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Annual reports

Annual report of the Australian National Poliovirus Reference Laboratory, 2006

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Abstract

The Australian National Poliovirus Reference Laboratory (NPRL), located within the Victorian Infectious Diseases Reference Laboratory, is the national laboratory for Australia, the Pacific Islands and Brunei Darussalam, and is accredited by the World Health Organization (WHO) as the Regional Reference Laboratory for the WHO Western Pacific Region. The NPRL, in collaboration with the Australian Paediatric Surveillance Unit, co-ordinates surveillance for acute flaccid paralysis (AFP), the major clinical presentation of poliovirus infection. After classification of AFP cases by the Polio Expert Committee, the non-polio AFP rate for Australia in 2006 was 1.1, meeting the WHO surveillance requirement of detecting more than one AFP case per 100,000 children aged less than 15 years. During 2006, 80 specimens were referred to the NPRL, 59 from AFP cases and 21 from other sources. Poliovirus type 3 was isolated from two patients without AFP and the isolates were characterised as Sabin-like using WHO accredited methodologies. Echovirus 30 was isolated from two cases of AFP and coxsackievirus B5 and adenovirus were isolated from individual cases. During 2006, 1,998 cases of poliomyelitis due to wild poliovirus infection were reported world-wide, of which, only 6.8% (127) were due to importation of wild poliovirus. *Commun Dis Intell* 2007;31:263–269.

Keywords: acute flaccid paralysis, disease surveillance, laboratory testing, poliomyelitis

Introduction

The World Health Organization's (WHO) polio eradication program is the largest public health initiative ever undertaken. In 1994, the Australian Government established the Australian National Poliovirus Reference Laboratory (NPRL) as part of Australia's commitment to the polio eradication program. Based within the Victorian Infectious Diseases Reference Laboratory (VIDRL), the NPRL is the WHO accredited facility for the isolation and characterisation of poliovirus from clinical specimens within Australia, the Pacific Islands and Brunei Darussalam. The NPRL is also designated as a Regional Reference Laboratory for the WHO Western Pacific Region.

In 1995, the Australian Federal Government initiated a surveillance program for the most serious clinical syndrome associated with poliovirus infection, acute flaccid paralysis (AFP). Since 2000, co-ordination of this surveillance program has been undertaken by the NPRL, in collaboration with the Australian Paediatric Surveillance Unit (APSU). All reported cases of AFP and suspected poliomyelitis are reviewed by the Australian Polio Expert Committee (PEC).

Polio vaccination in Australia is given at 2, 4 and 6 months and at 4 years of age, prior to school entry. From November 2005, the Australian immunisation program changed to exclusive use of inactivated poliovirus vaccine (IPV) in place of the live attenuated Sabin oral poliovirus vaccine (OPV).[1] Immunisation with OPV has been linked to vaccine associated paralytic poliomyelitis (VAPP), which is estimated to occur in one in 2.4 million doses. After administration of OPV, the recipient will shed live poliovirus intermittently for up to six weeks. In immunosuppressed persons who receive OPV, virus excretion can persist in excess of six weeks.[2,3] The exclusive use of IPV in the vaccination schedule eliminates the possibility of VAPP and the laboratory isolation of OPV polioviruses from recently vaccinated persons in Australia.[4] Any poliovirus isolated within Australia is now most likely indicative of importation and requires careful investigation.[5]

The performance of AFP surveillance in Australia and the laboratory activities of the NPRL in 2006 are described in this report.

Methods

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

- Clinicians reviewing patients presenting with AFP are advised to notify the NPRL. In keeping with WHO guidelines, the AFP surveillance program requires that all AFP cases involving children aged less than 15 years be reported. However, the NPRL tests specimens from cases of suspected poliomyelitis involving patients of all ages. Notification of AFP cases in children aged less than 15 years are also included on monthly report cards and emails submitted by paediatricians to the APSU.

- Two faecal specimens should be collected 24 to 48 hours apart and within 14 days of onset of paralysis.
- Faecal specimens are referred to the NPRL for testing.
- Reporting clinicians are supplied with a clinical questionnaire immediately upon notification of an AFP case.
- The PEC, convened by the Australian Government Department of Health and Ageing, reviews clinical and laboratory data for all notified cases of AFP, regardless of case eligibility.
  - The PEC case definition for AFP is: Any child under 15 years of age with acute flaccid paralysis (including Guillain-Barré syndrome) or any person of any age with paralytic illness if poliomyelitis is suspected.
  - In accordance with the WHO guidelines an ineligible case is a patient aged greater than 15 years, an overseas resident, or a case notified as AFP in error by a clinician.
- The PEC classifies cases of AFP as poliomyelitis due to wild poliovirus, vaccine-derived poliovirus (VDPV) or vaccine associated poliomyelitis; non-polio AFP; or non-AFP.
- A follow-up questionnaire is sent to notifying clinicians 60 days after the onset of paralysis in the patient if the PEC requires more information regarding the AFP case before a final classification can be made.
- Australian AFP data are forwarded to WHO for inclusion in the global AFP surveillance data published in the *Weekly Epidemiological Report,* (available from: http://www.who.int/wer/en/).
- At the end of each calendar year, a small number of eligible cases may remain un-classified by the PEC if no clinical or laboratory data were available from the notifying clinician.

Upon receipt at the NPRL, faecal specimens are extracted in a 7.7% v/v chloroform solution in Minimum Essential Medium containing 2% foetal bovine serum and inoculated onto a series of mammalian cell lines. In keeping with WHO requirements, cell lines used for the isolation of poliovirus are L20B (a transgenic mouse epithelial cell line expressing the human poliovirus receptor, CD155)[6,7] and RD-A (human rhabdomyosarcoma). These two cell lines are inoculated in duplicate to increase the sensitivity of virus isolation. The NPRL also utilises two additional cell lines for the isolation of poliovirus and non-polio enteroviruses (NPEVs): Hep2 Cincinnati (human epidermoid carcinoma) and HEL (human embryonic lung). Laboratories throughout Australia are encouraged to refer enteroviruses of unknown serotype to the NPRL for further characterisation. All polioviruses, whether isolated from AFP cases or other sources, undergo a process known as intratypic differentiation (ITD) to distinguish between wild and vaccine strains of poliovirus. ITD involves a nucleic acid detection method, [polymerase chain reaction (PCR)] and an antigenic method, [enzyme–linked immunosorbent assay (ELISA)]. These methods have been described in detail in previous annual reports.[8,9] In place of the ELISA, the NPRL is now sequencing portions of the poliovirus genome.

Two regions of the poliovirus genome are routinely sequenced from all poliovirus isolates. These regions are the VP1 capsid genomic region, where greater than 1% change compared to the prototype OPV strain is indicative of a vaccine-derived poliovirus as defined by WHO,[10] and the 3D genomic region, which is sequenced in order to determine whether the virus has undergone a recombination event with another poliovirus or enterovirus.

The NPRL is accredited as a Polio Regional Reference Laboratory, through proficiency testing and on-site inspections by WHO staff.

Results

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

In 2006, no AFP cases due to wild poliovirus, VDPV or VAPP were reported in Australia. A total of 48 eligible AFP cases were notified in Australia between 1 January and 31 December 2006 (Table 1).

Clinical and laboratory information was available for the PEC to review 43 of the 48 eligible AFP notifications. The WHO target for AFP surveillance in a polio non-endemic country is one case of AFP per 100,000 children aged less than 15 years. For Australia, this correlates to 40 cases per year (Table 1). Australia's non-polio AFP rate was 1.2, based on 48 eligible notifications. The non-polio AFP rate, based on the 43 eligible cases classified by the PEC, was 1.1 (Table 2).

The PEC was unable to provide final classification for five AFP notifications due to insufficient clinical information.

Notifications of acute flaccid paralysis by state or territory

New South Wales, Queensland and Victoria reached the expected WHO target of 1 case per 100,000 children aged less than 15 years for the reporting period (Table 1). This is the first time that Victoria has reached the WHO target since the initiation of AFP surveillance in Australia.

Faecal specimen collection from acute flaccid paralysis cases

WHO defines adequate specimens for laboratory testing, as two faecal specimens collected at least 24 hours apart and within 14 days of onset of paralysis. WHO recommends that specimens be tested in an accredited polio reference laboratory.

Faecal specimens were collected from 21 of the 43 eligible AFP cases with onset of symptoms in 2006 of which:

- Nine cases had two or more adequate specimens as defined by WHO.
- Seven cases had one specimen collected within 14 days of onset.
- Five cases had one or more specimens collected after 14 days of onset.
- No faecal specimens were referred to the NPRL from the remaining 22 eligible cases.

The proportion of eligible cases meeting the WHO criteria for adequate faecal specimen collection in the reporting period was 21% (9/43), well below the target of 80%.[11]

Laboratory testing of specimens

Acute flaccid paralysis cases

Forty-nine faecal specimens were received from 24 cases of AFP in Australian children less than 15 years of age. This included specimens from three AFP cases with onset of symptoms in late 2005, received by the laboratory in early 2006. An additional 10 specimens were referred from AFP patients aged greater than 15 years.

Table 1. Unique notifications of eligible acute flaccid paralysis cases by state or territory of residence with onset of symptoms between, 1 January to 31 December 2006

State or territory	Estimated population aged <15 years*	Expected number of cases/year	Unique notified eligible cases 1 January to 31 December 2006	Notification rate per 100,000 population aged <15 years
ACT	62,430	0.5	0	0.0
NSW	1,309,104	13	23	1.8
NT	50,674	0.5	0	0.0
Qld	816,566	8	11	1.4
SA	283,763	3	2	0.7
Tas	96,318	1	0	0.0
Vic	961,410	10	11	1.2
WA	404,349	4	1	0.3
Australia	3,984,614	40	48	1.2

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

Table 2. Acute flaccid paralysis surveillance compared with WHO indicator targets for children less than 15 years, Australia, 2006

WHO indicator target for AFP cases of children less than 15 years*	Australia's surveillance for AFP cases with onset in 2006	Australia's AFP surveillance rates for 2006
Non-polio AFP case rate of 1.0 per 100,000 children (40 cases for Australia in 2006).	48 unique cases of AFP notified 43 cases classified by the Polio Expert Committee as non-polio AFP	AFP notification rate: 1.2 per 100,000 children Non-polio AFP case rate: 1.1 per 100,000 children
More than 80% of notified AFP cases with 2 adequate stool specimens collected at least 24 hours apart, within 14 days of onset of paralysis.	9 AFP cases with 2 or more adequate specimens	Referral of adequate specimens from AFP cases: 21% (9/43) of the eligible cases

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

AFP Acute flaccid paralysis.

No polioviruses were isolated from the specimens of AFP cases in the reporting period. Non-polio enteroviruses were isolated from three cases of AFP: echovirus 30 was isolated from two cases and coxsackievirus B5 from one case. Adenovirus, which is not a member of the enterovirus family, was isolated from one case of AFP. No enterovirus was isolated from the faecal specimens of the remaining 17 eligible cases (Table 3).

A throat swab was received from an overseas resident aged greater than 15 years, who was admitted to hospital with AFP seven days after arriving in Australia. No enterovirus was isolated from the swab and the patient discharged themself from hospital without further follow-up.

Four rectal swabs and a faecal specimen from an overseas resident with AFP who was aged less than 15 years, were referred to the NPRL. Adenovirus was isolated from the faecal specimen.

No other virus isolations were reported from the specimens of the remaining AFP cases (Table 3).

Isolations from non- acute flaccid paralysis samples

In January 2006, five faecal specimens, a throat swab, a rectal swab, and cerebrospinal fluid were referred to the NPRL from an infant who had received routine immunisation of OPV in October 2005, followed by a booster of IPV in January 2006. Poliovirus type 3 (PV3) was isolated from one of the three faecal specimens initially forwarded to the NPRL. The PV3 was classified as Sabin-like using WHO approved methods for ITD. The VP1 genomic region was sequenced and had 99.4% nucleotide sequence identity to the prototype PV3 OPV strain. The isolation of a poliovirus, 107 days post-vaccination, is within the upper limits of 42–137 days for the excretion of poliovirus from a recently vaccinated patient.[2] No enteroviruses were isolated from a further two specimens that had been requested to confirm the clearance of the virus from the patient.

Although vaccine-associated paralytic polio (VAPP) was considered as a potential diagnosis by the PEC, the length of time between the administration of OPV and onset of symptoms (106 days) was outside the accepted range of 4–35 days for an OPV recipient. Acute and convalescent sera were also available for testing by the NPRL. There was evidence of immunity to all three poliovirus serotypes, with no detectable rise in titre observed between the acute and convalescent sera. The case, initially reported as post-trauma to a lumbar puncture, was subsequently diagnosed as osteomyelitis and classified as non-AFP by the PEC based on the available clinical information.

Two faecal specimens were received from a patient who was administered a low dosage of methotrexate and had received OPV. PV3 Sabin-like was isolated from one of the two initial specimens. A further three specimens were referred over a six week period to determine if there was prolonged virus excretion but no enterovirus was isolated from the specimens. A summary of enteroviruses tested at the NPRL between 1995 and 2006 is presented in Table 4.

Possible importation of wild poliovirus

On 19 October 2006, the importation of a wild poliovirus type 1 was reported in Kenya.[12] Virus genome sequencing and phylogenetic analysis traced the origin of this virus to Nigeria, an African country endemic for wild poliovirus. It was later determined that 12 people who arrived in Australia from Kenya between August and October, may have been in contact with the index case. Three people were tested as part of this investigation.

Table 3. Results from specimens referred to the Australian National Poliovirus Reference Laboratory, 2006

Result	Specimens from AFP cases*	Specimens from non-AFP referred samples	Total
Poliovirus Sabin-like type 3	0	2	2
NPEV†	4	2	6
Adenovirus	3	0	3
No virus isolated	52	16	68
Total	59	20	79

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

† NPEV: non-polio enterovirus. A coxsackievirus B5 (1 AFP case) and echovirus 30 (2 AFP cases) and coxsackievirus A17 (1 non-AFP case) were identified using either micro-neutralisation or molecular serotyping methods.

AFP Acute flaccid paralysis.

A non-polio enterovirus, coxsackievirus A17, was isolated from two faecal specimens from one of the people, while no enterovirus was isolated from the specimens collected from the others. Phylogenetic analysis of the VP1 nucleotide sequence with other coxsackievirus A17 sequences available through international databases did not identify a link with recent global isolations, thus providing no evidence as to whether the person was infected with the coxsackievirus before or after arrival in Australia.

Regional reference laboratory activities

In addition to the Australian samples, 155 specimens and isolates were received from countries of the Western Pacific Region. The referred samples included 30 specimens from 16 cases of AFP from the Pacific Islands with a non-polio enterovirus isolated from five of the cases. Ten specimens from five cases of AFP were referred from Brunei Darussalam and enterovirus 71, the cause of severe outbreaks of hand, foot and mouth disease in East and South Asia, was isolated from two cases of AFP. Fifty-nine specimens and isolates from Malaysia, and 32 specimens and isolates from the Philippines were also referred for ITD. A further 24 specimens and isolates from the National Polio Reference Laboratory of Papua New Guinea were tested in parallel as part of an ongoing laboratory quality assurance program.

Quality assurance program

As part of the accreditation procedure for a WHO polio reference laboratory, proficiency panels relating to the isolation, molecular detection and antigenic characterisation of poliovirus were received in February, June and November respectively. All proficiency panels were successfully completed. The annual laboratory accreditation site-visit to the NPRL was waived by WHO in 2006. The NPRL submitted documentation outlining the laboratory's activities to WHO Headquarters, Geneva and received notification that full accreditation status was retained.

Discussion

In 2006, Australia exceeded the WHO standard for AFP surveillance of one case of AFP per 100,000 children under the age of 15 years. Since the inception of the Australian AFP surveillance system in 1995, the WHO AFP surveillance standard has been achieved in 2000, 2001 and 2004. In 2006, adequate faecal sampling was obtained for only 21% of eligible AFP notifications, well below the 80% target established by WHO.

With the introduction of IPV into the standard immunisation schedule in Australia from November 2005, no further isolations of OPV strains of poliovirus are expected in Australian-born AFP cases without overseas travel. This was proven to be the case in 2006, with the last reported laboratory isolations of a poliovirus occurring after

Table 4. Summary of enterovirus testing at the Australian National Poliovirus Reference Laboratory, 1995 to 2006

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

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

† Two poliovirus isolates had discordant results by ITD. Sequencing confirmed the isolates as Sabin-like, with <1.0% variation from the parental Sabin strain.

two infants were vaccinated with OPV at the end of 2005. It is imperative that all poliovirus isolations after November 2005 be rigorously investigated, as they are potentially an importation from countries still using OPV or a wild poliovirus from one of the four endemic countries. While no polioviruses were reported to the Laboratory Virology and Serology Reporting Scheme in 2006, there were 101 untyped enteroviruses reported.[13] With pan-enterovirus PCR methods replacing routine cell culture in many diagnostic laboratories, the ability to determine enterovirus serotype is limited, thus increasing the risk of silent transmission of imported polioviruses and other enteroviruses of public health significance. As the characterisation of enteroviruses is both costly and time consuming, Australian virology laboratories are strongly encouraged to forward any untyped enteroviruses to the NPRL for further characterisation. Cases of imported VAPP and the isolation of VDPVs has been documented in countries that use IPV[14,15] and the monitoring of circulating enteroviruses in Australia is essential for the detection of such cases.

Globally, the number of poliomyelitis cases due to wild poliovirus infection in 2006 increased slightly to 1,998 in comparison to the 2005 case total of 1,979.[16] Although this may seem discouraging, the number of wild poliovirus cases reported by endemic countries in 2005 was 943 (47.7% of the total) and the number of imported cases was 1,036 (52.3%), which included a major outbreak in Indonesia.[17] In 2006, the number of endemic cases rose to 1,871 (93.6%), while the number of imported cases plummeted to 127 (6.8%).[16] This indicates that control measures instituted by WHO are proving successful in their capacity to contain poliovirus transmission within endemic countries and the focus now is to eradicate the last remaining pockets of circulating wild poliovirus.

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Annual report on surveillance of adverse events following immunisation in Australia, 2006

Glenda L Lawrence, Padmasiri E Aratchige, Ian Boyd, Peter B McIntyre, Michael S Gold

Abstract

This report summarises Australian passive surveillance data for adverse events following immunisation (AEFI) reported to the Adverse Drug Reactions Advisory Committee for 2006, and describes reporting trends over the seven-year period 2000 to 2006. There were 779 AEFI records for vaccines administered in 2006. This is an annual AEFI reporting rate of 3.8 per 100,000 population, the lowest since 2002 and a 10% decrease compared with 2005 (869 AEFI records; 4.3 records per 100,000 population). Dose-based AEFI reporting rates in 2006 were 1.9 per 100,000 doses of influenza vaccine for adults aged ≥18 years, 19.1 per 100,000 doses of pneumococcal polysaccharide vaccine for those aged ≥65 years and 12.5 per 100,000 doses of scheduled vaccines for children aged <7 years. Trend data showed transient increases in reporting of AEFI following the introduction of DTPa-IPV combination vaccines in November 2005 for children aged <7 years. The majority of the 779 AEFI records for 2006 described non-serious events while 11% (n=85) described AEFIs defined as serious. There was one report of death temporally associated with receipt of dTpa-IPV and typhoid vaccines in an adult with a history of a chronic medical condition. The most frequently reported individual AEFI was injection site reaction in children following a fourth or fifth dose of acellular pertussis-containing vaccine (70 reports per 100,000 doses). The data confirm the low rate of AEFI reported in Australia and demonstrate the ability of the system to detect and investigate signals such as those associated with changes in immunisation programs. *Commun Dis Intell* 2007;31:269–283.

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 Adverse Drug Reactions Advisory Committee (ADRAC) to 31 March 2007. The report focuses on AEFI reported for vaccines administered during 2006 and trends in AEFI reporting for the seven-year period 2000 to 2006.

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.[1–3] An 'adverse event following immunisation' is defined as any serious or unexpected adverse event that occurs after a vaccine has been given that may be related to the vaccine itself or to its handling or administration.[1] An AEFI can be *coincidentally* associated with the *timing* of immunisation without necessarily being caused by the vaccine or the immunisation process.

In Australia, AEFIs are notified to ADRAC (an expert committee of the Therapeutic Goods Administration) by state and territory health departments, health professionals, vaccine manufacturers and members of the public.[4] All reports received by ADRAC are evaluated using internationally consistent criteria[5] and are reviewed at regular meetings. Passive AEFI surveillance data have been collated in the ADRAC database since 2000 and are used to monitor trends, detect signals and generate hypotheses. Reports summarising national AEFI surveillance data have been published regularly since 2003.[6–13]

Several important changes to vaccine funding and availability occurred in 2005 and 2006 that impact on the AEFI surveillance data presented in this

report. Major changes to the funded Australian National Immunisation Program (NIP) Schedule[12] in November 2005 included:

(i) Inactivated poliovirus vaccine (IPV) replaced oral poliovirus vaccine 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 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.

(ii) 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.

In 2006, rotavirus (RotaTeq® and Rotarix®) and human papillomavirus (HPV) (Gardasil®) vaccines were registered by the Therapeutic Goods Administration and became available in the private market throughout Australia. In October 2006, the Northern Territory commenced a funded rotavirus immunisation program for infants. Both rotavirus and HPV vaccines were added to the funded NIP Schedule during 2007.[13]

Previous changes to the NIP Schedule in 2003 and 2005 also impact on the interpretation of trend data. On 1 January 2003, the meningococcal C conjugate immunisation program commenced when the vaccine was introduced into the routine schedule at 12 months of age with a catch-up program for all those born between 1984 and 2001.[13] Also in September 2003, the fourth dose of DTPa vaccine, given at 18 months of age, was removed from the immunisation schedule.[4] In January 2005, funded national pneumococcal immunisation programs commenced for infants at 2, 4 and 6 months of age, and for adults aged 65 years or over.[13]

Methods

Adverse events following immunisation data

De-identified information was released to the National Centre for Immunisation Research and Surveillance for all drug and vaccine adverse event notifications received by ADRAC between 1 January 2000 and 31 March 2007. Readers are referred to previous AEFI surveillance reports for a description of the AEFI surveillance system and methods used to evaluate AEFI reports received by ADRAC.[6,7]

ADRAC database records* 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 occurred between 1 January 2000 and 31 December 2006 *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 2006.

Study definitions of adverse events following immunisation outcomes and reactions

AEFIs were defined as 'serious' or 'non-serious' based on information recorded in the ADRAC database and criteria similar to those used by the World Health Organization[5] and the US Vaccine Adverse Events Reporting System (VAERS).[14] 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 or hospitalisation was prolonged; experienced a life-threatening event; or died.

Typically, each AEFI record listed several symptoms, signs and diagnoses that had been re-coded from the reporter's description into standardised terms using the Medical Dictionary for Regulatory Activities (MedDRA®).[15] 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 AEFIs listed and defined in *The Australian Immunisation Handbook* (8th edition).[4] 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 the SAS version 9 computer program.[16] The distribution of AEFI records was analysed by age, gender and jurisdiction. Average annual population-based reporting

* The term 'AEFI record' is used throughout this report because a single AEFI notification to ADRAC can generate more than one record in the database. For example, if a notification describes an injection site reaction plus symptoms and signs of a systemic adverse event (e.g. fever or generalised allergic reaction), two records will appear in the database: one record containing information relevant to the injection site reaction and one record for the systemic adverse event.

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 AEFIs 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'.

Dose-based AEFI reporting rates were estimated for influenza vaccine for adults aged ≥18 years, pneumococcal polysaccharide vaccine (23vPPV) for adults aged ≥65 years, and nine vaccines (i.e. DTPa-IPV, DTPa-IPV-HepB, DTPa-IPV-HepB-Hib, Hib, Hib-HepB, MMR, MenCCV, 7vPCV and varicella) funded through the NIP for children aged <7 years. The 2006 AEFI reporting rates per 100,000 doses of these vaccines were compared with those for 2005 and 2004 where denominator data were available.

Denominator data to estimate influenza and 23vPPV AEFI reporting rates were obtained from the 2006 draft national adult coverage survey report (unpublished) for adults aged ≥65 years and 18–64 years (influenza only). The number of administered doses of each of the nine childhood vaccines was calculated from the Australian Childhood Immunisation Register (ACIR), a national population-based register of approximately 99% of children aged <7 years.[17]

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

Notes on interpretation

Caution is required when interpreting the AEFI data presented in this report. Due to reporting delays and late onset of some AEFIs, the data are considered preliminary, particularly for the fourth quarter of 2006. Data published in previous reports for 2000–2005[6–11] differ to that presented in this report for the same period because the data have been updated to include AEFIs notified to ADRAC during 2006 for vaccines administered in previous years.

The information collated in the ADRAC database is intended primarily for signal detection and hypothesis generation. While reporting rates of AEFIs 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 AEFIs, and the variable quality and completeness of information provided in AEFI individual notifications.[6–12,18]

It is also important to note that this report is based on vaccine and reaction term information collated in a 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*.[4] These reaction categories are similar, but not identical, to the case definitions of adverse events.

The reported symptoms, signs and diagnoses in each AEFI record in the ADRAC database are temporally associated with vaccination but are not necessarily causally associated with a vaccine or vaccines. The causality ratings of 'certain', 'probable' and 'possible' 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. 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.

Results

Summary of data

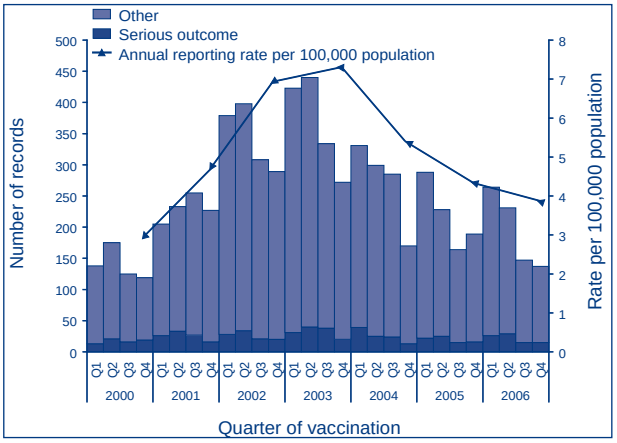
There were a total of 779 AEFI records in the ADRAC database where the date of vaccination (or onset of an adverse event, if vaccination date was not reported) occurred between 1 January and 31 December 2006. This is a decrease of 10% compared with 2005 when there were 869 AEFI records. In 2006, approximately 2% of AEFI notifications resulted in more than one AEFI record in the database (usually of an injection site reaction and a systemic reaction).

Eighty-five (11%) of the 779 AEFI records for 2006 were defined as 'serious' (i.e. recovery with sequelae, requiring hospitalisation, experiencing a life-threatening event, or death). A total of 345 (44%) AEFI records were assigned causality ratings of 'certain' (n=288, 37%) or 'probable' (n=57, 7%).

Adverse events following immunisation reporting trends

The AEFI reporting rate for 2006 was 3.8 per 100,000 population, down from 4.3 per 100,000 population in 2005 and the lowest since 2001 (Figure 1). The trends in AEFI notifications shown in Figure 1 are reflected in the trends in vaccines frequently suspected of involvement in reported AEFIs (Figure 2), and in the types of reactions frequently reported (Figure 3). Many of these changes correspond in time to changes in the funded NIP Schedule. Reports for meningococcal C conjugate vaccine (MenCCV) and pneumococcal conjugate vaccine (7vPCV) increased when the national routine and catch-up programs first commenced in January 2003 (MenCCV) and January 2005 (7vPCV), then stabilised over time. AEFI reports for DTPa-containing vaccines declined following the removal of the fourth dose from the immunisation schedule in the third quarter of 2003, and increased again following the introduction of the new DTPa-IPV containing multivalent vaccines in the fourth quarter of 2005.

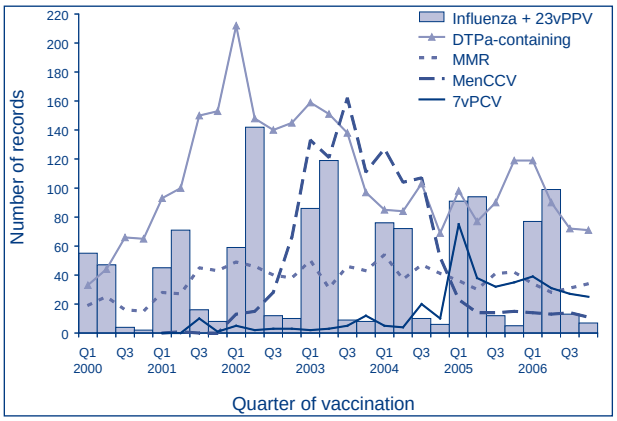
Figure 1. Adverse events following immunisation, ADRAC database, 2000 to 2006, by quarter of vaccination



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

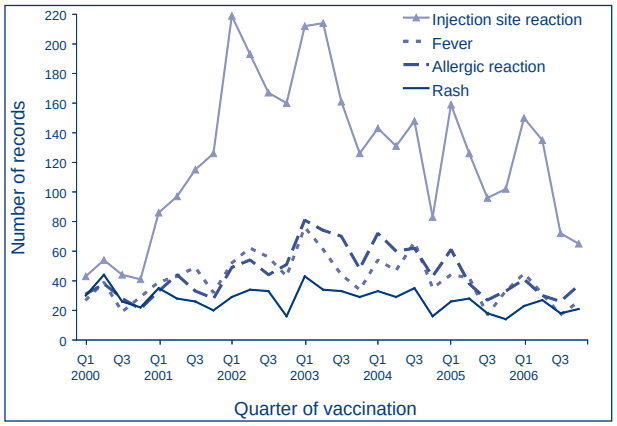
A seasonal pattern of AEFI reporting, seen in previous years, was apparent in 2006 with the highest number of AEFI notifications for vaccinations administered in the first half of the year (Figure 1). The seasonal peak corresponds to the months when more vaccinations are administered in Australia, particularly among 4 and 5-year-old children receiving measles-mumps-rubella (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 2. Frequently suspected vaccines, adverse events following immunisation, ADRAC database, 2000 to 2006, 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.

Figure 3. Selected frequently reported adverse events following immunisation, ADRAC database, 2000 to 2006, by quarter of vaccination

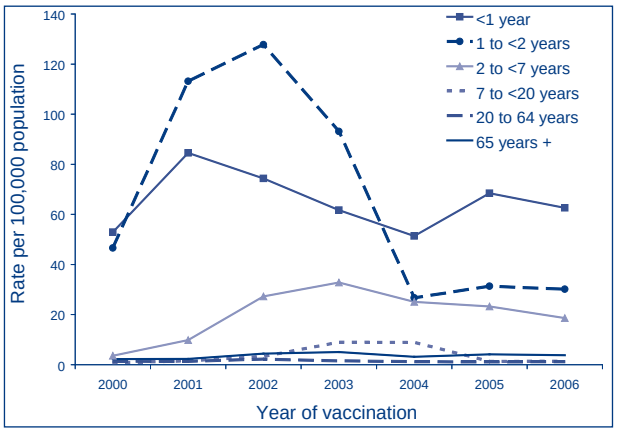


Age and gender distribution

In 2006, the highest AEFI reporting rate occurred in the <1 year age group, which received the highest number of vaccines (Figure 4). Compared with 2005, AEFI reporting rates declined in the <1 year (from 68.5 to 62.6 per 100,000 population), the 2 to <7 year (23.2 to 18.6 per 100,000) and ≥65 year (4.2 to 3.8 per 100,000) age groups and were stable for other age groups.

The overall male to female ratio was 1:1.2, similar to previous years. The gender ratio varied by age group with slightly lower AEFI reporting rates for females aged <1 year (male to female 1:0.8) and higher relative reporting rates for females aged ≥20 years (male to female 1:2.6).

Figure 4. Reporting rates of adverse events following immunisation per 100,000 population, ADRAC database, 2000 to 2006, by age group and year of vaccination



Geographical distribution

As noted in previous reports,[6,7,9,11] AEFI reporting rates varied between states and territories for vaccines received during 2006 (Table 1). The Northern Territory and the Australian Capital Territory had the highest reporting rates (18.2 and 16.6 per 100,000 population, respectively) while New South Wales and Queensland had the lowest rates (1.8 and 2.4 per 100,000 population, respectively). AEFI reporting rates either declined or were similar to those in 2005 in all jurisdictions except the Northern Territory where the reporting rate increased from 14.8 to 18.2 (30 to 37 records). The biggest decrease in reporting rates occurred in New South Wales, from 2.8 to 1.8 per 100,000 population in 2005 and 2006, respectively (188 to 125 records).

Adverse events following immunisation outcomes

Sixty-two per cent of reported AEFIs in 2006 were defined as 'non-serious' while 11% were defined as 'serious' (Table 2), similar to the percentages observed in the previous three years (59% and 9%, respectively). Fewer 'serious' AEFIs were assigned certain or probable causality ratings compared with 'non-serious' AEFIs (29% versus 50%) (Table 2). Vaccines listed in records where the outcome was defined as 'serious' are shown in Table 3.

One death was recorded as temporally associated with receipt of combined dTpa-IPV and typhoid vaccines in an adult with a chronic medical condition. The autopsy report indicated that the cause of death was not known.

Vaccines and adverse events following immunisation

Thirty-two vaccines were recorded as 'suspected' of involvement in the adverse events described in the 779 AEFI records for vaccines received in 2006 (Table 3). The percentage of records where only one 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'.

Table 1. Adverse events following immunisation (AEFI), ADRAC database, January to December 2006, by state or territory

State or territory	AEFI records		Annual reporting rate per 100,000 population*			
	n	%	Overall	'Certain' or 'probable' causality rating†	'Serious' outcome‡	Aged <7 years
Australian Capital Territory	54	7	16.6	3.7	0	112.8
New South Wales	125	16	1.8	0.9	0.15	8.5
Northern Territory	37	5	18.2	8.4	1.97	124.6
Queensland	94	12	2.4	1.1	0.23	18.1
South Australia	165	21	10.7	5.3	0.84	92.7
Tasmania	15	2	3.1	1.2	0.21	21.0
Victoria	187	24	3.7	1.6	0.46	26.4
Western Australia	61	8	3.0	1.4	0.45	27.1
Other§	41	5	na	na	na	na
Total	779	100	3.8	1.7	0.42	26.6

* Average annual rates per 100,000 population calculated using mid-2005 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 – Table 2).

§ Records where the jurisdiction in which the AEFI occurred was not reported or was unclear. Most (37/41) AEFI records in this category were notified by pharmaceutical companies while three were from the public and one from a nurse.

Vaccines containing diphtheria, tetanus and acellular pertussis antigens (including combination vaccines and dTpa) were suspected in 380 (49%) records (Table 3). DTPa-IPV was the most frequently suspected vaccine (278 records; 36%), followed by MMR (127 records;16%), 7vPCV (122 records; 16%) and 23vPPV (121 records; 16%). There were 10 reports of AEFI where rotavirus vaccine was suspected and two for HPV vaccine (Table 3).

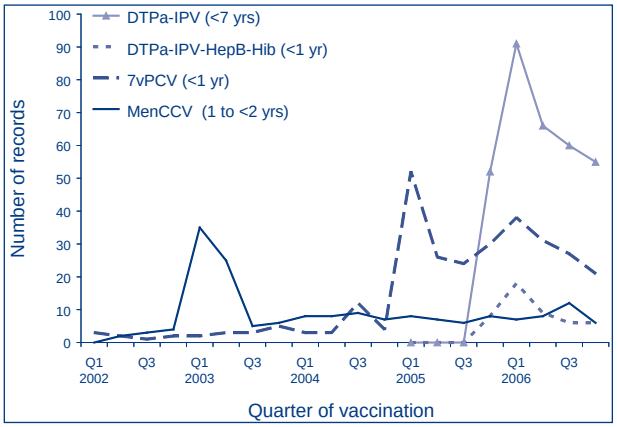
As described previously in this report, AEFI reporting trends differed by vaccine (Figure 2). Reports related to MMR vaccine remained relatively stable over time while AEFI reporting for vaccines recently introduced into the routine childhood schedule has stabilised over time, following peaks shortly after the programs commenced (Figure 5). This pattern has been evident following the introduction of the routine MenCCV dose at 12 months of age 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.

Adverse events following immunisation reactions

The distribution and frequency of reactions listed in AEFI records for 2006 are shown in Tables 4 and 5. In Table 4, only the reaction categories analogous to those listed in *The Australian Immunisation Handbook*[4] are shown. In Table 5, other reaction categories are listed in descending order of frequency.

The most frequently reported adverse events were injection site reaction (ISR; 54% of 779 AEFI records) followed by allergic reaction (17%), fever (15%) and rash (11%) (Table 4). Injection site reactions were the most commonly reported adverse event following receipt of 23vPPV (85%; 103/121), DTPa-containing vaccines (61%; 230/380), MMR (53%; 67/127) and influenza vaccine (38%; 39/103), administered alone or in combination with other vaccines.

Figure 5. Reports of adverse events following immunisation, ADRAC database, 2002 to 2006, for vaccines recently introduced into the funded National Immunisation Program*



* Meningococcal C conjugate vaccine (MenCCV) was introduced into the NIP on 1 January 2003, 7-valent pneumococcal conjugate vaccine (7vPCV) on 1 January 2005, and both DTPa-IPV and DTPa-IPV-HepB-Hib combination vaccines on 1 November 2005.

Table 2. Outcomes of adverse events following immunisation (AEFI), ADRAC database, 2006

Outcome	AEFI records		'Certain' or 'probable' causality rating†		Age group‡			
					< 7 years		≥ 7 years	
	n	%*	n	%§	n	%§	n	%§
Non-serious	484	62	240	50	310	64	171	35
Not recovered at time of report	158	20	60	38	72	46	85	54
Not known (missing data)	52	7	20	39	37	71	15	29
Serious:	85	11	25	29	56	66	28	33
recovered with sequelae	(1)		(1)		(0)		(1)	
hospital treatment – admission	(72)		(24)		(51)		(20)	
life-threatening event	(11)		(0)		(5)		(6)	
death	(1)		(0)		(0)		(1)	
Total	779	100	345	44	475	61	299	38

* Percentages relate to the total number of AEFI records (n=779).

† 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.

§ Percentages relate to the number of AEFI records with the specific outcome e.g. of 484 AEFI records with a 'non-serious' outcome, 50% had causality ratings of 'certain' or 'probable' and 64% were for children aged less than 7 years.

Table 3. Vaccine types listed as 'suspected' in records of adverse events following immunisation (AEFI), ADRAC database, 2006

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	%¶
DTPa-IPV**	278	141	51	133	48	28	10	272	98	0	–
MMR	127	22	17	19	15	11	9	122	96	5	4
7vPCV	122	3	2	8	7	22	18	122	100	0	–
23vPPV	121	87	72	74	61	6	5	6	5	114	94
Influenza	103	72	70	27	26	13	13	8	8	93	90
Hib-hepatitis B	97	7	7	11	11	20	21	96	99	1	1
MenCCV	52	5	10	6	12	7	13	50	96	2	4
DTPa-IPV-hepB-hib**	46	13	28	12	26	6	13	46	100	0	–
Hepatitis B	38	28	74	12	32	4	11	4	11	34	89
Varicella**	36	25	69	11	31	1	3	27	75	9	25
dTpa	25	17	68	13	52	0	–	1	4	24	96
Hib	23	1	4	3	13	4	17	23	100	0	–
DTPa	20	5	25	4	20	2	10	20	100	0	–
dT	17	12	71	7	41	3	18	0	–	17	100
Hepatitis A	12	2	17	1	8	1	8	6	50	6	50
Hepatitis A + B	11	7	64	6	55	4	36	1	9	10	91
Rotavirus††	10	8	80	3	30	2	20	10	100	0	–
BCG	8	0	–	7	88	3	38	8	100	0	–
DTPa-IPV-hepB**	7	0	–	1	14	1	14	7	100	0	–
Japanese encephalitis	7	6	86	2	29	0	–	0	–	7	100
IPV	7	1	14	0	–	1	14	3	43	4	57
Typhoid	7	1	14	1	14	0	–	0	–	7	100
Q fever	5	5	100	2	40	2	40	0	–	5	100
Men4PV	4	0	–	0	–	1	25	0	–	4	100
Rabies	4	1	25	0	–	0	–	0	–	4	100
dTpa-IPV	3	1	33	0	–	1	33	0	–	3	100
Cholera	3	0	–	2	67	0	–	0	–	3	100
Yellow fever	3	2	67	0	–	1	33	0	–	3	100
Hepatitis A-typhoid	2	0	–	0	–	0	–	0	–	2	100
HPV vaccine††	2	2	100	0	–	1	50	0	–	2	100
DTPa-hepatitis B	1	0	–	0	–	0	–	1	100	0	–
Tetanus	1	1	100	1	100	0	–	0	–	1	100
Total‡‡	779	486	62	345	44	85	11	475	61	299	38

* See appendix for abbreviations of vaccine names.

† AEFI records where only one 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 Table 2 also).

‖ 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. DTPa-IPV was 'suspected' in 278 AEFI records; this was the only suspected vaccine in 51% of the 278 AEFI records, 48% had 'certain' or 'probable' causality ratings, 10% were defined as 'serious' and 98% were for children <7 years.

** Varicella vaccine and combination vaccines containing inactivated poliovirus were added to the National Immunisation Program Schedule on 1 November 2005.[13]

†† Rotavirus vaccine and human papillomavirus vaccines were registered for use in Australia by the Therapeutic Goods Administration in 2006.

‡‡ 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 one vaccine.

Table 4. Reaction categories of interest* mentioned in records of adverse events following immunisation (AEFI), ADRAC database, 2006

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	422	260	61	281	67	266	63	152	36
Fever	119	1	1	39	33	76	64	43	36
Allergic reaction¶	117	22	19	30	26	72	62	45	38
severe allergic reaction¶	17	0	–	4	24	4	24	12	71
Rash	89	23	26	19	21	67	75	22	25
Abnormal crying	24	1	42	6	25	24	100	0	–
HHE**	20	7	35	2	10	20	100	0	–
Arthralgia	19	0	–	5	26	0	–	19	100
Convulsion	16	8	50	3	19	12	75	4	25
Lymphadenopathy/itis††	7	0	–	2	29	2	29	5	71
Abscess	6	2	33	5	83	5	83	1	17
Anaphylactic reaction	3	0	–	2	67	0	–	3	100
Arthritis	3	2	67	1	33	0	–	3	100
Guillain-Barré syndrome	2	0	–	0	–	0	–	2	100
Thrombocytopenia	2	2	100	0	–	1	50	1	50
Brachial neuritis	1	1	100	0	–	0	–	1	100
Death	1	0	–	0	–	0	–	1	100
Encephalitis	1	0	–	0	–	0	–	1	100
Encephalopathy	1	0	–	0	–	0	–	1	100
Orchitis	1	0	–	0	–	0	–	1	100
Parotitis	1	1	100	0	–	1	100	0	–
Acute flaccid paralysis	0	0	–	0	–	0	–	0	–
Meningitis	0	0	–	0	–	0	–	0	–
Osteomyelitis	0	0	–	0	–	0	–	0	–
Osteitis	0	0	–	0	–	0	–	0	–
Sepsis	0	0	–	0	–	0	–	0	–
SSPE‡‡	0	0	–	0	–	0	–	0	–
Toxic shock syndrome	0	0	–	0	–	0	–	0	–
Total§§	779	366	47	345	44	475	61	299	38

* Reaction categories were created for the AEFIs of interest listed and defined in *The Australian Immunisation Handbook*, (8th edition, p 22–23 and 271–275)[4] as described in the Methods section.

† AEFI records where only one 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 422 AEFI records listing injection site reaction, 61% listed only one type of reaction while 67% had a causality rating of 'certain' or 'probable' and 63% were for children aged less than 7 years.

¶ Allergic reaction includes skin and/or gastrointestinal (e.g. diarrhoea, vomiting) symptoms and signs.[4] The category 'severe allergic reaction' includes allergic reaction with involvement of the circulatory and/or respiratory system but not recorded in the ADRAC database as 'anaphylactic reaction'.[4]

** Hypotonic-hyporesponsive episode.

†† Includes lymphadenitis following BCG vaccination and the more general term of 'lymphadenopathy'.

‡‡ 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 one reaction term.

Table 5. 'Other'* reaction terms listed in records of adverse events following immunisation (AEFI), ADRAC database, 2006

Reaction term*	AEFI records	Only reaction reported†		Certain/probable causality rating‡		Age group§			
						< 7 years		≥ 7 years	
	n	n	%¶	n	%¶	n	%¶	n	%¶
Malaise	43	0	–	17	40	10	23	33	77
Oedema	39	3	8	18	46	28	72	11	28
Headache	39	0	–	12	31	3	8	36	92
Respiratory rate/rhythm change	35	1	3	6	17	22	63	11	31
Pallor	34	2	6	8	24	28	82	6	18
Irritability	29	0	–	6	21	29	100	0	–
Nausea	29	0	–	2	7	2	7	27	93
Anorexia	27	0	–	6	22	22	81	5	19
Heart rate/rhythm change	25	0	–	4	16	17	68	8	32
Myalgia	24	0	–	8	33	2	8	22	92
Increased sweating	23	0	–	8	35	3	13	19	83
Dizziness	22	0	–	5	23	0	–	22	100
Reduced sensation	18	1	6	8	44	0	–	18	100
Syncope	18	3	17	5	28	3	17	15	83
Pain	16	0	–	4	25	3	19	13	81
Erythema	14	2	14	0	–	10	71	4	29
Cough	9	0	–	2	22	5	56	4	44
Other	144	19	13	36	25	63	44	81	56
neurological	30	7	23	1	3	9	30	21	70
general non-specific	24	2	8	12	50	11	46	13	54
cardiovascular	22	1	5	7	32	5	23	15	68
respiratory	19	5	26	5	26	5	26	14	74
eye or ear	16	0	–	1	6	8	50	8	50
psychological	16	0	–	4	25	9	56	7	44
musculoskeletal	14	2	14	3	21	1	7	13	93
skin	11	2	18	4	36	6	55	5	45
gastrointestinal	8	0	–	1	13	5	63	3	37
infection	8	1	13	3	38	4	50	4	50
haematological	2	0	–	0	–	1	50	1	50
renal/urogenital	2	0	–	0	–	0	–	2	100
pregnancy/congenital	2	2	100	0	–	0	–	2	100
metabolic/endocrine	1	0	–	1	100	0	–	1	100

* Reaction terms not listed in the *Australian Immunisation Handbook*[4] 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.

Note: Please see Table 4 for the description of other footnotes.

More severe AEFIs included reports of anaphylactic reaction (n=3), severe allergic reaction involving the respiratory and/or circulatory system (n=17), hypotonic-hyporesponsive episode (HHE, n=20), thrombocytopenia (n=2), encephalitis (n=1) convulsion (n=16), Guillain-Barré syndrome (GBS; n=2) and death (n=1; described previously in this report). The two records coded as GBS were for a 63-year-old following receipt of influenza vaccine and 13-year-old following hepatitis B vaccine, although the diagnosis of GBS was apparently not confirmed for the latter report.

Two of the three reports of anaphylaxis occurred in adults following receipt of influenza vaccine and one occurred in a 13-year-old after receiving both HepB and Men4PV vaccines. Of the 16 reports of convulsion, 12 were in children aged <7 years following

routinely scheduled combinations of vaccines. The most commonly suspected vaccines were 7vPCV (n=5) and MMR (n=4). The majority (16/20) of HHE were reported by Victoria and South Australia, and the most commonly suspected vaccines were the ones used in these states including DTPa-IPV (n=14), 7vPCV (n=14) and Hib-HepB (n=13). DTPa-containing vaccines were listed for 18 of 20 reports of HHE.

Reactions shown in Table 5 include changes in respiratory rate/rhythm (n=35) and heart rate/rhythm (n=25). These include 16 reports of bradycardia combined with apnoea or respiratory depression in infants receiving vaccines due at two months of age. Eleven of these reports were for pre-term or very pre-term infants who had received their immunisations in a hospital setting at a chronological age of >8 weeks. An increase in reports of this type was observed following the introduction of the DTP-IPV containing vaccines in November 2005.[13] Before this time, an average of 2–3 reports of bradycardia combined with apnoea or respiratory depression in infants were received each year. The number of reports increased to a peak of six in the fourth quarter of 2005, then declined to four per quarter for the first half of 2006 and only one and two reports in each of the third and fourth quarters, respectively.

Reactions mentioned in fewer than 1% of AEFI records in 2006 are shown in the lower portion of Table 5, grouped by organ system categories. The most commonly reported categories were coded as 'neurological' and 'general non-specific' reactions, which included reaction terms such as 'feeling hot', 'feeling cold' and 'discomfort'.

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. 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 and 23vPCV.[6–12] The percentage of reports for 23vPPV that list ISR has increased over time, particularly for adults aged ≥65 years.[11] This has increased from 50% of 26 reports in 2001 to 88% of 82 reports in 2006.

Dose-based reporting rates of adverse events following immunisation

Influenza vaccine and adults aged ≥ 18 years

In 2006, influenza vaccine was suspected of involvement in 89 AEFI records for people aged ≥18 years. The dose-based AEFI reporting rates, by age group, are shown in Table 6. The AEFI reporting rate was 1.9 per 100,000 doses, similar to the rate in 2004 and 2005, while the reporting rate for serious AEFI declined. Both the overall and serious AEFI reporting rates were higher for vaccinees aged 18–64 years than among older vaccinees.

The most frequently reported adverse events were ISR, fever, headache and allergic reaction (0.8, 0.4, 0.4 and 0.3 per 100,000 doses, respectively). Rates of each of these reactions were higher in the 18–64 year age group. There was one report of GBS (in a 63-year-old) following influenza vaccination in 2006, the same as in previous years.[9]

Table 6. Reporting rate of adverse events following immunisation (AEFI) per 100,000 doses of influenza vaccine,* 18 years and over, ADRAC database, 2006

AEFI category†	Age group	AEFI records‡	Vaccine doses*	Rate per 100,000 doses§		
		n	n	2006	2005	2004
Overall	≥ 18 years	89	4,746,900	1.9	2.1	1.8
	18 to 64 years	65	2,626,400	2.5	2.8	2.4
	≥ 65 years	24	2,120,500	1.1	1.2	1.1
Serious	≥ 18 years	9	4,746,900	0.19	0.37	0.36
	18 to 64 years	7	2,626,400	0.27	0.49	0.46
	≥ 65 years	2	2,120,500	0.09	0.27	0.24

* 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 2006.

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

Pneumococcal vaccine and adults aged ≥ 65 years

It was estimated that approximately 429,500 doses of 23vPPV were administered to people aged >65 years in 2006 (unpublished). There were 82 reports of AEFI for this age group where 23vPPV was listed as suspected of involvement in the reported adverse event, with four reports coded as serious and 72 as ISR. The dose-based reporting rates were 19.1 AEFI reports per 100,000 doses, with 0.93 serious and 16.8 ISR reports per 100,000 doses of 23vPPV.

Scheduled vaccines for children aged <7 years

A total of 475 AEFI records for vaccines administered in 2006 were for children aged <7 years. Of these, 442 records listed one of the nine vaccines for which ACIR data could be used to estimate AEFI reporting rates per 100,000 vaccine doses, as the suspected vaccine (Table 7). Vaccines for which reliable denominator data were not available included rotavirus (n=10), BCG (n=8), influenza (n=8), 23vPPV (n=6), hepatitis A (n=6), and hepatitis B (n=4) (Table 3).

The AEFI reporting rates per 100,000 vaccine doses recorded on the ACIR were similar to, or lower than, those in 2005 for most vaccine types, including MenCCV, 7vPCV, MMR and DTPa-containing vaccines (Table 7). The apparent increase in the reporting rate for Hib-HepB and Hib vaccines may be related to reporting of AEFIs for the newer quadrivalent and pentavalent DTP-IPV combination vaccines among children aged <1 year, as the two vaccines are both given at 2 and 4 months of age.[13]

Reporting rates for the different DTPa-IPV combination vaccines varied by vaccine type. 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

Table 7. Reporting rates of adverse events following immunisation (AEFI) per 100,000 vaccine doses,* children aged less than 7 years, ADRAC database, 2006

	AEFI records‡	Vaccine doses*	Reporting rate per 100,000 doses§		
	n	n	2006	2005	2004
Vaccine†					
DTPa–containing vaccines	325	1,201,873	27.0	34.8	32.9
DTPa-IPV	272	827,510	32.9	–	–
Pentavalent (DTPa-IPV-HepB)	7	17,938	39.0	–	–
Hexavalent (DTPa-IPV-HepB-Hib)	46	356,425	12.9	–	–
Haemophilus influenzae type b	23	100,361	22.9	17.8	20.4
Haemophilus influenzae type b-hepatitis B	96	408,687	23.5	18.2	9.1
Measles-mumps-rubella	122	512,018	23.8	27.8	33.6
Meningococcal C conjugate	50	277,358	18.0	17.4	30.8
Pneumococcal conjugate	122	789,610	15.5	15.1	–
Varicella	27	233,912	11.5	–	–
Age group					
<1 year	144	1,850,721	7.8	6.6	5.5
1 to <2 years	72	1,089,218	6.6	7.2	6.9
2 to <7 years	226	583,972	38.7	31.7	33.6
AEFI category†					
Total	442	3,523,914	12.5	11.3	13.0
'Certain' or 'probable' causality rating	179	3,523,914	5.1	6.9	5.3
'Serious' outcome	45	3,523,914	1.28	0.71	0.97

* Number of vaccine doses recorded on the Australian Childhood Immunisation Register (ACIR) and administered between 1 January and 31 December 2006.

† 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 2006. More than one 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.

administered. The reporting rate for quadrivalent DTPa-IPV includes reports for children aged <1 year who were scheduled to receive the vaccine at 2, 4 and 6 months of age (reporting rate of 19.6 per 100,000 doses) and the 2 to <7 year age group (reporting rate of 78 per 100,000 doses). The reporting rate of ISR following DTPa-IPV in this older age group was 70 per 100,000 doses compared with 76–80 per 100,000 doses of DTPa vaccine over the four years 2002–2005.

Although the number of AEFI reports for children aged <1 year and 2 to <7 years was lower in 2006 than in 2005, AEFI reporting rates per 100,000 vaccine doses increased for children in these two age groups (Table 7). The reporting rate for AEFIs defined as serious also increased from 0.7 in 2005 to 1.3 in 2006. Reasons for these changes are discussed below and relate to a number of factors including a reduction in the denominator following the introduction of multivalent vaccines in November 2005.

Discussion

The data show a decrease in the number of AEFI reports received for 2006, the lowest since 2002. The reduction in AEFI reporting occurred mainly in the age groups that receive the most vaccines – the <1 year, 2 to <7 year and the ≥65 year age groups. The percentage of reports of serious AEFI increased slightly compared with previous years, from 9% to 11%, particularly among children aged <1 year. This appears to have been related to increased vigilance in reporting following the introduction of DTPa-IPV combination vaccines in November 2005, with a peak in the first quarter of 2006, and a reduction back to baseline later in the year.

An important contributor to the increase in serious AEFI reports in late 2005 and early 2006 was reports of bradycardia and respiratory depression among pre-term and very pre-term infants who received vaccines in hospital settings at around 8 weeks of age. Cardio-respiratory events are known and manageable AEFIs among hospitalised pre-term infants.[19–21] The total number of reports of serious AEFI is low and the increase in reports may be related to the usual increase in reporting following the introduction of new vaccines in Australia (Figure 5) and the United States of America (USA).[22,23] It may also be related to increased awareness among providers following published reports in Germany that suggested an increased risk of sudden unexpected death in children aged <2 years following receipt of a hexavalent vaccine marketed in Germany.[24,25] It is important to note that a large case-control epidemiological study found no link between the use of hexavalent vaccines and sudden unexpected death;[26] that the Global Advisory Committee on Vaccine Safety (convened by the World Health Organization) concluded that hexavalent vaccines are safe;[27] and that the German vaccine is not used in Australia.

The majority of AEFI reported to ADRAC in 2006 were mild transient and expected vaccine side-effects. Injection site reactions remain the most commonly reported AEFI. Two groups are of interest in this regard – children receiving a school entry booster dose of an acellular pertussis-containing vaccine[28,29] and adults receiving booster doses of 23vPPV.[30,31]

The 2006 AEFI data include the first cohorts of children (born after 1 April 2002) who received their fourth dose of acellular pertussis-containing vaccines at 4–5 years of age following the removal from the schedule, in September 2003, of the dose due at 18 months.[4] The rate of ISR following acellular pertussis-containing vaccines in the 2 to <7 year age group has declined slightly in 2006 to 70 per 100,000 doses, down from the consistent reporting rate of 76–80 per 100,000 doses for 2002–2005.[11] As more children receive their fourth dose at 4–5 years of age, it is expected that the reporting rate of AEFI will decline further.

The second group of interest regarding ISR are older adults who receive 23vPPV. Both the total number of reports and the proportion of reports of ISR following 23vPPV in adults aged ≥65 years has continued to increase since 2001. Although dose number is poorly recorded, approximately two-thirds of those where dose information was available indicated that the dose was not a first dose. Increased reporting of ISR following second and third doses of 23vPPV has been suggested previously,[30] however, a recent USA study found little difference in the rate of ISR for first versus subsequent doses.[31] Importantly, ISR does not represent a contraindication to revaccination for age groups that are recommended 23vPPV.[4,29,30]

Available unpublished data on the number of doses of 23vPPV administered in Australia to the ≥65 year age group allowed the first dose-based AEFI reporting rate to be calculated. The availability of 23vPPV coverage data from future regular adult vaccination surveys will allow monitoring of dose-based AEFI reporting rates over time.

The largest population group where dose-based AEFI reporting rates have not been included in this report is adolescents receiving funded vaccines through school-based programs. These programs have expanded considerably in recent years and include routine immunisation with HepB, dTpa and varicella vaccines and, from April 2007, HPV vaccine.[13] At this stage, coverage data are not routinely collated at a national level to allow routine

estimation of dose-based AEFI reporting rates for these vaccines among adolescents. It is anticipated that these data will become available in time.

Conclusion

The benefits of immunisation in reducing morbidity and mortality due to vaccine preventable diseases outweighs the risks of immunisation-related adverse events in Australia. Notification data show the impact of immunisation on reducing the number of cases of many severe infections,[32,33] including significant impacts on the incidence of both invasive meningococcal disease[34] and invasive pneumococcal disease[35] following the introduction of these national immunisation programs in 2003 and 2005.

During 2006, an estimated 9–10 million vaccine doses were administered in Australia and a total of 779 reports of AEFI were received by ADRAC. While under-reporting is a known disadvantage of passive surveillance systems,[1–3,18] the Australian national AEFI passive surveillance system is sufficiently sensitive to detect expected changes in AEFI reporting associated with changes in immunisation programs, and signals of rarer adverse events like the transient increase in reporting of bradycardia and respiratory depression among pre-term infants that occurred in late 2005 and early 2006. Processes are in place to investigate signals and monitor trends in AEFI reporting. 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 six months of 2007.

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Appendix

Abbreviations of vaccine types

7vPCV	7-valent pneumococcal conjugate vaccine
23vPPV	23-valent pneumococcal polysaccharide vaccine
BCG	Bacille Calmette-Guérin (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 (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

Articles

How reliable are Australian Childhood Immunisation Register coverage estimates for Indigenous children? An assessment of data quality and coverage

Claudia Rank, Robert I Menzies

Abstract

Low levels of reporting indigenous status to the Australian Childhood Immunisation Register (ACIR) in the past have resulted in reduced confidence in vaccination coverage data for Aboriginal and Torres Straight Islander children. This study shows that the reporting of indigenous status has improved from 42% of the estimated national cohort of Indigenous children aged 12 to 14 months in 2002 to 95% in 2005. Over that period diphtheria-tetanus-pertussis (DTP) vaccination coverage estimates for Indigenous children increased slightly from 86.0% to 86.9%. Data by state and territory or remoteness are also presented. ACIR vaccination coverage estimates for Indigenous children can now be used with confidence for program planning at the national and jurisdictional level. *Commun Dis Intell* 2007;31:283–287.

Keywords: vaccination coverage, Oceanic ancestry group, immunisation

Introduction

Accurate estimates of vaccination coverage are critical to determining the reasons for higher rates of some vaccine preventable diseases in Aboriginal and Torres Strait Islanders compared to non-Indigenous people.[1,2] The Australian Childhood Immunisation Register (ACIR) has been used to a limited extent to compare vaccination coverage between Indigenous and non-Indigenous children, but concerns remain about low reporting rates of indigenous status and the resultant unreliability of coverage estimates for Indigenous children.[2] One potential source of bias is more complete reporting of indigenous status from remote areas, where vaccination coverage has consistently been higher in the past.[3–7]

Several initiatives during 2003 and 2004 were expected to have resulted in an improvement in recording indigenous status; a promotion by the Health Insurance Commission in 2003 to encourage Aboriginal and Torres Strait Islander individuals to report their indigenous status, the commencement of regular transfer of demographic data from Medicare to ACIR records in 2003, and the commencement of transfer of data on indigenous status from immunisation registers in the Northern Territory in 2003 and Queensland in 2004.

The aims of this study therefore, were to conduct an analysis of ACIR data to determine whether:

- the reporting of indigenous status on the ACIR has improved since 1999;
- vaccination coverage estimates for Indigenous children have changed in association with changes in reporting of indigenous status; or
- there is substantial variation by jurisdiction or remoteness, in either reporting of indigenous status or coverage in Indigenous children.

Methods

Vaccination coverage

Data from the ACIR were obtained from the Health Insurance Commission. Birth cohorts corresponding to children aged 12–14 months in four consecutive years were studied utilising ACIR data as at 31 December of each year. The years chosen for this analysis and respective dates of birth in each cohort were 2002 (date of birth 1/7/2001 – 30/9/2001), 2003 (date of birth 1/7/2002 – 30/9/2002), 2004 (date of birth 1/7/2003 – 30/9/2003) and 2005 (date of birth 1/7/2004 – 30/9/2004). Vaccination coverage for each birth cohort was assessed by including only immunisations given on or before a child's first birthday. The third dose assumption was applied in the calculation of immunisation status for diphtheria-tetanus-pertussis (DTP) vaccine.[8] Therefore, children were considered fully immunised for DTP if a third dose of DTP vaccine was recorded on the ACIR by 12 months of age, irrespective of whether previous doses in the series had been recorded.

Remoteness

The Australian Standard Geographical Classification (ASGC) was used to analyse remoteness. This system was developed by the Australian Bureau of Statistics (ABS) and groups all areas in Australia into five classifications defined by their physical remoteness from goods and services.[9] Remoteness categories for ACIR data were derived from postcode, using 2001 Census based postal area/ASGC remoteness concordances.[10] Where a given postcode corresponded to more than one ASGC classification, the remoteness classification in which 50% or more of the postcode population resides was used.

Indigenous identification and population estimates

The completeness of reporting of indigenous status on the ACIR was assessed by comparing the number of children identified as Aboriginal or Torres Strait Islander on the ACIR with the low series Experimental Indigenous Population Projections at 30 June for the corresponding year, by age, derived from 2001 ABS Census data. Estimates by ASGC remoteness classification were available for 2001 only. Children for whom indigenous status was recorded as unknown or missing on the ACIR were analysed as non-Indigenous. Due to low Indigenous population estimates in the Australian Capital Territory relative to other states and territories, completeness and coverage data for the Australian Capital Territory and New South Wales were combined.

Statistical analysis

Vaccination coverage and completeness were calculated using SASv9.1.3.[11] The Kendall's Tau rank correlation coefficient[12] was used to test for correlation between annual estimates of vaccination status and completeness, by jurisdiction and remoteness category, using StatXact 4 for Windows.[13]

Results

The number of Indigenous and non-Indigenous children recorded on the ACIR in the four birth cohorts studied ranged from 1,269–2,970 and 62,544–62,739 respectively.

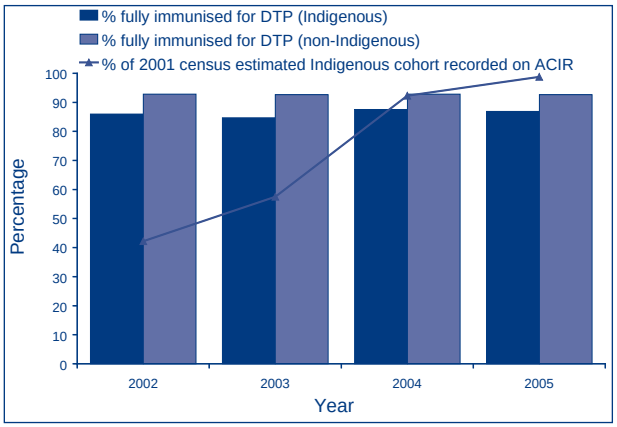
Completeness of Indigenous identification

The percentage of the ABS estimated Indigenous population of infants identified as Indigenous by the ACIR (indigenous identification completeness) steadily increased at the national level, from 42% of the ABS estimated Indigenous cohort in 2002 to 95% by 2005 (Table 1). Indigenous identification completeness increased substantially between 2002 and 2005 in all states and territories except South Australia and Western Australia, where it remained stable. By 2005, indigenous identification was over 90% complete in all jurisdictions except South Australia. In Victoria, Tasmania, Queensland and New South Wales/Australian Capital Territory completeness rose most notably between 2003 and 2004. The greatest increase was observed for Queensland, from 2% in 2002 to above 107% in 2005, due to the commencement of the electronic transfer of the indigenous status field from the state register (VIVAS) to the ACIR.

Vaccination coverage in Indigenous and non-Indigenous children

A comparison of DTP coverage at 12–14 months of age for Indigenous and non-Indigenous children in Australia is shown in the Figure. While immunisation coverage in non-Indigenous children has consistently remained at 93% in all years studied, coverage estimates for Indigenous children ranged from 85% to 88%.

Australian Childhood Immunisation Register data completeness of indigenous status reporting, and DTP coverage, for Indigenous and non-Indigenous children aged 12–14 months, Australia, 2002 to 2005



Data are analysed as at 31 December of each year.

Indigenous vaccination coverage estimates by state or territory

The percentage of Indigenous children fully immunised with DTP vaccine at 12–14 months of age varied between jurisdictions (Table 2). Gradual increases over time were evident in the Northern Territory and Tasmania. In South Australia, coverage estimates decreased steadily since 2002, falling to the lowest figure (76%) observed in all years by 2005. Tasmania exhibited the highest Indigenous DTP coverage from 2003 to 2005, and by 2005 all jurisdictions but Western Australia and South Australia had achieved coverage of 87% or higher.

Table 1. Increases in reporting of indigenous status to the ACIR,* for children aged 12–14 months,† 2002 to 2005, by state or territory

State or territory	% of ABS estimated Indigenous cohort recorded as Indigenous on ACIR		Correlation with coverage‡	
	2005	Annual increase 2002–2005	Correlation coefficient	P value
NSW/ACT	90	8.3	0.33	0.75
NT	98	29.1	0.33	0.75
Qld	108	35.1	–0.33	0.75
SA	72	–1.6	0.33	0.75
Tas	89	20.5	0.33	0.75
Vic	111	18.0	–0.33	0.75
WA	85	0.6	–1.0	0.08
All combined	95	17.7	0.04	0.80

* Australian Childhood Immunisation Register.

† July to September birth cohorts.

‡ Kendall's Tau coefficients of correlation between completeness of indigenous status and indigenous coverage, and P value for testing hypothesis that there was no correlation. Perfect positive correlation is indicated by an estimate of 1, no correlation by 0.

Table 2. DTP coverage for Indigenous children aged 12–14months, 2002 to 2005, by state or territory

State or territory	2002	2003	2004	2005
NSW/ACT*	88.1	87.4	87.8	88.7
Northern Territory	79.0	82.9	85.4	87.6
Queensland	90.0	79.8	88.3	88.0
South Australia	89.4	84.3	81.1	76.0
Tasmania	86.7	92.3	96.1	93.2
Victoria	90.9	89.2	91.8	88.4
Western Australia	80.2	80.6	84.9	80.0
Australia	86.0	84.7	87.6	86.9

Data are analysed as at 31 December of each year.

* New South Wales and the Australian Capital Territory combined.

Completeness of indigenous identification by remoteness

The number of Indigenous infants in the birth cohorts studied ranged from 459–935 in major cities, 277–673 in inner regional, 279–742 in outer regional, 127–243 in remote and 124–392 in very remote areas. In comparison, there were on average 43,010, 12,429, 5,842, 929 and 391 non-Indigenous children recorded on the ACIR in these areas respectively.

Indigenous identification completeness improved substantially in all remoteness categories from 2002 to 2005 (Table 3). From a low of 25% for very remote areas in 2002, by 2005 completeness was over 80% in all areas, and around 100% in major cities, inner regional and outer regional areas. Indigenous identification completeness was lowest in very remote areas in all years except 2003.

Indigenous vaccination coverage by remoteness

Vaccination coverage in very remote areas increased from 83.9% in 2002 to 88.8% in 2005 (Table 4). For other regions there was variation between years but no evident increasing or decreasing trend. Coverage was consistently lowest in remote areas. In comparison, non-Indigenous coverage estimates remained stable (92.3%–94.8%) in every area across all years except for the very remote classification, where a drop to 90.0% was observed in 2003 (not shown).

Correlation between data completeness and indigenous coverage

Kendall's Tau coefficients of correlation between indigenous status completeness and indigenous coverage are presented in Tables 1 and 3. No statistically significant correlation, or consistent pattern of negative or positive correlation, was found for jurisdictions or remoteness categories.

Discussion

This analysis has shown that the reporting of indigenous status to the ACIR has improved markedly from 42% of the estimated cohort of Indigenous infants in 2002 to 95% in 2005. By 2005 indigenous status reporting rates were more than 80% in all remoteness categories and more than 70% in all jurisdictions. During this period national vaccination coverage estimates for Aboriginal and Torres Strait

Table 3. Increases in reporting of indigenous status to the ACIR* for children aged 12–14 months,† 2002 to 2005, by ASGC remoteness category

Remoteness category	% of ABS estimated Indigenous cohort recorded as Indigenous on ACIR		Correlation with coverage‡	
	2005	Annual increase 2002–2005	Correlation coefficient	P value
Major cities	103	17.5	0.00	1.0
Inner regional	104	20.4	0.33	0.75
Outer regional	105	21.9	0.67	0.33
Remote	84	12.2	0.33	0.75
Very remote	81	18.4	0.67	0.33
All categories	95	17.7	0.22	0.18

* Australian Childhood Immunisation Register.

† July to September birth cohorts.

‡ Kendall's Tau coefficients of correlation between completeness of indigenous status and indigenous coverage, and P value for testing hypothesis that there was no correlation. Perfect positive correlation is indicated by an estimate of 1, no correlation by 0.

Table 4. DTP coverage reported to ACIR for Indigenous children aged 12–15 months, 2002 to 2005, by ASGC remoteness classification

Region	2002	2003	2004	2005
Major cities	88.0	85.0	88.9	86.5
Inner regional	85.6	84.1	88.4	87.5
Outer regional	85.3	82.5	85.9	86.1
Remote	81.9	80.1	83.1	80.9
Very remote	83.9	87.7	89.0	88.8

Data are analysed as at 31 December of each year.

Islander infants changed little, although coverage for the third dose of DTP was consistently 6%–8% lower in Indigenous compared to non-Indigenous children at 12 months of age. When analysed by jurisdiction or ASGC remoteness category, coverage estimates appeared to become more stable as indigenous status completeness increased, but there was no statistically significant trend of coverage increasing or decreasing as indigenous identification improved. Perhaps unexpectedly, the reporting of indigenous status was lower in the ASGC classified 'remote' and 'very remote' areas compared to other areas, and coverage estimates for Indigenous infants were lower in 'remote' areas compared to regional, urban and very remote areas.

A clear trend in coverage for Indigenous children by remoteness was not evident, as areas classified as 'very remote' generally had the highest coverage, and 'remote' areas consistently the lowest, with more urbanised areas in between. Coverage estimates for non-Indigenous children were consistently higher, with no apparent trend by remoteness. Previous estimates for Indigenous children have generally been higher in remote areas and lower in non-remote areas,[3–7,14,15] although there have been some exceptions.[16,17] Previous studies were limited to local areas, obtaining data from surveys or local registers, conducted between 10 and 25 years ago, when coverage estimates in general were much lower than currently. The definitions of remoteness used in previous studies varied, and fewer categories were used than the five ASGC categories used here. This analysis suggests that, if there was a relatively consistent trend in the past towards higher coverage in Indigenous children in remote areas and lower coverage in urban areas, this is no longer the case. The possibility of inaccurate data masking a real trend by remoteness cannot be excluded, but these data do not support the hypothesis that the ACIR coverage estimates for Indigenous children are biased by higher indigenous reporting rates in remote areas with higher coverage.

The use of ACIR coverage estimates for Indigenous children relies on the assumption that, in addition to the completeness of recording, the recorded indigenous status data are valid. While the validity of the data have not been formally assessed, previous analyses have found that children reported as indigenous on the ACIR were more likely to have been reported as receiving vaccines recommended only for Aboriginal and Torres Strait Islander children,[2] and that the ACIR coverage estimates were similar to those of a face-to-face survey.[18] This analysis has shown that the reporting of indigenous status has improved dramatically in recent years and is now high in all jurisdictions, and in remote as well as urban areas. The ACIR should now be used with more confidence by vaccination program managers and public health practitioners to estimate coverage in Indigenous children at the jurisdictional level.

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Prevalence of MRSA among *Staphylococcus aureus* isolated from hospital inpatients, 2005: Report from the Australian Group for Antimicrobial Resistance

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

Abstract

The Australian Group for Antimicrobial Resistance conducted a survey of the prevalence of antimicrobial resistance in unique clinical isolates of *Staphylococcus aureus* from patients admitted to hospital for more than 48 hours. Thirty-two laboratories from all states and territories collected 2,908 isolates from 1 May 2005, of which 31.9% were methicillin-resistant *Staphylococcus aureus* (MRSA). The regional prevalence of MRSA varied significantly (P<0.0001) from 22.5% in Western Australia to 43.4% in New South Wales/Australian Capital Territory. Prevalence of MRSA from individual laboratories varied even more from 4% to 58%. This variation was explained in part by distribution of age with the risk of MRSA significantly (P<0.0001) increasing with age. Other unmeasured factors including hospital activity and infection control practices in the individual institution may have also contributed. Further investigation is warranted as reductions in prevalence would reduce morbidity, mortality and healthcare costs. *Commun Dis Intell* 2007;31:288–296.

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

Introduction

Staphylococcus aureus remains a major bacterial pathogen and is associated with considerable morbidity and mortality. Manifestations of *S. aureus* infection range from mild to moderate skin and soft tissue infections such as impetigo and furunculosis to invasive and often life threatening infections such as osteomyelitis, necrotising pneumonia and infective endocarditis. Bacteraemia is also common. In the pre-antibiotic era the mortality of staphylococcal bacteraemia was as high as 90%.[1] With antibiotic treatment, mortality has fallen but remains a major issue. With methicillin-sensitive *S. aureus* (MSSA) the median associated mortality is 25% (range 4%–52%) while with methicillin-resistant *S. aureus* (MRSA) the median is 35% (range 0%–83%).[2] In Australia, as in most of the world, antimicrobial resistance in *S. aureus* is a major impediment to effective treatment. Hospital strains are frequently resistant to methicillin (and all other beta-lactams) and multiple other antimicrobials.[3]

Methicillin-resistant *S. aureus* was first reported in Australia in 1968.[4] This archaic strain of MRSA was not usually resistant to other non-beta-lactam antimicrobials and was not resistant to gentamicin. The emergence of MRSA resistant to gentamicin and other classes of antimicrobials was first noted in eastern Australia in 1976. Outbreaks of hospital infection due to multi-resistant MRSA (mMRSA) occurred in the state of Victoria in the late 1970s and early 1980s.[5,6] mMRSA became endemic in hospitals in the eastern Australian states in the late 1980s and 1990s with some spread to hospitals in South Australia, the Northern Territory and Tasmania.[3,7] However, these strains did not become established in Western Australian hospitals due to active screening and infection control policies.[3,8] Eastern Australian MRSA has now been shown to be one clone by multi-locus sequence typing – ST239-MRSA-III.[9] This is one of the most successful MRSA clones and is now found extensively in Europe, Asia, and South America. More recently, MRSA clones of overseas origin have also been found in Australia. Most notably the United Kingdom strain, EMRSA-15, has spread widely in Australia to become a major endemic cause of hospital sepsis.[9]

Vancomycin has been the mainstay of treatment for serious infections due to MRSA. However, there is evidence that vancomycin is less effective in the treatment of methicillin-sensitive *S. aureus* than anti-staphylococcal beta-lactams.[10,11] Failure of vancomycin treatment of MRSA has been associated with the emergence of strains with MICs to vancomycin in the intermediate range (VISA).[12,13] These strains have been described in many parts of the world including Australia.[14] Isolation of VISA follows failure of prolonged treatment with vancomycin. One recent study has suggested that treatment failure is related to slightly higher vancomycin MICs (1.0–2.0 mg/L versus ≤0.5 mg/L) in pre-treatment isolates of MRSA.[15] Few treatment options remain for multi-resistant

MRSA and resistance to linezolid, one of the few new anti-staphylococcal agents of recent years, is already being reported.[16]

While it is well known that *S. aureus* is a major cause of severe sepsis, few population based estimates of its incidence or prevalence are available. A recent Australian survey of *S. aureus* bacteraemia from 1999 to 2002 documented 3,129 episodes.[2] Approximately 51% of bacteraemic episodes had their onset in hospitals. MRSA caused 40% of hospital-onset and 12% of community-onset episodes. The authors estimated that approximately 6,900 episodes of *S. aureus* bacteraemia occur in Australia annually. This equates to 35 episodes per 100,000 population. Meta-analysis of the outcomes of *S. aureus* bacteraemia has shown that the relative risk of death due to MRSA bacteraemia is approximately twice that due to MSSA.[17,18] It is widely acknowledged that nosocomial MRSA infection represents an additional burden of disease not just replacement of MSSA infection.[19] The cost of these additional infections is substantial for hospitals, patients and society. While costs vary from country to country, annual additional hospital costs due to MRSA in the United States of America are estimated at between US$1.5 billion and US$4.2 billion.[19] In Australia, the additional hospital costs associated with nosocomial *S. aureus* bacteraemia alone are estimated at approximately $150 million.[2] Effective infection control measures have been shown to reduce nosocomial infection significantly and to result in substantial savings.[19]

The objective of this study was to determine the prevalence of antimicrobial resistance in clinical isolates of *S. aureus* throughout Australia in hospital inpatients admitted for 48 hours or more.

Methods

Thirty-two laboratories from all six states, the Australian Capital Territory and the Northern Territory participated in the *S. aureus* Australian Group for Antimicrobial Resistance (AGAR) survey. From 1 May 2005, each laboratory collected up to 100 consecutive significant clinical isolates from hospital inpatients (hospital stay >48 hours at the time of specimen collection). Only one isolate per patient was tested and no isolates from screening swabs were included. If *S. aureus* was isolated from more than one site, then the isolate from the most significant clinical site was tested. Specimens received for the purpose of gathering surveillance data were excluded.

Species identification

S. aureus was identified by morphology and positive results of at least two of three tests: slide coagulase test, tube coagulase test, and demonstration of deoxyribonuclease production.[20] Additional tests such as fermentation of mannitol or growth on mannitol-salt agar may have been performed for confirmation.

Susceptibility testing methodology

Participating laboratories performed antimicrobial susceptibility tests using the Vitek2® AST-P545 card (BioMerieux, Durham, NC). Antimicrobials tested were benzylpenicillin, oxacillin, cefazolin, vancomycin, rifampicin, fusidic acid, gentamicin, erythromycin, clindamycin, tetracycline, trimethoprim/sulphamethoxazole (cotrimoxazole), ciprofloxacin, quinupristin/dalfopristin (Synercid®), teicoplanin, linezolid, imipenem, and nitrofurantoin. Results were interpreted for non-susceptibility according to CLSI breakpoints.[22,23] Penicillin susceptible strains were tested for ß-lactamase production using nitrocefin. A cefoxitin disc diffusion test was used to confirm methicillin-resistance. Mupirocin and cefoxitin were tested by disc diffusion using the CLSI or CDS methods.[21–23] The minimum inhibitory concentration (MIC) of mupirocin resistant isolates was determined by Etest® (AB Biodisk, Solna, Sweden). The macro Etest® method was used to determine hetero-resistance to vancomycin.

Statistical analysis

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

Results

Participating laboratories (27 public and 5 private) were located in New South Wales (8), the Australian Capital Territory (1), Queensland (6), Victoria (6), Tasmania (2), the Northern Territory (1), South Australia (4) and Western Australia (4). To ensure institutional anonymity data were combined for New South Wales and the Australian Capital Territory; Tasmania and Victoria; and Queensland and the Northern Territory (Table 1). There were 2,908 isolates included in the survey with the majority (76.1%) of isolates contributed by New South Wales/Australian Capital Territory (28.4%), Victoria/Tasmania (24.9%) and Queensland/Northern Territory (22.8%).

Specimen source

The majority of *S. aureus* isolates (67.6%) were from skin and soft tissue infections (Table 2). Respiratory specimens were the second most common source (17.4%) followed by blood culture isolates, 6.7%, with significantly (P<0.0001) more isolates causing non-invasive (91.2%) than invasive (8.7%) infections.

Susceptibility results

Nationally, 31.9% of *S. aureus* isolates were MRSA (Table 3) with the proportion varying significantly between states and territories (X2 = 110.54, P<0.0001). The proportion of MRSA in New South Wales/Australian Capital Territory hospitals (43.4%) was significantly higher (P<0.001) than the Australian average of 31.9%. There was no significant difference in the proportion of MRSA isolates that caused invasive infections (20.0% to 41.2% respectively, P=0.267) while the proportion of non-invasive infections ranged from 22.8% in Western Australia to 43.7% in New South Wales/Australian Capital Territory (P<0.0001). There was a wide range in the proportions of MRSA isolated by institutions with 31.0%–58.0% in New South Wales/Australian Capital Territory, 19.0%–36.0% in Queensland/Northern Territory, 15.0%–29.0% in South Australia, 4.0%–53.5% in Victoria/Tasmania and 14.5%–29.2% in Western Australia (Table 4).

Resistance in MRSA to non-beta-lactam antimicrobials varied significantly between states with the exception of mupirocin (Table 5). Resistance with the widest range was identified for gentamicin (5.0% to 79.5%, P<0.0001), tetracycline (6.3% to 83.0%, P<0.0001), cotrimoxazole (7.5% to 80.8%, P<0.0001) and clindamycin (8.3% to 68.7%, P<0.0001). Resistance to ciprofloxacin was also common ranging from 42.5%–89.4% (P<0.0001). Resistance to fusidic acid across the states varied significantly (P=0.0023) with the highest proportion in South Australia (11.9%). There was no significant difference (P=0.713) in the low levels of mupirocin resistance. One isolate from Victoria/Tasmania had a quinupristin/dalfopristin MIC

Table 1. Isolates by region

Region	Number of institutions	Total	% 95%CI
New South Wales/ Australian Capital Territory	9	825	28.4 (26.7–30.0)
Queensland/ Northern Territory	7	664	22.8 (21.3–24.4)
South Australia	4	340	11.7 (10.5–12.9)
Victoria/Tasmania	8	724	24.9 (23.3–26.5)
Western Australia	4	355	12.2 (11.0–13.4)
Total	32	2,908	100

Table 2. Source of isolates

Specimen source	n	%
Skin and soft tissue	1,967	67.6
Respiratory	506	17.4
Blood	194	6.7
Urine	92	3.2
Eye	62	2.1
Sterile site	50	1.7
Ear	13	0.4
Cerebrospinal fluid	8	0.3
Other	11	0.4
Unknown	5	0.2
Total	2,908	
Invasive	252	8.7
Non-invasive	2,651	91.2
Not specified	5	0.2

Table 3. Proportion of methicillin-resistant *Staphylococcus aureus* for all isolates, invasive isolates and non-invasive isolates, by region

	All Isolates		Invasive		Non-invasive	
	n	%	n	%	n	%
NSW/ACT	358/825	43.4	35/85	41.2	323/739	43.7
Qld/NT	177/664	26.7	13/36	36.1	164/628	26.1
SA	84/340	24.7	10/34	29.4	73/304	24.0
Vic/Tas	229/724	31.6	23/59	39.0	206/664	31.0
WA	80/355	22.5	6/30	20.0	74/325	22.8
Aus	928/2,908	31.9	87/244	35.7	840/2,660	31.6
Difference across regions χ^2	81.01		5.20		78.81	
P value	<0.0001		0.267		<0.0001	

of >2 mg/L by broth micro-dilution and an Etest MIC of 6 mg/L. In addition, one result for quinupristin/dalfospristin was missing. One isolate from New South Wales/Australian Capital Territory had Vitek MIC results of 4 mg/L for vancomycin and teicoplanin (non-susceptible). The broth dilution MIC of both agents was 2 mg/L and the isolate was confirmed as a hetero-vancomycin intermediate *S. aureus* (hVISA) by the macro Etest method.

MSSA were generally susceptible to most non-beta-lactam antimicrobials with no significant difference in proportion across all regions with the exception of the level of resistance in tetracycline (P=0.0005) with New South Wales/Australian Capital Territory having the highest level at 3.6%, and gentamicin (P=0.0047) with Victoria/Tasmania having the highest level at 3.2% (Table 6).

Table 4. Proportion of methicillin-resistant *Staphylococcus aureus*, by institution

Region	Laboratory code	% MRSA
NSW/ACT	1	31.0
	2	50.0
	3	31.3
	4	47.0
	5	58.0
	6	51.0
	7	38.5
	8	46.0
	9	34.0
Qld/NT	10	30.0
	11	19.0
	12	20.0
	13	29.9
	28	23.2
	29	28.8
	30	36.0
SA	14	29.0
	15	29.0
	16	15.0
	17	27.5
Vic/Tas	18	4.0
	19	45.0
	20	23.1
	21	10.0
	22	43.0
	23	53.5
	31	35.0
	32	33.0
WA	24	14.5
	25	25.0
	26	22.0
	27	29.2
Australia		31.9

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

Patients with MRSA ranged in age from less than one year to 100 years, with a mean of 54.3 years. The distribution of age was skewed towards the elderly with the 25th percentile at 35 years, the 50th at 61 years and the 75th at 77 years. MSSA was significantly (P<0.0001) more common than MRSA in all five age groups; neonatal (<1–1 year), paediatric (2–16 years), adult (17–40 years), middle-age (41–61 years) and the older (62–100 years) (Table 7).

When the relationship between mean age and proportion of MRSA in institutions was examined, a significant (P two tailed = 0.02), but weak linear trend (r = 0.4195), was identified (Figure 1). The sample sizes contributed by the member hospitals were small with a wide dispersion of the mean age (Figure 2) across the 32 facilities. However, when age was categorised into five ranges for the aggregated data from all hospitals and odds ratio of MRSA cases for each age group was examined against the youngest, MRSA was significantly more likely to occur in patients in successively older age groups compared with MSSA (Table 8). Advancing age is a strongly significant risk factor for acquisition with patients aged between 62 years and 100 years being 10.33 (P<0.0001) times more likely to have MRSA (not MSSA) compared with babies.

Figure 1. Relationship of mean age and proportion of methicillin-resistant *Staphylococcus aureus* for 32 institutions

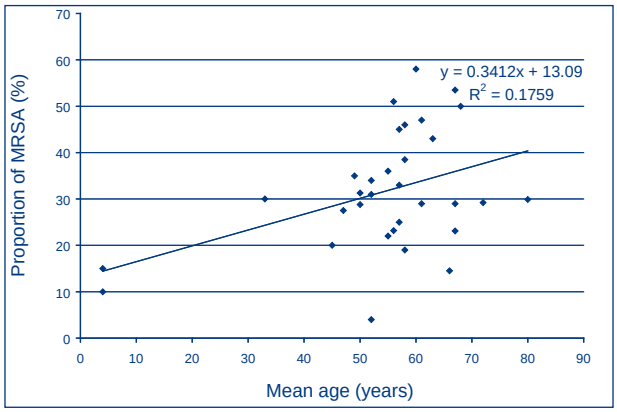


Table 5. Number and proportion non-susceptible methicillin-resistant *Staphylococcus aureus* isolates, by region

Region	Em		Cm*		Tc		Tmp-SXT		Cf		Gm		Fa		Mp	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
NSW/ACT	309/357	86.6	193/281	68.7	247/358	69.0	251/358	70.1	320/358	89.4	250/358	69.8	13/358	3.6	12/358	3.4
Qld/NT	129/177	72.9	74/177	41.8	79/177	44.6	91/177	51.4	111/177	62.7	98/177	55.4	10/177	5.6	4/177	2.3
SA	51/84	60.7	7/84	8.3	30/84	35.7	27/84	32.1	46/84	54.8	28/84	33.3	10/84	11.9	1/84	1.2
Vic/Tas	207/229	90.4	94/228	41.2	190/229	83.0	185/229	80.8	202/229	88.2	182/229	79.5	4/229	1.7	6/229	2.6
WA	46/80	57.5	8/80	10.0	5/80	6.3	6/80	7.5	34/80	42.5	4/80	5.0	3/80	3.8	1/80	1.3
Aus	742/927	80.0	376/850	44.2	551/928	59.4	560/928	60.3	713/928	76.8	562/928	60.6	40/928	4.3	24/928	2.6
Difference across regions χ^2	75.61		151.25		201.42		181.44		144.13		178.66		16.63		2.13	
P value	<0.0001		<0.0001		<0.0001		<0.0001		<0.0001		<0.0001		0.0023		0.713	

Em: erythromycin, Cm: clindamycin, Tc: tetracycline, Tmp-SXT: trimethoprim/sulphamethoxazole, Cf: ciprofloxacin, Gm: gentamicin, Fa: fusidic acid, Mp: mupirocin

* Constitutive resistance.

Table 6. Number and proportion non-susceptible methicillin sensitive *Staphylococcus aureus* isolates, by region

Region	Pc		Em		Cm		Tc		Tmp-SXT		Cf		Gm		Fa		Mp	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
NSW/ACT	405/467	86.7	60/467	12.8	8/448	1.8	17/467	3.6	12/467	2.6	18/466	3.9	5/467	1.1	13/467	2.8	4/467	0.9
Qld/NT	416/487	85.4	63/487	12.9	2/487	0.4	8/487	1.6	3/487	0.6	8/487	1.6	5/487	1.0	18/487	3.7	5/487	1.0
SA	219/256	85.5	22/256	8.6	3/256	1.2	7/256	2.7	3/256	1.2	6/256	2.3	2/256	0.8	7/256	2.7	2/256	0.8
Vic/Tas	406/495	82.0	66/495	13.3	8/495	1.6	25/495	5.1	8/495	1.6	10/495	2.0	16/495	3.2	18/495	3.6	6/495	1.2
WA	241/275	87.6	21/275	7.6	4/275	1.5	0/275	0.0	2/275	0.7	6/275	2.2	1/275	0.4	15/275	5.5	3/275	1.1
Aus	1,687/1,980	85.2	232/1,980	11.7	25/1,961	1.3	57/1,980	2.9	28/1,980	1.4	48/1,979	2.4	29/1,980	1.5	71/1,980	3.6	20/1,980	1.0
Difference across regions χ^2	6.17		9.37		4.37		20.15		7.88		5.75		15.01		4.20		0.47	
P value	0.187		0.052		0.358		0.0005		0.096		0.219		0.0047		0.379		0.977	

Em: erythromycin, Cm: clindamycin, Tc: tetracycline, Tmp-SXT: trimethoprim/sulphamethoxazole, Cf: ciprofloxacin, Gm: gentamicin, Fa: fusidic acid, Mp: mupirocin

* Constitutive resistance.

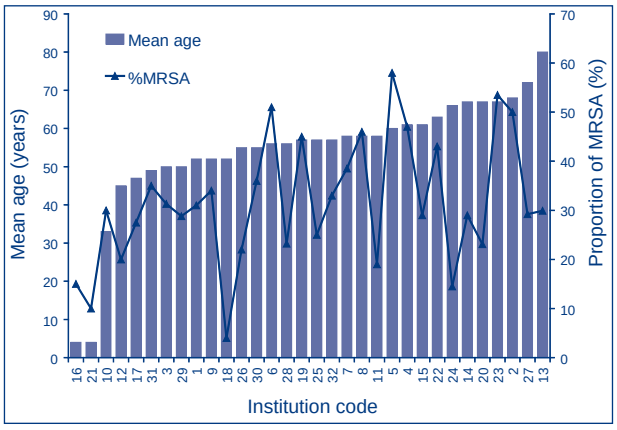
Table 7. Age by methicillin susceptibility of *Staphylococcus aureus*

Age	Total			MRSA			MSSA			Difference in isolates by age category (row)	
	n	%	95% CI	n	Row %	Column %	n	Row %	Column %	χ^2	P
0–1	264	9.1	8.1–10.2	17	6.4	1.8	247	93.6	12.5	400.76	<0.0001
2–16	132	4.5	3.8–5.4	29	22.0	3.1	103	78.0	5.2	82.97	<0.0001
17–40	426	14.7	13.4–16.0	113	26.5	12.2	313	73.5	15.8	187.79	<0.0001
41–61	642	22.1	20.6–23.6	207	32.2	22.3	435	67.8	22.0	161.94	<0.0001
62–100	1,443	49.6	47.8–51.5	562	38.9	60.6	881	61.1	44.5	1142.81	<0.0001
Total	2,907	100	–	928	31.9	100	1,979	68.1	100	103.96	<0.0001

Table 8. Risk of methicillin-resistant *Staphylococcus aureus*, by age groups

Age	Unadjusted Odds Ratio	95% CI	P	Adjusted Odds Ratio*	95%CI	P
0–1	1 (referent group)	–	–	1 (referent group)	–	–
2–16	4.09	2.06 – 8.16	<0.0001	4.25	2.22 – 8.11	<0.0001
17–40	5.25	2.99 – 9.32	<0.0001	5.72	3.22 – 9.85	<0.0001
41–61	6.91	4.02 – 12.04	<0.0001	7.37	4.36 – 12.46	<0.0001
62–100	9.27	5.49 – 15.86	<0.0001	10.33	6.21 – 17.10	<0.0001
	P<0.0001, χ^2 for linearity = 119.729			* Adjusted for state and territories		

Figure 2. Mean age compared with proportion of methicillin-resistant *Staphylococcus aureus* in participating institutions



Discussion

Surveys conducted by AGAR from 1986 to 1999 included all consecutive clinical isolates of *S. aureus* during the survey period regardless of acquisition.[3,7,24] Participating laboratories did not need to acquire any additional information to distinguish between inpatients and outpatients and so an overall MRSA prevalence was derived. Compliance with methodology was a potential issue particularly in the early days of the surveys but this simple data collection was reliably achieved. It also allowed for comparison of results over a prolonged period. The advent of community strains of MRSA during the 1990s[25,26] however, led to interest in studying the prevalence of MRSA in outpatient infections alone. AGAR responded by conducting biennial outpatient surveys from 2000 onwards.[9,27] Since then evidence has emerged that strains that initially were acquired almost exclusively in the community were now being acquired in the health care setting with increasing frequency.[28] Therefore, in 2005 a survey of hospital-acquired *S. aureus* infection was undertaken. The results provide us with the first accurate estimates at a national level of the proportion of hospital-acquired *S. aureus* infection that are due to MRSA.

In this survey 2,908 isolates were collected in 32 laboratories covering all states and territories. Overall, 31.9% of isolates were MRSA. While there was a significant difference in the proportion of MRSA between regions (from 22.5% in Western Australia to 43.4% in New South Wales), this may have been due in part to different age distributions. The overall proportion of MRSA in invasive (mainly bacteraemia) isolates was similar to that of non-invasive isolates (35.7% and 31.6% respectively, P=0.195. The high proportion of MRSA in invasive isolates is of concern as MRSA bacteraemia is associated with increased mortality compared with MSSA.[17,18,31] Direct comparison with prevalence in other countries is difficult due to methodological differences. For example, the

European surveillance system reports the proportion of MRSA in bacteraemia isolates in both inpatients and outpatients in 23 countries.[32] Even so, the overall proportion in Europe in 2005 varied from 1.7% in Denmark to 55% in Malta. The Netherlands and the Scandinavian countries have been consistently able to keep MRSA at very low levels in their hospitals over long periods.

Resistance to non-beta-lactams in MRSA was common for erythromycin, clindamycin, tetracycline, cotrimoxazole, ciprofloxacin and gentamicin and varied considerably from region to region. This regional variability is due to the differential distribution of MRSA clones in the major cities. For example, ST239-MRSA-III (AUS-2 and AUS-3 strains), which is resistant to multiple non-beta-lactams including gentamicin, erythromycin and tetracycline, is endemic in the eastern states but is less common in Western Australia and South Australia. ST22-MRSA-IV (UK EMRSA-15), which is resistant to ciprofloxacin and often erythromycin but susceptible to all other non-beta-lactams, is more common in Western Australia as are other non-multi-resistant strains.[9,27] Resistance of MSSA to non-beta-lactam antimicrobials was uncommon except for erythromycin. There was little variability between regions in the low levels of resistance to other agents, with the exception of tetracycline and gentamicin. Once again this may be due to regional variations in the prevalence of strains of MSSA carrying different combinations of resistance genes.

The prevalence of MRSA isolates varied from 4.0% to 58.0% between institutions. The high levels in some institutions are a cause for concern given the increased mortality, morbidity and cost associated with MRSA infection.[19,33] While it is generally accepted that the prevalence of MRSA in an institution reflects the effectiveness of infection control practice,[34] it is also true that age is a risk factor or proxy for MRSA infection.[35] Analysis of the 2005 survey data confirmed that risk of MRSA did increase significantly with age ($P<0.0001$). There was also a weak association between mean age and proportion of MRSA in institutions. The weakness of the association was due in part to the low sample size resulting in variability in the mean age. Equally, other factors such as variability in activity, acuity and infection control practice may also have contributed. Given the marked variability in prevalence between institutions it seems unlikely that mean age alone could explain the difference. Until other risk factors have been accurately identified, the elderly should be considered to be at highest risk when developing strategies for the control of MRSA. The possibility of controlling MRSA in the health care setting was demonstrated quite early in Australia.[8] There is now ample and consistent evidence that infection control strategies based on screening, isolation and decolonisation are successful and highly cost effective.[19] The reasons for significant variability between regional and institutional prevalence of MRSA is worthy of further study. Reduction of MRSA infection in high prevalence institutions is likely to result in lower levels of morbidity and mortality and in lower health care costs.

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

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Pneumonia cluster in a boarding school – implications for influenza control

Patrick Cashman, Peter Massey, David Durrheim, Fakhrul Islam, Tony Merritt, Keith Eastwood

Abstract

Streptococcus pneumoniae is a common cause of community acquired pneumonia (CAP). Influenza infection increases susceptibility to *S. pneumoniae* infection in adults but this link is less well described in children. We report on an outbreak of CAP affecting 25 previously well adolescents in a New South Wales boarding school. *S. pneumoniae* 1 was confirmed in two cases. During this period, the school also experienced an influenza outbreak with an influenza-like illness attack rate peaking at 27% in Year 8 students. A planned school closure may have contributed to controlling the outbreak. Boarding schools are vulnerable to outbreaks of respiratory illness and strategies for limiting this risk are required. *Commun Dis Intell* 2007;31:296–298.

Keywords: *Streptococcus pneumoniae*, influenza, boarding school, school closure

Introduction

Streptococcus pneumoniae is the most common cause of community acquired pneumonia (CAP).[1] Institutionalisation is a risk factor for pneumococcal clusters but these have generally been described in the elderly.[2] Serotype 1 has been associated with severe pneumonia in otherwise healthy children, has a propensity for invasive disease and has caused outbreaks in institutions.[3] This serotype remains highly susceptible to antibiotic therapy.[4]

Influenza infection frequently precedes pneumococcal pneumonia in adults but this relationship is less well documented in children.[3] Influenza virus may increase susceptibility to invasive pneumococcal disease through destroying the physical respiratory barrier, increasing virus adherence, decreasing mucociliary activity and disrupting immune system responses.[5]

Influenza and invasive pneumococcal disease are notifiable by pathology laboratories in New South Wales under the *NSW Public Health Act 1991*.[6]

We report on a cluster of 25 cases of CAP in previously well adolescents attending a boarding school in rural New South Wales and discuss implications for influenza surveillance and control.

Cluster report

In August 2006, Hunter New England Population Health was notified by a paediatrician at a rural referral hospital of the admission of five male students with pneumonia from a secondary boarding school. Three were boarders and two were day students. All had presented with fever, lethargy, chest pain and cough, and had a typical lobar pneumonia on chest X-ray. They responded rapidly to intravenous penicillin. A broad range of zoonotic infections were considered and excluded. *Streptococcus pneumoniae* was identified from one of the student's blood cultures. None of the students reported any recent overseas travel.

Enquiries to local general practitioners and the school sick bay identified a recent large increase in respiratory presentations amongst students from this school. Ongoing surveillance identified a further 20 students with lobar pneumonia. Thus a total of 25 of 600 students at the school were diagnosed with pneumonia, two of whom had *Streptococcus pneumoniae* serotype 1 isolated from blood cultures. Fifteen of these children required hospital admission, eight students were diagnosed clinically by general practitioners and two were treated as outpatients by the hospital emergency department. All hospitalised cases responded rapidly to intravenous penicillin with a median hospital stay of three days.

The pneumonia cases in previously healthy adolescents occurred in an environment of widespread influenza infection. The surveillance identified large numbers of students at the school who were presenting to the school sick bay with upper respiratory tract infection (URTI) and influenza-like illness (ILI). Influenza A H3N2 was isolated from respiratory specimens collected from two hospitalised students with pneumonia and from three students presenting to the sick bay at school with ILI. Two unimmunised hospital staff caring for student inpatients with pneumonia were also subsequently diagnosed with influenza.

Public health responses included implementing a 'testing and treatment algorithm' at the Emergency Department for CAP presentations and involving the local public pathology provider in ensuring prioritisation of investigations related to the outbreak with appropriate referral to reference laboratories. Increased respiratory hygiene measures were implemented throughout the school with students actively encouraged to cover coughs and sneezes with tissues and then dispose of tissues in the garbage after use. Handwashing after coughing, sneezing or nose-blowing was also promoted by the school nurses and staff. Information about the outbreak was distributed to parents in the school newsletter with advice to keep students with symptoms at home. The school nurses facilitated the separation of students with symptoms to their homes.

Structured interviews with students with pneumonia and their parents were conducted to attempt to identify specific common exposures by place, time, recreational or school activity and boarding status. No specific shared risk factor was found other than being a student at the school. Boarding status was not a risk factor as the proportion of boarding and day students with pneumonia was similar to those proportions in the whole school student population. However in the earlier part of the outbreak, more cases of pneumonia were noted amongst boarding students. Students with pneumonia were resident in both school dormitories.

School year-specific attack rates were calculated by examining presentations for URTI and ILI to the school sick bay and general practice, and presentations of pneumonia to general practice and hospitals (Figure, Table). Fifty per cent of all students at the school presented with some form of respiratory symptom. ILI presentations at the school sick bay were highest amongst Year 8 students (27%) but affected all school years.

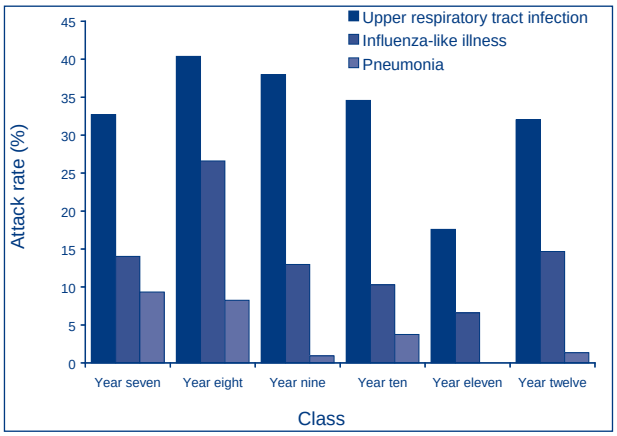
Discussion

Following the introduction of improved respiratory hygiene measures at the school and a pre-scheduled four day school closure, respiratory illness presentations to the sick bay decreased appreciably and returned to pre-outbreak levels within seven days of the school closure. This may indicate the success of social distancing in responding to respiratory outbreaks in institutions or may represent exhaustion of the influenza at-risk population.

Clusters of pneumonia in institutions amongst people of any age should alert clinicians to possible coinfection with influenza virus and *S. pneumoniae* and prompt appropriate laboratory investigations and notification to public health authorities.

Although influenza vaccination should primarily be targeted to traditionally high risk individuals, consideration should also be given to offering it in high-risk environments, including boarding schools.[7] The occurrence of influenza infection in hospital staff who cared for the children in this outbreak,

Attack rates for upper respiratory tract infection, influenza-like illness and pneumonia, August 2006, by year level for all students at the boarding school



	Year seven %	Year eight %	Year nine %	Year ten %
URTI	32.7	40.4	38.0	34.6
ILI	14.0	26.6	13.0	10.3
Pneumonia	9.3	8.3	0.9	3.7

All categories are exclusive.

adds weight to the current emphasis on protecting health staff and their patients with annual influenza immunisation.

Boarding schools, in common with other institutions where people live in close proximity, are vulnerable to outbreaks of respiratory illness. Strategies for limiting this risk are required and may include education on respiratory hygiene, guidelines for limiting overcrowding, consideration of annual influenza vaccination and guidelines for early detection and response to respiratory outbreaks.[8]

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Short reports

Importation of wild poliovirus into Australia, July 2007

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On 13 July 2007, the National Poliovirus Reference Laboratory (NPRL), which is part of the Victorian Infectious Diseases Reference Laboratory, confirmed infection with wild poliovirus serotype 1 in an overseas-born student who had recently returned from Pakistan. This is the first laboratory confirmed case of polio due to wild poliovirus reported in Australia since 1977. Pakistan is one of the four remaining polio endemic countries along with India, Afghanistan and Nigeria.[1] Wild poliovirus is currently circulating in six other countries – Angola, Chad, Democratic Republic of Congo, Myanmar, Niger and Somalia – due to importation from endemic countries.[1] The Australian wild poliovirus importation was characterised by the methods described in the accompanying annual report of the NPRL.[2]

The last wild poliovirus isolated from a patient with poliomyelitis in Australia prior to this case was an imported case from Turkey in 1977.[3] Based on phylogenetic analysis of archived poliovirus isolates, we believe the last endemic case of wild poliovirus infection in Australia to have occurred in the late 1960s, although an uncharacterised serotype 3 poliovirus was isolated from a case from Queensland in the early 70s, and wild poliovirus could not be excluded as the causative agent.

We have previously emphasised the need for vigilance in order to maintain Australia's polio free status.[4] This case reinforces that message. We continue to urge all clinicians who are consulted by a person of any age presenting with acute flaccid paralysis, to consider poliomyelitis in the differential diagnosis of the illness. Referral of stool samples to the NPRL at VIDRL is critical for complete characterisation of virus isolates.[2]

Importation of wild virus or vaccine-derived poliovirus into Australia is an extremely low probability event. The current case demonstrates that low probability events occur and confirms the requirement for vigilance until the world is declared free of circulating wild poliovirus. Although poliovirus serotype 2 has been eradicated from the world,[5] the eradication of serotypes 1 and 3 is still some years in the future.

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Haemolytic uraemic syndrome associated with a family cluster of enterohaemorrhagic *Escherichia coli*

Jeffrey N Hanna, Jan L Humphreys, Sian E Ashton, Denise M Murphy

Introduction

In early 1995 an outbreak of 23 cases of haemolytic uraemic syndrome (HUS) occurred in children (ranging from four months to 12 years of age) in South Australia.[1] Twenty of the cases were managed in a tertiary paediatric hospital in Adelaide, where 18 (90%) required dialysis.[2] A 4-year-old died and 12 months after discharge 5 of the surviving children still had significantly impaired renal function.[2]

In Australia HUS is usually caused by a subgroup of Shiga toxin-producing *Escherichia coli* known as enterohaemorrhagic *E. coli* (EHEC). The Shiga toxins cause cell damage and trigger an inflammatory process which initiates intravascular coagulation resulting in microthrombi forming in small blood vessels in the gut and kidney.[3] The natural reservoir of EHEC is the gut of animals, particularly cattle and sheep. Hence HUS can be caused by contact with animal faeces, either directly or via contaminated, inadequately cooked food, particularly meat and dairy products. Most of the cases in the 1995 outbreak of HUS in South Australia had consumed (in the week before the onset of illness) an uncooked fermented sausage manufactured in Adelaide.[1] Subsequent molecular studies revealed an identical EHEC in both faeces of the cases and samples of the sausage.[4]

Because EHEC infection, and therefore HUS, can be foodborne, it is of considerable public health concern. Following the South Australian outbreak, HUS became a notifiable disease in Queensland in mid-1996, and EHEC in mid-2001. However, a recent case of HUS in north Queensland has identified several shortcomings in the management and investigation of HUS and EHEC infections; some of these shortcomings were also identified in a previous cluster of HUS cases that occurred in north Queensland in 2004.[5]

The HUS case and subsequent investigations

In early January 2007, the Tropical Population Health Network (TPHN) was notified by an infection control practitioner that a 14-month-old Caucasian girl had been hospitalised the previous day with HUS; she had become unwell four days before being hospitalised. *Salmonella* Virchow was isolated from a diarrhoeal stool sample collected two days prior to her being hospitalised and from a stool sample collected on the day of admission.

The attending physician initially believed that the *Salmonella* infection was the cause of the HUS, and this led to problems in getting the (diarrhoeal) samples to the Queensland Health Scientific Services reference laboratory for screening for Shiga toxin (stx1 and stx2) gene (and therefore for EHEC). The initial sample was not forwarded, and the second sample was not forwarded frozen to the laboratory. When the latter sample was eventually screened stx genes were not detected. Therefore EHEC was never detected in the HUS case.

The child's parents were interviewed using the relevant OzFoodNet questionnaire; this did not reveal any suspect food items in the child's diet. However, it did reveal that the child and her two siblings had visited several commercial animal sanctuaries during the exposure period. At two of these the children had had direct contact with marsupials (particularly kangaroos and koalas) and apparently also with faeces from these animals. The parents stated that the children had no contact with other mammals at these sanctuaries, and no apparent contact with any bovine animals during the exposure period.

The child's two siblings attended a local child-care centre. Even though they were apparently asymptomatic, stool samples were collected (in mid-January) from both and the parents were requested by TPHN to keep them out of child care until the results of the stool tests were known.[6,7] The child's twin sibling's stool was positive for the stx2 (but not the stx1) gene and the eaeA gene (which encodes a virulence factor: intimin) upon screening, and was culture positive for *E. coli* O55:H80, *S.* Aberdeen and *S.* Chailey. The child's 3-year-old brother's stool was also positive for the stx2 (but not the stx1) gene and the eaeA gene upon screening, and was culture positive for *E. coli* O55:HR. ('R' indicates that the organism had become rough in sub-cultures; once an EHEC becomes rough, the H antigen cannot be typed.) Both parents then had stool samples collected, but neither had evidence of EHEC upon screening.

The two siblings were voluntarily excluded from child-care until they were clear of the Shiga toxin in weekly stool samples. The 3-year-old and the twin sibling were able to return to child-care (after having two successive stool samples collected at least 48 hours apart clear of any evidence of Shiga toxin or EHEC[7]) 3.5 and 4.5 weeks, respectively, after having been first identified as being infected with EHEC. This delay created considerable difficulties for the parents, and repeated explanations of the importance of their exclusion from child-care were necessary.

The two siblings (and presumably the case) were infected with Shiga toxin-producing *E. coli* (all presumably with O55:H80), and the twins were infected with three different *Salmonella* serovars. This array of pathogens supported the hypothesis, as suggested from the parent interview, that the EHEC was acquired via animal contact rather than via a particular food item. For this reason, an assessment of the facilities and signs at the two animal sanctuaries was undertaken (about 2.5 weeks after the onset of the HUS) by environmental health officers. The public was encouraged to handle animals at both sanctuaries but there were no signs recommending hand washing after handling the animals at either sanctuary. At one sanctuary there were no hand washing facilities near the animal-handling areas. There were several food outlets in close proximity to the animal facilities.

It appeared that the management of the sanctuaries had little understanding of the potential infectious hazards associated with such facilities, and were uncertain of their responsibilities to minimise the risk of such hazards. Indeed, there are no guidelines in Queensland on how to minimise these risks for managers of commercial facilities that encourage the public to handle animals.

There was no evidence of Shiga toxin in faecal samples from koalas and kangaroos at either of the facilities, however, the samples were collected about a month after the onset of the HUS.

Discussion

This report describes three siblings infected with a Shiga toxin-producing EHEC; two who remained asymptomatic (presumably both with *E. coli* O55:H80), and their sibling (presumably infected with the same EHEC) who developed HUS. Both the sxt2 and eaeA genes were detected in this EHEC; this combination of genes appears to be an important predictor of HUS.[8]

This is the second cluster of EHEC with HUS in north Queensland in three years.[5] The two clusters have identified several issues of concern (Box).

Salmonella infections do not cause HUS,[9] and the isolation of salmonellae from faecal samples from a HUS patient must be regarded as coincidental to the HUS. Screening for Shiga toxin and other virulence genes was undertaken on the *S.* Virchow isolated from the HUS child to prove this point to the attending physician; none of the genes were detected. Faecal samples from HUS cases must be forwarded promptly to a reference laboratory for screening for EHEC regardless of the isolation of *Salmonella* from the samples. Failure to do this may result in EHEC not being isolated from a case (as happened with the child with HUS in this cluster), and could impede the necessary investigations.

Issues of public health significance revealed by this family cluster of EHEC infections

- HUS is a notifiable disease. As it is a syndrome, notification has to come from clinicians: not only paediatricians but also haematologists, nephrologists, infectious diseases and intensive care physicians need to be aware of their responsibility to notify cases of HUS.
- As soon as HUS is diagnosed, a stool sample should be sent to the relevant reference laboratory for screening tests for Shiga toxins and EHEC.
- *Salmonella* infections do not cause HUS. Isolation of *Salmonella* from the stool of an HUS case must be considered as coincidental to the HUS.
- Stool samples being submitted for investigation for Shiga toxin and EHEC must be frozen soon after collection, and transported frozen to the reference laboratory.
- Children less than 5 years of age who attend child-care, and who are household contacts of a case of symptomatic EHEC infection, should be screened for EHEC even if they are asymptomatic. They should be excluded from child-care until two stool samples, collected at least 48 hours apart, are shown to be clear of the EHEC.
- Infectious disease hazards are associated with contact with animals in public facilities – 'petting zoos' – and the management of such facilities need to take measures to reduce the risk of these hazards.
- Guidelines on how these infectious hazards can be minimised need to be formally endorsed by the relevant agencies so that the management of petting zoos and other similar facilities can be made aware of their responsibilities.

The infecting dose of HUS is very low, and person-to-person transmission of EHEC is well documented, with transmission occurring among young children within families and in child care facilities.[10] For this reason it is essential to screen the young siblings of EHEC HUS cases for the organism even if they are asymptomatic, and older siblings (and other close contacts) if they have any relevant symptoms.[5] These individuals should be excluded from child care (or any workplace of concern) while the screening takes place, and may need to be further excluded should the screening indicate an EHEC infection.[6,7]

There does not appear to be any published information as to whether marsupials act as reservoirs of EHEC. However, it is well recognised that macropods (kangaroos, wallabies) can be infected with salmonellae,[11] and an outbreak of human salmonellosis associated with contact with wallabies in a petting zoo has been reported from the United States of America.[12] Several other zoonotic (mostly enteric) infections have occurred following handling animals in petting zoos.[12,13]

It is important that guidelines on how to minimise the risk of transmission of infections through handling animals be made readily available to those facilities that encourage the public to handle animals on-site. These guidelines should include educating the public; appropriate signage; providing hand washing facilities; ensuring adequate supervision of children; discouraging eating in animal contact areas; ensuring sick animals are not handled by the public; providing appropriate cleaning and infection of the animal holding area and ensuring the safe disposal of animal faeces.[13] Such guidelines are available in several countries,[14,15] and in South Australia;[16] these guidelines are being used as templates for the drafting, currently in progress, of petting zoo guidelines for use in Queensland.

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Current developments in varicella-zoster virus disease prevention

A report on the varicella-zoster virus workshop convened by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases on 16–17 November 2006

Anita E Heywood, Kristine K Macartney, C Raina MacIntyre, Peter B McIntyre

Introduction

On 16 and 17 November 2006, the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) hosted a workshop on varicella-zoster virus (VZV) disease. The workshop was aimed at presenting the latest information on the clinical, epidemiological, and diagnostic aspects of both primary varicella ('chickenpox') and herpes zoster (HZ or 'shingles') both in Australia and internationally, and to highlight important developments in the prevention of these diseases by vaccination. This workshop was held at a significant stage in the control of VZV disease in Australia with the recent addition of the varicella vaccine to the National Immunisation Program (NIP) schedule, the anticipated availability of combination measles-mumps-rubella-varicella (MMRV) vaccines for use in children, and the availability of a zoster vaccine for use in older adults to prevent reactivation of VZV causing HZ.

The workshop was attended by prominent international researchers and leading Australian experts. All state and territory jurisdictions were represented and participated in panel discussions, particularly with the regard to disease surveillance. The first day of the workshop was devoted to varicella disease with presentations on the clinical features, current epidemiology, the Australian varicella vaccination program, and the impact of varicella vaccination in the United States of America (USA). An overview of the development of the MMRV vaccines was provided, and the day closed with a panel discussion of the issues surrounding varicella vaccine scheduling.

The second day focused on HZ with presentations on the burden of disease in Australia, the pathologic mechanisms and diagnostics. An overview and update on data from the zoster vaccine clinical trials was presented. The day concluded with state and territory representatives presenting plans for disease surveillance, and a panel discussion focusing on the best approach for the control of VZV disease in Australia.

Day one – varicella

Clinical overview

Professor Margaret Burgess, NCIRS, began proceedings with a presentation on clinical features of primary VZV disease including varicella (chickenpox), and neonatal and congenital varicella. As she highlighted, varicella is usually a relatively mild disease of childhood, however, complications (such as pneumonia, secondary bacterial infections and neurologic conditions) occur in approximately 1% of cases, especially those most at risk such as neonates, the immunosuppressed, pregnant women, adolescents, adults and those with pre-existing co-morbidities.[1,2] Professor Burgess presented the results of community-based surveys and seroprevalence studies in Australia that indicate that the majority of the burden of varicella is in childhood and adolescence with almost 90% of cases occurring before the age of 20 years and the most common age of acquisition between 5–9 years of age.[3]

Congenital and neonatal varicella are rare in Australia with the Australian Paediatric Surveillance Unit (APSU) reporting 44 cases of neonatal varicella

and seven cases of congenital varicella syndrome (CVS) between 1995 and 1997.[5] Of the cases of CVS, maternal varicella infection occurred between 8 and 26 weeks gestation with sequelae including skin scarring, severe limb, heart and nervous system defects resulting in death, and zoster in infancy.[5] Studies of varicella immunity in women of childbearing age show that 8% of women over the age of 14 are susceptible to varicella.[4,5] Overall, since primary varicella infection still occurs in Australia, the risk of CVS remains.

Epidemiology and the varicella vaccination program in Australia

Epidemiological data on the burden of varicella in Australia prior to the inclusion of varicella vaccine on the NIP was presented by Dr Kristine Macartney, NCIRS. It was estimated that prior to the availability of varicella vaccine, the annual number of cases of varicella in Australia approximated the birth cohort with approximately 240,000 cases each year.[6] Approximately 1,500 cases were hospitalised each year (with a principal diagnosis of varicella), of which 10% were infants under 12 months of age, 30% children aged 1–4 years and 43% aged over 15 years.[7] On average 7–8 varicella-related deaths are recorded in Australia each year.[6]

Varicella vaccines have been available in Australia since 2000 and were recommended, but not publicly funded, for use in all children at 18 months, in September 2003.[8] In November 2005, varicella vaccine was included on the NIP with funding provided for a single dose for all children aged 18 months and for 'catch-up' immunisation at 10–13 years, administered through the school-based programs nationally, for those with no prior history of varicella or vaccination. Reliable rates of vaccine coverage prior to the inclusion of varicella vaccine on the NIP are not available, with reported estimates from national serosurveys, the Australian Childhood Immunisation Register (ACIR) and from a household survey ranging from 13.4%–48%.[6,9,10] Dr Macartney presented preliminary data from the ACIR which, as of September 2006, indicated that less than a year into the program, vaccine uptake is climbing nationally with approximately 45.3% of two-year-olds reported as having been vaccinated. Data on varicella hospitalisations since the vaccine has been on the NIP were not available, however, a decline in varicella hospitalisations from 2003–2005 has been observed especially in the 1–4 year age group where rates (assessed using a principal diagnosis of varicella) declined from 48.9 cases per 100,000 (95% CI 46.8–51.1%) during July 1999–June 2003 to 38.2 per 100,000 (95% Cl 35.6–41.1%) during July 2003–June 2005.[11]

Modelling the impact of a varicella vaccination program in Australia

Evaluation of the impact of childhood varicella vaccination on the incidence of VZV disease has been the focus of numerous studies internationally. Ms Heather Gidding, Centre for Infectious Diseases and Microbiology, presented information from a study that modelled the impact of an immunisation program in Australia, using similar assumptions to studies performed in Canada and the United Kingdom. Australian-based data, including that from national serological surveys, were used in the model to determine changes in the incidence and morbidity of varicella and HZ following universal varicella vaccination in the second year of life.[12] The model suggested that varicella vaccination resulted in a significant decrease in varicella associated-morbidity, especially once infant vaccination coverage is greater than 60%, albeit with a shift in morbidity to older age groups. However, total morbidity, including morbidity resulting from HZ reactivation (assuming that exposure to varicella boosts immunity to HZ for 20 years) increases in the first 8–52 years of the program (at 90% coverage in early childhood), after which there is a rapid decline in morbidity. Vaccination of adults may be required in such a scenario.

Varicella vaccination program in the United States of America

Professor Anne Gershon, Department of Pediatrics at Columbia University Medical Center, USA, has played a pivotal role in varicella vaccine research and development. In her first presentation at this workshop, Dr Gershon summarised the available data on the impact of the 10-year one-dose varicella vaccination program in the USA where over 50 million doses have been distributed since 1995. Overall vaccine safety has been excellent, with vaccine-virus transmission and cases of post-vaccination HZ being rare occurrences in healthy vaccinees. Disease surveillance has been undertaken at sentinel sites in the USA, which have reported a decline in the incidence of varicella of at least 84% from 1995 to 2000.[13] Hospitalisations and ambulatory visits have declined by 88%[14] and deaths from varicella declined by 66% across the USA between 1990 and 2001.[15] Vaccine effectiveness studies in the USA estimate that one dose of varicella vaccine in children is approximately 80%–85% protective against disease.

In the USA, investigation into factors associated with outbreaks in highly vaccinated populations have found that the waning of immunity may only partially explain this. In early studies of the Oka/Merck varicella vaccine, the presence of any detectable antibody by gpELISA test was used to determine seroconversion resulting in a high seroconversion

rate and a 4% primary vaccine failure rate. Evidence suggests that the gpELISA cut-off of 5 units is a better correlate with protection from varicella than any detectable antibody (reported as seroconversion). However, a more accurate surrogate marker of protection may be found with the fluorescent-antibody-to-membrane-antigen test (FAMA), with less than 2% of persons with a FAMA greater than 1:4 developing modified illness, known as 'breakthrough varicella', in a household study. Using FAMA, seroconversion after one dose of varicella vaccine may be as low as 76%–88%.[16] Additionally, some studies suggest that cases of breakthrough varicella are increasing over time, suggesting secondary vaccine failure (waning immunity). Both primary and secondary vaccine failure are likely to be overcome with the use of two doses of vaccine. A 10 year study comparing children who received one versus two doses of vaccine found that breakthrough varicella was 3.3-fold lower in children after two doses than after one dose of varicella vaccine (2.2% vs. 7.3%) ($P < 0.001$).[17] The results of these studies has led to the adoption of a recommendation for two doses of varicella vaccine in children the USA.[18]

In her presentation, Dr Gershon also summarised advances in understanding the role of the skin in the basic mechanisms of VZV infection, latency and immunity and how this may underpin changes in the approach to disease control. She demonstrated that VZV transmission to a susceptible host is dependent on the presence of enveloped cell-free virions in skin vesicles where mannose-6-phosphate receptors are absent and that VZV latency is established by these cell-free virions infecting sensory nerve endings in the epidermis. Studies have shown that vaccine virus transmission is associated with the appearance of skin lesions post-vaccination[19] and that HZ in leukaemic vaccinees is associated with post-vaccination rash.[20]

Measles-mumps-rubella-varicella vaccine development

Dr Barbara Kuter, Merck & Co. Inc, outlined the clinical development of Varivax® (varicella vaccine) and the subsequent development of the combination measles-mumps-rubella-varicella vaccine, ProQuad®. The development of ProQuad® has taken over 20 years with initial formulations limited by suboptimal immunogenicity to the varicella component compared with the monovalent varicella vaccine. Re-formulation of the vaccine, with increased VZV titre, has overcome this issue. In a total of five clinical trials of MMRV, 5,833 healthy children aged 12–23 months and 399 healthy children aged 4–6 years received one or two doses of ProQuad® with concomitant administration of MMR and monovalent varicella vaccine used as controls for most studies.[21] Both one and two doses of the MMRV formulation were found to be as immunogenic and well tolerated by 12–23-month-olds and 4–6-year-olds as the separate vaccines.

Dr Gershon then presented a comparison of the safety and immunogenicity of both MMRV vaccines; Pro-Quad® (Merck & Co. Inc. West Point, Pennsylvania, USA) and Priorix-Tetra® (GlaxoSmithKline Biologicals, Rixensart, Belgium). Both vaccines have an excellent safety profile and are highly immunogenic when compared to the MMR and varicella vaccines given at separate injections sites. As a result of suboptimal response rates to the varicella component, both products have higher titres of vaccine-strain VZV and both products result in similar rates of seroconversion, but higher geometric mean titres to varicella than the monovalent varicella vaccines. Vaccine efficacy has not been studied in clinical trials and licensure of both products is based on non-inferiority compared with existing component vaccines. Both MMRV vaccines are under consideration for licensure in Australia, and it is expected that application for funding of MMRV vaccine/s under the NIP will proceed.

Varicella vaccine scheduling

The first day of the workshop concluded with a presentation by Professor Terry Nolan, chair of the Australian Technical Advisory Group on Immunisation on issues around the funding and scheduling of vaccines in Australia. He presented a framework outlining the newly adopted immunisation policy advisory structures and discussed the role of the Pharmaceutical Benefits Advisory Committee in assessing the cost-effectiveness of vaccines. His talk highlighted that future considerations around a two-dose varicella schedule and the use of MMRV on the NIP would be considered under this structure. A discussion panel of various speakers from the day answered questions from audience, chaired by Professor Terry Nolan.

Day two – herpes zoster and varicella-zoster virus disease surveillance

Clinical overview

The opening presentation of Day 2 provided an overview of the burden of disease from HZ, particularly focusing on post-herpetic neuralgia (PHN). Dr David Gronow, Sydney Pain Management Centre and the Westmead Hospital Pain Services, highlighted the difficulties faced in the management of HZ and PHN, using a particularly detailed case study of zoster in a previously independent elderly woman who became bedridden and institutionalised as a result of post-herpetic neuralgia. Dr Gronow discussed that PHN is most commonly defined as pain lasting longer than three months

post-HZ rash and can affect 25%–50% of HZ cases in persons aged over >50 years, depending on use of antiviral therapies.[22] Risk factors for PHN include older age, severity of acute pain, severe prodromal pain, and severity of rash, being female and lack of timely antiviral therapy. Other HZ complications are many, including ophthalmic disease, Ramsay-Hunt syndrome and encephalitis.

Management of HZ and PHN may be very difficult and a variety of drugs of different classes are used, often in a multimodal approach. Evidence from randomised control trials of tricyclic antidepressants, anticonvulsants, antidepressants and topical applications, such as lidocaine patches, indicate varying degrees of effectiveness. There is limited evidence for other treatment options including botulinum toxin, nerve blockers and cognitive behavioural therapy.

Epidemiology

Professor Raina MacIntyre, National Centre for Immunisation Research and Surveillance, presented available data on the epidemiology of HZ in Australia in the context of an evolving surveillance system and a universal varicella program. Results from a 1999 serosurvey, prior to the availability of varicella vaccine, found that by 30 years of age more than 97% of the Australian population had primary varicella, and as such, are at risk of developing HZ.[3] Currently, the best available data on HZ in Australia is from the Australian Institute of Health and Welfare hospital morbidity database. However, this is subject to various limitations. Analysis shows that HZ is implicated in approximately 2.5 times more hospitalisations than varicella with longer length of stay and greater case-fatality rates.[23]

Data on clinical presentations to general practitioners have been analysed to determine the burden of HZ not requiring hospitalisation. Extrapolating to the Australian population suggest similar rates from two separate sources: 477 per 100,000 per year (calculated from the Bettering the Evaluation and Care of Health (BEACH) longitudinal data collection); and 491 per 100,000 per year (from the General Practice Research Network (GPRN) cross-sectional data collection). These results are not dissimilar to international studies[24] and indicate that approximately 100,000 cases of HZ occur in Australia each year. The community burden as assessed by prescriptions for antivirals on the Restricted Pharmaceutical Benefits Scheme (RPBS) is also considerable, with 59,200 prescriptions for anti-viral medication dispensed under the RPBS in 1999, rising to 76,000 prescriptions in 2005.[23] Approximately 60% of cases of HZ in both the BEACH and GPRN databases were treated with antivirals.

Varicella-zoster virus immunopathogenesis, diagnostics in Australia, and molecular studies

Three presentations discussed the immunopathogenesis of the VZV, the current approach to diagnosis, and the molecular tools available for both clinical diagnostics and VZV surveillance. Dr Allison Abendroth, Centre for Virus Research, Westmead Millennium Institute and the Department of Infectious Diseases and Immunology, University of Sydney, presented results from her laboratory's research, which aims to better determine how the VZV interacts with the immune system, particularly dendritic cells (DC), a specialised immune cell. This cell type appears critical in the immunopathogenesis of VZV disease. VZV interferes with the maturation of DC, prevents migration and antigen presentation to CD3+ T-cells and VZV infection alters the subsets of dendritic cells found in the skin. Productive VZV infection in primary human neurons has also been shown to be resistant to apoptosis. In addition, Dr Abendroth's laboratory has also explored the immune response to human ganglion cells following reactivation causing HZ and found that a predominantly non-cytolytic immune infiltrate. These findings make a contribution to understanding the pathogenesis of this complex virus and the best directions toward improvements in prevention and treatment.

Associate Professor Alison Kesson, medical virologist and infectious disease physician at the Children's Hospital at Westmead discussed laboratory diagnosis of VZV disease. She emphasised that the various methods of laboratory diagnosis, using either antigen or antibody detection, are primarily utilised when a patient is immunosuppressed; a neonate; in those presumed immune; or for unusual clinical cases. Differential diagnoses of varicella in children include Stevens Johnson Syndrome, enterovirus infection, herpes simplex, and a number of other conditions. The traditional diagnostic test for varicella has been the Tzank smear which detects intranuclear inclusions in multinucleated cells. However this test is not sufficiently sensitive or specific. The detection of the virus from culture of vesicle fluid takes 5–14 days and also has a low sensitivity (50%). Antigen detection using immunofluorescence is a more rapid and sensitive test, and nucleic acid detection (VZV PCR) is both sensitive and specific. Detection of IgM and IgA antibody can be utilised within 1–2 days of infection. However, absence does not exclude infection; IgM is also detected in HZ, and cross-reaction with HSV can occur.

Professor Judy Breuer, Centre for Infectious Disease, Barts and London School of Medicine and Dentistry discussed her work in VZV molecular diagnostics.

Molecular studies are useful for determining if vaccine virus or wild-type virus are responsible for rashes occurring after vaccination, for virus identification in the rare cases of possible disseminated disease from vaccine-virus and for identifying vaccine virus transmission. Professor Breuer presented the results of a genetic analysis comparing the Oka parent wild-type VZV (the virus originally isolated in Japan) with the attenuated Oka vaccine virus (now used in varicella vaccines), which identified 42 differences in the gene sequence. The vaccine virus is actually a mixture of viruses with only one of the vaccine viruses usually predominating in each vesicle of a vaccine-associated rash. Professor Breuer emphasised that the vaccine viruses from both the Merck and Co. and GSK varicella vaccines are indistinguishable. It was discussed that genomic analysis of VZV will become increasingly important in countries with established varicella vaccination programs as disease incidence declines. This was highlighted by an interesting case presentation in which samples from two separate episodes of HZ in the same individual were analysed and found to be caused by two genetically distinct wild-type varicella-zoster viruses.[25] Interestingly, this finding indicates that the individual had two separate primary varicella infections, a phenomenon not previously demonstrated by molecular methods.

The Shingles Prevention Study – the Veterans Zoster trial

Dr Myron Levin, University of Colorado and The Children's Hospital, USA, presented the results of the Shingles Prevention Study (SPS), a large clinical trial of the use of high titre live attenuated (Oka/Merck strain) VZV vaccine to prevent HZ in older adults (Zostavax®, Merck and Co. Inc.). The SPS involved 22 sites across the USA and included 38,500 subjects with a median age of 69 years. The occurrence of HZ or PHN in subjects was validated through a diagnostic algorithm, in which more than 93% of all cases of suspected HZ were confirmed using PCR. In addition to HZ and PHN (significant pain ≥90 days post-rash), the endpoints for the study also included a burden of illness (BOI) score, which is a sum of individual severity of illness scores of HZ cases. Vaccine efficacy was calculated as 61.1% (95% CI 51.1–69.1%) against HZ BOI, 66.5% (95% CI 47.5–79%) for PHN and 51.3% (95% CI 44.2–57.6%) for HZ incidence.[26]

Study of the persistence of zoster vaccine efficacy is still underway, however, preliminary data to 4 years post-vaccination indicate that the vaccine is most effective in the first year, with a slight but stabile decline in efficacy in the 2–4 years post-vaccination. Professor Levin also presented the results of the SPS sub-studies. The adverse events sub-study found no clinically meaningful differences in systemic adverse events between the two groups. In the vaccine group the most frequent adverse events at the injection site were erythema, pain or tenderness, swelling, and pruritus. In the USA, where the vaccine is now in use, post-marketing surveillance will be conducted to monitor adverse events. The immunology sub-study, assessing both antibody and various measures of cell mediated immunity (CMI), conducted assays at baseline and annually. The results indicate that immune response to the vaccine decreases with age, with the CMI response being 1%–2% lower for each additional year of life. This study was unable to determine a surrogate marker of protection, but further investigation is underway.

Following the morning's presentations, a panel discussion of the potential use and benefits of zoster vaccination occurred, with audience questions addressed by the speakers.

Economic modelling of zoster vaccine

Dr James Pellissier, Merck Research Laboratories described the complex economic modelling required to determine the cost-effectiveness of a zoster vaccine in the elderly. The model developed by the manufacturer included many considerations, such as rates of HZ, and PHN, complications avoided, healthcare costs and healthcare utilisation avoided, and the Quality Adjusted Life Years (QALYs) gained by use of this vaccine. Using this model, applied to the USA healthcare system (payer perspective), the cost of the zoster vaccine is $19,831 per QALY for all persons aged 60 years or older. This was compared to other preventative measures such as the influenza vaccine for the 50–64 year age group ($16,500 per QALY gained) and colon-cancer screening ($10,000–25,000 per QALY gained). The model was the most sensitive to vaccine price, age of vaccine recipient, the costs associated with PHN, duration of vaccine efficacy, QALY measurements associated with pain states, and the costs of complications. The model needs to be applied to an Australian perspective.

Varicella-zoster virus surveillance

The afternoon of Day 2 of the conference was dedicated to a discussion of surveillance mechanisms for varicella and HZ, both locally and internationally.

British Paediatric Surveillance Unit study

Professor Breuer presented data on the British Paediatric Surveillance Unit (BPSU) study of neonatal and congenital varicella and severe varicella requiring hospitalisation in children. Surveillance over 12 months in 2002–2003 identified 112 confirmed cases of hospitalised varicella in children aged less than 16 years at a rate of 0.82 per 100,000 per year, similar to the German Paediatric Surveillance Unit

figures. Most varicella cases hospitalised had complications of bacteraemia, pneumonia, encephalitis and ataxia with no clear high risk categories. The surveillance method has been modelled in a new VZV study adopted by the APSU, commencing in 2006.

Surveillance of zoster in the United States of America

Surveillance of both varicella and HZ in the USA was described by Professor Gershon. Active surveillance of varicella in the USA has been conducted by the Centers for Disease Control and Prevention in sentinel sites in the USA. Surveillance of HZ has been more challenging, but is important to determine if an increase in cases is occurring as VZV circulation declines. The results of studies of HZ incidence vary depending on the population and study methods with estimates ranging from 1 case per 1,000 person-years in adult varicella vaccinees and 2–4 in unvaccinated adults[27] to 14 per 1,000 person-years in adults aged greater than 75 years[28] and as high as 163 cases per 1,000 person-years in children with HIV.[29] Studies in the USA, including those in active surveillance sites, report conflicting rates of zoster prior to and since the commencement of the varicella vaccination program.[30]

Surveillance plans for Australia

The surveillance to be undertaken in Australia was described by Dr Paul Roche, Surveillance Branch, Office of Health Protection, Australian Government Department of Health and Ageing. The potential goals of VZV surveillance are to assess the impact of the varicella vaccination program, monitor changes in epidemiology, measure vaccine effectiveness, monitor trends in neonatal and congenital varicella and trends in hospitalisations and to measure population immunity. The proposed Australian surveillance methods include notification of cases of varicella and HZ to the National Notifiable Disease Surveillance System (NNDSS), surveillance of severe complications in children via APSU, national serosurveys undertaken by NCIRS, and continued assessment of hospitalisations. The APSU recommenced surveillance of CVS, neonatal varicella and varicella complications requiring hospitalisation in children aged 1 month to 15 years in May 2006.[31] Disease surveillance data would be complemented by information on adverse events following immunisation as reported to the Therapeutic Goods Administration, and vaccine coverage data.

The proposed NNDSS system will have three disease categories: chickenpox, zoster and varicella infection (unspecified). Confirmed cases are to require laboratory confirmation and clinical evidence, or an epidemiological link to a laboratory confirmed case, whereas probable cases will require clinical evidence only. Varicella (unspecified) will be reported for laboratory evidence of VZV without clinical correlation. Funding from the Commonwealth has been allocated to states and territories to establish VZV surveillance systems and approaches by each State and Territory differ. Five jurisdictions will be notifying VZV using passive notification from General Practitioners and laboratories (Australian Capital Territory, Northern Territory, Queensland, South Australia and Tasmania), and two jurisdictions will collect sentinel surveillance data in addition to passive notification data (Victoria and Western Australia). New South Wales will report VZV through use of Emergency Department syndromic surveillance data.

Data quality and the usefulness of data collections is affected by issues such as the delay in the implementation of surveillance well into the universal vaccination program and the diversity of populations in Australia. The under-estimation of vaccine effect due to incomplete reporting, and a variety of data sources across the states and territories may make the development of a national picture challenging.

Surveillance in South Australia

In anticipation of the widespread use of varicella vaccine, the state of South Australia implemented a notification system for both varicella and HZ in 2002.[32] Dr Rod Givney, South Australian Department of Health, presented data on the program indicating that a centralised collection of dual notifications from both medical practitioners and laboratories should provide the ability to track changes in childhood varicella, varicella cases in adolescents and adults, and any change in the age distribution of HZ since the implementation of a universal program in Australia. Data collection is proceeding, with notifications representing an estimated 4% of actual cases occurring for both varicella and HZ.[33]

Discussion panel 3 – jurisdictional surveillance and recommendations

The workshop concluded with representatives from all Australian jurisdictions participating in a discussion panel of the benefits of the proposed surveillance mechanisms, and future directions.

Presentations from both days of the workshop are available on the NCIRS website: http://www.ncirs.usyd.edu.au/newsevents/vzv_workshop_presentations_nov_06.doc

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Exercise Paton: A simulation exercise to test New South Wales emergency departments' response to pandemic influenza

Adam T Craig, Paul K Armstrong

Exercise Paton was a New South Wales-wide simulation exercise conducted on 30 November 2006, to test the response of New South Wales emergency departments (EDs), multi-purpose services* (MPSs), and public health units to the presentation of single cases of pandemic influenza during the early stages of a pandemic. The exercise followed the release of the New South Wales policy document to guide New South Wales hospitals' response to an influenza pandemic, titled *Hospital Response to Pandemic Influenza, Part 1: Emergency Department Response*.[1] The exercise was named after Dr Robert Paton, the New South Wales Director-General of Public Health during the 'Spanish influenza' pandemic of 1918–1919.

This report summarises the planned approach for clinical assessment of suspected pandemic influenza cases in New South Wales, describes activities during *Exercise Paton,* and lists key lessons to emerge from the exercise that could be of relevance to pandemic planners in other jurisdictions.

Planned approach for clinical assessment of suspected pandemic influenza cases in New South Wales

In keeping with the over-arching national response strategies for pandemic influenza,[2] New South Wales has incorporated the concepts of 'containment' and 'maintenance of social function' into state pandemic planning. In the containment stage, the emphasis is on slowing the spread of a pandemic to lessen the burden on the health system and to 'buy time' for the development of a pandemic vaccine. Containment measures include preventing cases from entering Australia, rapidly finding, isolating, and treating cases with antiviral medication, and tracing contacts

* Multi-purpose services provide acute, high and low level health and aged care services to rural and remote communities in New South Wales. While some multi-purpose services operate permanent emergency departments most respond to acute care needs as required.

of cases, quarantining them and providing them with antiviral prophylaxis. When containment measures are no longer effective and the disease begins to spread widely in the community, maintenance of social function becomes the priority.

In New South Wales, assessment and management of suspected cases will be coordinated through the public hospital system. General practitioners (GPs) will be encouraged to divert patients with potential pandemic influenza to the closest public hospital with an ED or MPS. This will be aided by a public messaging campaign. The rationale for this approach is to: (a) lessen the burden on GPs and their staff so that they can continue core primary care activities; (b) mitigate the risk of transmission of the pandemic virus within GP surgeries; (c) facilitate a timely public health response by integrated data collection and reporting systems throughout the public health system; (d) facilitate rapid transport and processing of laboratory specimens in public health laboratories; and (e) allow the secure dispensing of antiviral medications from the national medical stockpile to cases and contacts. Although case management will not be focussed in GP surgeries, GPs will play a key role in bolstering the public hospital workforce during the response to a pandemic, especially in the later stages when case numbers increase.

The method for assessment of suspected pandemic influenza cases at a hospital or MPS will vary according to the likelihood of a true case seeking care at that facility. In the very early stages of a pandemic, when the likelihood of someone presenting is small (e.g. when clusters of pandemic influenza are being reported overseas but not yet in Australia), 'enhanced ED triage' will be activated. This consists of screening every attendee of the ED or MPS based on their travel history and symptomatology. When the likelihood of pandemic influenza cases presenting increases, 'ED screening stations' will be established outside the waiting area to minimise the chance of transmission to other people in that waiting area. Once the number of cases exceeds the capacity of an ED to manage them, a stand-alone influenza clinic will be established. The influenza clinics will assess suspect pandemic influenza cases that are not in need of high-level ED care; EDs will continue to assess those cases who require a high level of care. The Table summarises the levels of ED and MPS responses to pandemic influenza and the drivers that determine an increase in the level of response.

Exercise Paton

Exercise Paton was conducted to test the ability to implement the 'enhanced ED triage' level of response to pandemic influenza, as described in the NSW Health *Hospital Response to Pandemic Influenza, Part 1: Emergency Department Response* guidelines. The exercise objectives were to ensure all EDs and MPSs in New South Wales were able to activate a prescribed screening process for pandemic influenza; to identify barriers to effective early containment of pandemic influenza; to test the interrelationship between ED/MPSs and public health activities; to evaluate the preparedness of EDs, MPSs and public health units to respond to an initial case of pandemic influenza; and to progress facility-based planning. *Exercise Paton* was not designed to test surge capacity of the health sector during an influenza pandemic or to test the interface between the health sector and other government and non-government sectors.

During *Exercise Paton*, all EDs and MPSs in New South Wales were required to prepare for, and activate, 'enhanced ED triage'. This entailed identifying and isolating patients; taking respiratory samples from, and treating suspected cases of pandemic influenza, and liasing with the public health unit to ensure contact tracing was undertaken. The quality of respiratory specimens (nose and throat swabs) collected and the time taken for transportation of these specimens from facilities to a specialist testing laboratory using conventional transport methods was also tested.

While all 210 EDs and MPSs in New South Wales were required to be prepared to receive a patient during *Exercise Paton*, mock patients were only deployed to 18 (8.6%) facilities. The sites to be tested were not revealed before the exercise. Half of the mock patients had a simulated symptom/clinical observation profile that would ordinarily result in admission to hospital, while the remaining half had a profile that would ordinarily result in being discharged home.

Performance against the exercise's objectives was assessed using observers at each of the tested sites, feedback from stakeholder debriefing sessions, and data collected from a state-wide participant questionnaire.

Lessons of relevance to other jurisdictions' biopreparedness planning

While many of the outcomes of *Exercise Paton* relate specifically to New South Wales facilities and processes, many of the lessons learnt may be of relevance to other jurisdictions. These are summarised below.

Method of exercising

The method used to test EDs and MPSs during *Exercise Paton* proved to be a valuable way to achieve the exercise's outcomes, in particular in progressing

Description, drivers for activation, and purpose of emergency department response to an influenza pandemic in New South Wales

Response	Description	Drivers for activation	Purpose
Enhanced ED triage initiated	Additional screening conducted at the usual ED triage point, based on travel history and symptomatology.	Declaration of World Health Organization overseas pandemic alert phase 4* (OS phase 4)— clusters with human-to-human transmission overseas— where the clusters are occurring in a relatively isolated region (if first clusters are in a major centre overseas, a move directly to pandemic influenza screening stations may be required.)	**Containment stage** To decrease the rate of transmission of pandemic influenza in the community, general practice surgeries, hospitals EDs, wards, and other health care facilities by: • ensuring rapid identification and isolation of suspected cases • allowing diagnosis and treatment of cases with antiviral agents, if indicated • providing a linkage with the public health response of contact tracing and provision of antiviral prophylaxis • allowing collection of epidemiological and clinical data to inform clinical management and public health decisions.
ED pandemic influenza screening station established	Pandemic influenza screening station established at the entrance to ED to identify patients who meet the pandemic influenza case definition before they enter the waiting room.	No cases in Australia (Australian pandemic alert phase 0–3) but outbreaks occurring in areas overseas from where it is likely that people will be travelling to Australia. Widespread outbreaks overseas. Significant morbidity and mortality from pandemic influenza overseas. Declaration of Australian pandemic alert phase 4 (i.e. clusters with human-to-human transmission in Australia).	**Containment stage** As for enhanced ED triage, and to allow a higher level of vigilance than provided by enhanced ED triage in light of an increased likelihood of pandemic influenza cases being encountered.
Stand-alone influenza clinic established	A separate influenza clinic facility established to identify and treat those who meet the case definition for pandemic influenza. Note: an influenza screening station at the entrance to ED will still need to be maintained.	**At containment stage** ED capacity to isolate and manage suspected cases is exceeded. **At 'maintenance of social function' stage** Inability to contain pandemic influenza outbreaks (resulting in declaration of 'maintenance of social function' stage). Declaration of influenza pandemic (Australian phase 6b).	**Containment stage** As for enhanced ED triage, and to allow effective management of an increased number of pandemic influenza patients. **'Maintenance of social function' stage** To provide standardised assessment, triage, and management of patients with suspected pandemic influenza. To reduce patient presentations to EDs and general practices, thereby allowing those facilities to continue their core business and reduce the risk of transmission within those settings. To collect epidemiological data to monitor progress of the pandemic and inform optimal resource allocation.

* This assumes that a pandemic starts overseas. If a pandemic starts in Australia, an elevated level of response will be immediately required.

facility-based pandemic planning. In a post-exercise questionnaire, 84% of facilities 'strongly agreed' or 'agreed' that *Exercise Paton* prompted their influenza pandemic planning. The strategy of involving all facilities across the state, not just those that received mock patients, maximised the impact of the exercise.

Infection control

Significant breaches in infection control practice were noted during *Exercise Paton* including incorrect use of personal protective equipment, poor hand hygiene and, in some cases, inappropriate packaging of specimens for transport. Compared with medical staff, nursing staff were generally

more aware of, and likely to comply with, infection control policies and procedures. A continued emphasis on the importance of infection control in preventing hospital acquired infections needs to be taken, which should include ongoing training and monitoring of infection control practice.

Decision making relating to patient disposition

The decision to either admit an ED patient to a ward or discharge them is normally based upon whether or not that patient is ill enough to require hospital care. When a patient poses a serious infectious risk to others, however, admission of a relatively well person into hospital isolation is also a clinically acceptable decision. Such a situation could arise in the early stages of a pandemic when the behaviour of the virus may not be well understood, and there is a risk of spreading pandemic influenza in the community from non-compliant cases in home quarantine.

Since *Exercise Paton,* New South Wales pandemic influenza ED response guidelines have been revised to include a stipulation that any decision to discharge a potentially infectious patient must be made in consultation with the public health unit and relevant medical specialists. In the very early stages of a pandemic, all patients suspected of having the illness will be admitted into isolation wards until the infectious period is over or an alternative diagnosis is made.

Communication and information management

Exercise Paton highlighted the importance of having multiple communication channels for an effective response. An in-house, password-protected Internet website was established during the exercise that enabled players to access all relevant documents. This proved an effective incident management tool and NSW Health intends building a more sophisticated system to help manage future real and simulated public health emergencies.

Another web-based data management system used during *Exercise Paton* was NetEpi, an open-source outbreak management software being developed by NSW Health and adopted (on an interim basis) nationally. The version used was much improved compared to previous versions and its use during the exercise will help inform development of the final version, due for release in September 2007.

Collection and transportation of clinical specimens

During the early stage of a pandemic, the pre-test probability of a person presenting with influenza-like symptoms having pandemic influenza will not be high. An important priority therefore, will be to obtain a laboratory diagnosis as rapidly as possible. The steps to ensure this occurs are first, to obtain an adequate specimen, second, to suitably package, process and transport the specimen, third, use a reliable, rapid test method, and finally, relay the result to the clinicians. *Exercise Paton* tested the first two of these steps.

Nineteen nose and throat swab specimens were collected during the exercise and the quality of all of them was deemed adequate by the laboratory. The quality of packaging was poor overall and clearer packaging guidelines will be developed. Using conventional specimen transportation methods, transit times for specimens ranged from 30 minutes to 4 hours in metropolitan areas, and 20 to 28 hours in rural areas. These transit times would have been longer if specimens had been taken outside of business hours. Given the urgency of confirming a clinical diagnosis during the early stages of a pandemic, strategies to expedite specimen transport from some areas to diagnostic laboratories need to be developed.

A comprehensive report, titled *Exercise Paton Evaluation Report,*[3] provides a more detailed evaluation of the activities and outcomes of the exercise and is available on the NSW Health website, www.health.nsw.gov.au In 2007–2008, NSW Health is planning to conduct further exercises to test containment policies and strategies.

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Quarterly reports

OzFoodNet quarterly report, 1 April to 30 June 2007

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 investigation of outbreaks of gastrointestinal illness and clusters of disease potentially related to food occurring in Australia from 1 April to 30 June 2007.

Data were received from OzFoodNet representatives in all Australian states and territories and a sentinel site in the Hunter/New England region of New South Wales. The data in this report are provisional and subject to change as the results of outbreak investigations can take months to finalise.

During the second quarter of 2007, OzFoodNet sites reported 334 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 significantly under-represent the true burden of these infections. In total, these outbreaks affected 6,664 people, of which 183 were hospitalised and 20 people died. The majority (72%, n=239) of outbreaks resulted from infections suspected to involve person-to-person transmission (Figure).

Foodborne disease outbreaks

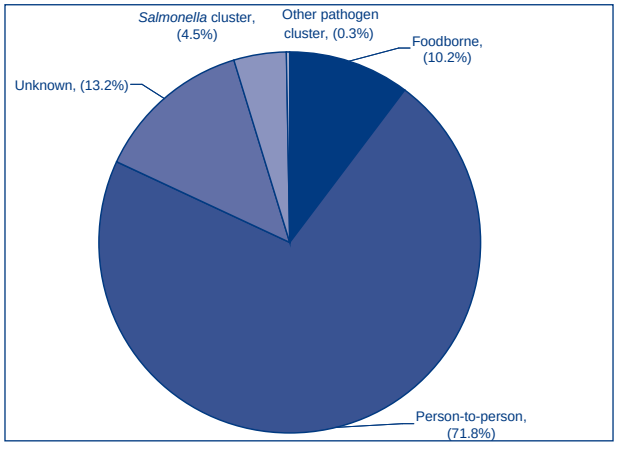
There were 34 outbreaks during the second quarter of 2007 where consumption of contaminated food was suspected or confirmed as the primary mode of transmission (Table). These outbreaks affected 360 people and resulted in 29 people being admitted to hospital. There were five deaths. This compares with 22 outbreaks for the second quarter of 2006 and 40 outbreaks in the previous quarter of 2007.

Salmonella was responsible for 10 outbreaks during the quarter, with *Salmonella* Typhimurium being the most common serotype. *S.* Typhimurium 9 was responsible for four outbreaks, while *S.* Typhimurium 44 and *S.* Typhimurium 135a were each responsible for two outbreaks. The other *Salmonella* serotypes causing outbreaks were *S.* Virchow 8 and *S.* Enteritidis 6A (a mixed infection with *Campylobacter* and rotavirus).

Norovirus was associated with one foodborne outbreak of illness during the quarter. There were five toxin-related outbreaks during the quarter including histamine poisoning (3 outbreaks) and ciguatera fish poisoning (2 outbreaks). The remaining 18 outbreaks were caused by unknown aetiological agents.

Fourteen outbreaks reported in the quarter were associated with food prepared by restaurants, four from food prepared in private residences, three from food prepared by takeaway outlets, and two outbreaks each from contaminated primary produce, commercial caterers, aged care facilities and a bakery. Single outbreaks were associated with food prepared by a hospital and a commercially manufactured food. There were three outbreaks where the food preparation setting was unknown as multiple foods from different sources could have caused the outbreak.

Mode of transmission for outbreaks of gastrointestinal illness reported by OzFoodNet sites, 1 April to 30 June 2007



Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 April to 30 June 2007

State	Month of outbreak	Setting prepared	Infection	Number affected	Evidence	Responsible vehicles
ACT	April	Restaurant	Unknown	29	A	Unknown
	May	Restaurant	Unknown	8	D	Unknown
NSW	April	Unknown	Unknown	9	A	Fruit, meringue and custard tart
		Takeaway	Unknown	5	D	hot dogs
		Restaurant	Unknown	7	D	Fried rice suspected
	May	Restaurant	Unknown	4	D	Unknown
		Takeaway	Unknown	6	D	Fresh fruit juices suspected
		Restaurant	*Salmonella* Typhimurium 9	12	M	Fried ice cream
		Restaurant	Unknown	6	D	Unknown
		Restaurant	Unknown	14	D	Mixed vegies, chicken, beef
	June	Takeaway	Unknown	2	D	Unknown
		Restaurant	Unknown	2	D	Unknown
NT	June	Unknown	*Salmonella* Enteritidis 6A/ *Campylobacter* species/rotavirus	11	D	Unknown
		Commercial manufactured food	Suspected histamine poisoning	2	D	Tinned tuna
Qld	April	Unknown	Unknown	21	D	Unknown
	May	Restaurant	*Salmonella* Virchow 8	15	D	Unknown
		Restaurant	*Salmonella* Typhimurium 135a	6	D	Unknown
		Private residence	Unknown	7	D	Wurst
		Contaminated primary produce	Ciguatera fish poisoning	3	D	Coral trout
		Contaminated primary produce	Ciguatera fish poisoning	2	D	Mackerel
		Bakery	*Salmonella* Typhimurium 135a	7	M	Cheesecake
	June	Private residence	Histamine poisoning	4	M	Tuna kebabs
SA	April	Commercial caterer	Unknown	12	A	Sushi
Vic	April	Restaurant	Histamine poisoning	2	D	Mahi Mahi fish
		Commercial caterer	Unknown	25	A	Suspected penne pasta salad
		Aged care	*Salmonella* Typhimurium 44	22	A	Unknown
	May	Restaurant	Unknown	9	D	Lasagne
		Private residence	*Salmonella* Typhimurium 9	3	D	Unknown
		Private residence	*Salmonella* Typhimurium 9	8	M	Chocolate mousse
		Hospital	*Salmonella* Typhimurium 9	4	D	Unknown
		Aged care	Unknown	17	D	Unknown
	June	Restaurant	*Salmonella* Typhimurium 9	5	D	Chicken massaman curry suspected
		Bakery	*Salmonella* Typhimurium 44	45	M	Pork rolls
WA	June	Restaurant	Norovirus	26	A	Unknown

* No foodborne outbreaks were reported in 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.

To investigate these outbreaks, sites conducted nine cohort studies and one case control study, and collected case series data on 23 outbreaks. There was one outbreak where no individual patient data was collected. Investigators obtained analytical epidemiological evidence in six outbreaks and microbiological evidence in five outbreaks. For the remaining 23 outbreaks, investigators obtained descriptive epidemiological evidence implicating the food vehicle or suggesting foodborne transmission.

Victoria reported 10 outbreaks of foodborne illness during the quarter, including three outbreaks of *S.* Typhimurium 9. The first outbreak involved 23% (8/35) of participants becoming ill after consuming various foods at a party, including a chocolate mousse containing raw eggs. One of the cases was confirmed to have an *S.* Typhimurium 9 infection and a leftover sample of the mousse was also positive for *S.* Typhimurium 9.

The second outbreak of *S.* Typhimurium 9 occurred in a group of seven people who attended a dinner party together where chocolate mousse containing raw eggs was served. Two dinner attendees were confirmed to have an *S.* Typhimurium 9 infection. A third case of *S.* Typhimurium 9 occurred in a person who did not attend the dinner but ate the chocolate mousse.

The third outbreak was detected during an investigation of clustering of cases of *S.* Typhimurium 9 where three inpatients of the same hospital were identified. An additional case in a food handler at the same hospital was also reported although no food source was identified. In addition, an outbreak caused illness in 36% (5/14) of attendees at a dinner party where foods were purchased from two takeaway stores. Eighty-three per cent (5/6) of people who ate a chicken curry became ill while none of the eight who didn't eat curry became ill. One case was confirmed to have an *S.* Typhimurium 9 infection and the other cases had symptoms consistent with salmonellosis.

Fifty-three guests attended a catered lunch party in Victoria and then 25 guests stayed for dinner, which consisted of leftovers from the lunch meal. A penne pasta salad was significantly associated with illness (RR 2.75 95% CI 1-7.53) with a food specific attack rate of 69%, which accounted for 88% (22/25) of the cases. Illness was consistent with a viral pathogen, with 80% and 76% of cases reporting vomiting and diarrhoea, respectively. The median incubation period was 1–2 days and median duration of illness was two days. Seven of the 25 ill guests only ate lunch. None of the food handlers reported illness and none of the attendees interviewed reported illness during the function. As the food was served in a buffet style it is possible that an asymptomatic food handler or attendee contaminated food.

Victoria reported that 45 people became ill, including eight who required hospital treatment, after eating pork rolls from a bakery. Samples of a leftover roll from a case's home and pate sampled from the premises were positive for *S.* Typhimurium 44. A raw egg mayonnaise was used as an ingredient in all rolls. Five cases ate rolls with the pate and egg butter that did not include any other meat products.

A Victorian aged care facility reported diarrhoea in 17 residents, with a clinical picture that was consistent with *Clostridium perfringens* enterotoxin. Fifteen of the 17 ill residents had consumed vitamised meals but no specific food was identified as the vehicle. Victoria also reported a small outbreak of histamine poisoning that affected two people after they ate a reheated meal of Mahi Mahi fish. Nine people, who were from four separate groups, reported diarrhoea and abdominal pains between 8 and 15 hours after dining at a Victorian restaurant. Each group ate lasagne purchased from the restaurant on the same night.

Queensland reported eight outbreaks of foodborne disease during the quarter. *S.* Virchow 8 caused illness among 15 participants of a four-day conference at a resort. OzFoodNet conducted a multi-state cohort study, which indicated that two food items consumed on the first night of the workshop were associated with illness – vegetables, and a tossed green salad. The local public health unit conducted an environmental inspection and collected food specimens for testing. Eggs collected from the resort were positive for *S.* Agona, *S.* Cerro, *S.* Ohio and *S.* Isangi. *S.* Cerro was also isolated from a chopping board used to cut red meat. The eggs appeared soiled with faecal matter and feather material (one of the eggs was cracked open). The source of infection was not identified, although there was some evidence that cross-contamination and/or poor food handling practices may have contributed to this outbreak.

Queensland investigated a cluster of *S.* Typhimurium 135a cases that had the same multiple-locus variable-number tandem-repeats analysis (MLVA) profile (1-3-6-13-3). Five of these cases plus another epidemiologically-linked case reported consuming meals from the same Brisbane restaurant over a period of six days, during May. An environmental health investigation identified multiple food hygiene breaches and that the facility was not licensed. The restaurant ceased operating until food safety issues were addressed and a license granted.

A cluster of 19 *S.* Typhimurium 135a cases with the same MLVA profile (1-4-5-13-3) were identified in the Gold Coast region. Of these, seven cases had consumed foods from the same bakery prior to infection. *S.* Typhimurium 135a, with the same MLVA profile (1-4-5-13-3), was detected in a sample of cheesecake taken from the premises. Environmental investigations further identified that the cheesecake was prepared using raw egg and was undercooked. No dirty or cracked eggs were identified on the premises at the time of inspection. Cheesecakes were subsequently removed from sale and destroyed. The bakery was instructed to use pasteurised eggs in place of raw eggs in foods that undergo minimal cooking.

Seven people from two Queensland families reported nausea, vomiting, fever, cramps and diarrhoea. All were ill after consuming garlic flavoured Wurst purchased from a local market and had no other foods or drinks in common.

Queensland investigated 21 cases of gastrointestinal illness of unknown aetiology among a group attending a conference. Based on the cases' clinical histories, a toxin was suspected to have caused this outbreak. An environmental inspection of the kitchen did not identify any food safety issues and no staff members reported illness.

Queensland reported three fish related toxin outbreaks during the quarter. Histamine poisoning affected four people after a meal of tuna kebabs, where 3,600 mg of histamine per kg was detected in a sample of the tuna.

Ciguatera fish poisoning was reported among three people who consumed coral trout at a restaurant. The fish was caught off Cairns. One case required hospital treatment for their illness.

Ciguatera fish poisoning was also reported among two people who had consumed mackerel purchased from a seafood outlet in Hervey Bay. A traceback identified that the fish was caught in Platypus Bay between Hervey Bay and Fraser Island, South East Queensland.

New South Wales reported 10 outbreaks of foodborne illness during the quarter. Twelve people were affected by *S.* Typhimurium 9 after a restaurant dessert of fried ice-cream. *S.* Typhimurium 9 was detected in the pre-prepared ice-cream. An aetiological agent was not identified for the remaining nine outbreaks. These outbreaks affected between two and 14 people.

Six outbreaks were associated with New South Wales restaurants, three with takeaway outlets and one setting was unable to be identified. Foods associated with illness included, commercially prepared tart, hot dogs, fried rice, and fresh fruit juices.

The Australian Capital Territory reported two outbreaks during the quarter including an outbreak of unknown aetiology that affected 29 restaurant patrons over several days. The cause of illness was unable to be identified in an investigation of eight people attending a planning day at a hotel. Many cases reported diarrhoea and a short incubation period of between two and six hours.

The Northern Territory reported two outbreaks during the quarter including one due to mixed infections of *S.* Enteritidis 6A, *Campylobacter* species, and rotavirus. The investigation commenced in June after notification of two cases of salmonellosis on board a ship returning from South East Asia. The source of the outbreak was not determined but may have involved transmission from infected persons after one or two cases acquired illness in Asia.

The Northern Territory reported two cases of suspected histamine poisoning in June. Cases worked at the same place, but had eaten two different brands of tinned tuna 10 days apart.

South Australia reported 12 cases of unknown aetiology in people attending a seminar during April. Lunch was provided by a caterer and included sandwiches, sushi and drinks. A cohort study showed that sushi was associated with illness after all cases reported consumption. Separate types of sushi were significantly associated with illness including cooked tuna sushi (RR 1.9, 95%CI1.0-3.8) and beef sushi (RR 3.4, 95%CI 1.6-7.1).

Western Australia reported one foodborne outbreak during the quarter. A company reported that a higher than expected number of staff were ill with gastroenteritis in early June. A case-control study showed that illness was strongly associated with eating at the staff cafe (OR 38, 95%CI 4.3-855.6). No association with a particular food was found. Faecal specimens from three affected employees were positive for norovirus.

Tasmania did not report any foodborne outbreaks during the second quarter of 2007.

Comments

In Australia, cases of *Salmonella* infection are more common in summer and autumn. However, the number of *Salmonella* infections were significantly increased during the quarter in several Australian states and territories, with the exception of Queensland, the Australian Capital Territory and Western Australia. In the first six months of 2007, there were 5,856 cases of *Salmonella* infection across Australia compared with the average for the previous five years of 4,704, an increase of 24%.

Some of the increase was probably due to several outbreaks in different jurisdictions that each affected large numbers of people. In the last six months, OzFoodNet recorded approximately 20 outbreaks which were suspected to be due to contaminated eggs. One outbreak of *Salmonella* infection affecting approximately 300 people in New South Wales was the largest outbreak in this State in several years. This outbreak was linked with pork and chicken rolls containing raw egg mayonnaise from a Vietnamese bakery. In response to these outbreaks, several health departments, including those in New South Wales and Victoria, issued media reports advising consumers to follow food safety practices for proper food storage and handling, and ensuring foods are fully cooked to prevent foodborne illnesses such as *Salmonella*.

Acknowledgements

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

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Communicable diseases surveillance

Highlights for 2nd quarter, 2007

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 with an onset in the 2nd quarter (1 April to 30 June) of 2007, compared with the 5-year mean for the same period.

Notifications were above the five-year mean for chlamydia, cholera, hepatitis B (unspecified), leprosy, mumps, Shiga-like toxin-producing *Escherichia coli*/verotoxin-producing *E. coli* (SLTEC/VTEC) and syphilis of less than two years duration. Notifications were below the 5-year mean for hepatitis B (incident), invasive pneumococcal disease, measles and pertussis.

Bloodborne disease

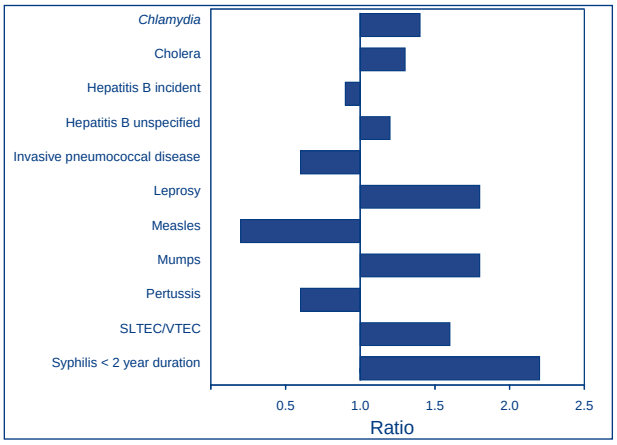
Hepatitis B unspecified

There were 1,802 cases of hepatitis B unspecified infections reported to NNDSS in the second quarter of 2007, giving a national notification rate of 35 cases per 100,000 population. The 30–34 year age group for males (75 cases per 100,000 population) and females (70 cases per 100,000 population) had the highest rate of notification.

Compared with the same period in 2006, hepatitis B (unspecified) notifications have increased by 22%. The major increases have been in New South Wales (29%), Victoria (17%) and Western Australia (46%). The Northern Territory recorded the highest notification rate with 83 cases per 100,000 population, however this was 70% less notifications compared with the same period in 2006. The increase in notifications is thought to be due to the detection of cases among refugee and humanitarian arrivals.

In contrast, hepatitis B (incident) notifications remain below the five-year mean. Rates of hepatitis B incident and hepatitis B unspecified are shown in Figure 2.

Figure 1. Selected* diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 April to 30 June 2007 with historical data*



* Selected diseases are chosen each quarter according to current activity. Five year averages and the ratios of notifications in the reporting period in the five year mean should be interpreted with caution. Changes in surveillance practice, diagnostic techniques and reporting, may contribute to increases or decreases in the total notifications received over a five 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 a list of all diseases.

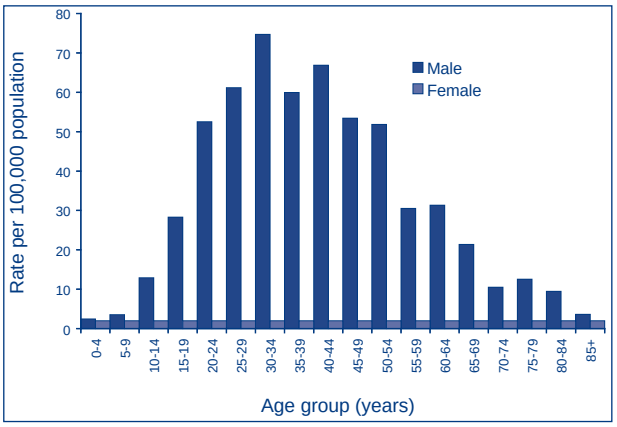
† Ratio of current quarter total to mean of corresponding quarter for the previous five years.

Vaccine preventable diseases

Measles

There were three cases of measles reported in the second quarter, one each from New South Wales, Queensland and Victoria. There were two males

Figure 2. Notification rates of incident hepatitis B and hepatitis B (unspecified), Australia, 1995 to 2007* by year†



* Annualised rate to 30 June 2007.

† Year of diagnosis for incident hepatitis B; year of notification for unspecified hepatitis B.

and one female reported with an age range between 19–30 years. Two cases had returned from an overseas trip (from Vietnam and India). One case had an unknown vaccination history and the other two were unvaccinated.

Mumps

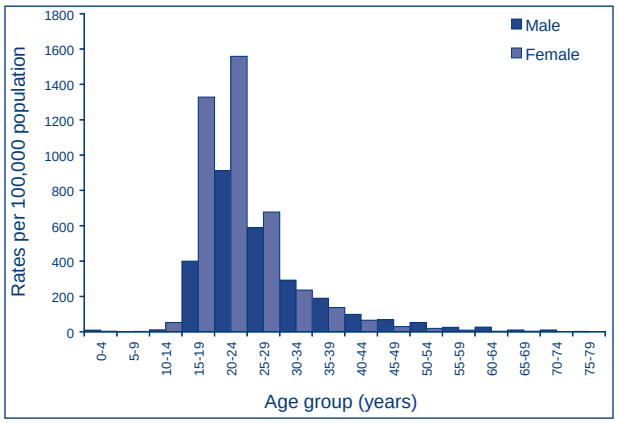
Eighty-seven notifications of mumps were notified during the quarter; this was 1.8 times the 5-year mean for the same period. The majority of notifications were from New South Wales with 53 cases (61%). The age ranged between 3 to 88 years, with the highest notification range in the 25–29 year age group.

Mumps in the 25–29 year age group probably represents a susceptible cohort of individuals who have not been immunised. Mumps vaccine was made available in Australia in 1980 for use at 12–15 months of age and was combined with the measles vaccine in 1982. Therefore, no childhood doses of mumps vaccine were available to individuals in the 25–34 year age group and uptake of vaccine in older individuals from the 15–24 year age group was likely to be poor.

Pertussis

There were 1,185 notifications of pertussis in the quarter, which was only 43% of the number of notifications in the same period in 2006 (2,727). Pertussis notifications have declined since the end of 2006 (Figure 3), largely in New South Wales in part due to changes in the cut-off for positivity in a widely used pertussis serological diagnostic test.

Figure 3. Notifications of pertussis, Australia, 1 January 2005 to 30 June 2007, by week of onset



Quarantinable diseases

Cholera

One case of cholera was notified in the second quarter of 2007. The case was a 44-year-old male from Sydney, New South Wales. The infecting organism was identified as *Vibrio cholerae* 01 El Tor, serotype Ogawa, and was acquired in India.

The average number of cholera cases notified over the last five years is 1.3 cases year-to-date (5 cases in 2002, 1 case in 2003, 5 cases in 2004, 3 cases in 2005, 3 cases in 2006 and 2 cases year-to-date 2007). Apart from the three cases in 2006, all cases since 2002 have been imported.

Cholera is one of seven human diseases subject to quarantine controls in Australia and is one of the diseases reportable to the World Health Organization.

Other bacterial infections

Leprosy

Five cases of leprosy were notified this quarter. Notifications were from New South Wales (1 case), Queensland (2 cases), South Australia (1 case) and Western Australia (1 case). There were four males and one female notified with an age range between 30–83 years. Two cases were in Indigenous people, two in non-Indigenous people and the indigenous status of the fifth case was unknown.

Acknowledgments

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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 33,300 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 April and 30 June 2007 (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 B (incident)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (incident)	All jurisdictions except Qld
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except NSW
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
SLTEC, VTEC	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Smallpox	All jurisdictions
Tularemia	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)	All jurisdictions
Syphilis <2 years duration	All jurisdictions
Syphilis >2 years or unspecified duration	All jurisdictions
Syphilis - congenital	All jurisdictions

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 infections (chickenpox)	All jurisdictions except NSW
Varicella infections (unspecified)	All jurisdictions except NSW
Varicella zoster infections	All jurisdictions except NSW
Vectorborne diseases	
Barmah Forest virus infection	All jurisdictions
Flavivirus infection (NEC)†	All jurisdictions
Dengue	All jurisdictions
Japanese encephalitis virus	All jurisdictions
Kunjin virus	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus	All jurisdictions
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssaviruses unspecified	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

* Laboratory confirmed influenza is not notifiable in South Australia but reports are forwarded to NNDSS.

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

Table 2. Notifications of diseases received by state and territory health authorities in the period 1 April to 30 June 2007, by date of onset*

Disease	State or territory								Total 2nd quarter 2007†	Total 1st quarter 2007	Total 2nd quarter 2006	Last 5 years mean 2nd quarter	Year to date 2007	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis B (incident)	2	15	5	14	0	1	18	12	67	76	83	83.6	143	165.4	0.9
Hepatitis B (unspecified)	11	788	43	276	85	5	433	161	1,802	1,991	1,488	1,518.4	3,793	3,073.0	1.2
Hepatitis C (incident)	1	9	0	NN	7	3	24	18	62	87	118	110.0	149	226.4	0.6
Hepatitis C (unspecified)	38	1,534	55	695	77	54	663	265	3,381	3,772	2,734	3,217.0	7,153	6,714.0	1.1
Hepatitis D	0	4	0	2	0	0	2	0	8	9	6	6.4	17	12.4	1.4
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	1	0	0.3	1	0.8	1.3
Campylobacteriosis§	104	NN	98	988	658	164	1,358	448	3,818	4,846	3,377	3,325.8	8,664	7,426.4	1.2
Cryptosporidiosis	2	81	36	85	81	7	149	165	606	1,122	937	625.4	1,728	1,812.8	1.0
Haemolytic uraemic syndrome	0	1	0	0	0	0	2	0	3	7	0	2.4	10	6.4	1.6
Hepatitis A	0	12	1	9	1	1	5	10	39	47	65	86.0	86	197.4	0.4
Hepatitis E	0	0	0	2	0	0	4	0	6	6	4	4.6	12	12.6	1.0
Listeriosis	0	5	0	0	0	1	2	0	8	16	7	16.2	24	34.0	0.7
Salmonellosis (NEC)	30	617	118	666	187	47	476	216	2,357	3,494	1,852	1,878.2	5,851	4,704.0	1.2
Shigellosis	0	16	39	10	13	2	25	25	130	145	136	138.8	275	314.4	0.9
SLTEC, VTEC‖	0	2	0	3	5	0	2	0	12	40	18	16.8	52	33.2	1.6
Typhoid	0	11	0	1	0	0	5	0	17	37	21	12.0	54	37.0	1.5
Quarantinable diseases															
Cholera	0	1	0	0	0	0	0	0	1	1	0	0.8	2	1.6	1.3
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
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Tularemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0

Table 2. Notifications of diseases received by state and territory health authorities in the period 1 April to 30 June 2007, by date of onset,* *continued*

Disease	State or territory								Total 2nd quarter 2007†	Total 1st quarter 2007	Total 2nd quarter 2006	Last 5 years mean 2nd quarter	Year to date 2007	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection¶	222	2,856	693	3,108	763	286	2,467	1,763	12,158	13,253	11,699	9,064.2	25,411	18,082.2	1.4
Donovanosis	0	0	0	0	0	0	0	0	0	1	3	2.8	1	7.0	0.1
Gonococcal infection	9	351	535	349	150	9	230	410	2,043	2,005	2,379	1,954.2	4,048	3,889.4	1.0
Syphilis (all)	6	311	89	96	12	10	195	48	767	726	655	220.0	1,493	478.2	3.1
Syphilis < two years duration	0	60	63	54	1	4	82	30	294	291	201	100.4	585	262.0	2.2
Syphilis >two years or unspecified duration	6	251	26	42	11	6	113	18	473	435	454	243.0	908	454.0	2.0
Syphilis - congenital	0	1	2	0	0	0	0	0	3	2	7	6.0	5	9.4	0.5
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	0	0	0	0	0	0	2	2	2	2	5.2	4	10.6	0.4
Influenza (laboratory confirmed)	6	152	7	205	10	14	33	95	522	337	397	444.6	851	624.4	1.4
Measles	0	1	0	1	0	0	1	0	3	4	96	27.8	7	40.0	0.2
Mumps	0	53	12	8	5	0	8	1	87	45	94	44.0	132	75.0	1.8
Pertussis	24	420	6	334	94	5	283	19	1,185	1,031	2,727	1,704.2	2,216	3,408.2	0.6
Pneumococcal disease (invasive)	8	109	17	69	34	3	74	30	344	190	425	539.0	534	833.8	0.6
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Rubella	1	5	0	4	1	0	3	1	15	8	17	20.4	21	42.2	0.5
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	0	0.4	0	0.8	0.0
Tetanus	0	0	0	0	0	0	0	0	0	0	0	0.6	0	2.4	0.0
Varicella infections (chickenpox)	NDP	NN	17	44	163	1	NN	40	265	350	168	NA	615	NA	NA
Varicella infections (unspecified)	NDP	NN	16	79	162	18	NN	69	344	1,134	174	NA	1,478	NA	NA
Varicella zoster infections	NDP	NN	2	719	73	7	NN	153	954	382	854	NA	1,336	NA	NA
Vectorborne diseases															
Barmah Forest virus infection	4	232	32	244	11	0	9	18	550	437	659	507.4	987	906.2	1.1
Dengue	1	16	3	42	4	0	2	10	78	90	56	95.2	168	254.6	0.7
Flavivirus infection (NEC)	0	0	0	6	0	0	1	0	7	12	10	12.8	19	33.6	0.6
Japanese encephalitis virus	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0.4	0.0
Kunjin virus	0	0	0	0	0	0	0	0	0	0	2	1.4	0	6.0	0.0
Malaria	1	17	9	61	11	2	38	28	167	153	203	158.2	320	362.2	0.9
Murray Valley encephalitis virus	0	0	0	0	0	0	0	0	0	0	1	0.2	0	1.2	0.0
Ross River virus infection	4	278	67	718	51	2	22	118	1,260	1,089	1,133	1,196.8	2,349	2,807.2	0.8

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 April to 30 June 2007, by date of onset,* *continued*

Disease	State or territory								Total 2nd quarter 2007†	Total 1st quarter 2007	Total 2nd quarter 2006	Last 5 years mean 2nd quarter	Year to date 2007	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	1	0	0.0	1	0.2	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	4	0	0	0	1	5	14	7	6.4	19	16.2	1.2
Leptospirosis	0	1	0	22	1	0	2	2	28	48	58	44.2	76	99.6	0.8
Lyssavirus unspecified	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Ornithosis	0	10	0	0	0	0	15	0	25	26	40	52.2	51	92.6	0.6
Q fever	0	47	1	30	11	0	9	2	100	113	85	131.2	213	271.8	0.8
Other bacterial infections															
Legionellosis	2	26	2	15	8	1	16	10	80	73	72	85.0	151	168.6	0.9
Leprosy	0	1	0	2	1	0	0	1	5	3	2	1.6	8	4.4	1.8
Meningococcal infection**	0	22	2	7	3	1	22	2	59	46	66	107.0	105	196.2	0.5
Tuberculosis	3	67	5	34	14	2	66	13	204	262	273	260.4	466	521.4	0.9
Total	479	8,072	1,912	8,952	2,696	646	6,664	4,156	33,300	37,534	33,210	28,319.3	70,834	59,146.0	1.2

* Date of onset = the true onset. If this is not available, the 'date of onset' is equivalent to the earliest of two dates: (i) specimen date of collection, or (ii) the date of notification to the public health unit. 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* (SLTEC/VTEC).

¶ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia which reports only genital tract specimens, Northern Territory 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.

NA Not applicable.

NDP No data provided.

Table 3. Notification rates of diseases, 1 April to 30 June 2007, by state or territory. (Annualised rate per 100,000 population)

Disease*	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis B (incident)	2.4	0.9	9.7	1.4	0.0	0.8	1.4	2.3	1.3
Hepatitis B (unspecified)	13.4	46.2	83.2	27.2	21.9	4.1	34.0	31.4	35.0
Hepatitis C (incident)	1.2	0.5	0.0	NN	1.8	2.5	1.9	3.5	1.5
Hepatitis C (unspecified)	46.2	89.9	106.4	68.6	19.8	44.2	52.1	51.7	65.6
Hepatitis D	0.0	0.2	0.0	0.2	0.0	0.0	0.2	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†	126.5	NN	189.7	97.5	169.3	134.2	106.7	87.4	110.8
Cryptosporidiosis	2.4	4.7	69.7	8.4	20.8	5.7	11.7	32.2	11.8
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.0	0.0	0.0	0.2	0.0	0.1
Hepatitis A	0.0	0.7	1.9	0.9	0.3	0.8	0.4	2.0	0.8
Hepatitis E	0.0	0.0	0.0	0.2	0.0	0.0	0.3	0.0	0.1
Listeriosis	0.0	0.3	0.0	0.0	0.0	0.8	0.2	0.0	0.2
Salmonellosis (NEC)	36.5	36.1	228.4	65.7	48.1	38.4	37.4	42.1	45.8
Shigellosis	0.0	0.9	75.5	1.0	3.3	1.6	2.0	4.9	2.5
SLTEC, VTEC‡	0.0	0.1	0.0	0.3	1.3	0.0	0.2	0.0	0.2
Typhoid	0.0	0.6	0.0	0.1	0.0	0.0	0.4	0.0	0.3
Quarantinable diseases									
Cholera	0.0	0.1	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
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tularemia	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§	270.1	167.3	1341.2	306.7	196.3	234.0	193.8	343.9	236.0
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	10.9	20.6	1035.4	34.4	38.6	7.4	18.1	80.0	39.7
Syphilis (all)	7.3	18.2	172.2	9.5	3.1	8.2	15.3	9.4	14.9
Syphilis <2 years duration	0.0	3.5	121.9	5.3	0.3	3.3	6.4	5.9	5.7
Syphilis >2 years or unspecified duration	7.3	14.7	50.3	4.1	2.8	4.9	8.9	3.5	9.2
Syphilis - congenital	0.0	0.1	3.9	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	0.0	0.0	0.0	0.0	0.0	0.0	0.4	0.0
Influenza (laboratory confirmed)	7.3	8.9	13.5	20.2	2.6	11.5	2.6	18.5	10.1
Measles	0.0	0.1	0.0	0.1	0.0	0.0	0.1	0.0	0.1
Mumps	0.0	3.1	23.2	0.8	1.3	0.0	0.6	0.2	1.7
Pertussis	29.2	24.6	11.6	33.0	24.2	4.1	22.2	3.7	23.0
Pneumococcal disease (invasive)	9.7	6.4	32.9	6.8	8.7	2.5	5.8	5.9	6.7
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3. Notification rates of diseases, 1 April to 30 June 2007, 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*									
Rubella	1.2	0.3	0.0	0.4	0.3	0.0	0.2	0.2	0.3
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 infections (chickenpox)	NDP	NN	32.9	4.3	41.9	0.8	NN	7.8	12.7
Varicella infections (unspecified)	NDP	NN	31.0	7.8	41.7	14.7	NN	13.5	16.5
Varicella zoster infections	NDP	NN	3.9	71.0	18.8	5.7	NN	29.8	45.7
Vectorborne diseases									
Barmah Forest virus infection	4.9	13.6	61.9	24.1	2.8	0.0	0.7	3.5	10.7
Dengue	1.2	0.9	5.8	4.1	1.0	0.0	0.2	2.0	1.5
Flavivirus infection (NEC)	0.0	0.0	0.0	0.6	0.0	0.0	0.1	0.0	0.1
Japanese encephalitis virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	1.2	1.0	17.4	6.0	2.8	1.6	3.0	5.5	3.2
Murray Valley encephalitis virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	4.9	16.3	129.7	70.9	13.1	1.6	1.7	23.0	24.5
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.0	0.0	0.4	0.0	0.0	0.0	0.2	0.1
Leptospirosis	0.0	0.1	0.0	2.2	0.3	0.0	0.2	0.4	0.5
Lyssavirus unspecified	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	0.0	0.0	0.0	1.2	0.0	0.5
Q fever	0.0	2.8	1.9	3.0	2.8	0.0	0.7	0.4	1.9
Other bacterial infections									
Legionellosis	2.4	1.5	3.9	1.5	2.1	0.8	1.3	2.0	1.6
Leprosy	0.0	0.1	0.0	0.2	0.3	0.0	0.0	0.2	0.1
Meningococcal infection‖	0.0	1.3	3.9	0.7	0.8	0.8	1.7	0.4	1.1
Tuberculosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

* 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* (SLTEC/VTEC).

§ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia which reports only genital tract specimens, Northern Territory 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.

NA Not appllicable.

NDP No data provided.

Laboratory Serology and Virology Reporting Scheme

There were 5,015 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 April to 30 June 2007 (Tables 4 and 5).

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 April to 30 June 2007, and total reports for the year†

	State or territory								This period 2007	This period 2006	Year to date 2007	Year to date 2006
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	–	–	–	1	–	–	–	–	1	41	7	52
Mumps virus	–	–	–	2	1	–	2	–	5	11	14	23
Rubella virus	–	–	–	1	–	–	–	–	1	6	9	8
Hepatitis viruses												
Hepatitis A virus	–	–	–	4	3	–	–	–	7	5	17	16
Hepatitis D virus	–	–	–	1	3	–	2	–	6	2	13	4
Arboviruses												
Ross River virus	–	8	18	307	31	1	1	8	374	189	675	977
Barmah Forest virus	–	7	–	111	6	–	1	–	125	85	265	229
Flavivirus (unspecified)	–	1	–	27	–	–	–	–	28	9	53	39
Adenoviruses												
Adenovirus not typed/pending	–	27	–	26	42	–	8	–	103	105	270	260
Cytomegalovirus	1	38	–	51	102	–	5	–	197	193	479	518
Herpes viruses												
Varicella-zoster virus	2	58	1	304	87	2	6	1	461	258	1,155	610
Epstein-Barr virus	–	8	22	268	106	1	5	106	516	275	1,193	819
Other DNA viruses												
Parvovirus	–	–	1	36	6	–	5	–	48	37	140	88
Picornavirus family												
Echovirus type 6	–	1	–	–	–	–	–	–	1	–	2	–
Echovirus type 9	–	1	–	–	–	–	–	–	1	–	1	–
Rhinovirus (all types)	–	37	–	–	3	–	–	–	40	25	107	42
Enterovirus not typed/pending	–	10	–	14	1	–	1	–	26	22	54	76
Picornavirus not typed	–	–	–	–	–	1	–	–	1	1	1	1
Ortho/ paramyxoviruses												
Influenza A virus	–	2	3	12	1	–	3	–	21	42	71	72
Influenza B virus	1	1	–	2	2	–	–	–	6	38	11	44
Parainfluenza virus type 1	–	–	–	–	–	–	1	–	1	36	4	58
Parainfluenza virus type 2	–	9	–	7	2	–	–	–	18	6	23	7
Parainfluenza virus type 3	–	10	–	6	5	–	1	–	22	13	54	25
Respiratory syncytial virus	–	160	1	88	4	1	10	–	264	457	447	561

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 April to 30 June 2007, and total reports for the year,† *continued*

	State or territory								This period 2007	This period 2006	Year to date 2007	Year to date 2006
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other RNA viruses												
HTLV-1	–	–	–	–	2	–	–	–	2		5	4
Rotavirus	1	7	–	–	22	–	–	–	30	68	73	132
Norwalk agent	–	5	–	–	–	–	45	–	50	491	93	681
Other pathogens												
Chlamydia trachomatis not typed	–	127	–	924	465	9	5	–	1,530	997	3,682	2,472
Chlamydia psittaci	–	–	–	–	1	–	9	–	10	16	31	26
Mycoplasma pneumoniae	–	5	5	138	21	5	30	11	215	262	528	607
Mycoplasma hominis	–	1	–	–	–	–	–	–	1		4	10
Coxiella burnetii (Q fever)	–	1	1	14	10	–	1	–	27	23	52	70
Rickettsia prowazeki	–	–	–	–	1	–	–	–	1	–	1	24
Rickettsia australis	–	–	–	–	–	–	1	–	1	–	1	–
Rickettsia tsutsugamushi	–	–	–	–	1	–	–	–	1	4	4	21
Rickettsia - spotted fever group	–	–	–	–	–	–	1	–	1	9	2	66
Streptococcus group A	–	5	28	157	–	–	1	1	192	127	424	264
Yersinia enterocolitica	–	–	–	1	–	–	–	–	1	1	2	4
Brucella species	–	–	–	1	–	–	–	–	1	1	2	3
Bordetella pertussis	–	2	2	77	55	–	4	–	140	242	320	671
Legionella pneumophila	–	3	–	–	–	–	5	–	8	11	12	19
Legionella longbeachae	–	–	–	–	1	–	–	–	1	2	4	10
Cryptococcus species	–	1	–	1	6	1	–	–	9	4	18	14
Leptospira species	–	1	–	11	–	–	–	–	12	4	35	11
Treponema pallidum	–	19	5	197	226	–	3	–	450	219	997	495
Entamoeba histolytica	–	–	–	1	–	–	–	–	1	–	5	–
Toxoplasma gondii	–	1	–	3	1	–	1	–	6	17	14	33
Echinococcus granulosus	–	–	–	–	10	–	–	–	10	–	13	3
Total	5	556	87	2,793	1,227	21	157	127	4,973	4,354	11,387	10,169

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

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

– No data received this period.

Table 5. Virology and serology reports by laboratories for the reporting period 1 April to 30 June 2007*

State or territory	Laboratory	April 2007	May 2007	June 2007	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	97	49	–	146
	New Children's Hospital, Westmead	85	106	–	191
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	13	46	–	59
	South West Area Pathology Service, Liverpool	21	0	–	21
Queensland	Queensland Medical Laboratory, West End	1,274	1,523	200	2,997
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	523	701	1	1,225
Tasmania	Northern Tasmanian Pathology Service, Launceston	4	15	1	20
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Monash Medical Centre, Melbourne	–	–	–	–
	Royal Children's Hospital, Melbourne	32	–	–	32
	Victorian Infectious Diseases Reference Laboratory, Fairfield	51	67	–	118
Western Australia	PathWest Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	86	78	–	164
Total		2,186	2,585	202	4,973

* The complete list of laboratories reporting for the 12 months, January to December 2007, will appear in every report regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.

– No data received this period.

Additional reports

Australian Sentinel Practice Research Network

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

The network consists of general practitioners who report presentations on a number of defined medical conditions each week. ASPREN was established in 1991 to provide a rapid monitoring scheme for infectious diseases that can alert public health officials of epidemics in their early stages as well as play a role in the evaluation of public health campaigns and research of conditions commonly seen in general practice. The aim of ASPREN is to also provide an indicator of the burden of disease in the primary health care setting and to detect trends in consultation rates.

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published. In 2007, four conditions are being monitored all of which are related to communicable diseases. They include influenza like illness (ILI), gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, *published in* Commun Dis Intell *2007;31:158.*

Reporting period 1 April to 30 June 2007

Sentinel practices contributing to ASPREN were located in all jurisdictions other than the Northern Territory and Tasmania. A total of 92 general practitioners contributed data to ASPREN in the second quarter of 2007. Each week an average of 56 general practitioners provided information to ASPREN at an average of 5,539 (range 3,500 to 7,463) consultations per week.

In the second quarter of 2007, influenza-like illness rates increased from mid-June (24.2 ILI per 1,000 consultations) (Figure 1). Two peaks were observed in mid-April (22.2 ILI per 1,000 consultations) and end of June (25.9 ILI per 1,000 consultations). In the corresponding period in 2006, ILI rates also peaked in mid-June, but at a slightly higher rate than in 2007 (29.1 ILI per 1,000 consultations) and decreased towards the end of June (between 14.8–16.3 ILI per 1,000 consultations).

Reports of gastroenteritis from 1 April to 30 June 2007 were lower compared to the same period in 2006 (Figure 2). During this reporting period, consultation rates for gastroenteritis showed a downward trend from mid-May onwards (between 4.6 to 9 cases per 1,000 consultations).

Reports of varicella infections were reported at a lower rate for the second quarter of 2007 compared with the same period in 2006 but there was no recognisable seasonal pattern. From 1 April to 30 June 2007, rates for chickenpox fluctuated between 0 to 2.4 cases per 1,000 consultations (Figure 3).

In the second quarter of 2007, rates for shingles fluctuated between less than 1 to 2.8 cases per 1,000 consultations (Figure 4).

Figure 1: Consultation rates for influenza like illness, ASPREN, 2006 to 30 June 2007, by week of report

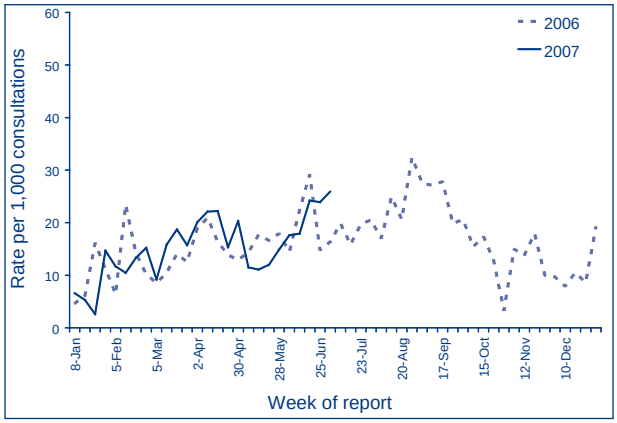


Figure 2. Consultation rates for gastroenteritis, ASPREN, 2006 to 30 June 2007, by week of report

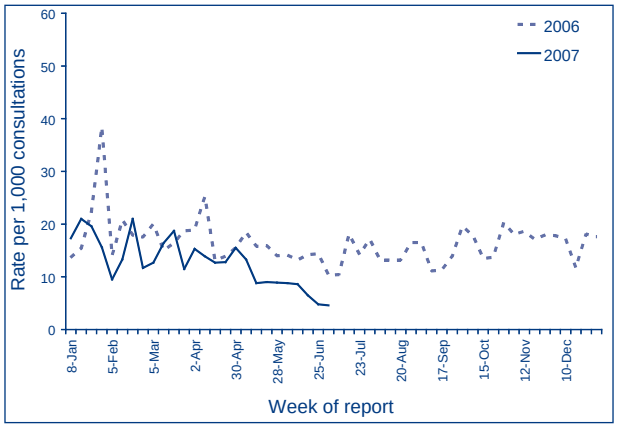


Figure 3. Consultation rates for chickenpox, ASPREN, 2006 to 30 June 2007, by week of report

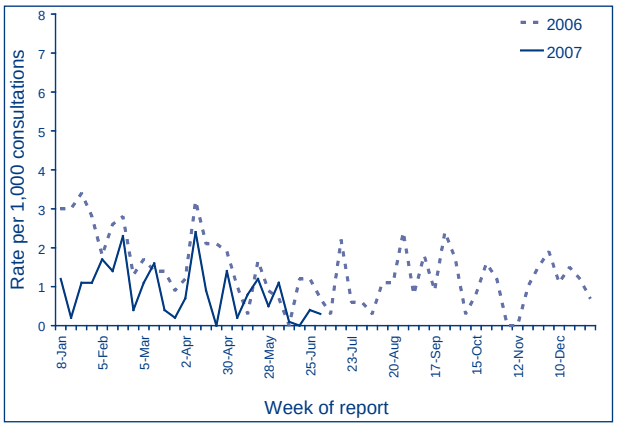
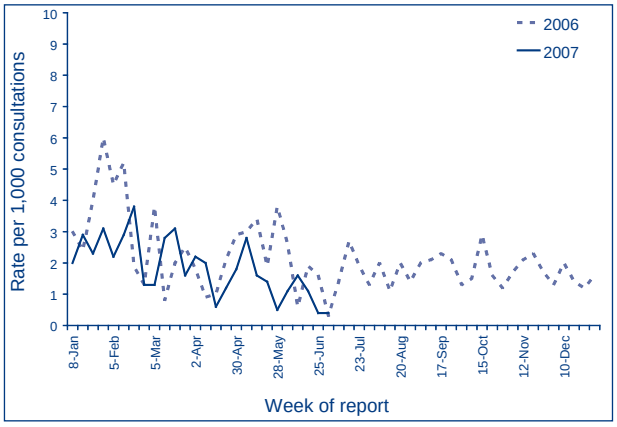


Figure 4. Consultation rates for shingles, ASPREN, 2006 to 30 June 2007, by week of report



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 *2007;31:162.*

Reporting period 1 January to 31 March 2007

The AGSP laboratories received a total of 856 isolates in this quarter of which 846 underwent susceptibility testing. This is considerably less than the 1,110 isolates reported in this period in 2006 and also less than the 985 reported in the first quarter of 2005. About 33% of this total was from New South Wales, 24% from Victoria, 12% from the Northern Territory, 11% from Queensland, 10% from Western Australia and 8% from South Australia. Small numbers of isolates were also received from Tasmania and the Australian Capital Territory.

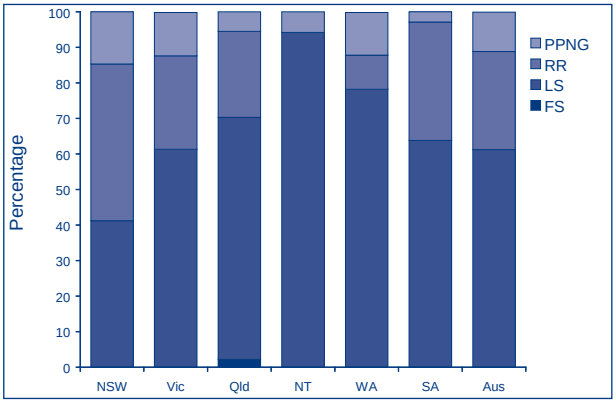
Penicillins

In this quarter, 327 (38.7%) isolates examined were penicillin resistant by one or more mechanisms. Ninety-four (11.1%) were penicillinase producing *Neisseria gonorrhoeae* (PPNG) and 233 (27.6%) were penicillin resistant by chromosomal mechanisms (CMRP). The proportion of all strains resistant to the penicillins by any mechanism ranged from 5.8% in the Northern Territory to 58.7% in New South Wales. These represent the highest rates of penicillin resistance seen to date in this surveillance system. In the corresponding quarter in 2006, 33.6% of isolates were penicillin resistant by any mechanism.

Figure 5 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 PPNG aggregated for Australia and by state or territory. A high proportion of those strains classified as PPNG or resistant by chromosomal mechanisms fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

The highest number and proportion of PPNG and CMRP were found in New South Wales where there were 42 PPNG (14.7%) and 126 CMRP (44%). Victoria had 54 (26%) CMRP and 25 (12.2%) PPNG. Western Australia had more PPNG (10, 12%) than CMRP (8, 9.6%) whereas CMRP were more prominent in Queensland (22, 24%) than PPNG (5, 5.5%). Six PPNG but no CMRP were found in the Northern Territory. South Australia had a high proportion of CMRP (23, 33%) and two (2.9%) PPNG. There were three PPNG reported from the Australian Capital Territory and one from Tasmania but no CMRP were seen in either of these jurisdictions.

Figure 5. Categorisation of gonococci isolated in Australia, 1 January to 31 March 2007, by penicillin susceptibility and region



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

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

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

PPNG Penicillinase producing *Neisseria gonorrhoeae*.

Ceftriaxone

Seven isolates with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected, five in New South Wales and one each in Western Australia and the Australian Capital Territory. This is the same number seen nationally in the first quarter of 2006.

Spectinomycin

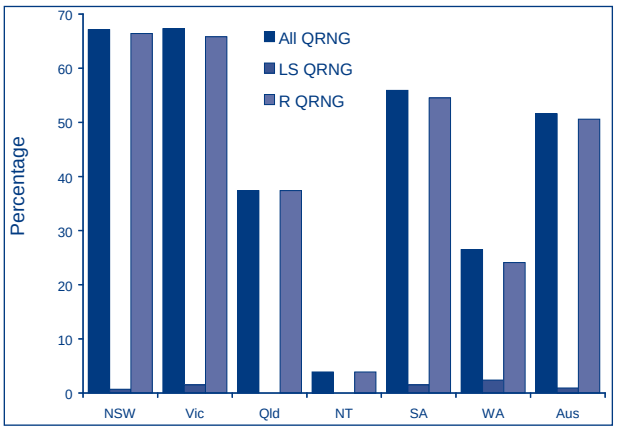
All isolates were susceptible to this injectable agent.

Quinolone antibiotics

The total number (436) and proportion (51.6%) of quinolone resistant *N. gonorrhoeae* (QRNG) were also the highest recorded for Australia for any quarter. In the corresponding period in 2006, there were 387 (35.5%) QRNG which was substantially higher than the corresponding figures in the first quarter of 2005 (283 QRNG, 29.7%), 2004 (188 QRNG, 20.5%) and 2003 (108 isolates, 11.5%). All but eight of the 436 QRNG detected in this quarter had ciprofloxacin MICs of 1 mg/L or more and 375 had ciprofloxacin MICs of 4 mg/L or more. QRNG are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06–0.5 mg/L) or resistant (MIC ≥ 1 mg/L) groups. Thus not only is there an increase in the number of QRNG but also an upward shift in resistance levels.

QRNG were present in all jurisdictions (Figure 6). The highest number of QRNG was found in New South Wales (191) which represented 66.8% of all isolates. The 138 QRNG in Victoria formed a slightly higher (67.3%) proportion of all isolates there. In South Australia, 38 QRNG represented 55.9% of all gonococci tested; in Queensland there were 34 (37.4%) QRNG, and in Western Australia 22 (26.5%) QRNG. Six QRNG were detected in the Northern Territory, two in Tasmania and five in the Australian Capital Territory.

Figure 6. The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 January to 31 March 2007, by jurisdiction



LS QRNG Ciprofloxacin MICs 0.06–0.5 mg/L.

R QRNG Ciprofloxacin MICs ≥1 mg/L.

High level tetracycline resistance

Nationally, the number (125) and proportion (14.8%) of high level tetracycline resistance (TRNG) detected increased when compared with the 2006 data (115 TRNG, 10.6%) but remained lower than in this period in 2005 (145 TRNG, 15.5%). TRNG were found in all states and territories except Tasmania and elsewhere represented between 9% (South Australia) and 23% of isolates (Western Australia) in mainland states. Five TRNG were present in the Northern Territory, and two in the Australian Capital Territory.

Reference

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

Childhood immunisation coverage

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

The data show the percentage of children fully immunised at 12 months of age for the cohort born between 1 January and 31 March 2006, at 24 months of age for the cohort born between 1 January and 31 March 2005, and at 6 years of age for the cohort born between 1 January and 31 March 2001 according to the National Immunisation Program.

For information about the Australian Childhood Immunisation Register see Surveillance systems reported in CDI, *published in* Commun Dis Intell *2007;31:163–164 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.

Immunisation coverage for children 'fully immunised' at 12 months of age for Australia increased marginally by 0.2 percentage points to 91.2% (Table 1), whilst there were no important changes in coverage for all individual vaccines due at 12 months of age. There were also no noteworthy movements in coverage for individual vaccines by jurisdiction.

Immunisation coverage for children 'fully immunised' at 24 months of age for Australia increased from the last quarter by 0.5 percentage points to 92.5% (Table 2). There were no significant changes in coverage in any jurisdiction for 'fully immunised' coverage or for coverage for individual vaccines. It is notable that the estimate for children 'fully immunised' at 24 months of age has been higher than the 12 months coverage estimate since the 18 month DTPa booster was no longer required from September 2003.

It is also notable that, for the two vaccines where no further doses are due between 6 months and 24 months of age (DTP and polio), coverage at the national level was 95.2% at 24 months versus 91.9% and 91.8% at 12 months. This suggests that delayed notification or delayed vaccination is making an important contribution to the coverage estimates at 12 months of age and that the 'fully immunised' estimate in particular is likely to be a minimum estimate.

Immunisation coverage for children 'fully immunised' at 6 years of age for Australia decreased marginally from the last quarter by 0.1 percentage point to 87.9% (Table 3). There were no important changes in coverage for all individual vaccines due at 6 years of age and no noteworthy movements in coverage for individual vaccines by jurisdiction.

Figure 7 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. It should be noted that currently, coverage for the vaccines added to the National Immunisation Program since 2003 (varicella at 18 months, meningococcal C conjugate at 12 months and pneumococcal conjugate at 2, 4, and 6 months) are not included in the coverage data.

Table 1. Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 January to 31 March 2006; assessment date 30 June 2007

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,201	22,789	910	14,440	4,545	1,581	16,420	6,999	68,885
Diphtheria, tetanus, pertussis (%)	94.8	91.9	91.3	91.8	91.5	91.6	92.9	89.4	91.9
Poliomyelitis (%)	94.8	91.8	91.3	91.7	91.5	91.5	92.8	89.4	91.8
Haemophilus influenzae type b (%)	96.6	94.8	95.8	94.0	94.7	95.6	95.0	93.5	94.6
Hepatitis B (%)	96.6	94.7	95.9	93.8	94.7	95.6	94.8	93.4	94.5
Fully immunised (%)	94.3	91.5	91.1	90.9	90.5	91.4	91.8	88.9	91.2
Change in fully immunised since last quarter (%)	+2.5	+0.3	+0.3	+0.1	+0.1	-1.1	+0.5	-0.9	+0.2

Table 2. Percentage of children immunised at 2 years of age, preliminary results by disease and state or territory for the birth 1 January to 31 March 2005; assessment date 30 June 2007[7]

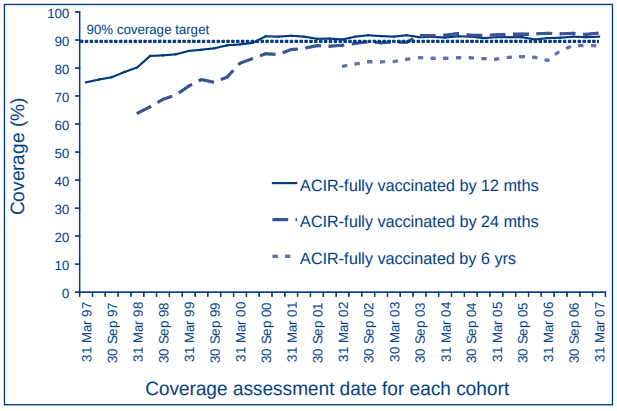
Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,082	22,126	855	13,938	4,477	1,381	15,628	6,821	66,308
Diphtheria, tetanus, pertussis (%)	94.7	95.2	96.6	94.8	95.5	97.0	96.0	94.0	95.2
Poliomyelitis (%)	94.7	95.1	96.1	94.7	95.4	97.0	95.9	93.9	95.2
Haemophilus influenzae type b (%)	93.6	94.3	93.9	93.7	94.1	96.7	94.7	93.0	94.1
Measles, mumps, rubella (%)	92.4	93.9	94.6	93.6	94.2	95.9	94.8	92.7	94.0
Hepatitis B(%)	95.0	95.9	97.4	95.6	96.3	97.3	96.6	95.1	96.0
Fully immunised (%)	91.9	92.3	92.5	92.2	93.0	95.2	93.8	90.6	92.5
Change in fully immunised since last quarter (%)	-1.1	+0.8	-0.7	+0.9	+1.4	+1.2	+0.4	+0.0	+0.5

* The 12 months age data for this cohort was published in *Commun Dis Intell* 2005;30:399

Table 3. Percentage of children immunised at 6 years of age, preliminary results by disease and state or territory for the birth cohort 1 January to 31 March 2001; assessment date 30 June 2007

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,039	21,920	938	13,560	4,582	1,577	15,785	6,710	66,111
Diphtheria, tetanus, pertussis (%)	90.1	88.5	85.8	88.6	86.4	90.4	91.1	85.1	88.7
Poliomyelitis (%)	90.4	88.4	85.7	88.9	86.3	90.2	91.4	85.3	88.8
Measles, mumps, rubella (%)	89.9	88.6	85.9	88.7	86.3	90.6	91.2	85.3	88.8
Fully immunised (%)[1]	89.4	87.7	84.8	87.8	85.7	89.8	90.6	84.2	87.9
Change in fully immunised since last quarter (%)	+0.8	-0.5	-1.5	+0.6	-0.2	-0.9	-0.4	+1.0	-0.1

Figure 7. Trends in vaccination coverage, Australia, 1997 to 2007, by age cohorts



HIV and AIDS surveillance

National surveillance for HIV disease is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with State and Territory health authorities and the Commonwealth of Australia. Cases of HIV infection are notified to the National HIV Database on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the State and Territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in 'HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia, annual surveillance report'. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Internet: http://www.med.unsw.edu.au/nchecr. Telephone: +61 2 9332 4648. Facsimile: +61 2 9332 1837. For more information see Commun Dis Intell *2005;29:91–92.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 January to 31 March 2007, as reported to 30 June 2007, are included in this issue of Communicable Diseases Intelligence *(Tables 4 and 5).*

Table 4. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 January to 31 March 2007, by sex and state or territory of diagnosis

	Sex	State or territory								Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2007	This period 2006	YTD 2007	YTD 2006
HIV diagnoses	Female	0	8	0	10	2	2	7	0	29	39	29	39
	Male	1	105	1	45	21	0	58	0	231	223	231	223
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	1	113	1	55	23	2	65	0	260	262	260	262
AIDS diagnoses	Female	0	0	0	0	0	0	0	0	0	4	0	4
	Male	0	10	2	2	0	0	8	1	23	46	23	46
	Total*	0	10	2	2	0	0	8	1	23	51	23	51
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	3	0	3
	Male	0	4	1	1	1	0	2	0	9	15	9	15
	Total*	0	4	1	1	1	0	2	0	9	19	9	19

* Totals include people whose sex was reported as transgender.

Table 5. Cumulative diagnoses of HIV infection, AIDS, and deaths following AIDS since the introduction of HIV antibody testing to 31 March 2007, and reported by 30 June 2007, by sex and state or territory

	Sex	State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	32	883	23	279	102	12	374	203	1,908
	Male	260	13,555	132	2,774	955	109	5,309	1,212	24,306
	Not reported	0	230	0	0	0	0	22	0	252
	Total*	292	14,697	155	3,062	1,058	121	5,727	1,422	26,534
AIDS diagnoses	Female	10	251	4	71	32	4	111	41	524
	Male	92	5,427	45	1,032	409	53	2,004	427	9,489
	Total*	102	5,696	49	1,105	442	57	2,127	470	10,048
AIDS deaths	Female	7	136	1	42	20	2	61	26	295
	Male	73	3,586	28	664	280	33	1,415	295	6,374
	Total*	80	3,733	29	708	300	35	1,485	322	6,692

* Totals include people whose sex was reported as transgender.

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 *2007;31:162.*

Laboratory confirmed cases of invasive meningococcal disease for the period 1 April to 30 June 2007, are included in this issue of Communicable Diseases Intelligence *(Table 6).*

Table 6. Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 April to 30 June 2007, by serogroup and state or territory

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD
Australian Capital Territory	07			0	1						1			0	2
	06					1	1							1	1
New South Wales	07			5	17	3	6	2	2	1	1	3	4	14	30
	06			13	22	1	2	1	1	0	1	0	3	15	29
Northern Territory	07				1	1	1							1	2
	06			1	2									1	2
Queensland	07			8	19	1	1					1	1	10	21
	06			10	25	3	4							13	29
South Australia	07			3	4									3	4
	06			3	6			1	1					4	7
Tasmania	07									1	1			1	1
	06			2	3	0	1							2	4
Victoria	07			15	21	2	2	3	3	1	1	1	1	22	28
	06			19	29	0	2	0	1	0	2			19	34
Western Australia	07			4	7									4	7
	06			4	9									4	9
Total	07			35	70	7	10	5	5	3	3	5	6	55	94
	06			52	96	5	10	2	3	0	3	0	3	61	117

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 six* Salmonella *laboratories in Australia.* Salmonella *isolates are submitted to these laboratories for typing by primary diagnostic laboratories throughout Australia.*

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

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

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

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

Reports to the National Enteric Pathogens Surveillance System of Salmonella infection for the period 1 April to 30 June 2007 are included in Tables 7 and 8. Data include cases reported and entered by 12 July 2007. 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 *2007;31:163–164.*

Reporting period 1 April to 30 June 2007

There were 2,232 reports to NEPSS of human *Salmonella* infection in the second quarter of 2007. This represents a seasonal decline in incidence after the first quarter, the final count for which was 3,249 reports, the highest quarterly count in more than 15 years. The 2,232 reports for the second quarter also represent the highest count in the second quarter for more than 15 years, 27% greater than the 10-year historical average.

During the second quarter of 2007, the 25 most common *Salmonella* types in Australia accounted for 1,387 cases, 62% of all reported human *Salmonella* infections. Twenty-three of the 25 most common *Salmonella* infections in the second quarter of 2007 were also among those most commonly reported in the preceding quarter.

The most notable features of the current data are the widespread outbreaks of various phage types of *S.* Typhimurium. These include *S.* Typhimurium phage type 9 (in New South Wales and South Australia), *S.* Typhimurium phage type 44 (in Victoria), *S.* Typhimurium phage type 29 (in South Australia and New South Wales), *S.* Typhimurium phage type U302 (in New South Wales), *S.* Typhimurium phage type U307 (in Western Australia and Queensland), *S.* Typhimurium phage type 197 (in Queensland), *S.* Typhimurium phage type 35 (in New South Wales), and *S.* Typhimurium phage type 120. More recently, an increase in *S.* Typhimurium phage type 135 has also become apparent in the eastern states.

Other salmonellae manifesting increases over historical averages and outbreaks include *S.* Mississippi (in Tasmania), *S.* Montevideo (in New South Wales), and *S.* Oslo (in the Northern Territory).

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 April to 30 June 2007, as reported to 12 July 2007

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total all *Salmonella* for quarter	24	635	91	585	171	53	480	193	2,232
Total contributing *Salmonella* types	17	120	40	108	52	18	102	69	231

Table 8. Top 25 *Salmonella* types identified in Australia, 1 April to 30 June 2007, by state or territory

National rank	*Salmonella* type	State or territory								Total 2nd quarter 2007	Last 10 years mean 2nd quarter	Year to date 2007	Year to date 2006
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
1	*S.* Typhimurium PT 9	1	125	0	12	29	0	43	6	216	115	553	225
2	*S.* Typhimurium PT 135	4	53	0	51	3	5	45	12	173	140	425	421
3	*S.* Typhimurium PT 44	1	15	0	4	6	0	73	0	99	15	282	119
4	*S.* Saintpaul	0	8	7	48	0	0	4	16	83	93	220	270
5	*S.* Typhimurium PT 29	1	31	0	8	33	0	2	0	75	3	124	11
6	*S.* Typhimurium PT U302	0	49	0	8	1	1	6	3	68	3	109	21
7	*S.* Typhimurium PT 170	0	24	0	6	0	1	34	0	65	72	189	246
8	*S.* Virchow PT 8	0	5	1	52	2	0	3	1	64	64	150	189
9	*S.* Typhimurium PT U307	0	4	0	13	0	0	2	40	59	5	80	27
10	*S.* Birkenhead	0	18	0	31	0	0	1	0	50	62	143	197
11	*S.* Typhimurium PT 197	0	10	0	25	1	0	6	0	42	21	136	63
12	*S.* Aberdeen	0	1	2	36	0	0	1	0	40	34	90	113
13	*S.* Mississippi	0	2	0	1	0	29	2	1	35	19	111	70
14	*S.* Montevideo	0	24	0	6	0	0	5	0	35	8	77	19
15	*S.* Infantis	0	13	3	3	6	0	8	1	34	35	94	120
16	*S.* Chester	0	4	2	15	3	1	2	6	33	40	104	99
17	*S.* Hvittingfoss	0	6	0	21	0	0	5	1	33	33	74	99
18	*S.* Waycross	0	12	0	20	0	0	0	0	32	31	68	107
19	*S.* Stanley	1	6	0	7	1	0	10	2	27	12	65	42
20	*S.* Typhimurium (PT pending)	3	1	0	1	1	1	20	0	27	0	30	0
21	*S.* Muenchen	0	5	3	14	2	0	0	2	26	36	84	104
22	*S.* Typhimurium PT 126	0	1	0	2	2	0	14	0	19	26	28	17
23	*S.* Typhimurium PT 12	0	3	0	2	3	0	3	7	18	23	65	72
24	*S.* Virchow PT 34	0	0	0	5	0	0	12	0	17	21	28	26
25	*S.* Typhimurium untypable	0	4	0	1	0	0	4	8	17	17	55	48

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 April to 30 June 2007

Avian influenza

The World Health Organization (WHO) confirmed 15 cases of human H5N1 with dates of onset between 1 April and 30 June 2007[1] compared with 33 cases including 23 deaths during the same period of 2006.[2] Nine of the 15 cases were fatal, resulting in a case-fatality rate (CFR) of 60%.[1] The 15 cases were reported from Cambodia, China, Egypt, Indonesia and Vietnam.[1]

Indonesia reported the highest number of cases (7 cases, including 6 deaths), and has reported the highest number of cases since the beginning of the global outbreak in November 2003 (102 cases including 81 deaths to 10 August 2007).[3]

Vietnam reported two non-fatal human cases of H5N1, which were the first cases in the country since November 2005.[4] Between May and August 2007, the Vietnamese Ministry of Health reported an additional five human cases of H5N1 (including 4 deaths), which have not yet been confirmed by the WHO.[5,6,7]

The source of infection for nearly all cases was established as exposure to sick and dead poultry.[1] Only one case, a 19-year-old soldier from China, had no clear source of infection identified and no obvious exposure to sick or dead poultry,[8] but there was no evidence of human-to-human transmission.

Chikungunya

There have been fewer outbreaks of chikungunya world-wide in 2007 than in previous years, but the disease is now common in Indonesia and India.

India

Major outbreaks of chikungunya have continued in India, with widening geographical incidence. Between January and early July 2007, there were approximately 19,000 probable cases of chikungunya fever in Kerala State, with 177 deaths thought to be at least partly due to the infection. An outbreak of chikungunya was also reported from Orissa State in May 2007, with 642 cases, including four deaths. There were also a number of cases reported in the capital, Delhi, but most were imported from areas outside the city.[9,10,11]

Indonesia

Indonesia reported 30 suspected cases (5 confirmed) of chikungunya from Central Jakarta during May 2007, the first in the Indonesian capital since 2004. In Lampung Province on the southern tip of Sumatra, health authorities reported a suspected outbreak of chikungunya fever, with 100 cases between April and May 2007.[12–16]

Dengue fever

Dengue fever is the most common viral illness world-wide and the global burden of disease due to dengue has increased more than fourfold in the last 30 years. Dengue fever is hyper-endemic (an endemic disease that affects a high proportion of the population at risk) in South East Asia and the Western Pacific, which are the regions most seriously affected by the disease.[17] Information on the extent of the disease in the Western Pacific Region is unreliable, with many cases and outbreaks not reported.[18] Outbreaks of dengue fever were reported across South East Asia during the reporting period, with major rises in incidence compared with previous years.

Cambodia has been one of the countries worst affected during the outbreaks, particularly due to the country's lack of resources to properly treat cases and implement control programs. The Cambodian Ministry of Health reported 27,265 cases of dengue fever, including 304 fatal cases (all of them children) between 1 January and 29 July 2007, a 60% increase over the number of cases that were reported for the whole of 2006 and nearly twice as many fatal cases.[19,20]

The outbreak of dengue in Singapore peaked in late June and early July, when 432 cases were reported in one week, crossing the epidemic threshold. The Ministry of Health reported 3,213 cases of dengue fever between 1 January and 30 June 2007.[21] The last epidemic of dengue in the country was in 2005 when 714 cases were reported in a single week.[22]

The Ministry of Health in Myanmar estimates that there were 30 fatal cases of dengue haemorrhagic fever between January and June 2007, a higher mortality rate than seen in 2006. The WHO has stated that the number of cases in the first half of 2007 is a 29% increase over the same period of last year.[23,24]

Other South-East Asian countries severely affected by outbreaks of dengue fever in 2007 include Thailand (a 17% increase in the number of cases between January and May 2007 compared with the same period of 2006),[25] Indonesia (100,000 cases including 1,100 deaths between January and July 2007, similar to 2006),[25,26] Vietnam (a 40% increase in the number of cases between January and June 2007 compared with the same period of 2006) and Malaysia (30,285 cases including 65 deaths between January and July 2007 compared with 20,258 cases including 49 deaths for the whole of 2006).[27]

Measles

Global update

Between 1 April and 30 June 2007, a number of countries reported outbreaks of measles including Canada, the Democratic Republic of the Congo, Japan, Norway, the Russian Federation, Switzerland, Taiwan and the United Kingdom. Cases of measles were also imported into Australia and the United States of America. The outbreak of measles in Japan was of particular concern to Australia because of the large number of people who travel between the two countries for tourism, education and business.

Japan

In February 2007, Japan's Infectious Disease Surveillance Centre (IDSC) reported an increase in the number of measles cases from Japan's southern Kanto region, including Tokyo and Saitama Prefecture. The outbreak spread to the nearby Prefectures of Chiba, Saitama and Kanagawa and then to the most northerly prefecture in the country, Hokkaido.[28] Young people were the most affected in the outbreak and a number of schools and universities were closed in an attempt to stop the infection spreading further.[29,30,31]

The outbreak of measles in most areas peaked between 21 and 27 May 2007[28] when the IDSC reported 215 paediatric cases and 387 adult cases from the sentinel surveillance system. The sentinel surveillance system collects data from approximately 3,000 paediatric healthcare facilities and 450 other hospitals across the country, which is only a proportion of all healthcare facilities (including approximately 10% of the paediatric facilities), so the total number of cases during the outbreak is not known. Between 1 January and 24 June 2007, the IDSC reported a total of 2,450 cases from the sentinel surveillance system compared with 545 cases of measles for the whole of 2006. It is estimated that only 10%-20% of cases each year are reported.[32]

One-dose measles immunisation rates in Japan for some of the most affected age groups are estimated to be 97% for 2 to 10-year-olds, 95% for 11 to 20-year-olds, 88.6% for 20 to 29-year-olds and 85% for 30 to 39-year-olds, but a single dose may not be sufficient to ensure strong lifetime immunity. Two-dose coverage in Japan is low (estimated at 40%), because two-dose immunisation was only introduced in 2006 for children starting school.[33,34,35] The Ministry of Health, Welfare and Labour will commence a catch-up measles vaccination campaign in the next academic year for school students. Doses will be administered to students in their first year of primary school and third year of high school commencing in the next academic year.[36,37]

Nipah virus

In early April 2007, six fatal cases of acute neurological syndrome were reported from the Kushtia region of western Bangladesh.[38] Three of the six cases tested positive for Nipah virus at the Institute of Epidemiological Disease Control and Research in Dhaka.[39] The International Centre for Diarrhoeal Disease Research identified a further 12 probable cases during the outbreaks, including five deaths that occurred between 21 January and 4 April 2007.[40] Seven of these probable cases were from the Thakurgaon region in northern Bangladesh.[40]

In mid-April 2007, an outbreak of Nipah was reported from the Nadia district of West Bengal (neighbouring the Kushtia region) with approximately 50 suspected cases, including three deaths, between February and mid-May 2007. Only the three fatal cases (all members of one family) were confirmed by the National Institute of Virology in Pune.[41,42,43] Some media sources reported a further two fatal cases (one was a relative of the earlier three fatal cases and one was a healthcare worker),[44] but there is doubt as to whether the deaths were due to Nipah virus infection or other causes of encephalitis.

Outbreaks of Nipah in south Asia have a strong seasonal pattern and a limited geographical range.[40] In 2005, there were 12 human cases (including 11 fatal) reported from the Tangail district of Bangladesh, most of them related to the consumption of fresh date palm juice contaminated by infected bats (thought to be the natural reservoir of the virus). However, in an outbreak between February and April 2004 with 36 cases (CFR 75%) in the Faridpur district, there was evidence of human-to-human transmission in some of the cases.[45] In particular, two of the fatal cases acquired the disease after having casual contact with a relative who was dying of the infection.[46] The 2004 outbreak was also unique in that six of the cases developed acute respiratory distress syndrome, rather than the neurological symptoms usually observed in Nipah virus cases.[46]

Health authorities were investigating a number of possible sources for the 2007 outbreaks. The probable index case in the Nadia outbreak visited the Kushtia region of Bangladesh in February 2007 and developed a fever within days of his return.[47] The wife of one of the three fatal cases in Nadia said that bats (which could be carrying the virus) are common in her village and even enter homes.[48]

Polio

Global update

There are currently four countries that are considered to have endemic wild polioviruses (India, Afghanistan, Pakistan and Nigeria), while another six countries (Angola, Myanmar, Chad, Democratic Republic of the Congo, Niger and Somalia) are considered to be re-infected, with active transmission of wild polioviruses following imported cases. Between 1 January and 17 July 2007, 239 cases of wild poliovirus have been reported from endemic countries and 55 cases from re-infected countries (each of the six re-infected countries listed above has reported cases in 2007) compared with 669 cases in endemic countries and 72 cases from re-infected countries in the same period of 2006.[49] There are a number of other countries (including Australia) that have had imported cases of polio since 2000, but none have had local transmission since 2003 and all are considered to be polio-free. Countries that have adequate surveillance systems are considered to be polio-free after three consecutive years without local transmission of wild polio viruses.

Pakistan

Polio in Pakistan is of particular interest following the case that was imported to Australia on 1 July 2007. Progress has been made towards the eradication of polio in Pakistan, but wild polioviruses continue to circulate in certain areas. The case of polio that was imported to Australia (a WPV 1 case) may have been acquired in the northern transmission zone. The virus typing indicated a genetic similarity to a strain that was circulating in the North West Frontier Province in 2006 and the case was known to have visited the Swat area in the North West Frontier Province.[50]

Between 1 January and 16 July 2007, Pakistan confirmed 10 cases of polio, compared with 40 cases of wild poliovirus in 2006.[51] In 2007, all cases of polio in Pakistan were located in two transmission zones, one of which borders Afghanistan. Cross-border transmission with Afghanistan remains a challenge in the drive to eradicate polio from Pakistan.[52] Four cases of wild poliovirus have been reported from the transmission zone in the North West Frontier Province in 2007. One of these cases (from the Nowshera district) was a WPV 3 case and the other three (1 each from the Kyber, Nowshera and Peshawar districts) were all WPV 1.[51,53] Cases in the other transmission zone, which spans the border of Sindh and Balochistan Provinces, are predominately WPV 3.[53] Only one WPV 1 case was reported (from the Khibaldia district of Sindh Province) from this zone.[51,53] All of the other cases were WPV 3, with cases in the Jocobabad (2 cases) and Khairpur (1 case) districts of Sindh Province and the nearby Nsirabad district (2 cases) of Balochistan Province.[51]

Tuberculosis

Human immunodeficiency virus and tuberculosis co-infection

A close association between human immunodeficiency virus (HIV) and tuberculosis (TB) is seen in a number of Western Pacific Region countries. TB is often asymptomatic, but co-infection with HIV and TB can lead to a large number of clinical cases, and HIV/TB co-infection is also the leading cause of deaths amongst HIV positive people.[54] The association is of most concern in Papua New Guinea (PNG) and Vietnam where the estimated prevalence of HIV among TB patients is 9.7% and 3% respectively (the prevalence of HIV overall in these countries is 1.8% and approximately 0.9%, respectively).[55–57] While the prevalence of HIV amongst TB patients in Cambodia is higher (9.9%), it has decreased from 11.8% in 2003.[57]

Prevalence and mortality rates of TB in PNG have dropped by 75% and 78% respectively between 2000 and 2005, with prevalence dropping to less than 0.5% (475 cases per 100,000 population),[57] while the prevalence of HIV is rising. The case detection rate of TB (the number of cases notified compared with estimated cases) in PNG is above the average for the Western Pacific Region (76% compared with 63%).[57] There are no data on drug susceptibility of TB in cases in PNG because the country does not have laboratories with the capacity for drug susceptibility testing.[57]

Drug resistant strains

In 2006, extensively drug resistant strains of TB (XDR-TB) with resistance to first line antibiotics and to at least one of the three injectable second-line antibiotics (amikacin, capreomycin or kanamycin) were reported from all regions of the world.[58] XDR-TB is a major threat to international public health, especially in areas with a high prevalence of HIV. In 2007, the WHO expects to treat 5,960 patients for XDR-TB, rising to 9,477 in 2008, which are only 25% and 43% respectively of the estimated number of global cases.[58]

United States of America and international travel

On 12 May 2007, a confirmed case of XDR-TB was found to have travelled on international flights between Atlanta, Europe and Canada, despite advice not to travel.[59,60,61] The US Centers for Disease Control and Prevention coordinated extensive contact tracing and investigation and the WHO released detailed information on the case and on the guidelines for the investigation.[61,62]

Thailand

In early June 2007, AID workers reported two cases of XDR-TB from the Thai border town of Mae Sot, both cases were immigrants from Myanmar. These are the first cases of XDR-TB ever reported from the country. Myanmar is unable to treat serious cases of TB, so many people travel across the border to Thailand to obtain treatment.[63]

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Other reports

New facility to help fight emerging infectious diseases

AusAID in collaboration with the Department of Health and Ageing and the Department of Agriculture, Fisheries and Forestry, has recently established AusReady: the Asia Pacific Emerging Infectious Diseases (EIDs) Facility.

AusReady will identify and mobilise expertise to undertake prevention and preparedness work in the Asia Pacific region. This work will support activities under the Pandemics and EIDs Strategy 2006–2010.

The AusReady database will provide a register of trained and qualified professionals across a range of relevant disciplines, available to work on short- and long-term projects in the Asia Pacific region.

Relevant disciplines include animal and human health, laboratory and clinical operations, epidemiology, health systems, social sciences, gender, capacity development, environment and natural resources, information systems and project/program management.

AusReady is encouraging professionals to register on the database. Application forms can be downloaded from the website or obtained from our Facility Officer via email at ausready@ausready.org.au

AusReady is also seeking expressions of interest from Australian Commonwealth Government agencies and AusAID accredited NGOs to act as Tasking Agencies. Tasking Agencies will identify and fund relevant assignments in the Asia Pacific region.

For more details see the AusReady website at http://www.ausready.org.au