* Typhimurium 9	11	4	0	D	Raw eggs	Egg-containing dish
	November	Private residence	*Salmonella* Typhimurium	8	2	0	D	Beef patties	Meat & meat products

Appendix. Foodborne outbreak summary for OzFoodNet sites, Australia, 2007 (n=149), *continued*

State	Month of outbreak	Setting prepared	Aetiology	Number affected	Hospitalised	Fatalities	Evidence	Vehicle	Vehicle category
NSW, *cont'd*	November	Private residence	*Salmonella* Typhimurium 9	3	0	0	M	Egg nog or undercooked chicken	Egg-containing dish
	November	Restaurant	Unknown	3	0	0	D	Unknown	Unknown
	November	Takeaway	Campylobacter	2	1	0	D	Meat kebab	Meat & meat products
	December	Commercial caterer	Unknown	28	0	0	A	Unknown	Unknown
	December	Private residence	*Salmonella* Virchow 8	22	7	0	D	Chicken or eggs, suspected	Mixed foods
NT	June	Unknown	Mixed	11	2	0	D	Unknown	Unknown
	June	Commercial manufactured food	Histamine	2	0	0	D	Tinned tuna	Fish
	July	Primary produce	Ciguatoxin	2	0	0	D	Reef cod	Fish
	August	Commercial caterer	*Salmonella* Oslo	3	0	0	D	Roast pork, suspected	Meat & meat products
	September	Commercial caterer	Norovirus	8	1	0	D	Unknown	Unknown
Qld	January	Unknown	*Salmonella* Typhimurium 197	25	6	0	M	Egg-based dish, suspected	Egg-containing dish
	January	Restaurant	*Salmonella* Typhimurium 197	3	1	0	M	Egg-based dish, suspected	Egg-containing dish
	February	Camp	*Salmonella* Saintpaul	24	0	0	M	Bore water	Water
	February	Restaurant	*Salmonella* Typhimurium 197	12	1	0	M	Egg-based dish, suspected	Egg-containing dish
	February	Restaurant	*Salmonella* Typhimurium 197	6	1	0	M	Egg-based dish, suspected	Egg-containing dish
	February	Private residence	Histamine	2	0	0	D	Imported Indonesian Tuna	Fish
	February	Primary produce	Ciguatoxin	2	0	0	D	Mackerel	Fish
	February	Restaurant	*Salmonella* Typhimurium 197	2	0	0	M	Egg-based dish, suspected	Egg-containing dish
	March	Institution – other	Norovirus	45	0	0	A	Ham; salad; bread	Mixed foods
	March	Restaurant	*Salmonella* Typhimurium U302	18	0	0	D	Egg-based dish, suspected	Egg-containing dish
	March	Primary produce	Ciguatoxin	6	0	0	D	Mackerel	Fish
	April	Other	Unknown	21	0	0	D	Unknown	Unknown
	May	Restaurant	*Salmonella* Virchow 8	15	1	0	D	Unknown	Unknown
	May	Bakery	*Salmonella* Typhimurium 135a	7	1	0	M	Cheesecake	Dessert
	May	Private residence	Unknown	7	0	0	D	Wurst	Meat & meat products
	May	Restaurant	*Salmonella* Typhimurium 135a	6	0	0	D	Unknown	Unknown
	May	Primary produce	Ciguatoxin	3	1	0	D	Coral trout	Fish
	May	Primary produce	Ciguatoxin	2	0	0	D	Mackerel	Fish

Appendix. Foodborne outbreak summary for OzFoodNet sites, Australia, 2007 (n=149), *continued*

State	Month of outbreak	Setting prepared	Aetiology	Number affected	Hospitalised	Fatalities	Evidence	Vehicle	Vehicle category
Qld, *cont'd*	June	Private residence	Histamine	4	0	0	D	Tuna kebabs	Fish
	August	Primary produce	*Shigella sonnei* biotype g	55	3	0	M	Baby corn	Fresh produce
	August	Restaurant	Norovirus	24	0	0	A	Mixed salad	Fresh produce
	August	Restaurant	*Salmonella* Typhimurium 135	8	0	0	M	Duck pate	Poultry
	September	Institution – other	Norovirus	35	0	0	D	Unknown	Unknown
	September	Primary produce	Ciguatoxin	5	0	0	D	Coral trout	Fish
	October	Aged care facility	*Salmonella* Kiambu	2	0	0	D	Unknown	Unknown
	November	Institution – other	*Salmonella* Typhimurium U307	6	4	0	D	Unknown	Unknown
	November	Takeaway	Bacillus cereus	3	0	0	M	Fried rice and honey chicken	Mixed foods
	November	Restaurant	*Salmonella* Typhimurium U307	3	0	0	D	Unknown	Unknown
	November	Primary produce	Ciguatoxin	2	0	0	D	Coral trout	Fish
	December	Restaurant	Norovirus	46	0	0	D	Unknown	Unknown
	December	Private residence	Norovirus	5	0	0	D	Salad, suspected	Fresh produce
	December	Primary produce	Ciguatoxin	2	0	0	D	Spanish mackerel	Fish
SA	March	Restaurant	*Salmonella* Typhimurium 9	46	0	0	A	Multiple food items	Mixed foods
	April	Commercial caterer	Unknown	12	0	0	A	Sushi	Sushi
	July	Restaurant	Norovirus	14	0	0	D	Unknown	Unknown
	July	Private residence	*Salmonella* Typhimurium 193	13	0	0	A	Unknown	Unknown
	August	Aged care facility	*Campylobacter*	6	0	0	D	Unknown	Unknown
	September	Other	Norovirus	24	0	0	D	Unknown	Unknown
Tas	January	Other	Unknown	19	0	0	D	Oysters, suspected	Seafood
	March	Other	*Salmonella* Typhimurium 135a	20	2	0	D	Eggs, suspected	Egg-containing dish
	September	Restaurant	*Salmonella* Typhimurium 135a	2	0	0	D	Unknown	Unknown
	October	Restaurant	Unknown	12	0	0	D	Unknown	Unknown
	October	Restaurant	*Salmonella* Typhimurium 135a	2	0	0	D	Unknown	Unknown

Appendix. Foodborne outbreak summary for OzFoodNet sites, Australia, 2007 (n=149), *continued*

State	Month of outbreak	Setting prepared	Aetiology	Number affected	Hospitalised	Fatalities	Evidence	Vehicle	Vehicle category
Vic	January	Takeaway	Unknown	17	0	0	A	Meat curry, suspected	Meat & meat products
	January	Restaurant	*Salmonella* Typhimurium 44	15	2	0	D	Caesar salad dressing	Egg-based sauce/dressing
	January	Private residence	*Salmonella* Typhimurium 44	11	4	0	A	Trifle	Dessert
	January	Private residence	*Salmonella* Typhimurium 44	10	1	0	A	Tiramisu	Dessert
	January	Private residence	*Salmonella* Typhimurium 44	4	2	0	M	Milkshake	Beverage
	January	Restaurant	Unknown	4	1	0	D	Unknown	Unknown
	March	Commercial caterer	Unknown	37	0	0	A	Passionfruit coulis, suspected	Dessert
	March	Camp	*Salmonella* Typhimurium 9	30	3	0	AM	Water	Water
	March	Camp	Unknown	19	0	0	D	Water, suspected	Water
	March	Restaurant	Unknown	10	0	0	A	Fetta cheese, suspected	Cheese
	March	Restaurant	Histamine	2	0	0	D	Tuna	Fish
	April	Commercial caterer	Unknown	25	0	0	A	Penne pasta salad, suspected	Pasta
	April	Aged care facility	*Salmonella* Typhimurium 44	22	8	5	A	Unknown	Unknown
	April	Restaurant	Histamine	2	0	0	D	Mahi Mahi fish	Fish
	May	Aged care facility	Unknown	17	0	0	D	Unknown	Unknown
	May	Restaurant	Unknown	9	0	0	D	Lasagne	Pasta
	May	Private residence	*Salmonella* Typhimurium 9	8	0	0	M	Chocolate mousse	Dessert
	May	Hospital	*Salmonella* Typhimurium 9	4	0	0	D	Unknown	Unknown
	May	Private residence	*Salmonella* Typhimurium 9	3	0	0	D	Chocolate mousse, suspected	Dessert
	June	Bakery	*Salmonella* Typhimurium 44	45	8	0	M	Pork Rolls	Mixed foods
	June	Restaurant	Unknown	5	0	0	D	Chicken massaman curry suspected	Poultry
	July	Aged care facility	*Clostridium perfringens*	30	0	0	D	Multiple food items	Mixed foods
	July	Restaurant	Norovirus	21	0	0	D	Unknown; ill food handler suspected	Unknown
	July	Aged care facility	*Campylobacter*	6	0	0	D	Unknown	Unknown
	August	Commercial caterer	Unknown	20	0	0	A	Roast chicken and/or stuffing	Poultry
	August	Aged care facility	*Campylobacter*	6	0	0	D	Unknown	Unknown

Appendix. Foodborne outbreak summary for OzFoodNet sites, Australia, 2007 (n=149), *continued*

State	Month of outbreak	Setting prepared	Aetiology	Number affected	Hospitalised	Fatalities	Evidence	Vehicle	Vehicle category
Vic, *cont'd*	August	Restaurant	*Salmonella* Dublin	6	0	0	D	Unknown	Unknown
	September	Restaurant	Norovirus	96	0	0	D	Unknown; ill food handler suspected	Unknown
	October	Commercial caterer	Norovirus	53	0	0	D	Unknown	Unknown
	October	Commercial caterer	Norovirus	34	1	0	D	Unknown	Unknown
	October	Commercial caterer	Norovirus	18	2	0	A	Fruit salad	Fresh produce
	October	Restaurant	*Salmonella* Typhimurium 44	16	7	0	M	Chicken foccacia with raw egg aioli	Egg-based sauce/dressing
	October	Aged care facility	*Salmonella* Saintpaul	3	0	0	D	Unknown	Unknown
	November	Private residence	*Salmonella* Typhimurium 44	13	0	0	D	Unknown	Unknown
	November	Institution – other	Clostridium perfringens	7	0	0	D	Unknown	Unknown
	December	Restaurant	*Salmonella* Typhimurium 44	14	0	0	M	Risottini, undercooked	Egg-containing dish
WA	Febuary	Restaurant	*Salmonella* Mbandaka	4	0	0	D	Unknown	Unknown
	March	Restaurant	*Salmonella* Typhimurium U307	75	6	0	A	Caesar salad	Fresh produce
	June	Restaurant	Norovirus	26	2	0	A	Unknown	Unknown
	August	Institution – other	*Salmonella* Typhimurium 44	8	1	0	D	Unknown	Unknown
	September	Takeaway	*Salmonella* Virchow 45	23	5	0	D	Sushi and Katsudon (made with eggs)	Sushi
	September	Grocery store/ delicatessen	Alkaloids (plant toxin)	2	2	0	D	Bitter lupin flour	Fresh produce
	November	Camp	*Salmonella* Tennessee	12	0	0	D	Drinking water	Water
	December	Restaurant	Norovirus	13	0	0	D	Unknown	Unknown
	December	Cruise/airline	Cyclospora cayetanensis	8	0	0	D	Unknown	Unknown

D Descriptive evidence implicating the vehicle.

A Analytical epidemiological association between illness and vehicle.

M Microbiological confirmation of aetiology in vehicle and cases.

Australian Rotavirus Surveillance Program annual report, 2007/08

Carl D Kirkwood, David Cannan, Karen Boniface, Ruth F Bishop, Graeme L Barnes, and the Australian Rotavirus Surveillance Group

Abstract

The National Rotavirus Reference Centre together with collaborating laboratories Australia-wide conducts a laboratory based rotavirus surveillance program. This report describes the types of rotavirus strains responsible for the hospitalisation of children with acute gastroenteritis during the period 1 July 2007 to 30 June 2008, the first complete year of surveillance following introduction of rotavirus into the National Immunisation Program. Six hundred faecal samples from across Australia were examined using a combined approach of monoclonal antibody immunoassays and reverse transcription-polymerase chain reaction. Of the 419 confirmed as rotavirus positive, serotype G1 was the dominant serotype nationally, representing 52% of specimens, followed by serotype G2 (19.8%), serotype G9 (12.2%), and serotype G3 (11%). No serotype G4 strains were identified. All G1, G3 and G9 strains assayed for P genotype contained the P[8] genotype, while all G2 strains contained the P[4] genotype, except one G2 strain which possessed a P[8]. Uncommon rotavirus genotypes, G8 (n=2) and P[9] (n=2) were identified during this study period. There was no evidence of unexpected changes in serotype distribution during the first 12 months of rotavirus vaccine use in the National Immunisation Program. *Commun Dis Intell* 2008;32:425–429.

Keywords: rotavirus, disease surveillance

Introduction

Rotavirus vaccine was introduced into the National Immunisation Program of Australia for all young infants from 1 July 2007. This is aimed to decrease the huge social and economic burden of rotavirus disease in Australia, which accounts for up to 50% of childhood hospitalisations for diarrhoea in Australia, and which represents 10,000 children hospitalised each year,[1] costing an estimated $30 million in direct costs.[2]

The 2 rotavirus vaccines, Rotarix® [GlaxoSmithKline] and RotaTeq® [Merck], have both been demonstrated to be safe and highly effective in the prevention of severe diarrhoea and hospitalisation due to rotavirus infections during large-scale phase III clinical and efficacy trials, each involving over 60,000 children worldwide.[3,4] In Australia, the state and territory health departments each made independent decisions on which vaccine to select; Victoria, South Australia, and Queensland selected RotaTeq®, while New South Wales, Western Australia, the Northern Territory, Tasmania and the Australian Capital Territory selected Rotarix®.

The Australian Rotavirus Surveillance Program has been reporting the changing annual pattern of dominant serotypes in the Australian population since 1999. Over this period our results highlight the diversity of rotavirus strains capable of causing disease in children, and provide the baseline information of the changing pattern of circulating strains, prior to vaccine introduction.[5–7]

The impact of these 2 widely used vaccines on the natural pattern of circulating rotavirus strains is unknown and difficult to predict, given the different components of each vaccine. Continuing serotype surveillance should identify the effects that each vaccine program has on circulating strains—in particular, whether changes occur in serotype incidence and whether increased proportions of rare or uncommon types result.

In this report we describe the surveillance and characterisation of rotavirus strains causing the annual epidemics of severe diarrhoea in young children in Australia for the period 1 July 2007 to 30 June 2008, the first 12 months in which rotavirus vaccine was available through the immunisation program.

Methods

Rotavirus positive specimens detected by enzyme immunoassay (EIA) or latex agglutination in collaborating laboratories were collected, stored frozen and forwarded to Melbourne together with relevant age and sex details. Specimens were then serotyped using an in-house monoclonal antibody (MAb) based serotyping EIA. The EIA employed a panel of MAbs specific for the major glycoprotein VP7 of the outer capsid of the 5 major group A human rotavirus serotypes (G1, G2, G3, G4 and G9).[8] Strains which could not be assigned a G serotype were genotyped by using a hemi-nested multiplex reverse transcription/polymerase chain reaction (RT-PCR), using G specific oligonucleotide primers.[9] P genotypes were determined by using a hemi-nested multiplex RT-PCR assay.[10]

Results

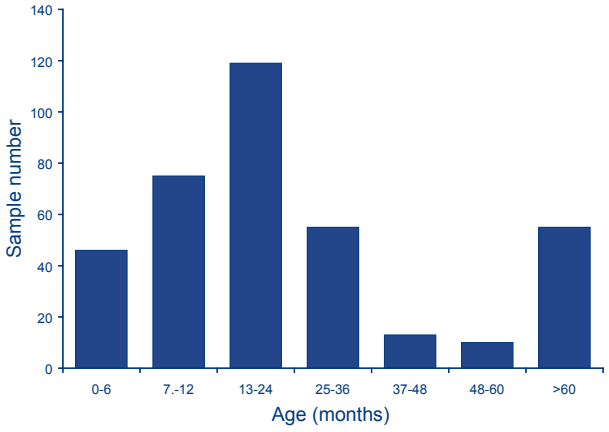
Number of isolates

A total of 600 specimens were received for analysis from Melbourne, Victoria and the collaborating centres in Western Australia, the Northern Territory, New South Wales and Queensland (Table). Specimens were not obtained from either South Australia or Tasmania. Four hundred and nineteen specimens were confirmed as rotavirus positive using our in-house EIA assay. The remaining 181 specimens contained either insufficient specimen for testing, or the specimens were not confirmed to be positive for rotavirus and were not analysed further.

Age distribution

The overall age distribution of children with acute rotavirus gastroenteritis is depicted in the Figure. In the reporting period, 11% of cases were from infants 0–6 months of age, 17.9% were from infants 7–12 months of age, 28.4% from patients 13–24 months of age, and 13.1% from patients 25–36 months of age. Overall, 86.9% of samples were from children aged 5 years or less.

Figure. Cases of rotavirus, Australia, 1 July 2007 to 30 June 2008, by age group



During the study period, slightly more specimens from male than female children (n=317 vs 247) were obtained for analysis.

Serotype distribution

The rotavirus serotypes identified in Australia from 1 July 2007 to 30 June 2008 are shown in the Table.

Table. Rotavirus G serotypes in Australia, 1 July 2007 to 30 June 2008

Centre	Total number	Serotype													
		G1		G2		G3		G4		G9		mix		NR	
		%	n	%	n	%	n	%	n	%	n	%	n	%	n
New South Wales															
Sydney (POW)	6	50	3	0.0	0	16.7	1	0.0	0	16.7	1	0.0	0	16.7	1
Sydney (Westmead)	33	39.4	13	27.3	9	21.2	7	0.0	0	0.0	0	3.0	1	9.1	3
Northern Territory															
Alice Springs	37	0.0	0	40.5	15	0.0	0	0.0	0	46	17	0.0	0	13.5	5
Darwin	18	0.0	0	61.1	11	11.1	2	0.0	0	22.2	4	0.0	0	5.6	1
Western Diagnostic (NT)	8	25	2	62.5	5	0.0	0	0.0	0	0.0	0	0.0	0	12.5	1
Queensland															
Brisbane	26	15.4	4	34.6	9	27.0	7	0.0	0	11.5	3	0.0	0	11.5	3*
Victoria															
Melbourne	82	64.6	53	5	6.1	18.3	15	0.0	0	3.7	3	0.0	0	7.3	6
Western Australia															
PathWest WA	134	68.7	92	11.2	15	3.7	5	0.0	0	11.9	16	0.0	0	4.5	5*
Perth	48	85.4	41	2.1	1	0.0	0	0.0	0	4.2	2	2.1	1	6.2	3
Western Darwin Pathology	27	0.0	0	48.2	13	33.3	9	0.0	0	18.5	5	0.0	0	0.0	0
Total	419	52.0	218	19.8	83	11.0	46	0.0	0	12.2	51	0.5	2	4.5	19

An additional 181 specimens were omitted from analysis due to insufficient sample or because the specimen was not confirmed to be rotavirus positive.

* Two samples were identified as genotype G8 (PerthWest and Brisbane).

Serotype G1 was the most common, representing 52% of all specimens, and was identified in Melbourne, Sydney, Perth and Brisbane, however only two of 90 rotaviruses identified in the Northern Territory were G1, both in Darwin. G1 was the dominant type in Melbourne, Sydney and Perth. Serotype G2 was the second most common type nationally, and represented 19.8% of specimens. It was identified in eight of the 10 collaborating centres and was the dominant type in Darwin and Brisbane. Strains belonging to serotype G9 were the third most common type identified, representing 12.2% of specimens. G9 was found in eight of the 10 centres, and was dominant in Alice Springs, prior to this surveillance period. This was probably related to the large rotavirus gastroenteritis outbreak which occurred in early 2007 in Alice Springs. Serotype G3 strains were identified in 7 centres during the study, and represented 11% of the total strains identified. No serotype G4 strains were identified in any centre. Two genotype G8 strains were identified during the study, one in Perth and one in Brisbane.

P genotype was determined for 116 of the rotavirus positive samples. All of the 25 G1 strains analysed were genotyped as P[8]. Similarly, all of the G3 and G9 strains analysed were also genotyped as P[8] (n=21 and 23, respectively). Thirty-eight of the 39 G2 strains analysed were associated with P[4]: 1 sample was typed as G2P[8]. Two G non-typeable strains were found to possess the P[9] VP4 gene.

Less than 0.5% of the rotavirus samples contained multiple serotypes. In 4.5% of the samples a serotype was not identified. The latter could be samples with virus numbers below the detection limits of our typing assays, or could have contained inhibitors in extracted RNA that prevent the function of the enzymes used in RT and/or PCR steps. Future studies will include further characterisation of the genes encoding the outer capsid proteins of these strains.

Faecal specimens were received from 12 children who developed gastroenteritis with 2 weeks of being vaccinated. No vaccine virus was identified in any of the cases, and in all cases wild type rotavirus strains were detected.

Discussion

In this report covering the period 1 July 2007 to 30 June 2008, we describe the annual epidemics and geographic distribution of the rotavirus types causing disease in Australian children during the first 12 months after national rotavirus vaccine introduction. Serotype G1 remained the dominant serotype nationally, comprising 52% of all strains characterised. It continues to be the dominant type on both sides of the country, in particular in Melbourne, Sydney and Perth, and was a minor strain in the Northern Territory. This survey continues to highlight the importance of serotype G1 as a major cause of disease in Australian children.

As noted in previous reports, multiple serotypes continue to co-circulate within the Australian population causing significant disease.[5–7] This year, G2, G3 and G9 were each identified in at least 7 locations during 2007–2008 and were all identified in greater than 10% of specimens.

The prevalence of G2 strains has increased during this survey, with G2 being the predominant type identified in the Northern Territory and Brisbane. During the 2004/05 and 2005/06 surveys G2 was a minor strain, identified in less than 2% of strains.[5,6] G2 has slowly increased in prevalence in Sydney and Melbourne, being the second most common strain during the 2006/07 period.[7] This year G2 predominated in the Northern Territory for the first time since it was responsible for a large outbreak of gastroenteritis in January 2004.[11]

In each of the G1, G3 and G9 strains analysed for VP4 genotype, the expected association with the P[8] VP4 protein was identified. Thus G1P[8], G3P[8] and G9P[8] combinations were the predominant strains identified in children during the current surveillance period similar to what is seen worldwide.[12,13] The G2 strains showed the expected association with the P[4] VP4 genotype, with 1 exception, the unusual G2P[8] strain that was identified in Brisbane. This is the first G2P[8] strain reported in Australia.

Both rotavirus vaccines used in Australia, RotaTeq and Rotarix have been shown to provide excellent protection against severe rotavirus gastroenteritis in large Phase III safety and efficacy trials.[2,3] RotaTeq® is a live attenuated bovine-human pentavalent vaccine. It contains rotavirus reassortants G1, G2, G3, G4 and P1[8] derived from rotaviruses infecting human and bovine species, which provides serotype specific protection against the most common rotavirus types. Rotarix® contains a live attenuated strain of human rotavirus (G1P[8]). Protection involves production of both specific and cross reactive antibodies, and has been demonstrated against serotypes G1, G3, G4 and G9. In a comparison of rotavirus types identified, based on vaccine usage in the various states, differences in the prevalence rates of G2, G3 and G9 were seen. G2 and G9 strains were more prevalent in states using Rotarix, whereas G3 strains were more prevalent in states using RotaTeq. These differences do not necessarily imply lack of protection by either vaccine against a particular type, but rather highlight the variation that can occur due to natural annual fluctuation in rotavirus strain prevalence.

Uncommon rotavirus types continue to be of worldwide interest because of the possible impact they may have on rotavirus vaccine programs. This year, 2 uncommon types have been identified in Australian children. Strains exhibiting a genotype G8 VP7 protein were identified in Perth and Brisbane, extending the previous identification of G8 in 2006–07 in Darwin. This continues the sporadic identification of G8 strains as a cause of acute gastroenteritis in Australian children.[7,14] The second uncommon type identified during this survey was the P[9] VP4 genotype in 2 strains. These strains were identified in Darwin, and represent the first report of P[9] in Australia. Previous reports of P[5] have been associated with G6 in the United States of America and Hungary, and G12 in Japan and Thailand.[12,13] Thus these reports of uncommon strains continue to highlight their low level existence in Australian children.

Prior to rotavirus vaccine introduction approximately 70% of clinical disease occurred among children less than 24 months of age.[7] During the previous survey (2006/07) 16% of faecal specimens were received from children admitted to hospital aged 0–6 months, and 23.9% of specimens were from infants aged 7–12 months. In comparison, the age distribution of children admitted to hospital during the first 12 months after implementation of the rotavirus vaccination programs has seen a slight reduction in the proportion of 0–6 and 7–12 month age groups affected, with 13% of specimens derived from infants aged 0–6 months, and 21% of specimens from infants aged 7–12 months. The potential impact of rotavirus vaccination is illustrated by the finding that no rotavirus positive specimens were received from infants aged 0–6 months in Darwin, despite faecal specimens being collected for serotype analysis from 54 children in Darwin during this survey period.

There have been no unexpected changes in the serotype distribution of rotavirus types causing disease in Australian children since the introduction of vaccination program nationwide. The rotavirus typing results from this survey, together with those of previous years, highlight the unpredictable nature of changes in the prevalence of rotavirus strains across Australia. In addition, the identification of rare or uncommon VP7 and VP4 genotypes further illustrate the diversity of strains capable of causing severe disease in Australian children. Understanding the fluctuations in rotavirus serotypes, using multicentre national surveillance, will provide valuable insight into vaccine efficacy over the next 3–5 years.

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Australian Paediatric Surveillance Unit annual report, 2007

Yvonne A Zurynski, Deepika Mahajan, Elizabeth J Elliott

Background

National active surveillance of rare diseases of childhood, including communicable and vaccine preventable diseases, genetic disorders, childhood injuries and mental health conditions is conducted by the Australian Paediatric Surveillance Unit (APSU). The study of communicable and vaccine-preventable diseases is supported in part by the Australian Government Department of Health and Ageing (DoHA) through its communicable diseases program. In 2007, APSU conducted national surveillance for 10 communicable or vaccine preventable conditions. The rationale for surveillance of each of these conditions is set out below:

1. Acute flaccid paralysis (AFP) is a major clinical presentation of poliomyelitis. The APSU provides clinical information on cases of AFP, via the Australian National Poliovirus Reference Laboratory, to the Australian Polio Expert Committee. The committee determines whether the cases are compatible with polio based on the clinical and laboratory information supplied. After each meeting, the Polio Expert Committee reports to the World Health Organization (WHO) Western Pacific Regional Office. These data are used to determine the polio-free status for Australia, which is reviewed each year by the Regional Commission for the Certification of the Eradication of Poliomyelitis in the Western Pacific Region, convened by WHO.
2. Congenital cytomegalovirus infection is a leading cause of congenital abnormality in Australia. Through this study, APSU aims to provide baseline data on which to base prevention and treatment strategies including trials of potential vaccines and antiviral drugs.
3. Congenital rubella is a vaccine preventable condition rarely seen in Australia. APSU provides a mechanism for identifying cases of congenital rubella, which in recent years have been due to missed school based immunisation or importation. APSU data are useful for monitoring the epidemiology and will inform the development of prevention strategies.
4. Perinatal exposure to HIV is the most frequently reported source of HIV infection in Australian children. The current study monitors new cases of perinatal exposure to HIV and HIV infection in children aged less than 16 years. Information collected through the APSU complements data collected through the National HIV Registry and the National AIDS Registry.
5. Neonatal herpes simplex virus (HSV) infection is a very rare, but serious infection with high morbidity and mortality. Through this study APSU will determine the incidence, mortality and morbidity of neonatal HSV infection in Australia.
6. Hepatitis C virus (HCV) infects an estimated 170 million people worldwide representing a viral pandemic with no vaccine yet available. This study aims to determine the reported incidence of newly diagnosed HCV infection and the prevalence of co-infection with hepatitis B virus and/or HIV in Australian children.
7. Non-tuberculous mycobacteria (NTM) are important environmental pathogens that cause a broad spectrum of diseases. The annual incidence of NTM infections in the developed world is believed to be increasing due to increasing awareness, better identification techniques and changing population groups. However, the magnitude of this problem in children is unquantified. APSU aims to expand on knowledge recently gained in Australia through laboratory surveillance by collecting data on the presentation, treatment and outcome of NTM infection.
8. Neonatal group B streptococcus (GBS) infection is the most common cause of life threatening infections in newborn babies. Early diagnosis and intervention can help decrease the risk of complications. This study aims to determine the incidence of early and late onset infections caused by GBS in Australia and to assess the effectiveness of intrapartum antibiotic use.

9–10 Routine, varicella vaccination is recommended in the latest Immunisation Program Schedule.[1] APSU surveillance for congenital and neonatal varicella provides a unique opportunity to compare current rates and the sources of infection in Australia, to rates reported by the APSU in 1995–1997. Surveillance for severe complications of varicella infection will allow us to describe serious presentations of varicella and through genotyping to identify varicella strains associated with severe complications. Genotyping will also

allow differentiation of vaccine strain from wild-type virus and determine true vaccine failures. These data will inform future vaccine and policy development.[2]

Methods

APSU study protocols are developed with collaborating investigators and/or institutions. Detailed protocols including case definitions for each condition under surveillance are available online.[3] APSU sends monthly report cards listing the conditions under surveillance to approximately 1,270 child health clinicians around Australia. Report cards are returned whether the clinician has a case to report or not, providing a measure of participation rates for the system. Sixty-five per cent of cards are sent and returned via e-mail, the rest via surface mail. All reported cases are followed-up by questionnaire requesting data on the presentation, treatment and short-term outcome.

The APSU aims to provide epidemiological information that is representative of the Australian population and maximal case ascertainment is a high priority. Despite a representative mailing list (93% of all paediatricians in active clinical practice in Australia participate in monthly surveillance) and high response rates (over 90% per annum since 1993), complete case ascertainment is unlikely.[4] This is particularly relevant in remote communities where children have limited access to paediatricians. However, for most conditions studied by the APSU no alternative national data are available to estimate completeness of ascertainment. APSU encourages the use of complementary data sources where available and reporting by a range of specialists to maximise case ascertainment. Reported rates for conditions ascertained through the APSU therefore represent a minimum estimate of the incidence of these conditions in the relevant Australian populations.

Results

All data provided in this report are accurate as at June 2008. It is possible that some notifications may be reclassified or the outcomes may change as additional clinical data are received. In 2007, 1,277 clinicians participated in the monthly surveillance of 16 uncommon childhood conditions, including the 10 communicable or vaccine preventable diseases described here. The report card return rate for 2007 was 92.3%. Enhanced data about diagnosis, clinical management and short-term outcome were available for more than 85% for cases notified. Table 1 shows the number of cases reported in 2007 and for the whole study period and the reported rate per 100,000 population.

Table 1. Confirmed cases identified for 2007 and for the total study period

Condition	Date study commenced	Questionnaire response (%) for total study period	Number of confirmed cases for 2007	Reported Rate for 2007 (per 10^5)	Number of confirmed cases for total study period	Reported rate for total study period (per 10^5per annum)
Acute flaccid paralysis	March 1995	89	26*	0.6†	438*	0.8†
Congenital cytomegalovirus	Jan 1999	68	12	4.5‡	87	3.8‡
Congenital rubella (with defects)	May 1993	95	Nil	Nil	50	0.1†
Perinatal exposure to HIV	May 1993	88	29	10.1‡	331	8.9‡
Neonatal herpes simplex virus infection	Jan 1997	95	7	2.6‡	95	3.4‡
Hepatitis C virus infection	Jan 2003	86	6	0.2†	47	0.2†
Non-tuberculous mycobacteria	July 2004	79	6	0.2†	44	0.3†
Neonatal group B streptococcus infection	July 2005	83	46	17.7‡	135	20.4‡
Congenital varicella	May 2006	100	1	0.4‡	2	0.5‡
Neonatal varicella	May 2006	89	5	1.9‡	13	3.3‡
Severe complications of varicella	May 2006	74	6	0.2†	18	0.3†

* All reported cases that have been classified by the Polio Expert Committee were 'non-polio acute flaccid paralysis' according to WHO criteria.

† Based on population of children aged ≤15 years as estimated by the Australian Bureau of Statistics.[6]

‡ Based on number of births as estimated by the Australian Bureau of Statistics.[6]

APSU data contribute significantly to the national surveillance effort, providing valuable information for clinicians, policymakers and the community.[4,6] The APSU is often the only source of national data that includes clinical and/or laboratory details, and data on both inpatients and outpatients.[4,6] The key findings for infectious diseases under surveillance by the APSU in 2007 are summarised in Table 2.

Studies concluding in 2007

Surveillance for NTM infection finished in September 2007. Adequate data have been collected in order to address the specific aims for this surveillance as determined by the investigators group leading this study, and a journal article is in preparation.

New surveillance studies started in 2007

Acute rheumatic fever

The acute rheumatic fever (ARF) surveillance study is a joint project of the Menzies School of Public Health, The National Heart Foundation of Australia and the APSU. Surveillance commenced in September 2007. The significant burden of ARF

Table 2. Results summary

Condition and principal investigator	Key findings
Acute flaccid paralysis (AFP) Dr Bruce Thorley, Victorian Infectious Diseases Reference Laboratory	In 2007, Australia failed to reach the WHO AFP surveillance target of 1 case per 100,000 aged less than 15 years per annum with only 26 confirmed cases. The primary causes of AFP are Guillain-Barré syndrome and transverse myelitis. Adequate faecal specimens were obtained for 58% of eligible cases which was an improvement on 2006 but below the 80% WHO target. In July 2007, the regional polio reference laboratory in Melbourne, Australia, reported isolation of a type 1 wild poliovirus, from a stool sample of a 22-year-old Pakistani man who had returned to Australia. This is the 1st case of wild polio in Australia in 30 years, and illustrates the need for continued vigilance to detect importations of poliovirus into Australia.
Congenital cytomegalovirus (cCMV) infection Prof. William Rawlinson, Virology Division, Department of Microbiology, Prince of Wales Hospital, Sydney	cCMV is the most common infectious cause of malformations in Australia. cCMV infection was not associated with maternal illness in approximately one third of cases, and should be considered regardless of maternal history. cCMV remains under-diagnosed. Although most cases are diagnosed by urine culture; use of polymerase chain reaction (PCR) for urinary screening for CMV may increase diagnostic yield. Universal neonatal hearing screening programs may also help identify new cases.
Congenital rubella (with defects) A/Prof. Cheryl Jones, The Children's Hospital at Westmead and Discipline of Paediatrics and Child Health, University of Sydney	There were no cases of congenital rubella with defects reported in 2007. As the risk of congenital rubella remains, particularly among immigrant women born in countries with poorly developed vaccination programs, such women should have serological testing for rubella after arrival in Australia, and vaccination when appropriate. Travel to rubella endemic countries in the 1st trimester by women with no prior rubella immunity poses a risk to the foetus of congenital rubella.
Perinatal exposure to HIV and HIV infection Ms Ann McDonald, National Centre in HIV Epidemiology and Clinical Research	In 2007, 29 cases of perinatal exposure to HIV were reported. Four children were born to women whose HIV infection was diagnosed postnatally. HIV infection has been confirmed in three of these children, while 1 child is HIV negative. Twenty-five children were born in Australia to women whose HIV infection was diagnosed antenatally. None of these children have been diagnosed with HIV infection. Twenty-three mothers used antiretroviral therapy in pregnancy and avoided breastfeeding. Antiretroviral treatment and mode of infant feeding were not reported for 2 women. Antenatal diagnosis of the mother's HIV infection and use of interventions continues to minimise the risk of mother-to-child HIV transmission.[7]
Neonatal herpes simplex virus infection (HSV) A/Prof. Cheryl Jones, Herpes Virus Research Unit, The Children's Hospital at Westmead and Discipline of Paediatrics and Child Health, University of Sydney	Over a half of neonatal HSV infections in Australia are caused by HSV type 1, in contrast to the United States of America where HSV type 2 predominates. Typical herpetic lesions of the skin, eye or mouth were not evident in half of infants identified with neonatal HSV infection, which makes early diagnosis difficult. Disseminated HSV infection in the newborn may be associated with the early onset of pneumonitis, in infants (in whom the chest X-ray may be normal). This is highly lethal unless antiretroviral therapy is initiated. Intrauterine HSV infection is rare. It manifests as chorioretinitis, intracerebral calcification, and birth defects.

Table 2: Results summary, *continued*

Hepatitis C virus infection (HCV) A/Prof. Cheryl Jones, The Children's Hospital at Westmead and Discipline of Paediatrics and Child Health, University of Sydney	Perinatal transmission is the main source of HCV infection in Australian children. In the APSU study, infected infants were born to mothers with hepatitis C who used intravenous drugs, had invasive procedures overseas or had tattoos. Most HCV-infected children were clinically asymptomatic with mildly elevated liver function tests at diagnosis, however, HCV induced chronic liver disease and liver failure have been reported among older children.[8] Given that 1%–2% of Australian women of childbearing age are infected with HCV, the reported rate of infected children is lower than predicted. This may be due to the lack of a consistent approach to screening to identify exposed children and HCV infection.[9]
Non-tuberculous mycobacterium infection (NTMI) Dr Pamela Palasanthiran, Paediatric Infectious Diseases Specialist, Department of Immunology and Infectious Diseases, Sydney Children's Hospital Randwick, NSW	This infection usually presents as lymphadenitis predominantly in immunocompetent children. *Mycobacterium avium intracellulare* and *Mycobacterium fortuitum* are the most common organisms isolated in Australian children. *Mycobacterium lentiflavum* is associated with higher relapse rates than other organisms. Microbiology (stain, culture or PCR) is 75.5% sensitive in identifying mycobacterial infection. Surgery is the most frequently offered therapy. Complete surgical excision is associated with a lower risk of relapse. There is marked heterogeneity in the type of antimicrobials used and course prescribed. Despite therapy relapse occurs in about 23% of cases.[10]
Neonatal and infant *Streptococcus agalactiae* (group B streptococcus – GBS) sepsis Prof. Lyn Gilbert Centre for Infectious Diseases and Microbiology, Institute for Clinical Pathology and Medical research, Westmead Hospital, Westmead NSW	Over a half (59%) of the reported cases have early onset of disease (at less than 8 days of age). The number of notifications received so far are consistent with other available data. Reported rates of confirmed GBS infection were higher in New South Wales/Australian Capital Territory than in other states during 2007. Pre-term birth is significantly associated ($p < 0.05$) with late-onset cases (infants aged 9 days or more). Group B streptococcus isolates have been collected for approximately 70% of cases and will be genotyped.
Severe complications of varicella infection Prof. Robert Booy National Centre for Immunisation Research and Surveillance, The Children's Hospital at Westmead, NSW	Six children were hospitalised with complications of varicella in 2007 (median age = 6 years; range 9 months to 12 years). Complications included bacteraemia, osteomyelitis, cellulitis, pneumonia, and ataxia. Median stay in hospital was 10 days (range: 5–18 days). All children were unvaccinated and family members were the infecting contacts. Severe complications of varicella pose a significant burden on affected children.
Congenital and neonatal varicella As above	One case of congenital varicella was reported in New South Wales in 2007. The infant was infected during the gestation period and the mother received anti-viral therapy. Five cases of neonatal varicella were reported. One neonate with pneumonitis was ventilated. Family members were the infecting contacts for both congenital and neonatal varicella.

has been recognised among Indigenous children and control programs and data collections in the top end of Australia have been invaluable. However, we know little about the incidence of ARF in the rest of Australia although the Australian Bureau of Statistics estimates that approximately 30% of Australia's Indigenous population lives in New South Wales.[11] The incidence of ARF in the non-Indigenous population is unknown, and this study may provide preliminary information on other high risk groups such as refugees. In order to improve surveillance coverage in rural and remote regions the APSU will recruit additional key clinicians from these areas. This is an important capacity building step for the APSU surveillance mechanism.

Intussusception

Surveillance for intussusception (IS) commenced in July 2007 after the introduction of rotavirus vaccination onto the Australian Immunisation Schedule,[1] and this study is led by Professor Julie Bines from the Department of Gastroenterology, Royal Children's Hospital, Melbourne. IS has been recognised as a potential complication of rotavirus vaccination,[12]

and the APSU study will provide information on the diagnosis and clinical management of intussusception and any temporal association between rotavirus vaccination and intussusception.

Influenza

In September 2007, APSU was commissioned to conduct rapid response surveillance for severe complications of influenza in children aged less than 5 years. The study was mounted within 10 days of commissioning and weekly rather than monthly surveillance was conducted. The results from this 1 month trial suggest that surveillance for severe complications of influenza is feasible and could again be conducted during the influenza season in 2008. If the study was repeated we would include children aged less than 15 years as there were several reports of very serious complications of influenza in this age group in 2007.[13]

Future directions

APSU surveillance provides valuable detailed clinical, treatment and outcome data on several infectious or vaccine preventable conditions simultaneously and is a valuable adjunct to other national surveillance systems. However, APSU surveillance of conditions such as AFP, where biological samples are required and timely identification of cases is essential, could be improved. To address these limitations APSU, in collaboration with the National Centre for Immunization Research and Surveillance, is currently piloting a Paediatric Active Enhanced Disease Surveillance (PAEDS) system in 4 tertiary paediatric hospitals in 4 states of Australia, with reporting by dedicated nurse specialists.[14]

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Peer-reviewed articles

Prospective surveillance of excess mortality due to influenza in New South Wales: feasibility and statistical approach

David J Muscatello, Patricia M Morton, Ingrid Evans, Robin Gilmour

Abstract

Influenza is a serious disease that seasonally causes varying but substantial morbidity and mortality. Therefore, strong, rapid influenza surveillance systems are a priority. Surveillance of the population mortality burden of influenza is difficult because few deaths have laboratory confirmation of infection. Serfling developed a statistical time series model to estimate excess deaths due to influenza. Based on this approach we trialled weekly monitoring of excess influenza mortality. Weekly, certified death information was loaded into a database and aggregated to provide a time series of the proportion of all deaths that mention pneumonia or influenza on the death certificate. A robust regression model was fitted to the time series up to the end of the previous calendar year and used to forecast the current year's mortality. True and false alarm rates were used to assess the sensitivity and specificity of alternative thresholds signifying excess mortality. Between 1 January 2002 and 9 November 2007, there were 279,968 deaths registered in New South Wales, of which 77% were among people aged 65 years or more. Over this period 33,213 (12%) deaths were classified as pneumonia and influenza. A threshold of 1.2 standard deviations highlighted excess mortality when influenza was circulating while providing an acceptable false alarm rate at other times of the year. Prospective and reasonably rapid monitoring of excess mortality due to influenza in an Australian setting is feasible. The modelling approach allows health departments to make a more objective assessment of the severity of seasonal influenza and the effectiveness of mitigation strategies. *Commun Dis Intell* 2008;32:435–442.

Keywords: influenza, surveillance, Serfling, excess mortality, robust regression, false alarm rate, sensitivity, specificity, seasonal

Introduction

Influenza, an acute viral disease of the upper respiratory tract, is a major threat to public health worldwide because of its capacity for distinct mutation (antigenic shift) that can result in rapid spread through populations and widespread morbidity and mortality.[1–3] Even in the absence of such pandemics, seasonal influenza epidemics cause substantial burden of morbidity and mortality annually.[4–9] More frequent, minor mutations (antigenic drift), cause substantial variability in the impact of seasonal epidemics.[2,3] The capricious nature of influenza demands constant vigilance to ensure seasonal vaccines are appropriate to current strains, and supplies of these and antiviral medications are sufficient for potential need. For these reasons, strong influenza surveillance systems are a priority for health departments.[10]

Assessing the population burden of influenza is difficult. Symptoms are non-specific and few clinical influenza diagnoses are laboratory confirmed. Hospitalisations and deaths from influenza are often due to secondary complications such as pneumonia that occur well after the initial influenza virus infection.[11] Therefore, influenza may not be listed on death certificates for many influenza related deaths because it is not recognised as the underlying cause of the condition.

Because of these difficulties, statistical models were developed to estimate the burden of mortality caused by influenza. As early as 1932, Collins determined that excess mortality during winter months in the United States of America (USA) was a consequence of epidemic influenza and therefore could be used as an indicator for the recognition of influenza outbreaks.[12] In 1963, Serfling described a regression model, based on the seasonal pattern of pneumonia and influenza (P–I) deaths, to infer excess deaths due to influenza. Since then, Serfling's model has been applied in a number of temperate countries (USA,[13,14] France,[14] Australia,[14] Italy[15]) to demonstrate that excess mortality occurring during winter months is associated with pandemic and seasonal epidemics of influenza.

The US Centers for Disease Control and Prevention (CDC) took the approach further and established the 122 Cities Surveillance System to provide timely, prospective information of excess mortality due to influenza. The system collects and reports weekly

counts by age of all deaths registered, usually within a week of the date of death, in 122 cities around the country. Deaths due to P–I are also counted, so that the weekly proportion of all deaths due to pneumonia and influenza can be monitored. The system covers approximately one third of all deaths in the USA and allows epidemiologists to determine an early quantitative estimate of the severity of an influenza epidemic.[16,17]

In Australia, prospective seasonal surveillance of influenza is largely based on: targeted laboratory surveillance, specific subtype and strain identification of circulating influenza viruses by the World Health Organization Collaborating Centre for Research on Influenza in Victoria; and general practice or emergency department consultation rates for influenza-like illness (ILI).[18] The New South Wales Influenza Surveillance Program runs from May to September each year and has primarily monitored the proportion of influenza positive specimens from all respiratory specimens from the major public health laboratories, the proportion of emergency department visits diagnosed with influenza, and outbreaks of influenza reported to Public Health Units by residential care facilities.[19]

This paper describes the introduction of prospective monitoring of excess P–I mortality due to influenza in New South Wales and demonstrates a sound and repeatable statistical approach to the selection of a threshold for signalling excess mortality due to influenza.

Methods

Data source

Under the *New South Wales Public Health Act 1991,* the Registrar of Births, Deaths, and Marriages is required to make death registry information available for inspection by the NSW Department of Health.[20] Prior to 2007, incremental, password-protected updates in the form of a data file were transmitted monthly to the Department of Health's Population Health Division. Each update was saved on a secure, password-protected server[21] using SAS statistical software[22] and was only accessible by authorised departmental officers. In early 2007 the Registry of Births, Deaths and Marriages commenced providing weekly updates of the information.

The data file contains an electronic copy of the Medical Certificate of Cause of Death (death certificate) for each death certified in New South Wales. The certificate includes a section describing the disease or condition directly leading to death, and any antecedent causes, co-morbid conditions, or other significant contributing conditions. Deaths are required to be certified within 7 days. Deaths referred to a Coroner are not immediately certified and are therefore not available until completion of the coronial inquest.

For the period the data were provided weekly, we assessed the timeliness of the mortality data by calculating the median interval between the date of each death and the date it was provided to the Department of Health.

Identification of pneumonia and influenza deaths

The SAS program that loads the data includes a module that scans the cause of death text for the text strings 'PNEUMONIA' or 'INFLUENZA', including some common misspellings (available on request). The words 'HAEMOPHILUS INFLUENZAE' and 'ASPIRATION' (for aspiration pneumonia) are excluded. The resulting dataset includes the date of death and a flag indicating whether either of the 2 conditions was mentioned. The data are then aggregated into weekly counts by date of death to provide a time series of the proportion of all deaths that mention pneumonia or influenza on the death certificate.

To validate the automatic classification of P–I deaths using the SAS program above, we created 3 samples of death certificates from the complete death certificate dataset from 1 January 2002 to 9 November 2007: 299 deaths that were automatically categorised as influenza or pneumonia, 120 deaths categorised as influenza and 299 deaths categorised as neither influenza or pneumonia. There were insufficient deaths mentioning influenza to obtain a sample of 299. The 'uniform' function in SAS software was used to select the random samples. Author PM manually compared the cause of death text with the categorised value. Any inconsistency was defined as a misclassification.

Statistical analysis

The time series of the proportion of P–I deaths is strongly seasonal with a winter peak. Serfling's method involved fitting a seasonally cyclical linear regression model to the time series of weekly proportions of P–I deaths. The details of the model specification are described below. Because the influenza epidemics cause excess deaths, or outliers, in the observed data, the model fitting could be overly influenced by the epidemic behaviour it is trying to detect. For this reason, Serfling manually excluded past epidemics from the model fitting. An upper threshold was chosen to define the upper limit of the expected proportion of P–I deaths in the absence of an influenza epidemic. The threshold was the predicted proportion in a given week plus a constant multiple of the standard error of the time series of

differences between each value predicted by the model and the actual observed values (the 'model residuals'). Serfling chose the constant multiple to be 1.64 standard errors, and considered 2 consecutive weeks above the threshold to indicate epidemic behaviour.[23]

During the 2007 influenza season we implemented a simplification of Serfling's method to demonstrate the feasibility of weekly, prospective excess P–I mortality monitoring. Each week, we fit a cyclic linear regression model to the full time series including data for the previous 5 calendar years and the current year-to-date. The threshold indicating an excess proportion of deaths due to pneumonia or influenza was defined as the upper 95% confidence limit of the expected mean proportion of P–I deaths predicted by the model, as output by PROC REG in SAS statistical software.[23] Graphs of the raw time series and the upper threshold using a model fit to the latest available data were incorporated into weekly seasonal influenza surveillance reports.

This paper concentrates on a more sound variation of the above approach similar to that adopted by the US CDC.[24] The approach involves fitting the same cyclic linear regression model, but using a 'robust' estimation procedure for fitting the model. Robust regression down-weights the influence of extreme observations (outliers) in the model-fitting procedure.[25] The model is fit to the 5-year time series up to the end of the previous calendar year and the model is used to forecast expected behaviour for the current year. Forecasting the current year's time series from data up to the end of the previous year ensures the threshold is consistent from week to week in the current year.

The cyclic regression model includes: a linear time term, t, with values 1, 2, 3,... for each week of the time series, and the square of the time term, t^2, to accommodate long-term linear and curvilinear changes in the background proportion of P–I deaths arising from factors such as population growth or improved disease prevention or treatment. Also included are annual seasonal harmonic variables to describe the cyclical seasonal background pattern. The harmonic variables are functions of the week number, t, and the periodicity in the same units – in this case, yearly (52.18 weeks). The 2 harmonic variables in this case are: $sine(2\pi t/52.18)$ and $cosine(2\pi t/52.18)$.

The final model was:

$$\text{Expected(proportion)} = A + Bt + Ct^2 + D\,sine(2\pi t/52.18) + E\,cosine(2\pi t/52.18)$$

where A, B, C, D, and E

To evaluate the approach, we fitted the model to the 5-year time series from 1 January 2002 to 31 December 2006 using PROC ROBUSTREG in SAS Software with the simplest, default 'M estimation' method.[26] We then used the PROC SCORE procedure to forecast values for the 2007 year.[23]

Threshold identification

To identify a threshold at which to signal excess mortality, an estimate of the expected variability of the weekly proportion of P–I deaths is required. If an observed proportion exceeds the expected range of variability, then excess mortality can be signalled. Because PROC ROBUSTREG only offers limited statistical output from the model-fitting procedure, one of us (DM) wrote a SAS program (available on request) to calculate the 'standard error of prediction' of the model. The formula is equivalent to that of the 'STDI' parameter available from other regression procedures in SAS.[26]

The standard error of prediction is a more logical choice for assigning the threshold of excess mortality than the root mean square error (standard error of the model residuals) because it incorporates not only the variance of the residuals but also the variance of the model parameter estimates.[27] This provides an estimate of the expected variability of the observed values in the absence of influenza epidemics.

To determine the best threshold of excess mortality due to influenza, we varied the constant factor by which the standard error of prediction was multiplied from 0.1 to 5 standard errors in increments of 0.1. We then calculated the true positive and false positive signalling rates (threshold exceedences) of each threshold. A true positive signal was a weekly exceedence occurring during an influenza season and a false positive was a weekly exceedence occurring outside the influenza season in each year. We defined influenza seasons to be periods where 4-week moving average counts of laboratory-notified influenza cases from the New South Wales notifiable diseases database[21] were above 30. This was the lowest level that clearly discriminated seasonal from unseasonal activity in the study period. The true and false positive rates of surveillance signals are equivalent to the sensitivity (the true positive rate) and specificity (=1-false positive rate) of a laboratory test. Thus a graph describing the relationship between these 2 quantities at different thresholds ('receiver operating characteristics' (ROC) curve) can be plotted to assist in choosing a threshold that provides the surveillance system with the most useful balance between sensitivity and specificity. The complete time series available at the time of the study, 1 January 2002 to 9 November 2007, was used

in assessing the true and false positive rates with the weekly threshold determined from the combined fitted model and 2007 forecast described above.

Ethics approval was not required for this study as it used data collected and used in accordance with New South Wales legislation for the purpose of health protection. Identifying variables and codes that could be used for re-identifying individuals were excluded from the study data.

Results

Timeliness of the data

For the period when data were supplied weekly to the Department of Health, May to November 2007, the median interval between the date of death and the date of registration was 10 days, and the median interval between registration and receiving the information at the Health Department was an additional 5 days.

Descriptive analysis of mortality statistics

Between 1 January 2002 and 9 November 2007 there were 268,048 deaths registered in New South Wales, of which 33,220 (12%) were classified as P–I deaths. The age distribution of P–I deaths was older than that of all deaths combined, with 79% of P–I deaths in persons aged 75 years or more, compared with 64% in all deaths combined (Table 1). Influenza was mentioned only rarely in the cause of death text. Of the P–I deaths, 61 (0.2%) mentioned influenza and not pneumonia in the cause of death text, 59 (0.2%) mentioned both pneumonia and influenza, while the remaining 33,100 (99.6%) mentioned pneumonia only.

Applying the 4-week moving average criterion to influenza notifications from laboratories, influenza seasons occurred in the periods: 14 June 2002 to 6 September 2002, 25 July 2003 to 12 September 2003, 20 August 2004 to 22 October 2004, 1 July 2005 to 23 September 2005, 28 July 2006 to 8 September 2006, and 22 June 2007 to 4 September 2007. During these periods combined, a somewhat lower proportion of P–I deaths were in persons aged 45–54 years or 65–74 years, while a greater proportion were aged 85 years or more (Table 2).

Validation of automated classification of pneumonia and influenza deaths

From the 3 random samples: (1) deaths categorised as flu or pneumonia; (2) those categorised as influenza; and (3) those classified as neither influenza or pneumonia, there were 1 (0.33%), 0 (0.00%), and 1 (0.33%) misclassifications, respectively. These were a result of spelling errors in the cause of death text resulting in the wrong category being assigned. This indicates that misclassification was negligible.

Routine surveillance reporting, 2007

During most of the 2007 influenza surveillance reporting season, data were provided weekly by the New South Wales Registry of Births, Deaths, and Marriages and a graph similar to Figure 1 was included in the weekly surveillance report.[19] Clear

Table 1. Age distribution of all deaths and pneumonia and influenza deaths, 1 January 2002 to 1 November 2007

Age group (years)	All deaths		Pneumonia & influenza deaths	
	n	%	n	%
0-1	2,475	0.9	31	0.1
2-4	296	0.1	16	0.1
5-14	555	0.2	45	0.1
15-24	2,281	0.9	70	0.2
25-34	3,604	1.3	86	0.3
35-44	6,236	2.3	241	0.7
45-54	12,802	4.8	588	1.8
55-64	23,985	9.0	1,614	4.9
65-74	43,872	16.4	4,143	12.5
75-84	86,062	32.1	11,350	34.2
85 or more	85,800	32.0	15,029	45.2
Total	268,048*	100.0	33,220*	100.0

* Age-specific deaths do not sum to the total, due to 80 deaths (including 7 pneumonia and influenza deaths) with missing age information.

Table 2. Contribution of each age group to pneumonia and influenza deaths during influenza and non-influenza seasons, 1 January 2002 to 1 November 2007

Age group (years)	Non-influenza season % (95% confidence interval) (n=23,774)		Influenza season % (95% confidence interval) (n=9,446)	
0-1	0.08	(0.05-0.12)	0.13	(0.07-0.22)
2-4	0.04	(0.02-0.08)	0.06	(0.02-0.14)
5-14	0.12	(0.08-0.17)	0.18	(0.10-0.29)
15-24	0.21	(0.16-0.28)	0.20	(0.12-0.31)
25-34	0.27	(0.21-0.35)	0.22	(0.14-0.34)
35-44	0.72	(0.61-0.83)	0.75	(0.59-0.95)
45-54	1.90	(1.73-2.08)	1.45	(1.22-1.71)*
55-64	4.97	(4.70-5.25)	4.58	(4.17-5.03)
65-74	12.90	(12.5-13.4)	11.30	(10.7-12.0)*
75-84	34.40	(33.8-35.0)	33.60	(32.7-34.6)
85 or more	44.40	(43.8-45.0)	47.40	(46.4-48.5)*

* The proportion differs significantly between the influenza and non-influenza seasons.

Figure 1. Example of simple mortality surveillance implemented during the 2007 influenza season, deaths due to pneumonia and influenza per 1,000 deaths

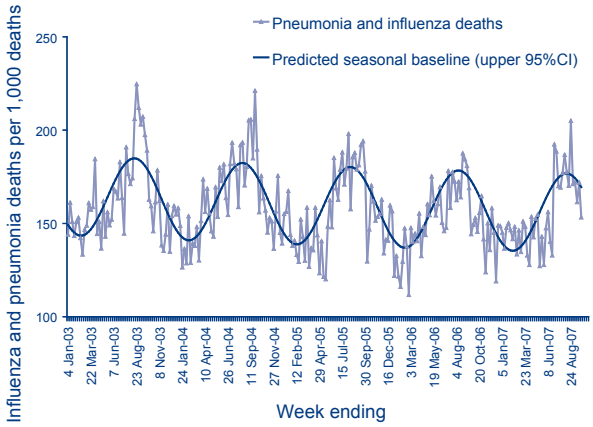
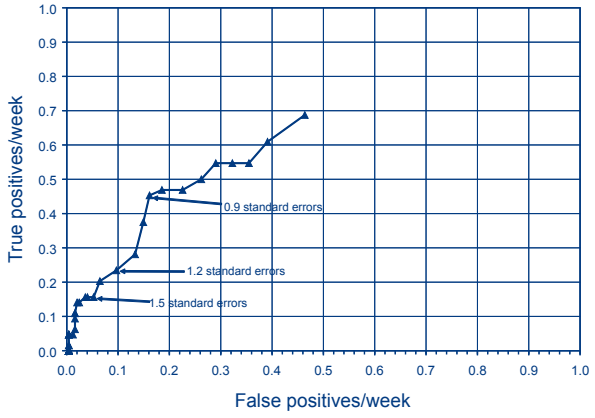


Figure 2. Relationship between the epidemic threshold and the true and false positive rates, indicating the rate of threshold exceedences per week during influenza and non-influenza seasons, respectively



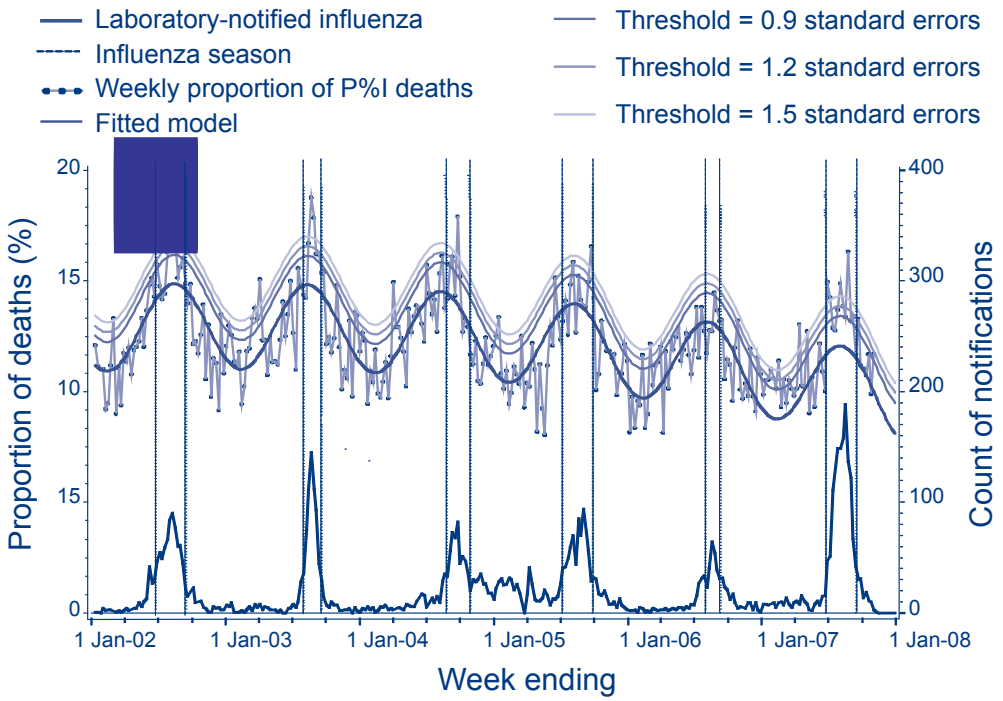
influenza season behaviour could be observed in 2003 and 2004. However, using a threshold of the upper 95% confidence interval of the predicted mean resulted in many weekly exceedences outside of the influenza seasons (false positives). This made the graphs difficult to interpret.

Improved method of modelling and threshold setting

Figure 2 shows the true and false positive rate of threshold exceedences in response to changing the threshold in multiples of the number of standard errors of prediction. Three thresholds are indicated, 0.9, 1.2 and 1.5 standard errors that provide a false positive rate of 0.16, 0.10 and 0.04 threshold exceedences per week, respectively. These equate to approximately 8, 5 or 2 weeks per year in which false alarm (non-influenza season) threshold exceedences occur, respectively. The corresponding true positive rates of 0.45, 0.23 and 0.16, respectively, are low and indicate many weeks during influenza seasons in which threshold exceedences do not occur.

Figure 3 shows the result of the robust regression modelling, along with the 3 thresholds highlighted above. The excessive number of non-influenza weeks in which the threshold is exceeded when 0.9 standard errors is used, is evident. This threshold would offer limited discriminatory power between influenza and non-influenza seasons. A threshold of 1.5 standard deviations excludes all but a few peaks during influenza seasons. A threshold of

Figure 3. Time series of raw weekly pneumonia and influenza mortality proportions, the fitted robust regression model, and 3 epidemic threshold curves. Weekly counts of influenza notifications from laboratories and the boundaries of the seasonal influenza periods



1.2 standard deviations appears to be the best compromise, and clearly highlights a high proportion of deaths occurring in at least 1 week of each of the 2002, 2003, 2004, 2005 and 2007 seasons.

Discussion

We found that prospective, largely automated monitoring of excess influenza mortality is possible in the Australian setting. Further, we demonstrated a sound statistical approach based on Serfling's methods for defining a surveillance threshold. A threshold of 1.2 standard errors of prediction provides an acceptable false positive rate that limits false alarms to 5 weeks per year while being sensitive enough to highlight some excess mortality in the years we studied. Epidemic sensitivity appears to be quite low, however, which could reflect either high levels of vaccine coverage in the population at risk, or relatively low virulence of circulating strains during the years studied. Another explanation for some relatively short-lived peaks in excess deaths could reflect different strains with varying virulence circulating at different times within seasons.[28,29]

An important question is whether to analyse the time series by date of death or date of death registration. In prospective surveillance the earliest available date closest to the exposure that caused infection is most representative of when the disease is circulating. In our case, the date of death is the earliest available date. While analysing by date of registration gives a date closer to the date of surveillance reporting, there is a risk of shifting the perceived onset of the epidemic forward in time. We found the interval between date of death and date of registration to be a median 10 days, and from date of death to date of receipt at the Health Department to be 15 days. With more real-time registry data extraction we could reduce the interval from registration to reporting, but this would still be more than 1 week from the date of death. In these circumstances, excluding the last data point from the time series prior to analysis and reporting is sensible.

We found that the majority of P–I deaths occurred in those aged 85 years or over and the contribution of this age group to deaths increased slightly but statistically significantly during influenza seasons. This finding is consistent with that of Thomson, et al with 23 years of USA mortality data.[8] Future work could evaluate whether applying these methods to age-specific data would improve detection of excess mortality due to influenza.

Clearly, mortality is the most extreme outcome of disease, and prospectively monitoring excess mortality due to influenza provides an objective perspective on the virulence of circulating strains or seasonal vaccine effectiveness. It could well prove valuable in assessing the impact of a pandemic.

Limitations

Unlike in the USA,[30] New South Wales influenza mortality data does not show clear peaks indicating influenza epidemics. This may partly be due to the smaller population and thus greater statistical variability; in 2007, the New South Wales population was around 7 million while the USA population was over 300 million. Another explanation could be higher influenza vaccination coverage for persons aged over 65 years in New South Wales (75% in 2006)[31] compared with the USA (64% in 2006).[32] Given that the majority of deaths are in the elderly, this could have some influence.

The best threshold we estimated is based on a limited time period that included a mix of observed and forecast data. In practice, it would be best to determine the threshold by calculating the true and false positive rates in a real, prospective scenario over many years. Also, some of the exceedences that we designated as false alarms may have been caused by respiratory syncytial virus, which, while estimated to have one-third the mortality risk of influenza, still does contribute to seasonal excess mortality.[8] Another factor that could be considered in future work is the delay between infection with influenza and death, which could be several weeks. While the moving average we used for the laboratory time series may have limited this problem, lagging the mortality time series relative to the laboratory time series could have improved the true alarm rate. In addition, we used the default settings in the PROC ROBUSTREG procedure. The procedure does allow the degree of weighting of outliers to be controlled, and provides alternative model estimation procedures. These options could be evaluated in future work.

The completeness and accuracy of the death registration information received by the NSW Department of Health needs to be evaluated. The laboratory notification data used to define influenza seasons also has some limitations. In recent years, increasing use of rapid influenza diagnostic tests may have inflated influenza notifications. Also, in late 2004 to early 2005 there was a known problem of false positive influenza notifications, evident in Figure 3.

Conclusions

Prospective and reasonably rapid monitoring of excess mortality due to influenza in an Australian setting is feasible and can provide valuable information on the impact of influenza on the population. Appropriate statistical methods for automatically identifying excess mortality are available and can be applied. The additional information can help health departments make a more objective assessment of the severity of seasonal and possibly pandemic influenza as well as the effectiveness of mitigation strategies.

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We are grateful to William Thompson and colleagues at the National Centre for Immunization and Respiratory Diseases, US Centers for Disease Control and Prevention, who provided helpful advice on the development of this study. We are also grateful to the New South Wales Registry of Births, Deaths and Marriages for consistently providing the death information, and to Tim Churches for establishing the death certificate data collection in HOIST. We also thank the peer reviewers for their instructive and helpful comments on the work.

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Influenza immunisation of doctors at an Australian tertiary hospital: immunisation rate and factors contributing to uptake

Jonathan Kaufman, Joshua Davis, Vicki Krause

Abstract

Immunisation of health care workers against influenza reduces influenza-related morbidity and mortality of hospital inpatients and staff absenteeism. Uptake of influenza vaccination amongst hospital doctors is generally inadequate, and factors contributing to influenza vaccine uptake among doctors have not been well defined. We performed an audit of doctors at an Australian hospital to establish the rate of and the factors contributing to influenza immunisation uptake. The audit was conducted by delivering a survey to doctors for self-completion at major departmental meetings. Of 243 doctors employed at the hospital, 150 completed the survey (response rate 62%), of whom only 28% received influenza immunisation in 2007 and 44% in any prior year. Doctors immunised in 2007 were of an older age (39.1 vs. 34.7 years, P=0.01) and level of seniority (odds ratio for consultant vs. more junior staff=2.9, P=0.02) than those not immunised. Doctors who had ever been immunised had a better knowledge about influenza than those never immunised (odds ratio for high knowledge score 4.2, P<0.001). The most common reasons cited for not being immunised in 2007 were being too busy, immunisation not being offered conveniently and not being aware how to access the vaccine. Immunisation rates among doctors in this study are inadequate. A perceived lack of convenience of the immunisation service and poor knowledge about influenza vaccination are the major contributing factors. Efforts to improve influenza immunisation uptake amongst hospital doctors should focus on education, and on innovative strategies to make immunisation more convenient and accessible specifically for doctors. *Commun Dis Intell* 2008;32:443–448.

Keywords: influenza vaccine, immunisation programs, physicians, hospital medical staff

Introduction

Around 1,500 Australians die each year from influenza related complications.[1] Doctors are often exposed to influenza during the course of their work, resulting in substantial rates of clinical and subclinical infection during the influenza season.[2] They may then act as vectors, passing infection to patients, staff, family and friends. It is important that influenza is recognised as a significant nosocomial illness, with high mortality in vulnerable groups.

There is good evidence that immunising health care workers (HCWs), including doctors, against influenza is cost-effective in reducing staff absenteeism,[3] and may also reduce morbidity and mortality in high-risk patients under their care.[3] In healthy adults aged under 65 years, immunisation has 70%–90% protective efficacy against influenza virus infection when the antigenic match between vaccine and circulating virus is close.[4]

The standard of care for influenza immunisation in Australia is unambiguous: annual influenza immunisation is recommended for all contacts of high risk patients such as HCWs, as stated in national guidelines such as *The Australian Immunisation Handbook*.[4] While a 100% uptake is desirable, it is not realistically achievable by even the most efficient hospital immunisation services. Studies have shown that 60% coverage provides significant protection,[5] therefore aiming for 80% coverage strikes a balance between the ideal and the achievable.

Bull et al found that in 2005 only 29% of doctors in Victorian public hospitals (with more than 100 beds) received influenza immunisation, according to a survey of hospital infection control staff, although this study collected data at a health service rather than individual level.[6] Other previous studies demonstrate a range of immunisation rates of overseas doctors, from 38%–82%.[7,8] This study sought to establish the rate of influenza immunisation uptake among doctors at an Australian tertiary referral hospital and the factors contributing to the uptake. A preliminary report of this audit has been previously published in a local bulletin.[9]

Methods

Setting

The Royal Darwin Hospital (RDH) is a 300 bed tertiary hospital and is the main teaching hospital in the Northern Territory of Australia. Influenza is a recommended (but not mandatory) immunisation for all HCWs. Annually updated influenza vaccine is available free of charge and promoted throughout March and April, with occasional catch-up clinics

in May. Immunisation is available from a drop-in staff clinic between 1 pm and 3 pm most days or by appointment on Friday afternoons. Mobile clinics are also conducted for 1–2 days on each ward, but immunisation is not available outside of business hours. The immunisation campaign is publicised with flyers in common areas and loudspeaker announcements.

Data collection

An anonymous, written survey was given to RDH doctors at the end of the 2007 hospital influenza immunisation campaign. The survey was delivered face-to-face at major meetings for each department, and self-completed independently.

The main information sought is summarised in Table 1. A knowledge score was obtained from 5 true or false questions derived from information in *The Australian Immunisation Handbook*.[4]

Table 1. Summary of survey questions

Had they received influenza immunisation in 2007, or in the past?
If they had (in 2007), what reasons motivated them?
If they had not, what were the perceived barriers?
A knowledge score about influenza immunisation
What might facilitate more doctors getting immunised?
What level of doctor (intern, RMO, registrar, consultant), speciality and age were they?

Statistics

Continuous variables were compared using student's t test if normally distributed, and Mann Whitney U test was used for non-parametric variables. Categorical variables were compared using chi^2 test. Multivariate analysis for the outcome of vaccination was done using logistic regression analysis, using a backward stepwise approach. All analyses were performed using Stata 10 (Statacorp, California). P values of <0.05 were considered significant.

Table 2. Survey respondents by level of seniority

	Total number at Royal Darwin Hospital	Number surveyed	% surveyed
Interns & RMO's	68	48	71
Registrars	94	56	60
Consultants	81	46	57
Total	243	150	62

Results

Characteristics of respondents

Of 243 doctors who work at RDH, 150 (62%) completed the survey. Of the 150 doctors surveyed, the distribution by level of seniority was representative of that of the hospital as a whole (Table 2), and proportionate for each major department (medicine, surgery, paediatrics, emergency, obstetrics and gynaecology, critical care, psychiatry). Overall, the knowledge about influenza infection and immunisation was good, with 84% of respondents scoring four or five out of five, and 16% scoring three or less.

Immunisation status

Of 150 respondents, 42 (28%) reported having received influenza immunisation in 2007. This was well below our desired standard of 80%. Of the 108 doctors (72%) not immunised in 2007, 66 (44%) had received influenza immunisation in any year prior to 2007. Given that annual immunisation is recommended for all doctors, it was surprising to find that 42 (28%) had never been immunised.

Predictors of immunisation in 2007

On univariate analysis, the only significant predictors of having been immunised in 2007 were higher age and level of seniority (Table 3). On multivariate analysis (controlling for age, area of work and knowledge score) only level of seniority remained an independent predictor.

Table 3. Predictors of having received influenza immunisation in 2007

	Immunised in 2007 (n=42)	Not immunised in 2007 (n=108)	P value	Adjusted OR* (95% CI)
Mean age	39.1	34.7	0.01	
Consultant (%)	45	25	0.02	2.9 (1.2–7.1)
Knowledge score of 5/5 (%)	71	56	0.08	1.8 (0.8–4.0)

* Adjusted using multivariate logistic regression, controlling for work area (e.g. medicine, surgery, ED etc), level of seniority and knowledge score. Age was not included in the model, as it strongly correlated with level.

Predictors of ever immunised

The only significant predictor of having ever received immunisation was a high knowledge score. Those with a knowledge score of five out of five were 4.2 times more likely to ever have been immunised as those with a knowledge score of four or less (Table 4).

Factors facilitating influenza immunisation uptake in 2007

Among doctors immunised in 2007, most did so to protect themselves, their patients, family and friends against influenza (Table 5).

All respondents, regardless of whether they had received immunisation or not, were also asked an open-ended question: what they thought might facilitate and encourage more doctors to receive influenza immunisation at RDH. Of the 112 respondents who answered this question, 50 (45%) suggested that the immunisation service needed to be more convenient, and 25 (22%) suggested there needed to be more reminders about when immunisation was available. Additionally, 15 (13%) suggested more education was required, 10 (9%) suggested bribery, nine (8%) suggested making immunisation compulsory and three (3%) suggested peer pressure would help influence their decision.

Impediments to immunisation uptake in 2007

Among doctors who did not receive influenza immunisation in 2007, the most common reasons given related to accessing the immunisation service, with 30% being too busy, 29% saying immunisation was not offered conveniently and 26% being unaware of how to access the vaccine (Table 6). Reasons relating to the actual vaccine were far less common. No respondents reported not receiving the vaccine due to a contra-indication.

Previous adverse reactions to influenza immunisation

Rates of adverse reactions were consistent with rates suggested in *The Australian Immunisation Handbook*[4] and were minor in nature. The majority of respondents (87%) had never had a previous adverse reaction to influenza immunisation. Fifteen (10%) reported experiencing a mild flu-like illness and four (3%) reported local soreness after previous immunisation.

Location of immunisation

Of the 42 doctors who received influenza immunisation in 2007, 38 (91%) did so through RDH staff immunisation service (65% in staff clinic, 26% on the wards). Only three (7%) received their vaccine elsewhere and 1 respondent had self-administered it.

Table 4. Predictors of ever having received influenza immunisation

	Immunised ever (n=108)	Never immunised (n=42)	P value	Adjusted OR* (95% CI)
Mean age	36.3	35.8	0.22	
Consultant (%)	32	29	0.7	0.83 (0.3–2.0)
Knowledge score of 5/5 (%)	70	35	<0.001	4.2 (1.9–9.1)

* Adjusted using multivariate logistic regression, controlling for work area (e.g. medicine, surgery, ED etc), level of seniority and knowledge score. Age was not included in the model, as it strongly correlated with level.

Table 5. Reasons given for receiving influenza immunisation in 2007*

Reason immunised	Number citing reason	Percentage citing reason
Protect self	38	90
Protect patients	30	71
Protect family/friends	24	57
Offered conveniently	24	57
Reduce sick-leave	18	43
Encouraged by peers	11	26
Recommended in guidelines	7	17

* More than 1 reason could be cited by each respondent.

Table 6. Reasons given for not receiving influenza immunisation in 2007*

Reason not immunised	Number citing reason	Percentage citing reason
Too busy	32	30
Not offered conveniently	31	29
Unaware how to access vaccine	28	26
Concern re: flu-like illness	16	15
Forgot	15	14
Other	15	14
Concern re: adverse reaction	12	11
Unlikely to catch flu	10	9
Don't believe evidence/guidelines	8	7
Unlikely to spread to patients	3	4
Unlikely to spread to family/friends	3	3

* More than 1 reason could be cited by each respondent.

Discussion

We have found a disappointingly low immunisation rate among RDH doctors for influenza in 2007 (28%), and a significant number of doctors who have never received influenza immunisation (28%). The only significant predictor of having been immunised in 2007 were higher age and more senior level, and the most common reasons cited for not being immunised were lack of time and inconvenience. We also found a higher level of knowledge about influenza immunisation was strongly associated with ever having received immunisation.

Access and convenience

Some current strategies to increase the accessibility of immunisation, such as mobile ward-based clinics at limited times, may not be as effective for doctors as for staff who have more reliable breaks such as allied health professionals and nurses. Offering immunisation at times and places specifically convenient for doctors, such as at major meetings and education sessions, has been shown to improve compliance.[10] Increasing the availability of drop-in clinics and extending the service to out-of-hours is also likely to be beneficial.

Some respondents who were not immunised in 2007 (26%) were not aware of how to access the immunisation service, and 14% just forgot. Effective publicity thus appears important to ensure all doctors are aware of the immunisation program and how to access it. Several reminders may be needed, such as sending targeted messages to doctors who have not yet received immunisation.

When all doctors (not just those immunised in 2007) were asked what might facilitate more doctors receiving influenza immunisation, their responses that greater convenience (45%) and more reminders (22%) were needed further reinforce these notions.

Motivation and education

The findings that protection of self (90%) and others (patients 71%) (family/friends 57%) are the major factors motivating doctors to receive influenza immunisation is consistent with studies overseas: 93% of the 205 American doctors surveyed by Wodi et al cited self-protection as a reason for receiving immunisation.[7]

Knowledge about influenza immunisation was good overall with 84% of all respondents having high levels of knowledge scores (4/5 or 5/5), but higher levels of knowledge about influenza and its consequences was strongly associated with ever-having received immunisation. This result is also consistent with other studies: Wodi et al. found that doctors ever-immunised had higher knowledge scores than doctors never-immunised.[7] Martinello et al however found in their study that knowledge influences immunisation uptake for nurses but not for doctors.[8]

Study limitations

We have assumed that the convenience sampling method we used would give us a representative sample of the cohort of doctors at RDH. It is possible though that doctors who are less likely to attend meetings are also less likely to get immunised against influenza, and we might thus overestimate the immunisation rate. It is, however, reassuring that the characteristics of the survey respondents (level of seniority, area of work) were similar to doctors in the hospital overall.

Immunisation rates in this study were derived from self-reported data. It is unlikely that the 2007 immunisation rates would be inaccurate, but the respondents' recall of previous vaccination may have been prone to error and consequent under-reporting.

Implications

HCWs and health care systems have an ethical and moral duty to protect vulnerable patients from influenza.[11] The disappointing immunisation rate suggests that successful immunisation campaigns must implement specific strategies that target doctors and take into account the busy nature of their work. Effective multi-faceted interventions can significantly improve immunisation uptake as demonstrated clearly by Cooper and O'Reilly's Australian study,[12] where immunisation rates of staff in contact with patients improved from 8% to an impressive 81%. New innovation is required, such as was demonstrated in an American hospital which significantly improved the immunisation rate of HCWs by conducting a bioemergency pandemic influenza drill incorporating staff immunisation.[13]

Since it appears that most doctors receive their immunisation through staff immunisation clinics, this is an ideal opportunity for Infection Control Departments to keep accurate records of doctors' influenza immunisation status (as they do for other immunisations). The Commonwealth Government is currently considering a 'Whole of Life Register', similar to the Australian Childhood Immunisation Register (which records immunisations given up to the age of 7 years for all Australian children). This would ensure documentation of influenza immunisation status for all Australians, and would thus assist with enhancement of coverage.[14]

There are many difficulties in meeting standards for influenza immunisation in hospitals, including the annual turnover of junior staff, further emphasising the need for effective annual campaigns that make immunisation convenient and accessible. Where these strategies are unsuccessful, a mandatory influenza immunisation policy (or an opt-out policy) could be considered.

Based on the data from our study and those of others,[6,7,8] influenza immunisation rates are generally insufficient among hospital doctors. Efforts to improve influenza immunisation uptake among hospital doctors should focus on education about influenza emphasizing the protective benefits of immunisation, as well as strategies to make immunisation more convenient and accessible.

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Angela Brannely, Nursing Director, Infection Control Department, Royal Darwin Hospital, and all the doctors who completed the influenza immunisation survey.

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Pertussis in infants: how to protect the vulnerable?

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Abstract

In terms of adverse outcomes, infants remain the group most vulnerable to severe pertussis disease. Adult household contact is thought to be the main source of transmission to infants. This study reviews exposure history, vaccination status, admission outcome and quality of discharge coding of hospitalised infants with pertussis at a tertiary paediatric hospital. We identified cases between 1997 and 2006 from 2 sources: hospital discharge coding and positive *Bordetella pertussis* results from the hospital laboratory database. We assessed the completeness of each of these sources, compared with the dataset of all identified cases. We identified 55 hospitalised infants with pertussis. The 35 cases (64%) less than 3 months of age had greater risk of Intensive Care Unit admission, higher mortality, and were more likely to have parents as an identified source. On admission, only 5 cases (9%) were more than 2 weeks overdue for their previous scheduled pertussis vaccination. Discharge coding was more sensitive for identifying cases than the laboratory database. Nine cases (16%) had incorrect discharge coding. Even infants up to date for pertussis vaccine can have severe disease requiring hospitalisation. Immunising parents planning to have, or who have had, a newborn baby may help to prevent pertussis in infants. *Commun Dis Intell* 2008;32:449–456.

Keywords: whooping cough, *Bordetella pertussis*, infants, household transmission

Introduction

Infants are the group most vulnerable to severe pertussis disease, with higher rates of hospitalisation and death. Acellular pertussis vaccines replaced locally produced whole-cell vaccine in a primary course of vaccination at 2, 4 and 6 months of age in Australia in 1999 and the coverage rate for the 3 primary doses of acellular pertussis vaccination at 12-months of age has been over 90% since 2001.[1] Nevertheless, hospitalisation rates of infants with pertussis remain high.[2–4]

Adolescent and adult household contacts are thought to be the most important source of pertussis transmission to infants.[3–12] Currently, adults aged over 20 years represent over 80% of notified pertussis cases in Australia.[13] Adolescents and adults with pertussis may present with non-specific symptoms, making diagnosis difficult and delayed.

Acellular pertussis vaccines that are formulated for adolescents and adults are available and have been shown to be effective.[14] A publicly-funded booster pertussis vaccination program for adolescents was introduced in Australia in 2003. Since that time, the pertussis notification rate amongst teenagers has fallen but the notification rate for pertussis among the older adult population continues to increase.[2,4,13,15]

In Australia, a booster dose of pertussis vaccine is recommended for adults who work with young children. It is also recommended for parents planning a pregnancy, or as soon as possible after delivery of a baby.[16] A universal pertussis vaccination program for adults is difficult to justify due to problems with accurate estimation of pertussis incidence and on cost effectiveness grounds.[17,18] However, parental vaccination at the birth of a child could be cost effective.[19,20]

We performed a retrospective case-series study of hospitalised infants at a tertiary paediatric hospital in Brisbane, Australia. The survey targeted infants aged less than 12 months who had a diagnosis of pertussis in the decade between 1997 and 2006. The main aim of the study was to review the exposure history, vaccination status, and the quality of discharge International Classification of Diseases (ICD) coding of admission episodes of infants hospitalised with pertussis and to explore implications for future prevention.

Methods

We identified hospitalised infants who were aged less than 12 months and had a diagnosis of pertussis between January 1997 and December 2006 from 2 sources. First, cases were identified from the Royal Children's Hospital (RCH) inpatient database, using discharge ICD codes including whooping cough due to:

- *Bordetella pertussis* (ICD 9.033.0, ICD 10.A37.0);
- an unspecified organism (ICD 9.033.9, ICD 10.A37.9);
- *Bordetella parapertussis* (ICD 9.033.1, ICD 10.A37.1); or
- other *Bordetella* species (ICD 9.033.8, ICD 10.A37.8).

The last 2 codes were included to capture *B. pertussis* that may have been incorrectly coded. Second, *B. pertussis* positive results at RCH were identified from the AUSLAB (2000 to 2006) and the PARIS (1997 to 1999) laboratory databases used by Pathology Queensland.

Using the dataset containing infant pertussis cases identified through both sources, we assessed the completeness of each source compared to the complete dataset. The dataset of all infants diagnosed with pertussis during the period was cross-checked with cases notified to the Communicable Diseases Branch (CDB) of Queensland Health using 4 primary linkage fields: first name, last name, sex, and date of birth. If only three out of 4 primary fields matched, then 1 secondary field, either date of admission for hospitalised cases or postcode, was used to confirm the link.

For this study we reviewed the following information from medical records of hospitalised infants using a standard data collection form: name; medical record number; sex; date of birth; ethnicity; immunisation status; contact history; admission date and discharge date; admission outcome; and diagnostic methods. Disease severity was assessed by intensive care unit (ICU) admission, ventilation support, and mortality. The diagnosis of pertussis was defined by the case definitions of the National Notifiable Diseases Surveillance System (Box). Due to small numbers, 2-tailed Fisher's exact tests were used for comparative analysis and a p-value of less than 0.05 was considered statistically significant.

The study was approved by the Human Research and Ethics Committee of the Royal Children's Hospital and Health Services District, Brisbane.

Results

Study population

We identified 59 cases of hospitalised infants aged less than 12 months with a possible diagnosis of pertussis infection between 1997 and 2006. These cases were identified from inpatient database (22 cases) and positive *B. pertussis* results from a hospital laboratory database (2 cases), or both (35 cases). One case was not reviewed as the patient's medical chart could not be found. Among the 58 reviewed cases, 55 cases met criteria for inclusion in this study. The 3 excluded cases were coded as having 'whooping cough due to unspecified organism' (ICD 9.033.9), but had alternative laboratory-confirmed diagnoses, including 2 cases of respiratory syncytial virus (RSV) infections and 1 case of parainfluenza virus type 3 (PIV3) infection.

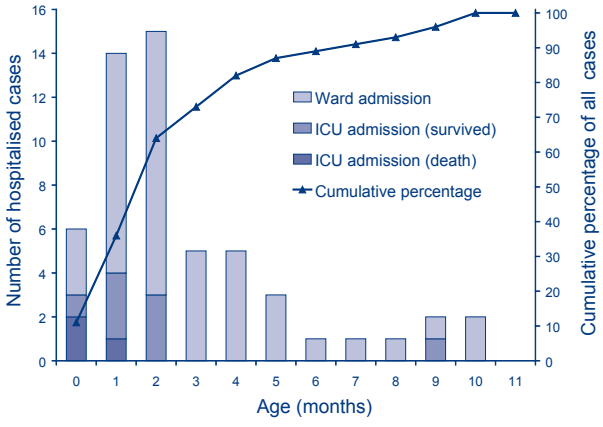
Of the 55 cases that met inclusion criteria, 45 were confirmed cases of whooping cough with detection of *B. pertussis* from laboratory tests. Seven were probable cases meeting only the clinical component of the case definition and had no other pathogen detected by laboratory testing.

The remaining 3 cases were diagnosed by their medical teams and the patients had symptoms including paroxysms of coughing or inspiratory whoop or post-tussive vomiting. However, at the time of discharge, the duration of the ongoing coughing illnesses in these cases had not reached 2 weeks. No further details about duration of cough post-discharge were available in the medical notes. No pathogens, including *B. pertussis*, were isolated by laboratory testing.

Demography and seasonality

Thirty-five cases (64%) of hospitalised infants with pertussis were aged less than 3 months (Figure 1). Male infants accounted for 60% of all cases. According to admission records, 5 (9%) cases were identified as Aboriginal with four of these aged less than 2 months. All other cases were identified as non-Indigenous. Two peaks in the number of hospitalised infants with pertussis were seen (1997–1998 and 2001–2002), closely matching the epidemic pattern seen in overall disease notifications in Queensland over the review period (Figure 2).[15] There were 22 cases hospitalised between January 1997 and December 1998 and 14 cases hospitalised between January 2001 and December 2002. More cases (18/55, 33%) occurred in spring (September to November) than other seasons.

Figure 1. Hospitalisation and outcome according to age for infants with pertussis, Royal Children's Hospital, Brisbane, 1997 to 2006



Box. Pertussis case definition

Notifiable case definition of pertussis from January 2004:[4]

Reporting

Both **confirmed cases** and **probable cases** should be notified.

Confirmed case

A confirmed case requires either:

- **Laboratory definitive evidence**

OR

- **Laboratory suggestive evidence** AND **clinical evidence**

OR

- **Clinical evidence** AND **epidemiological evidence**

Laboratory definitive evidence

1. Isolation of *Bordetella pertussis*

OR

2. Detection of *B. pertussis* by nucleic acid testing.

Laboratory suggestive evidence

1. Seroconversion or significant increase in antibody level or fourfold or greater rise in titre to *B. pertussis* in the absence of recent pertussis vaccination)

OR

2. Single high IgA titre to whole cells

OR

3. Detection of *B. pertussis* antigen by immunofluorescence assay (IFA).

Clinical evidence

1. A coughing illness lasting two or more weeks

OR

2. Paroxysms of coughing OR inspiratory whoop OR post-tussive vomiting.

Epidemiological evidence

An epidemiological link is established when there is:

1. Contact between two people involving a plausible mode of transmission at a time when:
   a. one of them is likely to be infectious (from the catarrhal stage, approximately 1 week before, to 3 weeks after onset of cough)

AND

   b. the other has an illness which starts within 6 to 20 days after this contact

AND

2. At least one case in the chain of epidemiologically linked cases (which may involve many cases) is a confirmed case with at least laboratory suggestive evidence.

Probable case

A probable case requires **clinical evidence** only.

Clinical evidence

1. A coughing illness lasting two or more weeks

AND

2. Paroxysms of coughing OR inspiratory whoop OR post-tussive vomiting.

Notifiable definition of pertussis in use prior to 2004:[21]

- Isolation of *B. pertussis* from a clinical specimen

or

- Elevated *B. pertussis*-specific IgA in serum or the detection of *B. pertussis* antigen in a nasopharyngeal specimen using immunofluorescence with a history of clinically compatible illness

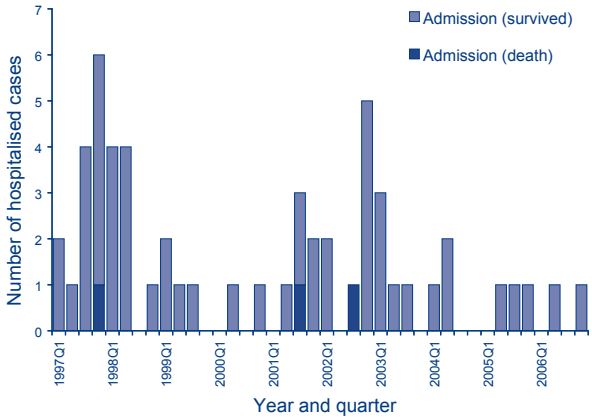
or

- An illness lasting 2 weeks of more with one of the following: paroxysms of coughing or inspiratory whoop without other apparent causes or post-tussive vomiting

or

- an illness characterised by a cough lasting at least 2 weeks in a patient who is epidemiologically linked to a laboratory-confirmed case.

Figure 2. Quarterly Royal Children's Hospital infant pertussis admissions, 1997 to 2006



Immunisation history

Documentation of immunisation status was available in all cases. Almost half (49%, 27/55) of all hospitalised infants had received no vaccine against *B. pertussis*, with 25 (93%) of these aged less than 3 months. In 29% (16/55) of hospitalised infants 1 dose of pertussis-containing vaccine had been received and 13% (7/55) had received 2 doses. Only 5 (9%) cases, all aged over 8 months, had completed a primary course of 3 doses of vaccine as required to protect against pertussis.[16]

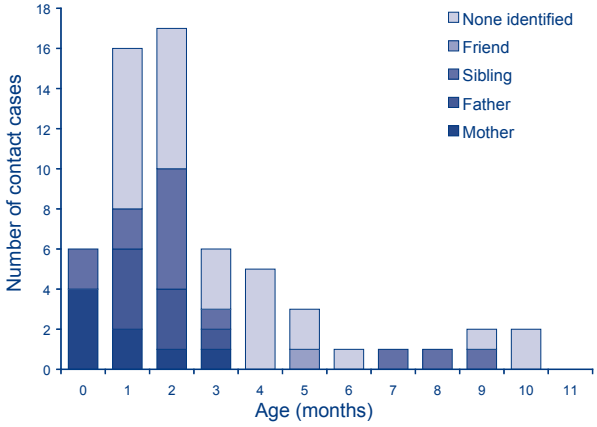
Despite this, at the time of admission only 5 hospitalised infants (9%) were more than 2 weeks overdue for their most recent scheduled pertussis vaccination, with all of them between 3 months to 7 months of age: 1 case overdue for the 1st scheduled pertussis vaccine dose, one for the 2nd dose and two for the 3rd dose. One case had received no pertussis vaccine doses at 5 months of age.

Contact history

Of the 26 infants (47%) with at least 1 documented identifiable source of pertussis, 3 cases had 2 identifiable sources and 1 case had 3 identifiable sources (Figure 3). Of a total of 31 identifiable sources, 16 (52%) were parents and 11 of these parents reported a preceding prolonged cough illness. Of the hospitalised infants (13/15, 87%) where a parent was identified as a source the majority were aged less than 3 months.

Twenty-seven identified sources for 24 laboratory-confirmed infant pertussis cases, had symptoms compatible with pertussis. The remaining 4 identified sources for 2 clinically-diagnosed infant pertussis cases, had a cough illness lasting 2 weeks or more. Overall, 7 (23%) identifiable sources had positive laboratory tests: *B. pertussis* IgA serology or polymerase chain reaction (PCR) on nasopharyngeal aspirate (NPA) and four of these were parents.

Figure 3. Contact history of hospitalised cases of pertussis according to age of infants



Cases that have more than 1 potential contact are represented multiple times in this figure.

Admission duration and disease severity

The duration of admission ranged from two to 115 days, with a median duration of 6 days. When separated into age groups, infants aged less than 3 months had a longer admission with a median duration of 7 days versus 4 days for older infants.

Clinically, 51 cases (93%) had one or more of the following symptoms documented: paroxysms of coughing; respiratory whoop; or post-tussive vomiting. The other 4 cases did not have these symptoms, but had a cough illness lasting two or more weeks and had laboratory-confirmed *B. pertussis* infection. Overall, 30 cases (55%) had documented cough illness lasting two or more weeks.

Severe pertussis disease was more common in hospitalised infants aged less than 3 months (Figure 1). Of 35 hospitalised infants in this age group, 10 (29%) required ICU admission, and eight of these needed ventilation support, including all 3 deaths identified in this case series. In comparison, only 1 (5%) of the 20 hospitalised infants aged 3 months or older, required ICU admission ($P=0.042$) and ventilation.

All 3 deaths in this case series were male infants aged less than 2 months and were laboratory confirmed with *B. pertussis* infection. Two of these cases were aged less than 1 month, had received no doses of pertussis vaccine, and had a parent identified as a source. The other case had received the 1st dose of

pertussis vaccine at 6 weeks of age–a week before presenting with clinical pertussis–and did not have an identified source.

A total of 9 hospitalised infants with pertussis (16%) had other concomitant infections. Of these cases, 5 cases were co-infected with another respiratory virus (2 cases of RSV, 2 cases of PIV1 and 1 case of PIV3). One case had co-infection with an adenovirus and cytomegalovirus. Other concomitant infections included 1 case of rotavirus gastroenteritis, 1 case of *Escherichia coli* urinary tract infection and 1 case with *Enterobacter cloacae* sepsis due to central venous line infection that developed after admission.

Laboratory investigations

Tests on NPAs were the dominant laboratory diagnostic method used to detect *B. pertussis* in hospitalised infants. Between 1997 and 1999, 24 cases had a NPA immunofluorescence test for the detection of IgA antibody (IFA) performed and 15 cases (63%) were positive. From 2000 onwards, PCR replaced IFA as the standard laboratory test and 26 of 28 cases (93%) tested using this method, had positive results.

A total of 39 cases had NPA culture performed with 15 (38%) being positive. Seven of eight of these culture-positive cases also had positive NPA IFA and six of 6 cases had positive NPA PCR. A single culture-positive case only had culture performed, without IFA or PCR testing. Of the 24 culture-negative cases, six of 14 cases had positive NPA IFA and nine of 10 cases had NPA PCR positive. One culture-negative case had negative NPA IFA but positive IgA serology.

Overall, 34 cases had IgA serology tests performed with only 2 cases (6%) being positive. Both cases did not have other pathogens identified by laboratory testing. One case was a 1-month-old unvaccinated infant. The other case was a 10-month-old infant who had received the third dose of pertussis vaccine at 6 months of age. In this case the initial IgA serology on admission was negative, but the repeat test 10 days later, was positive.

During the period under review, the IgA serology test was decreasingly used as a method for diagnosis in hospitalised infants. Between 1997 and 1999, 21 of 26 cases (81%) had this test performed, compared with 13 of 29 cases (45%) between 2000 and 2007.

Database completeness

Among the 55 cases that met inclusion criteria for this study, 53 cases (96%) were identified from the inpatient database through the discharge codes of whooping cough and 37 cases (67%) were identified from positive *B. pertussis* results from the hospital laboratory database.

The reasons for the low sensitivity of laboratory database detection include tests performed at other laboratories (7 cases) and clinically-diagnosed cases with negative laboratory test results (10 cases). Only 1 case with a positive *B. pertussis* test result performed at the hospital laboratory did not appear on the computer generated search list from the hospital laboratory database. This was due to an unexplained error during the search process.

Incorrect discharge ICD coding of pertussis cases (9/55, 16%) was not uncommon. Eight cases with *B. pertussis* detected from various laboratory tests were incorrectly coded: four as 'whooping cough due to unspecified organisms'; one as 'whooping cough due to *B. parapertussis';* one as 'whooping cough due to other *Bordetella* species'; and two as unrelated discharge codes. One case of whooping cough did not have an organism identified and was incorrectly coded as a case due to *B. pertussis*.

Notifications to the Communicable Diseases Branch

Of the 55 hospitalised infant pertussis cases, 52 met the pertussis case definition in use at the time. Of 45 laboratory-confirmed cases of *B. pertussis*, 38 cases (84%) were notified to CDB; 36 of these were linked on 4 primary fields, and two linked on 3 primary fields and 1 secondary field. None of the 7 clinically-diagnosed pertussis cases were notified. Overall, only 73% of cases were notified.

Of the 7 laboratory-confirmed cases that were not notified, 6 cases occurred between 1997 and 1999 and they were recorded in the PARIS laboratory database. The other case that was not notified occurred in 2000 and was recorded in the current AUSLAB laboratory database.

Discussion

Even in communities with high vaccination coverage, infants remain the most vulnerable to severe pertussis disease. In Australia, it has been suggested that many infants diagnosed with pertussis are overdue for immunisation time points.[3,5] However, on admission, less than 10% of infants in our case-series were more than 2 weeks overdue for their most recent scheduled pertussis vaccination. None of these were less than 3 months of age. An accelerated schedule of vaccination commenced at 6 weeks of age would not have prevented the deaths in our series, as all were aged under 2 months.

Options available to reduce the infant burden of pertussis include modifying the existing childhood schedule, or boosting immunity in parents and other infant contacts. A recent study in Germany showed neonatal immunisation with acellular pertussis vaccine given at birth, resulted in earlier antibody responses without inducing immunologic tolerance to pertussis antigens, but appeared to dampen response to a full primary course of *Haemophilus influenzae* type b vaccines at 7 months of age.[22] The second Global Pertussis Initiative meeting in 2005 recommended the implementation of a strategy involving immunising the household family members and close contacts of a newborn.[20] This 'cocoon strategy' may provide indirect benefit to protect infants against pertussis.

Our study suggests that parents remain the most important source of pertussis infection for infants. Educating and immunising parents planning a pregnancy, or who have recently had a baby may help to prevent pertussis infection in infants. Both of the two most recent editions of *The Australian Immunisation Handbook* have recommended this strategy.[16,23] Routine postpartum pertussis vaccination of women has recently been recommended by the Advisory Committee on Immunization Practices in the United States of America.[24] However, the uptake of the pertussis booster in parents is likely to be poor without a publicly funded program or more focused attempts to educate parents perinatally. Education campaigns will be required to increase clinician awareness and to target parents about pertussis infection.[25] Pertussis education at antenatal visits and in maternity units during the post-natal period may help to encourage the uptake of the pertussis booster in parents. Printed material could be included in the information pack parents receive in hospital, and pertussis vaccine could be offered to mother or both parents prior to discharge.

The period of our review saw a switch from whole cell to acellular pertussis vaccine and immunisation of adolescents with a booster dose added to the National Immunisation Program Schedule in Australia. Despite high vaccination coverage rates in infants since 2001, pertussis has remained endemic with epidemic cycles. Nearly two-thirds of hospitalised infants in our study were aged less than 3 months and had the greatest risk of ICU admission; the highest mortality; and were more likely to have a parent identified as the source of infection. Our findings confirm those of previous studies.[5,12,26–29]

Our study also shows the change in laboratory diagnostic methods from 1997 to 2006. For a variety of infectious diseases, PCR has been shown to be a more sensitive testing modality than traditional detection methods, such as serology and culture.[30] Our findings are consistent with trends in age-specific pertussis diagnostic methods identified between 2000 and 2005.[15] A recent study showed positive NPA PCR results could be obtained up to 3 weeks following onset of catarrhal symptoms and up to 2 weeks following onset of paroxysmal cough.[31] NPA PCR will remain an important diagnostic tool for infant pertussis infection. By way of contrast, IgA serology tests for pertussis do not appear sensitive in infants, as they fail to develop measurable antibodies.[32]

With the broader ICD coding search criteria used in our study, we were able to identify 96% of infant pertussis cases from the inpatient database. However, nearly one in 6 cases had incorrect coding. Some of this misclassification may be explained by the unavailability of final laboratory results at the time of coding. However, the hospital discharge ICD codes of whooping cough are still considered reasonably reliable to monitor trends in infant pertussis.[33]

Over one-quarter of infant pertussis cases (27%) identified were not notified to the CDB; a finding similar to that of a previous New South Wales study.[33] All 7 laboratory-confirmed cases that were not notified occurred prior to 2001. Complete notification of laboratory-confirmed cases from 2001 is likely to be due to streamlined laboratory reporting processes incorporating automatic electronic notification of laboratory-confirmed results from the AUSLAB system. None of the 7 clinically-diagnosed cases were notified, despite this being required under Queensland legislation. A low notification rate of clinically-diagnosed pertussis cases was reported in a previous study in New South Wales.[33] Efforts to improve clinical notification of pertussis in infants are required, particularly in hospital settings where follow-up and chemoprophylaxis of vulnerable contacts are likely to be needed.

As with all observational studies, limitations must be considered before interpreting results. In this study these include the study's retrospective nature, a lack of detailed contact history and inclusion of 3 clinically-diagnosed cases that did not meet the diagnostic criteria for pertussis at the time of discharge. Not all identifiable sources had laboratory tests (23%), meaning some of the clinically-diagnosed sources may have been caused by other organisms producing a pertussis-like illness.

Pertussis remains a serious threat to Australian infants, particularly the very young, and parents continue to be an primary source of infection. More effort should be made to improve the public awareness of pertussis, particularly among parents who should also be targeted for vaccination in the perinatal period.

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Surveillance summaries

Interim estimates of human papillomavirus vaccination coverage in the school-based program in Australia

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Abstract

In November 2006, the Australian Government announced the National HPV Vaccination Program, consisting of a course of prophylactic human papillomavirus (HPV) vaccine for all Australian females aged 12–26 years. Females aged 12–18 years are vaccinated through school-based programs. The school-based component commenced in April 2007, with the school years targeted varying across jurisdictions. Each jurisdiction maintains comprehensive records of HPV doses delivered in the school-based programs although how this is captured varies. This report presents interim coverage estimates for Year 1 (2007) of the program. Both New South Wales and Victoria achieved coverage of 70% or more among almost all school cohorts vaccinated in the program. Some of the variation in coverage achieved may reflect different levels of experience with school-based programs, and varying methods for school-based vaccine delivery and recording of doses administered. Except for some doses in South Australia, these interim coverage estimates do not include catch-up doses delivered by general practitioners or persons who were vaccinated prior to the onset of the program. Therefore, these data should be considered minimum estimates of coverage. The 1st year of the school-based HPV vaccination program should be considered a success, given time and resource constraints. Public sector immunisation providers across Australia should be commended for planning and implementing a new national immunisation program in approximately 4 months. *Commun Dis Intell* 2008;32:457–461.

Keywords: human papillomavirus immunisation, immunisation coverage, school-based immunisation, Australia, vaccination.

In November 2006, the Australian Government announced the National HPV Vaccination Program, consisting of a course of human papillomavirus (HPV) vaccine for Australian females aged 12–26 years. The program will provide ongoing vaccination to future cohorts of girls in the 1st year of high school (aged 12–13 years), with the catch-up component (ages 14–26 years) running for 2 years (i.e. 2007/08). Females aged 12–18 years are vaccinated through school-based programs, established in all states and territories of Australia, with women aged 18–26 years and those aged 12–18 years not in school, vaccinated through primary care services; primarily general practitioners (GPs). A complete course requires 3 doses of vaccine to be given over a 4–6 month period. The Federal Government is responsible for the development and dissemination of communication materials, funding of the vaccine (including most of the resources for service delivery), and the establishment of the National HPV Vaccination Program Register. States and territories are responsible for program implementation including vaccine delivery, provider education, and implementing school-based programs, including production of local program resources, such as consent forms and information for parents and data collection. The school-based component commenced in April 2007.

The school-based programs provide free vaccination to female students within both public and private schools by teams of trained immunisation providers. Information and consent forms are provided to parents and, in some jurisdictions, older students through the school. Parental consent is required for students under 18 years of age to receive the vaccine at school except in South Australia and Queensland, where consent may be accepted from students aged 16 years or older if they are assessed as competent to give consent. Consent is also obtained for inclusion of details of the vaccination on the National HPV Vaccination Program Register. If a student missed a scheduled dose during school-based delivery, parents/students were requested to obtain the remaining doses of the course either through immunisation clinics or their GP.

Due to the need to complete the vaccine course within the school year, in Year 1 of the program an accelerated schedule of 0, 1, and 4 months was considered acceptable where necessary, based on data provided by the manufacturer to the Australian

Technical Advisory Group on Immunisation (the usual schedule is 0, 2, and 6 months). In 2007, the only HPV vaccine available for use in the Program was the quadrivalent HPV vaccine, Gardasil®.

The National HPV Vaccination Program Register, established under the *National Health Amendment Act 2007 (National HPV Vaccination Program Register),* will maintain a record of HPV vaccinations given in Australia. The Register will receive data (retrospectively initially) from all states and territories and from all types of vaccination providers. Until the register is fully operational, no estimates of HPV vaccination coverage in women targeted for the program are available. However, all states and territories maintain comprehensive records of HPV doses delivered in the school-based programs although how this is captured varies across jurisdictions. In some jurisdictions the number of doses administered are collated at a local level (e.g. council level) before being forwarded to a central database, whereas in others (e.g. New South Wales) doses administered are reported to a central database on a daily basis. There is also no standard approach for estimating the denominator population. Some jurisdictions use the enrolled school population at the time each dose is administered (e.g. Western Australia and New South Wales) and others use start of year enrolments as the denominator throughout (e.g. South Australia, Tasmania and Victoria).

The Table shows interim coverage estimates by jurisdiction, vaccine dose and school year for Year 1 of the school-based HPV vaccine program. The cohorts targeted for the 1st year of the program varied by jurisdiction.

Australian Capital Territory

The HPV school-based program commenced in the Australian Capital Territory in April 2007 offering the vaccine to girls in Years 7, 10, 11 and 12 (n=~9,400). In 2007, the HPV vaccine was not co-administered with any other vaccine. Dose 3 coverage rates for Year 7 were 68.3%, while the Year 10, 11 and 12 catch-up years had a combined coverage rate of 61.3% (Table). The 1st dose HPV coverage rates are comparable to the 2007 coverage rates for single dose dTpa (79.8%), which in the Australian Capital Territory is administered to Year 9 students.

The HPV program was implemented through the existing school-based vaccination program run by ACT Health. The school program allows for missed doses to be administered through a general practice program at the end of the school year. The catch-up program is continuing in 2008 with girls in Years 7, 9 and 10 being offered the HPV vaccine.

New South Wales

In New South Wales, the school-based program commenced in late April 2007 and targeted females in Years 10, 11 and 12 (ages 15–18 years, n=~114,000). HPV vaccine was not co-administered with other vaccines and catch-up of missed doses is ongoing through targeted clinics and GPs. School-based immunisation is delivered by area health service or public health unit (n=8) based teams of immunisation nurses.

Surveillance of adverse events following immunisation (AEFI) in the school-based program in New South Wales in 2007 noted an apparently higher rate of anaphylaxis than documented from other vaccination programs. An investigation confirmed 7 reported cases (2.6 per 100,000 doses)[1] but this apparently increased rate was not observed in other areas of Australia.

Northern Territory

In the Northern Territory, the school-based program commenced in April 2007 and targeted females in Years 10, 11 and 12 (ages 15–18 years) at 21 schools (n=~2,800 students). HPV vaccination was co-administered with pneumococcal vaccine for Indigenous students in Year 10, where possible. School-based immunisation was delivered by health promoting school nurses in public schools and by public health unit immunisation nurses in private schools. Across all schools and Years 10, 11 and 12, the coverage for the 1st dose was 80% (2,260), second dose 71% (2,000) and third dose 64% (1,795).

Queensland

In 2007, the Queensland Health School-based Vaccination Program offered the HPV vaccine to approximately 79,000 girls in Years 10, 11 and 12 across 560 private and public schools. The program commenced in April 2007. HPV vaccine for Year 10 female students was co-administered with dTpa vaccine.

Initial data indicates approximately 74% of students (58,824) in Years 10, 11 and 12 received dose 1, 69% (55,082) received dose 2, and 62% (49,416) received dose 3 in the school program. Vaccination coverage for school students is expected to be higher, given that some students completed the series through their GP. GP data are currently being analysed at Queensland Health.

The school-based program is provided by qualified vaccination teams from Queensland Health, the local council or another health provider contracted by Queensland Health. The program is managed at a state-wide level with Zonal Coordinators located

in Southern, Central and North Queensland. Catch-up vaccination is offered by the vaccination teams at follow-up visits to schools or through community clinics. Students are also able to access free catch-up vaccine through their GP.

Tasmania

In Tasmania, the school-based HPV program is predominantly being delivered by local government councils. There are 29 council municipalities across the state; all have participated in the HPV program. The program is being coordinated, and the data collected centrally, through the Department of Health and Human Services, Communicable Diseases Prevention Unit.

The school-based program commenced in May 2007. The recommended target groups for 2007 were Grades 6 or 7, and 10 through 12 (ultimately however only a few schools vaccinated Grades 11 and 12 in 2007). Approximately 176 schools participated. In 2008, the target group will be Grades 6 or 7, 8 and 9, and 11 and 12. Some local councils chose to do all grades in 2007 and this is reflected in the coverage data (estimated target population in 2007 approximately 16,000). HPV vaccinations were co-administered with hepatitis B and varicella vaccines in Grade 6 and 7 and dTpa in Grade 10. Hepatitis B coverage was slightly higher in Grade 7 students than HPV coverage (hepatitis B vaccine dose 1 and 2 coverage of 79% and 71% respectively)

Almost 32,000 doses of vaccine were administered in the school-based program in 2007 and it is anticipated that coverage will increase as the program continues in 2008. Currently, catch-up of missed doses is ongoing through council school programs, immunisation clinics, targeted HPV clinics, and through GPs.

South Australia

In South Australia, the 1st year of high school commences at Year 8 (13 years of age). It was considered impractical to offer the vaccine to 12 year olds in Year 7 as there are significantly more primary than high schools. South Australia elected to deliver both the ongoing program and the entire school catch-up program over 1 year. In 2007, the HPV vaccine was offered to 50,191 girls in Years 8 through 12. Coverage for the full series was highest in Year 8 (Table). Students who were not vaccinated on the day the immunisation team visited, attended local council clinics or general practice for vaccination.

Providers who deliver the vaccine to individuals outside of the school setting are asked to report on a special 'follow up' card. These data are added to the school year level data. Providers were also asked to obtain consent on specifically designed forms from all individuals who wished their details to be sent to the HPV Register. Until the Register is operational, providers were asked to send the completed register forms to the South Australian health department. As some GPs considered the register forms as replacing the 'follow up' cards, data on some doses were included on the HPV Register forms but not on the 'follow up' cards. This explains why the school coverage may have a lower figure for dose 2 compared with dose 3.

Victoria

In Victoria, the HPV vaccine secondary school program commenced on 16 April 2007. Females in Years 7, 10, 11 and 12 (ages 12 and 15 to 18 years) were targeted (n=~125,000). This program was coordinated and provided by 78 local councils (1 council has no secondary schools). The HPV vaccine was administered at the same time as other scheduled vaccines due in Year 7 and Year 10. Coverage rates for co-administered vaccines were stable or improved from 2006 (e.g. diphtheria tetanus and acellular pertussis [dTpa] Year 10 coverage was 78% for both years).

A cluster of AEFI reported in May 2007 led to mass media coverage regarding HPV vaccine safety.[2] The Surveillance of Adverse Events Following Vaccination in the Community service provided a clinical investigation of the reported AEFI. The final assessment of the HPV-related adverse events (that they were cases of mass psychogenic illness) did not alter the administration or safety profile of the vaccine program.

Western Australia

In Western Australia the school-based HPV program is coordinated by the Communicable Disease Control Directorate (CDCD). It is delivered by either community school nurses employed by area health services (in 7 rural and 1 metropolitan region), or contracted out to local government authorities (5 in total in the metropolitan area) who engage nurses to deliver the immunisation program in schools. CDCD is responsible for the overall coordination of the HPV program to the target group aged 12–18 years.

The school-based HPV program commenced in May 2007 and was offered to females in Years 10, 11 and 12 in all schools in Western Australia. (n=~39,000). In 2008 the HPV vaccine is being offered to females in Years 7, 8, 9 and 10 in all schools.

Table. Quadrivalent human papillomavirus vaccine coverage (doses administered to enrolled population) in school-based programs, 2007,* by state or territory

Dose	Year 6	Year 7	Year 8	Year 9	Year 10	Year 11	Year 12
Australian Capital Territory					**(Years 10 to 12 combined)**		
Dose 1	NA	82%	NA	NA	79%		
Dose 2	NA	79%	NA	NA	74%		
Dose 3	NA	68%	NA	NA	61%		
New South Wales							
Dose 1	NA	NA	NA	NA	84%	82%	84%
Dose 2	NA	NA	NA	NA	81%	79%	81%
Dose 3	NA	NA	NA	NA	75%	72%	75%
Northern Territory					**(Years 10 to 12 combined)**		
Dose 1	NA	NA	NA	NA	80%		
Dose 2	NA	NA	NA	NA	71%		
Dose 3	NA	NA	NA	NA	64%		
Queensland					**(Years 10 to 12 combined)**		
Dose 1	NA	NA	NA	NA	74%		
Dose 2	NA	NA	NA	NA	69%		
Dose 3	NA	NA	NA	NA	62%		
South Australia							
Dose 1	NA	NA	83%	69%	70%	64%	66%
Dose 2	NA	NA	78%	64%	64%	57%	60%
Dose 3	NA	NA	77%	65%	64%	57%	55%
Tasmania							
Dose 1	76%	72%	70%	67%	73%	64%	57%
Dose 2	71%	67%	65%	63%	67%	58%	52%
Dose 3	64%	61%	58%	55%	57%	50%	44%
Victoria							
Dose 1	NA	85%	NA	NA	82%	81%	82%
Dose 2	NA	81%	NA	NA	76%	76%	78%
Dose 3	NA	75%	NA	NA	69%	70%	71%
Western Australia					**(Years 10 to 12 combined)**		
Dose 1	NA	NA	NA	NA	71%		
Dose 2	NA	NA	NA	NA	67%		
Dose 3	NA	NA	NA	NA	60%		

* Does not include catch-up doses delivered in general practice/community health settings, except in South Australia where these are partially recorded.

NA Not applicable as this year cohort was not targeted in 2007.

The 2007 school-based HPV program for Years 10, 11 and 12 in Western Australia achieved a combined vaccine uptake of 71% (28,590) for dose 1, 67% (27,235) for dose 2, and a 60% (24,520) uptake for dose 3. Reports of adverse events were minimal; the majority of reports were arm soreness, arm tingling and general malaise for 24 hours.

Summary

Public sector immunisation providers in all Australian jurisdictions should be commended for planning and implementing a new national immunisation program in approximately 4 months. This achievement is even more remarkable considering that Australia was the 1st country in the world to implement a universal HPV vaccination program delivered to females through the school system.

HPV vaccination coverage in the school-based programs reached a high of 77% among Year 8 students in South Australia. Both New South Wales and Victoria achieved coverage of 70% or more among almost all school cohorts vaccinated in the 1st year

of the program. Some of the variation in coverage achieved reflects different levels of experience with school-based vaccination delivery across jurisdictions, with those jurisdictions that have had such programs in place for many years benefiting from parental and student familiarity with the process. It should be noted that these coverage estimates record doses delivered in schools and do not (except for some doses in South Australia) include catch-up doses delivered outside the school system if doses were missed. In addition, anyone who had been vaccinated prior to the onset of the program would not be included. Therefore, these data should be considered minimum estimates of coverage.

While reported coverage varied by jurisdiction, it also varied by school year and tended to be inversely proportional with increasing age, as has been documented in other school-based vaccination programs in Australia. As many jurisdictions started the program among the older school year cohorts, coverage could be expected to increase in Year 2 of the program, targeting the lower enrolment years. There is, however, an ongoing risk of coverage rates being adversely affected by media reporting of adverse events following HPV immunisation or by the activities of anti-immunisation proponents.

Although coverage was less than optimum among some cohorts of students, the 1st year of the school-based HPV vaccination program should be considered a success, given time and resource constraints. It is anticipated that this success will continue, and perhaps be surpassed, during the second year. However, the school-based program is only 1 component of the overall National HPV Vaccination Program; the other is vaccination of women aged 18–26 years through primary care services. Coverage will be much more difficult to capture. However, as the National HPV Program Vaccination Register is implemented, a more accurate estimate of coverage among the entire cohort of women targeted for the program is anticipated.

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Short reports

An outbreak of *Clostridium perfringens* and the enforcement of food safety standards

Megan K Young, Peta Smith, Jack Holloway, Rod P Davison

Abstract

Investigation and management of a possible foodborne outbreak notified to the Brisbane Northside Population Health Unit aimed to determine the likely source of the outbreak and prevent the same scenario from recurring. Environmental health officers inspected the implicated premises and collected legal samples prior to the 1st outbreak control team meeting. Interview evidence was carefully documented. Inspection revealed large quantities of meat dishes being allowed to cool at room temperature overnight. Microbiological results implicated the meat dishes as a source of *Clostridium perfringens,* consistent with the cause of illness in notified cases. When educational measures failed to alter food handling practices, the restaurant owner was successfully prosecuted under the *Food Act 2006*. Education and voluntary compliance with food safety standards must form the foundation of sustainable behaviour change among food handlers. When these fail, prosecution is justified to mitigate the risk to public health. Immediate inspection, sampling left over food, and attention to formal interview technique and evidence collection can assist the investigation of outbreaks of foodborne illness and help to ensure any necessary court proceedings are a cost effective use of resources. *Commun Dis Intell* 2008;32:462–465.

Keywords: Foodborne outbreak, *Clostridium perfringens,* enforcement, food safety, legal proceedings

Access to safe food and water is fundamental to maintaining public health, and expected by Australian consumers. Within public health infrastructure, the interplay of health promotion, food safety education, national food standards, state legislation, local licensing and communicable disease surveillance, investigation and control strives to achieve this expectation. Yet, with an estimated 5.4 million cases of gastroenteritis in Australia per year costing around AU$811 million,[1] foodborne illness is both common and costly.

Outbreaks of foodborne illness contribute significantly to the total burden. In each year from 2002 to 2005, there were around 100 reported outbreaks of foodborne illness.[2–5] While not the most common foodborne pathogen at an estimated 43,000 cases of gastroenteritis per year,[6] *Clostridium perfringens* was still responsible for between 3 and 8 outbreaks of foodborne illness annually between 2002 and 2005.[2–5]

Symptoms typical of food poisoning by *C. perfringens* include epigastric pain, nausea, and watery diarrhoea lasting 12 to 24 hours after an incubation period of 8 to 24 hours.[7] The elderly and hospitalised populations are at risk of more severe disease. A cytotoxin is thought to be responsible for the symptoms in most cases.[7]

Cooking inactivates many foodborne pathogens including the vegetative cells of *C. perfringens*, but the spores of this bacteria may survive typical cooking temperatures and germinate and multiply as the cooked product cools. The optimal growth temperature is generally between 43 and 45 degrees Celsius.[8] Published outbreak investigations have usually implicated meat or poultry cooked on a large scale with improper attention to temperature regulation of the cooked product.[9–12]

Here we report on a foodborne outbreak of *Clostridium perfringens* that resulted from allowing large quantities of meat dishes to cool for prolonged periods of time at room temperature. We then discuss the implications for public health practice in the investigation of potential outbreaks of foodborne illness.

Outbreak summary

In mid-2006, the Brisbane Northside Population Health Unit was notified that a minimum of 7 people from a function of 25 had gastrointestinal symptoms after eating at a local restaurant. Contact details were obtained for as many of those known to be unwell as possible and specimen collection kits were delivered to their homes shortly after the outbreak was notified.

Within hours of the notification, environmental health officers from the Population Health Unit attempted to inspect the restaurant in question, finding the premises closed. They returned the following day to discover that large quantities of cooked food were being left at room temperature to

cool for more than 12 hours at a time. Food from the same batch eaten by function attendees (and that had undergone this same cooling process) was still present in the cold room. Samples were taken in accordance with the legislative requirements under the *Food Act 2006* and the owner of the restaurant was informed of the nature and possible consequences of the breach of food safety. Environmental health officers counselled the owner and staff on safe food practices at this time.

Whilst environmental health officers and communicable disease control officers were in consultation with each other from the time of the notification, the result of circumstance was that sample collection was initiated prior to the 1st outbreak control team meeting.

Further symptom and meal specific food histories were taken from symptomatic function attendees following the 1st outbreak control team meeting. As the host of the function had travelled overseas soon after its completion, no contact details were available for a number of the function attendees. A total of 11 cases were interviewed with a further 2 known cases unable to be contacted. One asymptomatic function attendee was contacted. Because of the availability of only a single control, epidemiological enquiry was based only on the food histories of the cases. The available data indicated that it was possible that one of the pre-prepared meat dishes that had been sampled was among the likely sources of infection (Table).

Table. Proportion of interviewed cases (n=11) who ate specific food types from the restaurant on the night previous to the complaint

Food type	Proportion of cases who ate the food type (%)
Beef/lamb kebab	91
Bread and dips	91
Lamb curry (pre-prepared)	91
Water	91
Chicken kebab	82
Rice	73
Salad	64
Chicken curry (pre-prepared)	46
Turkish delight	36
Vegetarian dish	9

Laboratory results confirmed this hypothesis, with counts of *C. perfringens* greater than 2.5×10^7 per gram in the pre-prepared meat dish samples ($>10^5$ per gram is considered a public health risk[13]) and spore counts between 2.1×10^5 and 8.7×10^6 per gram in the faecal samples obtained from the function attendees (a median of $>10^6$ per gram is considered consistent with *C. perfringens* induced diarrhoea[13]).

Environmental health officers returned to the restaurant upon receipt of the laboratory results for the food samples, to find that large quantities of cooked meat dishes were still being allowed to cool for hours at room temperature. In light of the public health risk incurred by this practice, the relevant food was seized and destroyed after further samples were taken. The result of laboratory testing showed that the seized food had indeed been a risk to public health with *C. perfringens* counts of at least 1.6×10^5 per gram.

The owner of the restaurant was successfully prosecuted under section 35(2) of the *Food Act 2006* for selling food that was unsafe, and was fined in the order of $20,000.

Discussion

In Australia and elsewhere, foodborne outbreaks still occur as the result of poor food handling.[14] Therefore, the key to decreasing the incidence of outbreaks lies, at least in part, in changing the behaviour of food handlers. As with any behaviour change at either the population or individual level, this is not an easy task.

Safe food handling is supported in Queensland in a number of ways. These include education and training, food safety guidelines and standards, and the enforcement of legislation at both a local and state level. There are a number of providers of food handling training. These include TAFE (Technical and Further Education) colleges, registered training organisations, industry associations and private tutors. A number of local councils also run training courses. The Australian Institute of Environmental Health has developed an in-house training program for food handlers.[15]

Under the Foods Standards Code, it is now a requirement that all food handlers have the appropriate skills and knowledge to handle food safely.[16] These skills and knowledge may be gleaned from a formal food handling course, or by in-house processes such as information provision or operating rules.

In Queensland, the *Food Act 2006* authorises local governments to licence food premises and conduct regular inspections to ensure compliance with licensing requirements. The Act also requires that all food sold should meet safety standards and that food premises operators comply with safety standards.

In the case reported here, previous local government inspections had not discovered the inappropriate food handling practice that is believed to have led to the outbreak. When failure of preventive measures like this occur, local investigation and management, possibly including enforcement of state legislation, is the appropriate recourse. As in this case, specific advice on the changes required to meet food safety standards, support as necessary to implement the changes, and follow up to ensure compliance with these should precede legislative action.

As a result of this case, 2 points of procedure from the investigation have been adopted into local policy:

1. If preliminary enquires are suggestive of a foodborne source for the illness (rather than person to person transmission), environmental health officers now routinely conduct an inspection of the suspected premises and collect left-over food samples as soon as possible after the complaint is taken (this will often be prior to the 1st outbreak control team meeting); and

2. Both samples and interviews are undertaken formally, so as to be admissible in a court proceeding if required.

Outbreak management protocols have been altered to support these procedures.

The reasoning behind this change is not punitive. Education and voluntary compliance with food safety standards will remain the foundation of sustainable behaviour change among food handlers. Indeed, most food businesses readily comply with advice after a foodborne illness outbreak.

However, in instances of significant public health risk, where educational measures fail to alter behaviour, prosecution is justified to mitigate the risk. To be successful and a cost effective use of resources, a prosecution's case must be built on evidence admissible in court.

For the charge of selling unsafe food, the evidence must show that:

1. the food was potentially unsafe
2. the food was sold by the vendor to the victim/s
3. the food caused harm when consumed.

To prove this offence, it is necessary to demonstrate the bacteria in the food was the same as that in biological samples, and that the person or persons affected bought and consumed the contaminated food from the vendor. To fulfil the first of these requirements, samples of left-over food and biological samples from the victims of the food poisoning are a prerequisite; hence the purpose of sampling as a priority over the 1st outbreak control meeting. To fulfil the second of these requirements, in the absence of detailed receipts held by the victims from the vendor, witnessed statements from the victims and vendor are necessary; hence the change to the interview procedure.

Since the successful prosecution of the offending restaurant as outlined above, no further cases of gastrointestinal illness linked to the restaurant have been notified. It is our recommendation that other public health authorities adopt similar procedures for the investigation of potential foodborne illness.

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Recurring outbreaks of *Salmonella* Typhimurium phage type 135 associated with the consumption of products containing raw egg in Tasmania

Nicola Stephens, David Coleman, Kathleen Shaw

Abstract

Large egg-associated outbreaks of *Salmonella* Typhimurium 135 (STm135) that were associated with inadequate food safety practices but also linked to a common poultry farm occurred in Tasmania in 2005. A series of public health interventions were implemented to prevent further occurrences but 2 more egg-associated outbreaks in Tasmania in March 2007 and January 2008 led to a further 66 cases of STm135. This report describes these outbreaks and their links to the common source associated with the outbreaks in 2005. *Commun Dis Intell* 2008;32:466–468.

Keywords: salmonellosis, foodborne illness, outbreak, cohort studies, surveillance, eggs, Typhimurium 135

Introduction

In recent years in Australia, eggs and dishes containing eggs have been the most common food vehicle identified in *Salmonella* outbreaks.[1] Uncooked or lightly cooked foods containing raw egg as an ingredient accounted for 14% of foodborne outbreaks in 2006,[1] 13% of foodborne outbreaks in 2007 (OzFoodNet Working Group. OzFoodNet unpublished data) and 28% of foodborne outbreaks in the 1st quarter of 2008.[2] All jurisdictions, except the Northern Territory, reported egg-related outbreaks due to various strains of *Salmonella* Typhimurium in 2006 and 2007.[1]

In Tasmania, one of the largest egg-associated outbreaks of foodborne illness in Australia for many years occurred between June and December 2005. During this time, 5 outbreaks of *Salmonella* Typhimurium phage type 135 (STm135) were identified in Tasmania, leading to 125 laboratory-confirmed cases.

Public health investigations included case and food handler interviews, cohort studies, environmental health investigations of food businesses, microbiological testing, traceback, and inspections and drag swabbing of an egg farm. These investigations enabled the identification of foods containing raw egg or foods contaminated through inadequate food handling and/or storage procedures as possible vehicles for exposure. A particular poultry farm that supplied a large share of the catering and restaurant market was reported as the common source of eggs. Interventions targeting the general public and food handlers to promote better handling of egg products, and advice to egg producers regarding harm minimisation strategies led to the series of outbreaks being brought under control.[3]

In March 2007 and January 2008, another 2 point-source outbreaks of STm135 occurred in Tasmania. These outbreaks, and their links to the egg-associated outbreaks reported above, are described in this paper.

Outbreak 1 – March 2007 (OB1)

Between 19 March and 2 April 2007, a total of 18 individuals were identified as having a microbiologically-confirmed *Salmonella* infection following the reported consumption of products originating from a bakery in the north-west of Tasmania (the bakery). A further 2 individuals were identified as having a microbiologically-confirmed *Salmonella* infection following the consumption of eggs or dishes containing eggs purchased from retail businesses that sourced their eggs from the same egg supplier as that which supplied the bakery. All cases were interviewed and were questioned about foods consumed as far back as 7 days prior to onset of symptoms, with additional questions asked about places of purchase of foods prepared both in and out of the home, illness in contacts, and non-food (environmental) exposures.

Food businesses identified in the food histories of more than 1 case were investigated by local and State government environmental health officers (EHOs). In each case food handling practices were reviewed, and samples were collected for microbiological investigation from food products, raw ingredients, food preparation surfaces and equipment. Following a series of case interviews, it became apparent that the bakery was the only food business reported by all cases. It was subsequently hypothesised that bakers, who were the only staff that handled raw egg product for glazing could have transmitted salmonellae

from raw egg to ingredients in sandwiches or rolls or to the sandwich-making area at the front of the bakery. Rolls and other ready-to-eat products were typically placed in a refrigerated display cabinet prior to sale and when measured by probe thermometer, EHOs found temperatures ranging from 9 to 11 degrees Celsius—or twice the appropriate food storage temperature required for potentially hazardous foods, under the *Food Act 2003*. Several food samples were taken from this display cabinet and submitted for laboratory analysis.

Traceback of ingredients confirmed that the bakery had been supplied eggs from the same egg farm (Farm A) that was implicated in previous outbreaks of STm135.[3] The microbiological tests of food and environmental samples from the bakery all returned negative results for the presence of *Salmonella*. Some of the eggs supplied to the bakery were also found to have been co-mingled with eggs from a non-authorised producer (Farm B) which was later closed. A second authorised egg producer (Farm C) was also found to be co-mingling the local eggs with eggs from Farm A. As a precautionary measure and in light of epidemiological evidence pointing to eggs as the most likely original source of STm135, all remaining products supplied by Farm A through Farm B and Farm C prior to the outbreak were voluntarily withdrawn from retail sale by the egg producers.

Outbreak 2 – January 2008 (OB2)

Between 30 January and 12 February 2008, a total of 47 individuals were identified as having a microbiologically-confirmed *Salmonella* infection following the consumption of products originating from a restaurant in southern Tasmania (the restaurant). Cases were interviewed and were defined as belonging to 1 of 4 cohorts linked to either eating at the restaurant or eating foods that had been provided by the restaurant. Cohorts 1 to 3 were made up of attendees of 3 separate catered functions (2 funerals and 1 workplace meeting). Cohort 4 was made up of restaurant attendees, and restaurant staff and their family members. A breakdown of the estimated numbers in each cohort is shown in the Table. Cases were questioned about foods consumed at the funerals or restaurant using questionnaires developed from the restaurant and catering menus. Analysis of foods consumed by cases found that chicken sandwiches containing aioli resulted in the highest attack rate for illness in catered cohorts 1, 2 and 3 and that all cases from cohort 4 had eaten restaurant items that contained aioli. A very strong association was found between eating any product containing aioli and becoming ill (OR 511, 95%CI 90–4709).

Food Safety Officers and local council EHOs visited the restaurant. A number of food safety issues were identified including: a failure to monitor temperature; concerns about hygiene arising from a failure to maintain handwashing stations and to provide paper towel; and evidence of inadequate cleaning and sanitation of the mixer used to blend aioli and in the production area.

Fourteen food samples and a range of environmental samples were obtained for microbiological testing. Culture results confirmed the presence of *Salmonella* Typhimurium 135 in 4 foods, one of which was aioli. The recipe for aioli included raw egg yolks. Traceback revealed that the eggs used were again supplied by the same egg farm (Farm A) that was implicated in previous outbreaks of STm135[3] and in outbreak 1 described above.

Table. Cohorts – outbreak 2

Cohort	Number of attendees*	Number. Interviewed†	Number ill	Number laboratory-confirmed	% of ill laboratory-confirmed
Cohort 1 – (funeral 1)	99	66	30	16	53
Cohort 2 – (funeral 2)	153	65	17	10	59
Cohort 3 – (workplace meeting)	7	7	4	1	25
Cohort 4 – (restaurant attendees, restaurant staff and their family members)	74+	72	29	20	69
Total	333+	210	80	47	59

* Total numbers for catered functions 1 and 2 were determined from signed guest lists at the 2 funerals and from details provided by the immediate family and other social networks of the deceased person. For these 2 cohorts the total numbers are likely to be close to the true numbers who attended. The total number for catered function 3 was supplied by the organiser of the meeting and is accurate. The total number for restaurant attendees was determined by the restaurant booking lists and from members of the pubic who contacted the Communicable Diseases Prevention Unit. This total is therefore an estimate.

† Interviews were conducted until it was determined that enough evidence had been gathered to inform the investigation.

Microbiology

All human and non-human isolates from both OB1 and OB2 were sent to the Microbiological Diagnostic Unit, Public Health Laboratory in Victoria for serotyping and phage typing. All 20 cases from OB1 were microbiologically-confirmed as STm135. In OB2, 46 of 47 isolates were reported as STm135, and 1 was reported as *Salmonella* Typhimurium untypable. As described previously, all STm135 isolates were found to be antigenically identical and exhibited a phage reaction pattern that is designated as *S.* Typhimurium 135a by the Institute of Medical and Veterinary Science in South Australia.[3]

Discussion

There was very strong epidemiological evidence indicating the point source of infection in both outbreaks. The evidence suggested that *Salmonella* was introduced into the food preparation environment implicated in each outbreak, on eggs (or possibly in cracked eggs) and the organism was then able to multiply in foods and/or spread through cross-contamination, thus becoming a public health risk and leading to an outbreak of STm135.

The lack of confirmatory microbiological testing of egg and food samples in OB1 in no way undermines these conclusions as positive *Salmonella* culture results in such circumstances are useful corroboration but negative results do not indicate absence of the organism from these or other foods. OB1 also underscores the need to improve egg industry packaging and labelling compliance and to implement measures to provide better traceability of eggs in the marketplace.

These 2 outbreaks and previous egg-related outbreaks of *Salmonella* Typhimurium 135 in Tasmania[3] emphasise the need for a through-chain approach in managing food safety risks. It is clear that vigilance must be exercised at all points in the food supply chain to remove as far as possible on-farm risks and to ensure that appropriate interventions are in place to remove remaining risk prior to sale for human consumption.

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Quarterly reports

OzFoodNet quarterly report, 1 July to 30 September 2008

The OzFoodNet Working Group

Introduction

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

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

During the third quarter of 2008, OzFoodNet sites reported 534 outbreaks of enteric illness, including those transmitted by contaminated food. Outbreaks of gastroenteritis are often not reported to health agencies or the reports are delayed, meaning that these figures under-represent the true burden of enteric illness. In total, these outbreaks affected 7,446 people, of which 210 were hospitalised and 31 people died. The majority (90.0%, n = 480) of outbreaks were due to person-to-person transmission (Table 1).

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

Transmission mode	Number of outbreaks	Per cent
Foodborne	17	3.2
Person-to-person	480	90.0
Unknown	28	5.2
Salmonella cluster	1	0.2
Other pathogen cluster	8	1.5
Total	534	100

Foodborne disease outbreaks

There were 17 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as the primary mode of transmission (Table 2). These outbreaks affected a total of 229 people and resulted in eight being admitted to hospital. There were 8 deaths associated with foodborne disease outbreaks during the quarter. This compares with 25 outbreaks in the second quarter of 2008[1] and 36 outbreaks for the third quarter of 2007.[2]

Clostridium perfringens was the most common cause of foodborne outbreaks during the quarter and was responsible for 4 outbreaks. Three of these occurred in institutional settings. *Salmonella* was implicated in 3 outbreaks during this quarter, with *S.* Typhimurium 44 causing 2 outbreaks and *S.* Anatum causing one.

There were 3 foodborne outbreaks of norovirus during this quarter. Two outbreaks of ciguatera fish poisoning were both from Queensland. There was 1 outbreak due to *Staphylococcus aureus*. The remaining 4 outbreaks were caused by unknown aetiological agents.

Seven outbreaks were associated with food prepared in restaurants, five with food prepared in institutional settings, three with food prepared by commercial caterers and 2 outbreaks associated with primary produce. To investigate these outbreaks, sites conducted 6 cohort studies and 1 case control study. Case series data were collected for 6 investigations. Investigators obtained analytical epidemiological evidence in 2 outbreaks and microbiological evidence in 5 outbreaks. For the remaining 10 outbreaks, investigators obtained descriptive evidence implicating the food vehicle or suggesting foodborne transmission.

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

Table 2. Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 July to 30 September 2008 (n=17)

State	Month of outbreak	Setting prepared	Infection	Number affected	Evidence	Responsible vehicles
NSW	Jul	Restaurant	Norovirus	4	M	Oysters
	Jul	Restaurant	Norovirus	10	M	Oysters
	Jul	Restaurant	Unknown	3	D	Fried rice
	Aug	Institution	*Clostridium perfringens*	25	A	Macaroni bolognaise
	Aug	Commercial caterer	Unknown	8	A	Mixed sandwiches
	Sep	Restaurant	*Staphylococcus aureus*	7	M	Chicken
	Aug	Restaurant	Unknown	3	D	Multiple foods
	Jun	Aged care	*Clostridium perfringens*	69	D	Unknown, possibly pureed food
	Aug	Aged care	*Salmonella* Typhimurium 44	10	AM	Chocolate mousse with raw eggs
	May	Restaurant	*Salmonella* Anatum	11	M	Chicken
	Sep	Restaurant	Unknown	5	D	Barramundi, lamb & salad
Qld	Jul	Primary produce	Ciguatera	4	D	'Yellow king' – Samson fish
	Jul	Primary produce	Ciguatera	6	D	Red throat emperor/reef snapper
SA	Sep	Commercial caterer	Norovirus	5	D	Unknown
Vic	Aug	Institution – other	*Clostridium perfringens*	15	D	Savoury mince
	Sep	Aged care	*Salmonella* Typhimurium 44	14	D	Vitamised food
WA	Jul	Commercial caterer	*Clostridium perfringens*	30	D	BBQ Asian chicken

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

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

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

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

New South Wales

New South Wales reported 11 foodborne outbreaks during this quarter.

There were 2 outbreaks of *Clostridium perfringens* intoxication in institutional settings. The 1st outbreak occurred in an aged care facility where 53% (69/131) of residents experienced episodes of gastroenteritis over a 1 month period. Forty-two per cent (29/69) of residents experienced more than 1 episode of gastroenteritis (predominately manifested as diarrhoea). The median age was 83 years (range 60–100 years) and 74% (69/51) were female. Epidemiological investigation found that during the outbreak, residents in the nursing home were almost 3 times more likely to be ill than residents in the hostel (RR 2.7, 95%CI 1.7–4.1). Residents on milled or pureed diets were twice as likely to be ill as those who were not on a milled or pureed diet (RR 2.3, 95% CI 1.62–3.1). Laboratory testing identified *C. perfringens* enterotoxin type A in seven out of 10 stool samples submitted for testing. No other pathogens were found in the 10 stool samples collected. The NSW Food Authority (NSWFA) found that food handling, temperature control and kitchen hygiene were all satisfactory. The epidemic curve suggests that there were 2 foodborne point source outbreaks within the outbreak period, with the possibility of some environmental contamination and/or person-to-person spread.

The second outbreak occurred in August in an institution for disabled adults, where 6% (25/450) of residents experienced diarrhoea. *C. perfringens* enterotoxin type A was detected in three of 6 stool samples taken from residents. A cohort study demonstrated a strong association between consuming macaroni bolognese and illness (RR = 12.1, 95% CI 1.7–86.8). The NSWFA did not find any food safety breaches and could not identify any temperature control issues that would explain the outbreak.

In August 2008, 22% (10/46) of residents of another aged care facility in rural New South Wales developed gastroenteritis, with eight confirmed as *S.* Typhimurium 44. The median age of cases was 82 years with cases' illness spread over an 8-day period. In this outbreak, chocolate mousse made with raw eggs was the likely source of infection with *S.* Typhimurium 44 detected on the shell of an egg supplied to the facility by a local farm. The eggs used in the facility kitchen were substandard and there were several potential food safety issues within the facility and kitchen environment.

In July 2008, there was an increase in *S.* Anatum, with 9 cases notified between 13 May and 2 July 2008. The median age was 26 years and 4 cases were male. Three of the 5 cases contacted for interview reported eating a chicken meal from the same restaurant. The NSWFA conducted an environmental investigation at the premises, with food and environmental samples taken. One sample collected from a stainless steel bench in the food preparation area was positive for *S.* Anatum, although the source of contamination of the environment was not identified.

On the north coast of New South Wales, there were 2 outbreaks of norovirus due to consumption of contaminated oysters that affected at a total of 14 people. Norovirus was detected in 2 oysters sampled from the lease. The river was closed for harvesting by the NSWFA. The source of contamination remains unclear.

Two separate groups of people reported illness within 3 hours of consuming chicken kebabs at a commercial premise. Illness was consistent with foodborne intoxication caused by *Staphylococcus aureus*, which was isolated from cold cooked chicken stored in a cool room. Food handler contamination was the suspected cause of this outbreak.

Public health units in New South Wales also investigated a further 4 small outbreaks of gastroenteritis where foodborne transmission was suspected. Less than 10 people were affected in each of these outbreaks, and the aetiology was unknown.

Queensland

Queensland reported 2 outbreaks of foodborne illness during the third quarter of 2008, both of which were due to ciguatoxin contaminated fish.

In July, 4 cases of suspected ciguatera fish poisoning occurred after consuming steaks from a single 2–3 kilogram Samson fish. Cases experienced symptoms of nausea, vomiting, diarrhoea, abdominal cramps and numbness or tingling of hands, feet and mouth. The cases were aged between 5 and 62 years. The fish was caught off Saumarez Reef, north-east off Gladstone and purchased from a local fish market in Brisbane.

In late July and early August, 6 cases of suspected ciguatera fish poisoning were associated with fish purchased from a single distributor in Brisbane. Cases were aged 25–63 years. The median incubation period was 13 hours (range 3–18 hours) and symptoms included diarrhoea, vomiting, reversed temperature sensation and numbness or tingling of hands, feet and mouth. One case reported eating Reef Snapper while the remaining cases all ate Red Throat Emperor. These cases were associated with 2 different mixed catches from the same fishing location, Capel Bank, approximately 400 kilometres east-north-east of Brisbane. In total, the 2 catches consisted of 8,654 kilograms of fish and approximately 700 kg of fish was also distributed to New South Wales; no cases were reported from New South Wales. Retailers disposed of all remaining Red Emperor from the implicated batch of fish.

South Australia

South Australia reported a single outbreak of suspected foodborne illness during the quarter. The outbreak occurred amongst participants of a 5-day catered training course in metropolitan Adelaide during September. Of 5 course attendees ill with gastroenteritis, three provided specimens that were positive for norovirus. One of the catering staff developed gastroenteritis during the 5-day course, which was also confirmed as norovirus infection. This catering worker was involved in food preparation prior to becoming ill, but was not involved in serving foods. A cohort study was conducted, but no foods were significantly associated with illness.

Victoria

Victoria reported 2 outbreaks of foodborne illness during the quarter, both of which occurred in institutional settings.

In August, a foodborne outbreak was reported in a supported residential facility. Fourteen residents and 1 staff member became ill with diarrhoea within a 2-hour period of each other. Three faecal specimens were positive for *C. perfringens* enterotoxin. Savoury mince served for dinner the previous night, was suspected as the cause of the illness. Cooling and reheating of foods was identified as a problem at the facility.

An outbreak of gastroenteritis in an aged care facility was notified in September 2008. There were 11 residents and 3 staff members who were affected and six of the residents had specimens positive for *S.* Typhimurium 44. The staff members cared for

ill residents and were likely to have been secondary cases. A food specific source was unable to be identified but consumption of vitamised food was associated with illness (RR 9.47 95% CI 3.1–28.9).

Western Australia

Western Australia reported a single outbreak of foodborne illness this quarter in mine workers. In July, at least 30 mine workers were ill with diarrhoea and abdominal pain following a company barbeque meal. The pattern of illness was consistent with *C. perfringens* intoxication. A total of 662 people were reported to have eaten at this meal, and 88 were interviewed in a case control study of 30 cases and 58 controls. Analysis of the results demonstrated a minimum attack rate of 34% (30/88). Consumption of chicken and steak was associated with illness, but did not reach statistical significance. Faecal specimens from four of the 5 mine workers were positive for *C. perfringens,* with two of the samples having an indistinguishable pulsed field gel electrophoresis pattern. Asian barbeque chicken was the most likely source, as chicken was the food item consumed by the highest proportion of cases and was possibly held at incorrect temperatures for long periods of time.

Comments

During the quarter, the number of outbreaks reported was lower than previous quarters in 2008, although the number of outbreaks spread from person to person remained high. There were only 3 foodborne outbreaks of salmonellosis this quarter, compared to 11 outbreaks each for the 1st and second quarters of 2008.

The 2 outbreaks of ciguatera poisoning in Queensland associated with commercially-available fish highlight the potential for large reef fish to cause illness. It is unusual to receive reports associated with commercial fisheries, as affected reefs are well known and avoided by fishermen.

The 2 outbreaks associated with commercial caterers highlight 2 major food safety issues that lead to outbreaks in these operations: ill food handlers and poor temperature control of foods. It is vital that people preparing and serving food do not work while they have symptoms of gastroenteritis, as it can lead to outbreaks of norovirus.[3] Similarly, catering services can lead to food being held at inappropriate temperatures for long periods of time allowing bacteria such as *C. perfringens* to grow.

There were increased numbers of toxin-related outbreaks reported during this quarter, including a large highly publicised outbreak in New South Wales. The unusually long outbreak of *C. perfringens* with 2 apparent point sources demonstrated that foodborne illnesses may have an unusual course or presentation in elderly people.[4] There were 5 foodborne institutional outbreaks during the quarter, including three in aged care facilities. While identifying a specific food vehicle in these outbreaks is difficult, the association between illness and consumption of pureed food is striking and highlights the need to improve food handling associated with foods for vulnerable populations.[5]

Acknowledgements

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Communicable diseases surveillance

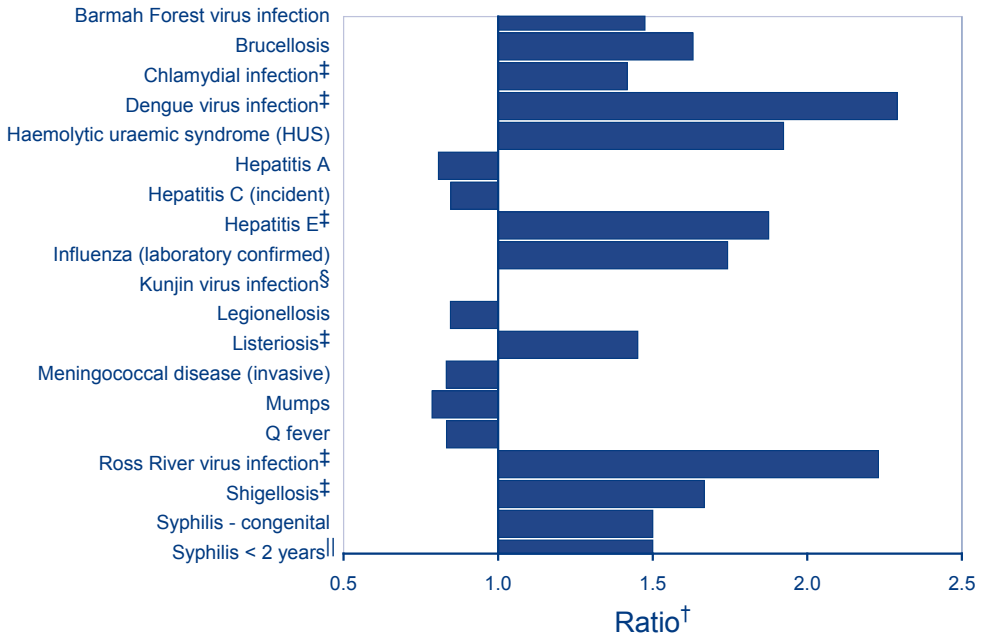
Highlights for 3rd quarter, 2008

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

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

Figure 1 shows the changes in selected disease notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a diagnosis in the 3rd quarter (1 July to 30 September) 2008, in comparison with the 5-year mean for the same period. Notifications were above the 5-year mean for the corresponding period and exceeded 2 standard deviations from the 5-year mean for: chlamydial infection, dengue virus infection, hepatitis E, Ross River virus infection and shigellosis. Notifications were equal to or below the 5-year mean for Barmah Forest virus infection, brucellosis, congenital syphilis, cryptosporidiosis, haemolytic uraemic syndrome (HUS), hepatitis B (unspecified), influenza (laboratory confirmed), listeriosis, pertussis, rubella, salmonellosis, STEC/VTEC, infectious

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



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

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

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

§ Significant. First case diagnosed in this quarter in 6 years.

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

syphilis <2 years, syphilis >2 years or unspecified duration, typhoid, varicella zoster (shingles), and varicella zoster (unspecified).

Gastrointestinal diseases

Hepatitis E

Between 1 July and 30 September 2008, there were 9 notifications of hepatitis E in Australia, 1.9 times the mean notifications for the corresponding period over the last 5 years. Hepatitis E cases in Australia are commonly imported, and of the 9 notifications during the 3rd quarter of 2008, three were known to have been acquired overseas, three were thought to have been locally acquired, while the travel status of the remaining 3 cases was unknown.

Shigellosis

Between 1 July and 30 September 2008, there were 201 notifications of shigellosis in Australia, representing an annualised rate of 3.8 notifications per 100,000 population. The number of shigellosis notifications represent a 23% increase over the number reported during the corresponding quarter of 2007, and is 1.7 times the 5-year mean of notifications for the corresponding period.

The highest notification rate was in the Northern Territory, where 34 cases were notified, representing an annualised rate of 63.3 cases per 100,000 population. Notification rates for shigellosis in the Northern Territory are usually high compared with other Australian states and territories, with an annual rate of 80.5 cases per 100,000 in 2007 compared with 2.8 cases per 100,000 population nationwide.[1]

There were 2 large clusters of shigellosis reported during the 3rd quarter of 2008 that contributed to the observed increase in notifications compared with previous years. The first was amongst members of the Jewish community in Melbourne, Victoria and the other cluster, with cases reported from 4 states (Queensland, New South Wales, Victoria and Western Australia) was reported amongst adult men, many of whom identified as men who have sex with men.

Sexually transmissible infections

Chlamydial infection

Nationally, chlamydial infections continue to be the most frequently notified infection to the NNDSS, at a rate of 277 cases per 100,000 population (annualised). During the 3rd quarter of 2008 there were 14,531 notifications received, which was 41% higher than the 5-year mean for the corresponding quarters of previous years and exceeded 2 standard deviations from this mean. All jurisdictions reported cases, with the majority notified from Queensland (n=3,714, 26%), New South Wales (n=3,534, 24%), Victoria (n=3,112, 21%) and Western Australia (n=2,131, 15%).

The notification rate was highest in the Northern Territory at 918 cases per 100,000 population (annualised), although there were only 493 cases of chlamydial infections. The rates of chlamydial notification in the Northern Territory during the quarter were substantially higher compared with other jurisdictions such as Western Australia at 405 and Queensland at 355 cases per 100,000 population (annualised).

The total number of cases for the 3rd quarter (n=14,531) was comparable with the previous quarter (n=15,156). For the first 3 quarters of 2008 there were 44,195 notifications, which was 11.9% higher compared to the same period in 2007 (n=39,479) and was 41.3% higher than the 5-year mean for the corresponding periods (n=31,284).

For the 3rd quarter, 37% (5,315) of the total number of infections occurred in the 20–24 year age group and 25% (3,595) in the 15–19 year age group. The highest rates of chlamydial infection was in females aged 20–24 years (443 cases per 100,000 population) and in the 15–19 year age group (385 cases per 100,000 population). The highest rate in males was 270 cases per 100,000 population in the 20–24 year age group. Overall, the ratio of male to female notifications for the quarter was 1:1.5, which was similar to previous years.

Figure 2 shows the epidemic curve of chlamydial infection notifications received by the NNDSS since 2003 by jurisdiction. The increasing trend in chlamydial notifications is most likely associated with increased screening and promotion programs; and the use of less-invasive and more sensitive diagnostic tests.

Vaccine preventable diseases

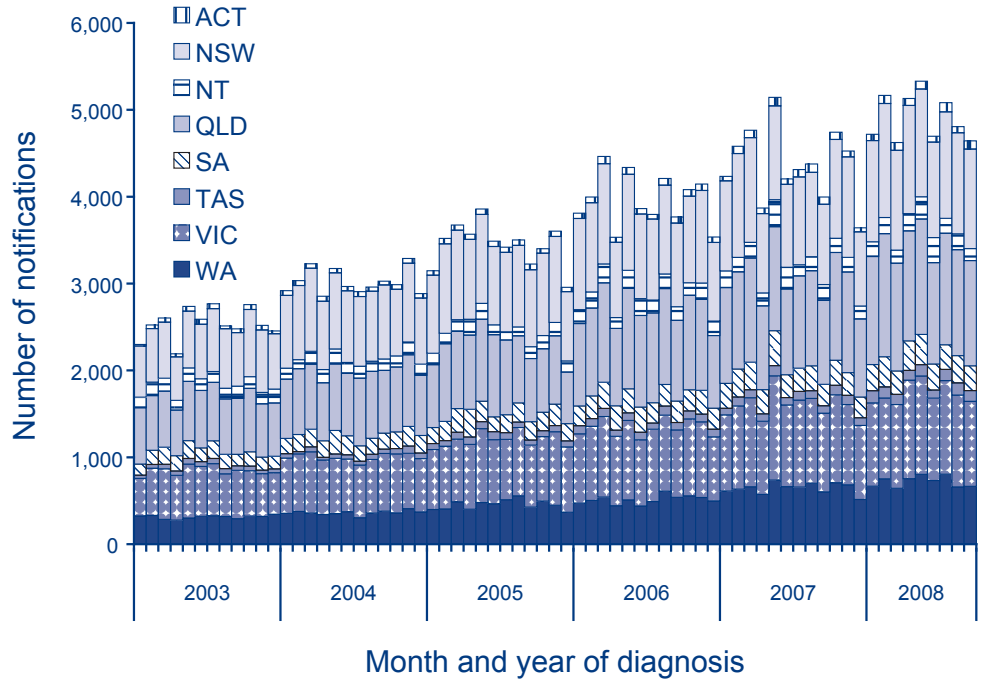
Influenza

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

The 2008 influenza season began in mid-July following a very gradual increase in notifications since mid-June (Figure 3). The 2008 season commenced several weeks late compared to previous influenza seasons – approximately 5 weeks later than the start of the 2007 season, with notifications peaking in early September, approximately 4 weeks later than 2007.

The total number of laboratory-confirmed influenza notifications to NNDSS for the 3rd quarter

Figure 2. Epidemic curve of notifications of chlamydial infection, Australia, 1 January 2003 to 30 September 2008, by month of diagnosis and state or territory

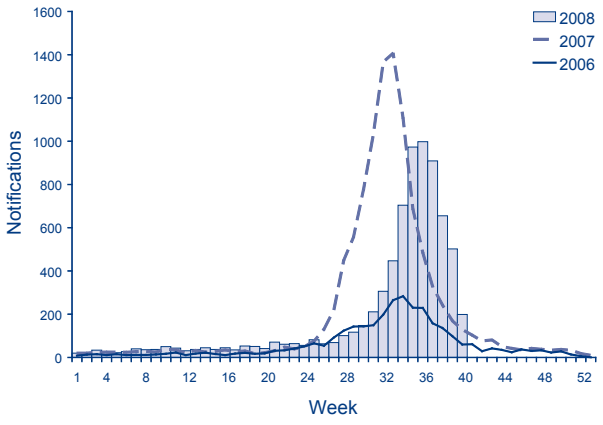


was 6,324 cases (84.7% of year-to-date notifications); this was 1.7 times the 5-year mean for the corresponding period. The majority of notifications were from Queensland with 2,823 cases (44.6%).

During the 3rd quarter of 2008, the highest rate of notifications occurred in Queensland, followed by Tasmania, the Northern Territory and the Australian Capital Territory (Table). The Australian rate of influenza infection was 30 cases per 100,000 population during this quarter.

Nationally, age-specific notification rates during the 3rd quarter of 2008 were highest in children under 1 year of age, at approximately 156 cases per 100,000 population for males and 116 cases per 100,000 for females.

Figure 3. Number of influenza notifications to the National Notifiable Diseases Surveillance System, Australia, 2006, to 2008 (to 30 September), by week of diagnosis



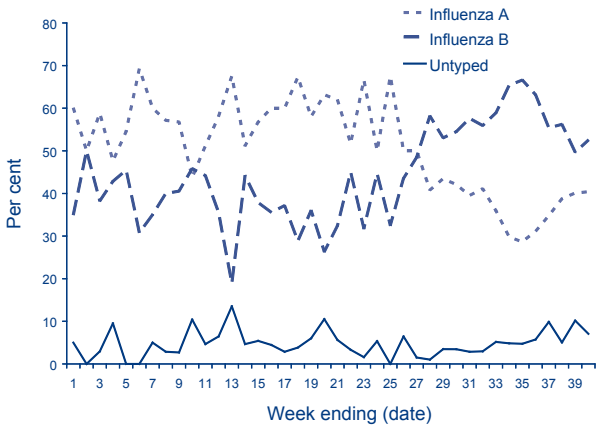
Influenza notifications to NNDSS were predominantly type A in the first 2 quarters of 2008. From the start of July, and throughout the 3rd quarter, notifications were predominantly type B (Figure 4). There has not been a predominantly type B season in Australia since influenza became nationally notifiable in 2001.

Table. Number and rate of laboratory-confirmed influenza notifications to the National Notifiable Diseases Surveillance System, 1 July 2008 to 30 September 2008, by date of diagnosis, and state or territory

State	Cases	Percentage of total notifications	Rate per 100,000 population*
ACT	164	2.6	48.3
NSW	1,028	16.3	14.9
NT	104	1.6	48.4
Qld	2,823	44.6	67.5
SA	234	3.7	14.8
Tas	291	4.6	59.0
Vic	979	15.5	18.8
WA	701	11.1	33.3
Aus	6,324	100	30.1

* This is a quarterly rate and has not been annualised due to seasonality of influenza. For the annualised rate please see Table 3.

Figure 4. Typing characteristics of notifications of laboratory-confirmed influenza, National Notifiable Diseases Surveillance System, Australia, 1 January to 30 September 2008, by week of diagnosis



In the period between 7 July and 26 September 2008, a total of 344 samples were typed by the WHO Collaborating Centre for Reference and Research in Influenza. Of these, 22 (6.4%) were A/Brisbane/59/2007-like (H1N1), 75 (21.8%) were A/Brisbane/10/2007-like (H3N2), 158 (45.9%) were B/Florida/4/2006-like and 89 (25.9%) were B/Malaysia/2506/2004-like.

During the 2nd and 3rd quarters of 2008, the World Health Organization reported that of those Australian H1N1 isolates that underwent resistance testing, 80% (47 of 59) tested resistant to oseltamivir. Resistant isolates were also detected in 20 of 27 countries that submitted data.

Vectorborne diseases

Dengue virus infection

Between 1 July and 30 September 2008, there were 93 notifications of dengue virus infection in Australia, representing an annualised rate of 1.8 notifications per 100,000 population. The number of dengue notifications represent a 43% increase over the number reported during the same quarter of 2007, and was 2.3 times the 5-year mean of notifications for the corresponding period. All 93 cases were imported from overseas. The number of cases notified in this quarter was the exactly the same as notified in the previous quarter.

New South Wales (n=37), Queensland (n=21) and Western Australia (n=21) reported the highest number of cases. The number of cases reported in New South Wales represented a 118% increase over the number of cases (n=17) reported for the corresponding quarter in 2007, while the number of cases reported in Western Australia represented a 62% increase over the number of cases (n=13) reported for the corresponding quarter in 2007.

The increase in notifications of imported dengue for this quarter has been attributed to an outbreak of dengue in the pacific region, in particular Fiji, Kiribati, New Caledonia, Samoa, Tonga, Wallis and Futuna and Vanuatu.

Kunjin virus infection

One sporadic case of Kunjin was notified in Queensland between 1 July and 30 September 2008. This was the only case notified in Australia during this quarter and was the first time Kunjin has been reported in this period for at least 6 years. The date of diagnosis (15 July) was very early in the reporting period.

Queensland has averaged 1.9 Kunjin notifications per annum since 2001 (when Kunjin became notifiable). The average number of Kunjin notifications in Australia since 2001 is three per annum.

Ross River virus infection

Between 1 July and 30 September 2008, there were 768 notifications of Ross River virus infections (RRV) in Australia, representing an annualised rate of 14.6 notifications per 100,000 population. The number of RRV notifications represent a 17% increase over the number reported during the corresponding quarter of 2007, and was 2.2 times the 5-year mean of notifications for the corresponding period. The number of cases notified this quarter was 34% lower than the number of cases notified in the previous quarter.

With the exception of Tasmania, all jurisdictions reported cases, with the majority notified from Queensland. In Queensland there were 393 notifications, which represented 51% of the number of notifications for Australia for this period. Higher rainfall and warmer than usual conditions in Queensland during this period was likely to have contributed to higher mosquito numbers and the increased transmission of RRV.

Acknowledgements

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

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Tables

National Notifiable Diseases Surveillance System

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 41,853 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 July 30 September 2008 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1. Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis (NEC)	All jurisdictions
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (incident)	All jurisdictions except Queensland
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except New South Wales
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
STEC, VTEC	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Highly pathogenic avian influenza in humans	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis (all)	
Syphilis <2 years duration	All jurisdictions
Syphilis >2 years or unspecified duration	All jurisdictions except South Australia where data is not collected
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

* 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 July to 30 September 2008, by date of diagnosis*

Disease	State or territory								Total 3rd quarter 2008†	Total 2nd quarter 2008	Total 3rd quarter 2007	Last 5 years mean 3rd quarter	Year to date 2008	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0.0
Hepatitis B (incident)	0	15	1	12	1	2	25	17	73	63	60	195	226.8	71.8	1.0
Hepatitis B (unspecified)	24	782	34	220	112	21	517	162	1,872	1,681	1,719	5,214	4,687.0	1,592.4	1.2
Hepatitis C (incident)	1	7	3	0	8	5	33	25	82	85	107	249	279.0	96.8	0.8
Hepatitis C (unspecified)	54	1,276	41	649	119	83	569	292	3,083	2,925	2,990	9,070	9,405.8	3,117.2	1.0
Hepatitis D	0	2	0	1	0	0	5	0	8	15	10	35	24.4	11.2	0.7
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0	1.2	0.2	0.0
Campylobacteriosis§	71	0	66	1,103	440	78	1,165	441	3,364	3,417	3,555	11,471	11,435.4	3,666.6	0.9
Cryptosporidiosis	0	78	8	84	13	20	93	15	311	480	262	1,550	1,882.4	269.0	1.2
Haemolytic uraemic syndrome	0	2	0	2	1	0	0	0	5	6	2	19	9.8	2.6	1.9
Hepatitis A	1	18	0	13	4	0	17	3	56	86	43	228	238.8	69.4	0.8
Hepatitis E	0	4	0	2	0	0	2	1	9	11	2	34	18.6	4.8	1.9
Listeriosis	2	8	0	3	0	0	1	4	18	14	10	60	44.4	12.4	1.5
Salmonellosis	21	376	76	267	132	16	256	177	1,321	1,967	1,498	6,187	6,144.4	1,247.8	1.1
Shigellosis	1	32	34	14	30	0	53	37	201	184	163	627	433.8	120.6	1.7
STEC, VTEC‖	0	2	0	7	4	0	2	0	15	19	13	65	50.6	13.4	1.1
Typhoid	0	7	0	2	0	0	9	1	19	26	21	80	55.0	14.8	1.3
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	0	0	0	2.2	0.4	0.0
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0.0

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

Disease	State or territory								Total 3rd quarter 2008†	Total 2nd quarter 2008	Total 3rd quarter 2007	Last 5 years mean 3rd quarter	Year to date 2008	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection¶	270	3,534	493	3,714	885	392	3,112	2,131	14,531	15,156	12,685	44,195	31,284.0	10,251.0	1.4
Donovanosis	0	0	0	0	0	0	0	0	0	1	1	1	7.4	2.2	0.0
Gonococcal infection	3	342	322	373	74	3	227	390	1,734	2,356	1,830	6,166	5,921.4	1,803.0	1.0
Syphilis (all)	18	350	59	77	16	10	199	78	807	830	856	2,476	256.4	670.3	1.2
Syphilis < 2 years duration	1	62	10	48	16	4	77	59	277	350	377	998	645.8	188.0	1.5
Syphilis >2 years or unspecified duration	17	288	49	29	NDP	6	122	19	530	480	479	1,478	1,266.3	372.6	1.4
Syphilis - congenital	0	3	0	0	0	0	0	0	3	2	0	5	10.2	2.0	1.5
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0.0
Haemophilus influenzae type b	0	2	0	1	1	0	0	0	4	11	5	20	13.4	6.4	0.6
Influenza (laboratory confirmed)	164	1,028	104	2,823	234	291	979	701	6,324	705	8,823	7,463	4,264.6	3,630.0	1.7
Measles	0	1	0	2	1	0	0	1	5	26	4	64	47.6	8.6	0.6
Mumps	0	11	20	2	6	1	4	12	56	55	149	255	166.8	71.2	0.8
Pertussis	33	1,630	148	550	364	48	401	89	3,263	2,021	1,534	6,804	6,123.2	2,848.4	1.1
Pneumococcal disease (invasive)	1	209	19	149	41	14	130	58	621	432	606	1,263	1,473.4	719.0	0.9
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	1	0	0.2	0.2	0.0
Rubella	0	7	0	3	0	0	1	2	13	9	5	26	35.6	9.8	1.3
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	0	0	1.2	0.4	0.0
Tetanus	0	0	0	0	0	0	0	0	0	1	2	4	2.2	0.6	0.0
Varicella zoster (chickenpox)	0	0	29	166	144	9	0	74	422	278	495	966	496.5	248.3	0.8
Varicella zoster (shingles)	4	0	32	99	172	28	0	107	442	444	332	1,421	332.0	166.0	1.3
Varicella zoster (unspecified)	24	0	1	858	114	9	0	197	1,203	1,033	1,084	3,209	1,047.0	418.8	1.1
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	0	4	0	0	2	0	6	3	3	16	32.8	7.8	0.8
Barmah Forest virus infection	0	87	19	221	11	0	2	34	374	486	318	1,692	1,233.0	253.6	1.5
Dengue virus infection	2	37	5	21	4	3	0	21	93	93	65	339	308.0	40.6	2.3
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0	0.4	0.0	0.0
Kunjin virus infection	0	0	0	1	0	0	0	0	1	0	0	1	2.8	0.0	0.0
Malaria	5	31	3	42	5	2	42	22	152	128	115	405	515.4	148.4	1.0
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	1	0	2	0.8	0.0	0.0
Ross River virus infection	3	149	43	393	31	0	19	130	768	1,159	654	4,717	3,384.4	344.4	2.2

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

Disease	State or territory								Total 3rd quarter 2008†	Total 2nd quarter 2008	Total 3rd quarter 2007	Last 5 years mean 3rd quarter	Year to date 2008	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0	0.4	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	1	0	14	0	0	0	0	15	13	10	36	25.6	9.2	1.6
Leptospirosis	0	4	0	9	0	0	0	1	14	30	10	87	114.2	22.4	0.6
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0.0
Ornithosis	0	11	0	2	0	0	12	0	25	33	16	81	128.6	43.8	0.6
Q fever	0	46	1	29	4	0	7	0	87	69	110	266	339.0	104.4	0.8
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0.0
Other bacterial infections															
Legionellosis	0	13	1	10	4	0	10	18	56	76	50	194	236.6	66.2	0.8
Leprosy	0	0	1	0	0	0	0	0	1	2	1	6	7.4	2.0	0.5
Meningococcal infection**	2	41	3	29	8	0	24	9	116	65	123	224	299.6	139.4	0.8
Tuberculosis	3	112	1	40	10	2	90	22	280	281	292	858	777.6	280.4	1.0
Total	707	10,258	1,567	12,011	2,993	1,037	8,008	5,272	41,853	36,778	40,634	118,346	97111.7	33,161.0	1.3

* 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 date of notification.

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

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

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

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

¶ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only genital tract specimens; Queensland and the Northern Territory, which excludes 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 July to 30 September 2008, by state or territory. (Annualised rate per 100,000 population)

Disease*	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (incident)	0.0	0.9	1.9	1.1	0.3	1.6	1.9	3.2	1.4
Hepatitis B (unspecified)	28.3	45.4	63.3	21.0	28.3	17.0	39.7	30.8	35.6
Hepatitis C (incident)	1.2	0.4	5.6	0.0	2.0	4.1	2.5	4.7	1.6
Hepatitis C (unspecified)	63.6	74.1	76.3	62.1	30.0	67.3	43.7	55.5	58.7
Hepatitis D	0.0	0.1	0.0	0.1	0.0	0.0	0.4	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis†	83.6	NN	122.8	105.5	111.1	63.2	89.5	83.8	95.3
Cryptosporidiosis	0.0	4.5	14.9	8.0	3.3	16.2	7.1	2.8	5.9
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.2	0.3	0.0	0.0	0.0	0.1
Hepatitis A	1.2	1.0	0.0	1.2	1.0	0.0	1.3	0.6	1.1
Hepatitis E	0.0	0.2	0.0	0.2	0.0	0.0	0.2	0.2	0.2
Listeriosis	2.4	0.5	0.0	0.3	0.0	0.0	0.1	0.8	0.3
Salmonellosis	24.7	21.8	141.4	25.5	33.3	13.0	19.7	33.6	25.1
Shigellosis	1.2	1.9	63.3	1.3	7.6	0.0	4.1	7.0	3.8
STEC, VTEC‡	0.0	0.1	0.0	0.7	1.0	0.0	0.2	0.0	0.3
Typhoid	0.0	0.4	0.0	0.2	0.0	0.0	0.7	0.2	0.4
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§	317.9	205.2	917.5	355.3	223.5	317.8	239.2	404.7	276.6
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	3.5	19.9	599.3	35.7	18.7	2.4	17.4	74.1	33.0
Syphilis (all)	21.2	20.3	109.8	7.4	4.0	8.1	15.3	14.8	15.4
Syphilis <2 years duration	1.2	3.6	18.6	4.6	4.0	3.2	5.9	11.2	5.3
Syphilis >2 years or unspecified duration	20.0	16.7	91.2	2.8	NDP	4.9	9.4	3.6	10.1
Syphilis - congenital	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	0.0	0.1	0.3	0.0	0.0	0.0	0.1
Influenza (laboratory confirmed)	193.1	59.7	193.6	270.1	59.1	235.9	75.2	133.1	120.4
Measles	0.0	0.1	0.0	0.2	0.3	0.0	0.0	0.2	0.1
Mumps	0.0	0.6	37.2	0.2	1.5	0.8	0.3	2.3	1.1
Pertussis	38.9	94.7	275.4	52.6	91.9	38.9	30.8	16.9	62.1
Pneumococcal disease (invasive)	1.2	12.1	35.4	14.3	10.4	11.4	10.0	11.0	11.8

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

Disease*	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Vaccine preventable diseases, *continued*									
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.4	0.0	0.3	0.0	0.0	0.1	0.4	0.2
Rubella - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Varicella zoster (chickenpox)	0.0	NN	54.0	15.9	36.4	7.3	NN	14.1	18.9
Varicella zoster (shingles)	4.7	NN	59.6	9.5	43.4	22.7	NN	20.3	19.8
Varicella zoster (unspecified)	28.3	NN	1.9	82.1	28.8	7.3	NN	37.4	53.9
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	0.0	0.4	0.0	0.0	0.2	0.0	0.1
Barmah Forest virus infection	0.0	5.1	35.4	21.1	2.8	0.0	0.2	6.5	7.1
Chikungunya virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Dengue virus infection	2.4	2.1	9.3	2.0	1.0	2.4	0.0	4.0	1.8
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Malaria	5.9	1.8	5.6	4.0	1.3	1.6	3.2	4.2	2.9
Murray Valley encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	3.5	8.7	80.0	37.6	7.8	0.0	1.5	24.7	14.6
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	1.3	0.0	0.0	0.0	0.0	0.3
Leptospirosis	0.0	0.2	0.0	0.9	0.0	0.0	0.0	0.2	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	0.6	0.0	0.2	0.0	0.0	0.9	0.0	0.5
Q fever	0.0	2.7	1.9	2.8	1.0	0.0	0.5	0.0	1.7
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial infections									
Legionellosis	0.0	0.8	1.9	1.0	1.0	0.0	0.8	3.4	1.1
Leprosy	0.0	0.0	1.9	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection‖	2.4	2.4	5.6	2.8	2.0	0.0	1.8	1.7	2.2
Tuberculosis	3.5	6.5	1.9	3.8	2.5	1.6	6.9	4.2	5.3

* 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; Queensland and the Northern Territory, which excludes 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.

Laboratory Serology and Virology Reporting Scheme

There were 9,524 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 July to 30 September 2008 (Tables 4 and 5).

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

	State or territory								This period 2008	This period 2007	Year to date 2008	Year to date 2007
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	–	–	–	3	3	–	–	–	6	1	30	18
Mumps virus	–	–	2	1	2	–	–	–	5	12	39	41
Rubella virus	–	–	–	3	–	–	–	–	3	–	12	15
Hepatitis viruses												
Hepatitis A virus	–	–	–	5	4	–	–	–	9	4	44	31
Hepatitis D virus	–	–	–	1	1	–	–	–	2	–	20	20
Hepatitis E virus	–	–	–	1	–	–	1	–	2	–	8	1
Arboviruses												
Ross River virus	–	3	1	178	24	–	1	2	209	59	1,266	887
Barmah Forest virus	–	4	2	82	17	–	1	–	106	34	483	409
Flavivirus (unspecified)	–	1	–	12	–	–	–	–	13	7	55	81
Adenoviruses												
Adenovirus not typed/ pending	2	80	–	269	216	–	16	1	584	177	1,299	893
Herpesviruses												
Cytomegalovirus	1	62	–	172	111	1	3	1	351	121	971	945
Varicella-zoster virus	6	134	–	528	186	5	13	1	873	267	2,195	2,125
Epstein-Barr virus	–	19	13	316	233	7	1	29	618	230	1,837	1,965
Other DNA viruses												
Parvovirus	–	3	–	67	6	–	8	–	84	49	207	289
Picornavirus family												
Coxsackievirus A9	–	3	–	–	–	–	–	–	3	–	4	–
Coxsackievirus A16	–	4	–	–	–	–	–	–	4	–	4	–
Echovirus type 11	–	5	–	–	–	–	–	–	5	–	10	–
Rhinovirus (all types)	–	45	–	–	2	–	–	–	47	38	138	245
Enterovirus not typed/ pending	–	7	–	10	4	–	1	–	22	10	122	128
Picornavirus not typed	–	1	–	–	–	1	–	–	2	3	9	7
Ortho/ paramyxoviruses												
Influenza A virus	4	106	2	292	79	2	20	–	505	264	643	2,227
Influenza B virus	6	152	1	333	240	6	38	–	776	39	867	131
Parainfluenza virus type 1	–	2	–	5	20	–	5	–	32	11	181	39
Parainfluenza virus type 2	–	1	–	3	1	–	–	–	5	2	25	59
Parainfluenza virus type 3	–	60	–	70	35	–	9	–	174	131	198	373
Respiratory syncytial virus	1	232	–	163	355	23	71	2	847	284	1,791	2,063

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

	State or territory								This period 2008	This period 2007	Year to date 2008	Year to date 2007
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other RNA viruses												
HTLV-1	–	–	–	–	24	–	–	–	24	1	42	12
Rotavirus	–	37	–	–	34	2	5	–	78	111	275	366
Norwalk agent	–	42	–	–	–	4	–	–	46	219	74	767
Other												
Chlamydia trachomatis not typed	32	291	–	1,301	545	19	8	2	2,198	692	6,594	6,326
Chlamydia pneumoniae	–	–	–	–	–	1	–	–	1	–	2	1
Chlamydia psittaci	1	2	–	1	–	1	19	–	24	1	79	39
Chlamydia species	–	–	–	–	–	–	1	–	1	–	3	2
Mycoplasma pneumoniae	–	13	6	129	75	5	74	3	305	120	729	989
Mycoplasma hominis	–	3	–	–	–	–	–	–	3	2	7	7
Coxiella burnetii (Q fever)	3	5	–	18	18	–	9	–	53	25	205	183
Rickettsia - spotted fever group	–	2	1	1	4	–	2	–	10	9	100	98
Streptococcus group A	–	10	48	260	–	–	–	–	318	106	768	825
Yersinia enterocolitica	–	5	–	–	–	–	–	–	5	1	10	7
Brucella species	–	–	–	11	–	–	–	–	11	3	28	7
Bordetella pertussis	–	99	2	140	350	1	1	–	593	67	1,039	666
Legionella pneumophila	–	–	–	–	–	–	1	–	1	4	12	28
Legionella longbeachae	–	–	–	–	2	–	2	–	4	–	9	6
Cryptococcus species	–	1	–	5	3	–	–	–	9	15	24	42
Leptospira species	–	1	–	14	1	–	–	–	16	9	70	52
Treponema pallidum	–	63	13	268	168	–	9	–	521	176	1,629	1,785
Entamoeba histolytica	–	–	2	1	–	–	1	–	4	–	8	6
Toxoplasma gondii	–	–	–	3	3	–	–	–	6	1	12	21
Echinococcus granulosus	–	–	–	–	4	–	2	–	6	1	28	16
Total	56	1,498	93	4,666	2,770	78	322	41	9,524	3,306	24,205	25,243

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

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

– No data received this period.

Table 5. Virology and serology reports by laboratories for the reporting period 1 July to 30 September 2008*

State or territory	Laboratory	July 2008	August 2008	September 2008	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	118	125	180	423
	New Children's Hospital, Westmead	140	127	128	395
	Repatriation General Hospital, Concord	–	–	–	0
	Royal Prince Alfred Hospital, Camperdown	38	43	53	134
	South West Area Pathology Service, Liverpool	63	75	72	210
Queensland	Queensland Medical Laboratory, West End	1,376	1,804	1,992	5,172
	Townsville General Hospital	–	–	–	0
South Australia	Institute of Medical and Veterinary Science, Adelaide	829	893	1,040	2,762
Tasmania	Northern Tasmanian Pathology Service, Launceston	27	19	24	70
	Royal Hobart Hospital, Hobart	–	–	–	0
Victoria	Australian Rickettsial Reference Laboratory	23			23
	Monash Medical Centre, Melbourne	58	52	23	133
	Royal Children's Hospital, Melbourne	–	–	–	0
	Victorian Infectious Diseases Reference Laboratory, Fairfield	48	65	41	154
Western Australia	PathWest Virology, Perth	–	–	–	0
	Princess Margaret Hospital, Perth	–	–	–	0
	Western Diagnostic Pathology	48	–	–	48
Total		2,768	3,203	3,553	9,524

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

– No data received this period.

Additional reports

Australian Sentinel Practices Research Network

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

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

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published. In 2008, 4 conditions are being monitored. They include influenza like illness, gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, *published in* Commun Dis Intell *2008;32:135.*

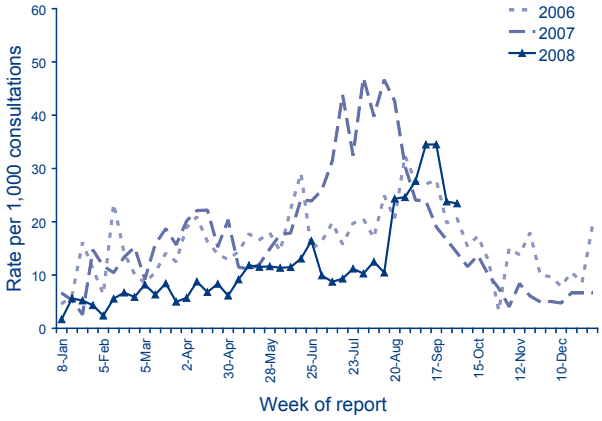
Data on influenza-like illness, gastronenteritis, chickenpox and shingles from 1 July to 30 September 2008 compared with 2007, are shown as the rate per 1,000 consultations in Figures 1, 2, 3 and 4, respectively.

Reporting period 1 July to 30 September 2008

Sentinel practices contributing to ASPREN were located in all jurisdictions other than the Northern Territory. A total of 104 general practitioners contributed data to ASPREN in the second quarter of 2008. Each week an average of 80 general practitioners provided information to ASPREN at an average of 7,804 (range 6,850 to 8,464) consultations per week.

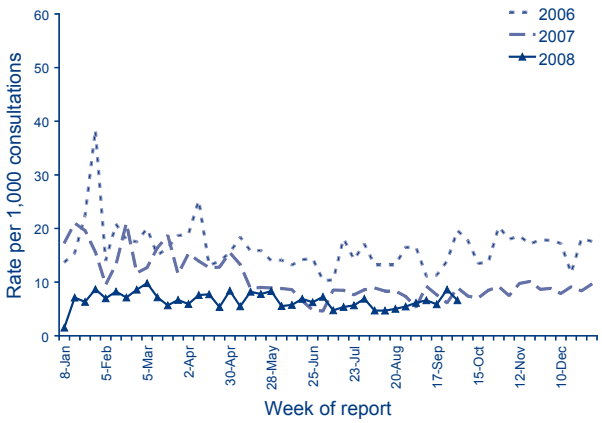
Influenza-like illness (ILI) rates reported from 1 July to 30 September 2008 were lower (9–35 cases per 1,000 consultations) compared with the same reporting period in 2007 (30–47 cases per 1,000 consultations. The rise in ILI rates in the third quarter of 2008 in mid-August occurred later compared with 2007 (beginning July). The peak ILI rate of 35 cases per 1,000 consultations occurred in mid-September (Figure 1).

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



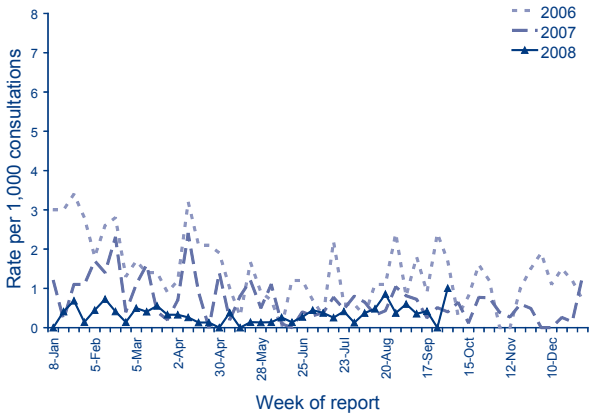
Reports of gastroenteritis from 1 July to 30 September 2008 were lower compared with the same period in 2007 (Figure 2). During this reporting period, consultation rates for gastroenteritis ranged from 5 to 9 cases per 1,000 consultations.

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



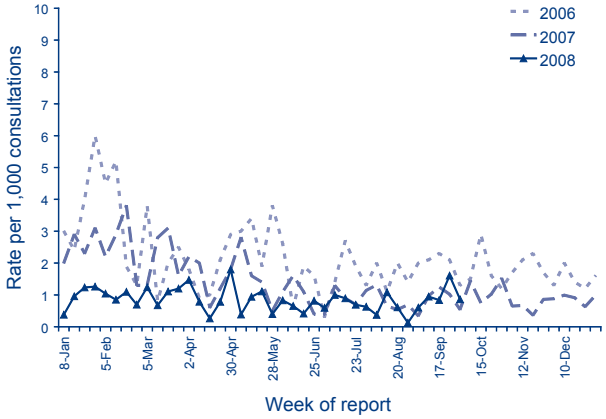
Reports of varicella infections were reported at a similar rate for the third quarter of 2008 compared with the same period in 2007. From 1 July to 30 September 2008, recorded rates for chickenpox were between 0 to 1 case per 1,000 consultations (Figure 3).

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



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

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



Australian childhood immunisation coverage

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

The data show the percentage of children fully immunised at 12 months of age for the cohort born between 1 April and 30 June 2007, at 24 months of age for the cohort born between 1 April and 30 June 2006, and at 5 years of age for the cohort born between 1 April and 30 June 2002 according to the National Immunisation Program Schedule. However from March 2002 to December 2007, coverage for vaccines due at 4 years of age was assessed at the 6-year milestone age.

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

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

'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of 3 doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of *Haemophilus influenzae* type b (Hib) vaccine, and 2 or 3 doses of hepatitis B vaccine. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of 3 or 4 doses of a DTP-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of Hib vaccine, 2 or 3 doses of hepatitis B vaccine and 1 dose of a measles, mumps and rubella-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 remained unchanged at 91.2% (Table 1). The only important changes in coverage for any individual vaccines due at 12 months of age occurred in the Northern Territory, where coverage for all vaccines decreased by 2 percentage points and fully immunised coverage dropped just below 90% for the 1st time since mid-2004.

Immunisation coverage for children 'fully immunised' at 24 months of age for Australia decreased by 0.3 of a percentage point to 92.5 (Table 2). There were no important changes in coverage for any individual vaccines due at 24 months of age or by jurisdiction.

Immunisation coverage for 'fully immunised' at 5 years of age for Australia decreased for the third consecutive quarter, by 0.5 of a percentage point, to 86.8% (Table 3). Coverage for all individual vaccines also decreased by 0.6 of a percentage point for Australia, however there were no important changes in coverage for any jurisdiction. This decrease in coverage is likely due to the change in the coverage calculation algorithm, which, since the beginning of 2008, now calculates coverage for vaccines due at 4 years of age at the 5-year milestone, not the 6-year milestone. This means late immunisations given to a child aged between 5 and 6 years are no longer included in the assessment.

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

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

Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,145	24,196	996	15,320	4,695	1,524	17,163	7,387	72,426
Diphtheria, tetanus, pertussis (%)	94.1	91.8	90.3	91.4	92.1	91.8	92.7	90.5	91.8
Poliomyelitis (%)	94.0	91.8	90.3	91.4	92.1	91.8	92.7	90.5	91.8
Haemophilus influenzae type b (%)	96.0	94.8	93.8	93.8	94.3	95.3	94.7	94.3	94.5
Hepatitis B (%)	95.6	94.8	94.2	93.6	94.2	95.2	94.5	94.2	94.4
Fully immunised (%)	93.5	91.5	89.8	90.7	91.4	91.6	91.6	90.0	91.2
Change in fully immunised since last quarter (%)	-0.1	+0.2	-1.8	-0.1	+0.4	+0.6	-0.3	-0.1	-0.0

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

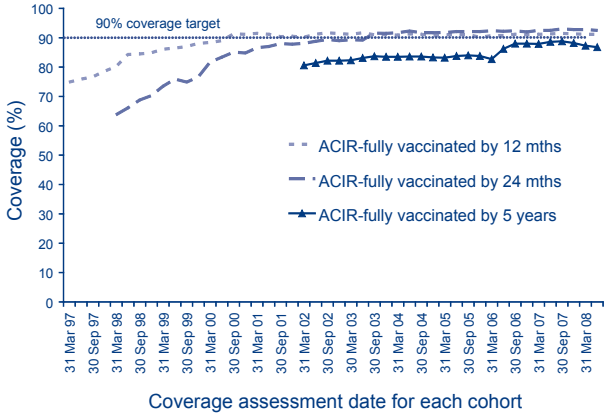
Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,137	22,861	946	14,549	4,518	1,403	16,684	7,154	69,252
Diphtheria, tetanus, pertussis (%)	97.2	94.8	95.9	94.5	94.5	95.4	95.5	93.7	94.8
Poliomyelitis (%)	97.2	94.7	95.9	94.4	94.5	95.2	95.4	93.7	94.8
Haemophilus influenzae type b (%)	97.0	95.2	94.3	93.4	93.3	95.4	94.4	93.5	94.4
Measles, mumps, rubella (%)	96.1	93.7	95.2	93.5	93.4	94.4	94.6	92.9	93.9
Hepatitis B (%)	97.4	95.6	97.2	95.1	95.1	95.9	95.9	94.4	95.5
Fully immunised (%)	94.9	92.4	93.6	91.9	92.4	93.5	93.4	91.2	92.5
Change in fully immunised since last quarter (%)	+0.1	-0.0	-1.2	-0.7	-0.9	+0.1	-0.3	+0.0	-0.3

* The 12 months age data for this cohort was published in *Commun Dis Intell* 2007;31:348.

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

Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	998	22,128	937	13,897	4,482	1,362	15,877	6,719	66,400
Diphtheria, tetanus, pertussis (%)	91.3	86.5	87.1	87.2	86.2	89.7	90.6	84.3	87.5
Poliomyelitis (%)	91.2	86.3	87.0	87.1	86.0	89.8	90.5	84.2	87.4
Measles, mumps, rubella (%)	90.7	86.1	87.1	86.9	86.2	89.7	90.3	84.2	87.2
Fully immunised (%)	90.6	85.7	86.5	86.4	85.7	89.2	89.9	83.4	86.8
Change in fully immunised since last quarter (%)	+1.7	-0.7	-1.5	-0.9	+1.6	-0.6	-0.5	-0.7	-0.5

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



Gonococcal surveillance

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

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

Reporting period 1 April to 30 June 2008

The AGSP laboratories received a total of 854 isolates in this quarter, a slight increase over the 823 isolates seen in the corresponding period in 2007. Of these, 831 remained viable for susceptibility testing. About 26.5% of this total was from New South Wales, 17% from Victoria, 16% from South Australia, 15% from Queensland, 13% from Western Australia and 11% from the Northern Territory. There was a single isolate from the Australian Capital Territory and 3 from Tasmania. There was a decline in numbers examined in some states but a large increase in South Australia.

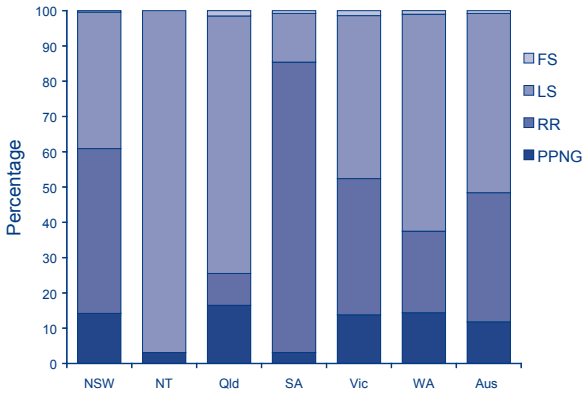
Penicillins

In this quarter, 402 (48.4%) of all isolates examined were penicillin resistant by one or more mechanisms. Ninety-eight (11.8%) were penicillinase-producing *Neisseria gonorrhoeae* (PPNG) and 304 (36.6%) resistant by chromosomal mechanisms, (CMRP). These proportions were greatly increased from those recorded in this quarter in 2007, when 259 (32.1%) of 806 isolates examined nationally were penicillin resistant. PPNG increased from the 79 (9.8%) seen in 2007, and CMRP from 180 (22.3%) isolates in 2007. The proportion of all strains resistant to the penicillins by any mechanism ranged from 3% in the Northern Territory to 85.4% in South Australia. High rates of penicillin resistance were also found in New South Wales (61%), Victoria (52.4%), Western Australia (37.5%) and Queensland (25.5%).

Figure 6 shows the proportions of gonococci fully sensitive (MIC ≤ 0.03 mg/L), less sensitive (MIC 0.06–0.5 mg/L), relatively resistant (MIC ≤ 1 mg/L)

or else PPNG aggregated for Australia and by state and territory. A high proportion of those strains classified as PPNG or CMRP will fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

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



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

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

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

PPNG Penicillinase producing *Neisseria gonorrhoeae.*

Most of the resistance in South Australia was related to CMRP—107, comprising 82.3% of isolates—whereas only 4 (3.1%) isolates were PPNG. In New South Wales and Victoria most of the penicillin resistance was also due to CMRP. In New South Wales 105 (46.7%) isolates were CMRP with 32 PPNG (14.2%) and in Victoria 56 (38.6%) were CMRP and 20 (13.8%) PPNG. In Western Australia 24 CMRP were detected accounting for 23% of isolates with 15 PPNG accounting for 14.4% of isolates. In Queensland, PPNG were more prominent (16.5%, 21 isolates) with 9% CMRP. Three PPNG were noted in both Tasmania and the Northern Territory, but no CMRP were detected. The single isolate from the Australian Capital Territory was chromosomally resistant.

Ceftriaxone

Five isolates with decreased susceptibility to ceftriaxone (MIC 0.06 and 0.12 mg/L) were detected, one each in Queensland and South Australia and three in New South Wales.

Spectinomycin

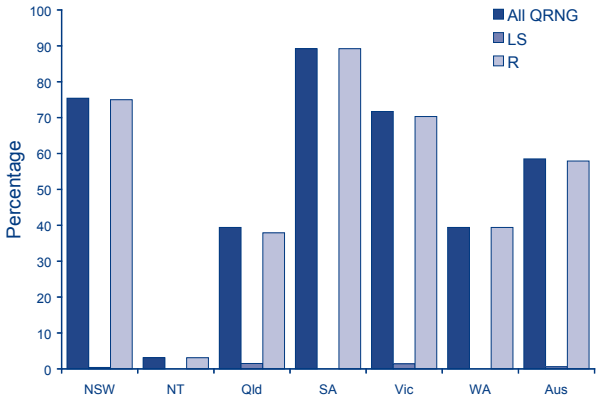
All isolates were susceptible to this injectable agent.

Quinolone antibiotics

QRNG are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06–0.5 mg/L) or resistant (MIC ≤ 1 mg/L) groups.

A total of 486 quinolone resistant *N. gonorrhoeae* (QRNG) were detected during this quarter and represented 58.5% of all gonococci tested nationally. This was a further increase in the proportion of QRNG when compared with the 44.5% in this quarter in 2007, 33.7% in 2006 and 30% in 2005. The great majority of QRNG in the current period (99%) continued to exhibit higher-level resistance (ciprofloxacin MICs 1mg/L or more) (Figure 7).

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



LS QRNG Ciprofloxacin MICs 0.06–0.5 mg/L.

R QRNG Ciprofloxacin MICs ≥1 mg/L.

QRNG were detected in all states and territories. The highest proportion of QRNG was present in South Australia where 116 QRNG accounted for 89.2% of all isolates. A high number (169) and proportion (75%) of QRNG were also found in New South Wales, Victoria (104 QRNG, 72%), Queensland (49 QRNG, 39%), and Western Australia (41 QRNG, 39%). Three isolates from Tasmania and the Northern Territory were QRNG as was the single strain from the Australian Capital Territory.

High level tetracycline resistance

The number (145) of high level tetracycline resistance (TRNG) detected this quarter was greater than the 121 found in the corresponding quarter in 2007 and represented 17.5% of all isolates. The

highest proportion of TRNG in any jurisdiction (37%) was in Western Australia and the highest number (44) was detected in New South Wales. TRNG were present in all states and territories except the Australian Capital Territory.

Reference

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

Meningococcal surveillance

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

The reference laboratories of the Australian Meningococcal Surveillance Programme report data on the number of laboratory confirmed cases confirmed either by culture or by non-culture based techniques. Culture positive cases, where a Neisseria meningitidis is grown from a normally sterile site or skin, and non-culture based diagnoses, derived from results of nucleic acid amplification assays and serological techniques, are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions. Data contained in the quarterly reports are restricted to a description of the number of cases per jurisdiction, and serogroup, where known. A full analysis of laboratory confirmed cases of IMD is contained in the annual reports of the Programme, published in Communicable Diseases Intelligence. For more information see Commun Dis Intell *2008;32:135.*

Laboratory confirmed cases of invasive meningococcal disease for the period 1 July to 30 September 2008, are included in this issue of Communicable Diseases Intelligence *(Table 4).*

HIV and AIDS surveillance

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

Table 4. Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 July to 30 September 2008, by serogroup and state or territory

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD
Australian Capital Territory	08			0	2	1	1							1	3
	07			1	3						1			1	4
New South Wales	08			14	27	1	4	1	3	1	2			17	36
	07			35	52	1	7	2	4	0	1	3	7	41	70
Northern Territory	08			3	3	0	2							3	5
	07			0	1	0	1							0	2
Queensland	08			11	52	2	4					11	11	24	67
	07			24	43	4	5	1	1	2	2		1	31	52
South Australia	08			5	12					1	1			6	13
	07			5	9	1	1					1	1	7	11
Tasmania	08													0	0
	07			2	2			1	1		1			3	5
Victoria	08			20	44	1	1	0	1			3	6	24	52
	07			14	35	0	2	1	4	1	2	3	4	19	47
Western Australia	08			8	16							0	1	8	17
	07			8	15									8	15
Total	08			61	156	5	12	1	4	2	3	14	18	83	193
	07			89	160	6	16	5	10	3	6	7	13	110	205

ties to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available 3 months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in 'HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia, annual surveillance report'. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Internet: http://www.med.unsw.edu.au/nchecr. Telephone: +61 2 9332 4648. Facsimile: +61 2 9332 1837. For more information see Commun Dis Intell *2005;29:91–92.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 January to 31 March 2008, as reported to 30 June 2008, are included in this issue of Communicable Diseases Intelligence *(Tables 5 and 6).*

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

	Sex	State or territory								Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2008	This period 2007	YTD 2008	YTD 2007
HIV diagnoses	Female	0	11	0	5	2	1	6	3	28	33	28	33
	Male	5	85	0	43	9	3	68	10	223	253	223	253
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	5	96	0	48	11	4	74	13	251	286	251	286
AIDS diagnoses	Female	0	1	0	0	0	0	1	0	2	3	2	3
	Male	1	8	1	7	1	0	16	2	36	35	36	35
	Total*	1	9	1	7	1	0	17	2	38	38	38	38
AIDS deaths	Female	0	1	0	0	0	0	0	0	1	0	1	0
	Male	0	4	0	1	0	0	3	0	8	15	8	15
	Total*	0	5	0	1	0	0	3	0	9	15	9	15

* Totals include people whose sex was reported as transgender.

Table 6. Cumulative diagnoses of HIV infection, AIDS, and deaths following AIDS since the introduction of HIV antibody testing to 31 March 2008, and reported by 30 June 2008, by sex and state or territory

	Sex	State or territory								Australia
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
HIV diagnoses	Female	32	939	23	303	113	13	406	224	2,053
	Male	274	13,908	137	2,940	988	115	5,569	1,284	25,215
	Not reported	0	228	0	0	0	0	22	0	250
	Total*	306	15,105	160	3,252	1,102	128	6,019	1,515	27,587
AIDS diagnoses	Female	10	264	4	73	32	4	117	42	546
	Male	94	5,520	46	1,062	413	55	2,068	436	9,694
	Total*	104	5,802	50	1,137	446	59	2,198	480	10,276
AIDS deaths	Female	7	139	1	43	20	2	64	29	305
	Male	73	3,601	30	677	280	33	1,429	299	6,422
	Total*	80	3,751	31	722	300	35	1,502	329	6,750

* Totals include people whose sex was reported as transgender.

National Enteric Pathogens Surveillance System

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

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

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

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

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

Reports to the National Enteric Pathogens Surveillance System of Salmonella *infection for the period 1 July to 30 September 2008 are included in Tables 7 and 8. Data include cases reported and entered by 21 October 2008. Counts are preliminary, and subject to adjustment after completion of typing and reporting of further cases to NEPSS. For more information see* Commun Dis Intell *2008;32:137.*

Reporting period 1 July to 30 September 2008

There were 1,018 reports to NEPSS of human *Salmonella* infection in the third quarter of 2008, approximately 40% fewer than in the second quarter of 2008. Limited third quarter data from Western Australia were available at the time of preparing this report. Taking this into account, the overall count of cases for the remainder of Australia was similar to the recent historical mean number of reports for this time of each year. The nadir in the annual cycle of human salmonellosis in Australia typically occurs in August–September.

During the third quarter of 2008, the 25 most common *Salmonella* types in Australia accounted for 617 cases, 61% of all reported human *Salmonella* infections. Sixteen of the 25 most common *Salmonella* infections in the third quarter of 2008 were also among those most commonly reported in the preceding quarter.

The most conspicuous feature of the national data was the predominance of various phage types of *S.* Typhimurium, which comprised 6 of the 8 most common salmonellae. Among these, the increase in *S.* Typhimurium phage type 9 above the historical average was due to increased cases in New South Wales, Victoria and South Australia. The increase in *S.* Typhimurium phage type 44 was mostly due to cases in Victoria and New South Wales. Increases of *S.* Typhimurium phage type 29 (mostly in Queensland and South Australia) and *S.* Typhimurium phage type 193 (South Australia) were more geographically restricted. *S.* Stanley was moderately elevated (most cases in New South Wales and Victoria).

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

Table 7. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 July to 30 September 2008, as reported to 21 October 2008

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA*	Australia
Total all *Salmonella* for quarter	23	324	58	211	130	13	249	10	1,018
Total contributing *Salmonella* types	16	89	30	74	54	10	87	5	179

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

Table 8. Top 25 *Salmonella* types identified in Australia, 1 July to 30 September 2008, by state or territory

National rank	*Salmonella* type	State or territory								Total 3rd quarter 2008	Last 10 years mean 3rd quarter	Year to date 2008	Year to date 2007
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
1	*S.* Typhimurium PT 9	4	44	2	9	12	0	25	0	96	63	364	608
2	*S.* Typhimurium PT 135	0	20	1	16	5	0	17	0	59	96	724	528
3	*S.* Typhimurium PT 44	0	18	0	13	1	0	24	0	56	11	282	332
4	*S.* Typhimurium PT 170	2	34	0	6	0	0	13	0	55	28	223	224
5	*S.* Stanley	2	13	0	2	3	1	13	0	34	21	88	100
6	*S.* Virchow PT 8	0	4	7	12	1	0	2	0	26	30	146	184
7	*S.* Typhimurium PT 29	0	3	2	9	11	0	0	0	25	1.8	62	136
8	*S.* Typhimurium untypable	1	5	0	4	9	0	5	0	24	10	60	69
9	*S.* Enteritidis PT 6a	0	5	0	5	2	2	8	0	22	12	49	55
10	*S.* Infantis	0	8	0	3	7	0	2	0	20	27	143	145
11	*S.* Saintpaul	0	3	4	8	1	1	2	0	19	49	186	280
12	*S.* Chester	0	1	3	8	2	0	4	0	18	22	112	127
13	*S.* Enteritidis PT 1	1	3	0	1	2	0	8	1	16	9	41	22
14	*S.* Birkenhead	1	12	0	1	0	0	0	0	14	23	142	163
15	*S.* Weltevreden	0	4	2	2	2	0	4	0	14	10	71	49
16	*S.* Typhimurium PT 193	0	0	1	0	12	0	1	0	14	4.3	37	42
17	*S.* Anatum	0	6	5	0	1	0	1	0	13	12	62	60
18	*S.* Typhimurium PT 12	0	7	0	0	2	0	4	0	13	11	41	78
19	*S.* Singapore	0	11	0	0	0	0	2	0	13	8	66	55
20	*S.* Agona	1	5	0	5	1	0	0	0	12	13	42	44
21	*S.* Aberdeen	0	0	4	8	0	0	0	0	12	12	66	107
22	*S.* Javiana	0	4	0	4	0	0	3	0	11	5	25	37
23	*S.* Montevideo	0	4	0	6	0	0	1	0	11	5	67	97
24	*S.* Typhimurium PT 197	0	3	0	5	1	0	1	0	10	19	83	158
25	*S.* Typhimurium PT 6 var 1	1	2	0	1	0	0	0	6	10	2.8	38	20

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

OVERSEAS BRIEF

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

Reporting period 1 July to 30 September 2008

Chikungunya in India

The number of probable cases of chikungunya continued to rise in India during the third quarter of 2008, July through September. The total number of probable cases to date for 2008 has now increased to 70,740.[1] These have yet to be laboratory confirmed. This compares with the 59,536 cases reported for 2007.[2]

The resurgence and geographic spread of chikungunya in recent years shows our vulnerability to vectorborne, emerging infectious diseases and emphasises the importance of sustained control programs to maintain health security.

Dengue in the Pacific Region

During this quarter the World Health Organization (WHO) has declared a dengue pandemic in the Pacific Region and is calling for a co-ordinated regional response.[3]

For 2008, year-to-date cases of dengue in ongoing outbreaks have been reported in:

- New Caledonia, 1,000;[4]
- American Samoa, 162;[4]
- Fiji more than 1,800;[5]
- Kiribati, 831;[6]
- Samoa, 427.[3]

The WHO has warned of a spreading threat of dengue outbreaks in the Asia Pacific Region with further outbreaks expected unless a more comprehensive approach to mosquito control is urgently taken. Often the disease attracts attention only during outbreaks, when it is usually too late for effective remedial action.

The *Aedes aegypti* mosquito, the principal vector for dengue virus, is spreading to geographical areas that were previously unaffected. Of an estimated 2.5 billion people at risk globally, about 1.8 billion—or more than 70%—reside in Asia Pacific countries.

Over the past 3 decades in the Asia Pacific Region, the *Aedes aegypti* mosquito has been thriving. Human activities, such as rainwater collection and inappropriate disposal of used tyres and containers where water can collect has allowed mosquitoes to breed. Recent changes in weather patterns and the rapid growth of urban areas are also expanding the habitat range of the dengue mosquito.

The WHO has urged countries to ensure high level political commitment so that adequate resources are made available for dengue prevention and outbreak response, including exploring possible opportunities to build on existing public health initiatives.

The Regional Committee for the Western Pacific, WHO's governing body in the region, endorsed the Dengue Strategic Plan for the Asia Pacific Region 2008–2015 on 26 September 2008. The move is considered a critical step towards securing the required political commitment and allocation of resources by Member States to address the increasing threat from dengue. The Regional Dengue Strategic Plan was developed with the active involvement of WHO Member States, the WHO Regional Offices for the Western Pacific and South East Asia and several leading experts in the field.[7]

Influenza (avian)

The WHO confirmed 2 human cases of H5N1[8,9] with onset dates between 1 July and 30 September 2008. Both cases were fatal. Nine cases were confirmed during the same period in 2007, of which seven were fatal (CFR 78%). The 2 WHO confirmed cases are not known to be linked epidemiologically, though they both were from Tangerang city, Banten Provence, Indonesia. Both cases were were known to have had contact with free roaming poultry, and 1 case is reported to have slaughtered and consumed sick birds within a week of disease onset.[9]

There was no evidence of human-to-human transmission of avian influenza during the reporting period.

Influenza (seasonal)

During the third quarter (which includes the Southern Hemisphere influenza season), influenza B viruses dominated in New Zealand,[10] and WHO reported influenza A and B viruses circulating in Argentina, Chile and China (Hong Kong SAR), and influenza A viruses in Brazil, and New Caledonia.[11,12]

WHO recommended the following strains for the Southern Hemisphere 2009 influenza vaccine: A/Brisbane/59/2007(H1N1)-like virus; A/

Brisbane/10/2007(H3N2)-like virus; and B/Florida/4/2006-like virus.[13] The selected strains are unchanged from those recommended for the 2008–2009 Northern Hemisphere vaccine.

Oseltamivir resistance

Between the second quarter 2008 and 22 September 2008, WHO reported 35% (324 of 931) of H1N1 isolates tested were resistant to oseltamivir (containing the H274Y mutation) by phenotypic and/or genotypic analysis.[12] Resistant isolates were detected in 20 of 27 countries that submitted data. The percentage of H1N1 oseltamivir resistant isolates detected increased from 10.8% reported in the second quarter to 35% in the third quarter. The percentage of H1N1 oseltamivir resistant isolates detected in each country varied from zero to greater than 90%; as seen in South Africa and the Philippines.

Polio

As of 30 September, there have been 1,228 reported cases of polio from the endemic countries of India (449), Nigeria (692) Pakistan (67) and Afghanistan (20). In India the highest priority is being given to stopping the wild poliovirus type 1 (WPV1) outbreak in western Uttar Pradesh. The state had been free of endemic WPV1 for more than 12 months prior to local spread of polio originally imported from Bihar.[14]

On 14 September, 2 doctors on WHO duty preparing logistics for a regional polio campaign, and their driver were killed by a vehicle-borne suicide bomber in Kandahar province, southern Afghanistan. UNESCO and UNICEF called on all parties to the regional conflict to allow safe access to vaccinators on the International Day of Peace (21 September).[15]

In Pakistan, in response to the recent increase and geographical spread of polio cases, a large-scale campaign was conducted on 15–17 September in the highest risk areas of North West Frontier Province, Balochistan, Punjab and Islamabad. The campaign aimed to reach more than 28 million children under the age of 5 years.[14]

In Nigeria, operational challenges continue to contribute to significant vaccination coverage gaps, with upwards of 60% of children remaining underimmunised.[14] The current WPV1 outbreak affecting northern Nigeria threatens to spread further. Transmission of the virus remains intense, and large-scale population movements are expected for the upcoming Hajj season (pilgrimage to Mecca, Saudi Arabia).[16] The Expert Review Committee for Polio Eradication and Routine Immunization in Nigeria (ERC), which met in July, welcomed the establishment of the several high-level bodies to respond urgently to the current outbreak.[17]

Salmonellosis in the United States of America

In a large outbreak of salmonellosis in the United States of America associated with fresh produce, public health authorities reported 1,442 cases between 16 April and 11 August. All cases were infected with a strain of *Salmonella* Saintpaul that was indistinguishable by pulsed-field gel electrophoresis. The outbreak peaked in late May and early June 2008, with the number of new cases declining from 35 per day between 1 and 7 June 2008 to 12 per day between 19 June and 3 July 2008.[18] Preliminary epidemiological and microbiological evidence support the conclusion that jalapeño peppers were a major vehicle by which the pathogen was transmitted. Serrano peppers were also a vehicle and tomatoes were thought to be a possible vehicle, particularly early in the outbreak.[19]

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The 2009 Communicable Disease Control Conference

The Communicable Diseases Network Australia (CDNA) and the Public Health Laboratory Network (PHLN) will host a 3 day Communicable Diseases Control Conference (CDC 2009) from Monday 4 to Wednesday 6 May 2009 in Canberra at the Hotel Realm.

CDC 2009 aims to promote evidence-based discussion around communicable disease control themes, including:

- communicable diseases considered to be under control;
- communicable diseases that are poorly controlled, re-emerging or newly emerging;
- communicable disease issues of importance to indigenous peoples, and
- the impact of communicable disease locally, regionally and globally.

Keynote and invited speakers

The following noted experts will draw on their experiences to provide keynote addresses introducing each theme to conference participants, from an international perspective:

- Prof. David Salisbury, Director of Immunisation, Department of Health, London
- Assoc. Prof. Julie Hall, Team Leader Emerging Infectious Disease, WHO Western Pacific Office, Manila; and
- Prof. Rick Speare, Director, Anton Breinl Centre for Public Health and Tropical Medicine, James Cook University.

The inaugural Aileen Plant Oration will be presented by Professor John McKenzie, Australian Biosecurity CRC, Curtin University of Technology.

Various research leaders will also provide plenary addresses on each day of the conference. These include:

- Dr Graham Tallis, Communicable Diseases Surveillance and Response Team Leader, WHO Indonesia;
- Dr Naomi Pomat, Paediatrician Daru Hospital, Papua New Guinea;
- Prof. Bart Currie, Professor of Medicine, Northern Territory Clinical School, Flinders University;
- Prof. Cindy Shannon, Director of the Centre for Indigenous Health, University of Queensland; and
- Dr Vicki Krause, Program Director, Disease Control Program, Northern Territory Department of Health and Community Services.

Program

- Pre-conference workshops – Sunday 3 May
- Public health nurses and public health officers workshop
- Zoonoses – presented by Prof. Rick Speare
- Threats to our regional area – presented by Assoc. Prof. Julie Hall
- How to sell vaccination programs – presented by Prof. David Salisbury

Plenary sessions

- Regional issues in infectious diseases
- Infectious diseases in Indigenous health
- Old infectious diseases

Concurrent sessions

- International and globally mobile populations
- Lessons from a mass gathering
- Pandemic flu
- Antimicrobial resistance
- Indigenous health
- Vaccines
- Laboratory aspects of public health
- Modelling and seasonal flu
- Zoonoses and arboviruses
- Infections in aged care
- Foodborne diseases
- Surveillance methods
- Bloodborne and sexually transmitted infections
- Respiratory diseases

For more information on the conference including speaker's biographies and a detailed conference program visit www.diseases.consec.com.au

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