Australian Government
Department of Health and Ageing

Communicable Diseases Intelligence

Volume 31 Number 1 Quarterly report March 2007

Annual reports

Articles

Short reports

Quarterly reports

Other reports

ISBN 0725-3141

Communicable Diseases Intelligence aims to diseminate information on the epidemiology and control of communicable diseases in Australia. *Communicable Diseases Intelligence* invites contributions dealing with any aspect of communicable disease epidemiology, surveillance or prevention and control in Australia. Submissions can be in the form of original articles, short reports, surveillance summaries, reviews or correspondence. Instructions for authors can be found in *Commun Dis Intell* 2007;31:158–162.

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

Website

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

Subscriptions and contacts

Communicable Diseases Intelligence is produced every quarter by:
Surveillance Branch
Office of Health Protection
Australian Government Department of Health and Ageing
GPO Box 9848, (MDP 6)
CANBERRA ACT 2601;
Telephone: +61 2 6289 8245
Facsimile: +61 2 6289 7100
Email: cdi.editor@health.gov.au

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

Disclaimer

Printed by Union Offset, Canberra

Annual Reports

Australia's notifiable diseases status, 2005: annual report of the National Notifiable Diseases Surveillance System

Rhonda Owen, Paul W Roche, Kirsty Hope, Keflemariam Yohannes, April Roberts, Conan Liu, Stefan Stirzaker, Fiona Kong, Mark Bartlett, Basil Donovan, Iain East, Gerard Fitzsimmons, Ann McDonald, Peter B McIntyre, Robert I Menzies

With contributions from:

National organisations

Communicable Diseases Network Australia and subcommittees

Australian Childhood Immunisation Register

Australian Gonococcal Surveillance Programme

Australian Meningococcal Surveillance Programme

Australian Sentinel Practice Research Network

Australian Quarantine Inspection Service

National Centre in HIV Epidemiology and Clinical Research

National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

National Enteric Pathogens Surveillance Scheme

World Health Organization Collaborating Centre for Reference and Research on Influenza

State and territory health departments

Communicable Diseases Control Unit, Australian Capital Territory Department of Health and Community Care, Australian Capital Territory

Communicable Diseases Surveillance and Control Unit, NSW Health Department, New South Wales

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

Communicable Diseases Unit, Queensland Health, Queensland

Communicable Diseases Control Branch, South Australian Department of Human Services, South Australia

Communicable Disease Prevention Unit, Department of Health and Human Services, Tasmania

Communicable Diseases Section, Department of Human Services, Victoria

Communicable Diseases Control Directorate, Department of Health, Western Australia

Abstract

In 2005, 60 diseases and conditions were nationally notifiable in Australia. States and territories reported a total of 125,461 cases of communicable diseases to the National Notifiable Diseases Surveillance System: an increase of 10% on the number of notifications in 2004. In 2005, the most frequently notified diseases were sexually transmissible infections (51,557 notifications, 41% of total notifications), gastrointestinal diseases (29,422 notifications, 23%) and bloodborne diseases (19,278 notifications, 15%). There were 17,753 notifications of vaccine preventable diseases; 4,935 notifications of vectorborne diseases; 1,826 notification of other bacterial infections (legionellosis, leprosy, meningococcal infections and tuberculosis) and 687 notifications of zoonotic diseases. *Commun Dis Intell* 2007;31:1–70.

Keywords: Australia, communicable diseases, epidemiology, notifiable diseases, surveillance

NNDSS annual report contents

Cont'd next page

Annual report contents, *continued*

Tables

Figures

Cont'd next page

Figures, continued

Cont'd next page

Figures, continued

Maps

Abbreviations used in this report

ABL	Australian bat lyssavirus
AIDS	Acquired immune deficiency syndrome
AFP	Acute flaccid paralysis
AGSP	Australian Gonococcal Surveillance Programme
ASPREN	Australian Sentinel Practice Research Network
BFV	Barmah Forest virus
CDI	*Communicable Diseases Intelligence*
CDNA	Communicable Diseases Network Australia
CSF	Cerebrospinal fluid
DENV	Dengue virus
DoHA	Australian Government Department of Health and Ageing
DSS	Dengue shock syndrome
DTP	Diphtheria-tetanus-pertussis vaccine
HBV	Hepatitis B virus
Hib	*Haemophilus influenzae* type b
HIV	Human immunodeficiency virus
HPAIH	Highly pathogenic avian influenza in humans
HUS	Haemolytic uraemic syndrome
ICD10-AM	International Classification of Diseases, version 10, Australian Modification
IPD	Invasive pneumococcal disease
JEV	Japanese encephalitis virus
KUNV	Kunjin virus
LabVISE	Laboratory Virology and Serology Reporting Scheme
MIC	Minimum inhibitory concentration
MMR	Measles-mumps-rubella vaccine
MVE	Murray Valley encephalitis virus
NNDSS	National Notifiable Diseases Surveillance System
NCHECR	National Centre in HIV Epidemiology and Clinical Research
NEC	Not elsewhere classified
NIP	National Immunisation Program
NN	Not notifiable
RRV	Ross River virus
SARS	Severe acute respiratory syndrome
SLTEC	Shiga-like toxin-producing *Escherichia coli*
STI(s)	Sexually transmissible infection(s)
TB	Tuberculosis
VPD(s)	Vaccine preventable disease(s)
VTEC	Verotoxigenic *Escherichia coli*
WHO	World Health Organization

Introduction

Australia's notifiable diseases status, 2005, is an annual surveillance report of nationally notifiable communicable diseases. Communicable disease surveillance in Australia operates at the national, state and local levels. Primary responsibility for public health action lies with the state and territory health departments. The role of communicable disease surveillance at a national level includes:

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

Methods

Australia is a federation of six states (New South Wales, Queensland, South Australia, Tasmania, Victoria and Western Australia) and 2 territories (the Australian Capital Territory and the Northern Territory). State and territory health departments collect notifications of communicable diseases under their public health legislation. The Australian Government Department of Health and Ageing (DoHA) does not have any legislated responsibility for public health apart from human quarantine. States and territories voluntarily forward data on a nationally agreed set of communicable diseases to DoHA for the purposes of national communicable disease surveillance.

Sixty communicable diseases (Table 1) agreed upon nationally through the Communicable Diseases Network Australia (CDNA) are reported to the National Notifiable Diseases Surveillance System (NNDSS). The system is complemented by other surveillance systems that provide information on various diseases, including some that are not reported to NNDSS.

The national dataset included fields for unique record reference number; notifying state or territory; disease code; age; sex; indigenous status; postcode of residence; date of onset of the disease; death, date of report to the state or territory health department and outbreak reference (to identify cases linked to an outbreak). Where relevant, information on the species, serogroups/subtypes and phage types of organisms isolated, and on the vaccination status of the case was collected. While not included in the national dataset, additional information concerning mortality and specific health risk factors for some diseases was obtained from states and territories.

Notification rates for each notifiable disease were calculated using 2005 mid-year resident population supplied by the Australian Bureau of Statistics (Appendix 1). Where diseases were not notifiable in a state or territory, national rates were adjusted by excluding the population of that jurisdiction from the denominator. For some diseases age adjusted rates were calculated using the indirect method of standardisation, with 2001 census data as the standard population.

The geographical distribution of selected diseases was mapped using ArcGIS (ESRI, Redlands, CA, USA) software. Maps were based on the postcode of residence of each patient aggregated to the appropriate Statistical Division (Map 1). Rates for the different Statistical Divisions were ordered into six groups — the highest value, the lowest value above zero, those equal to zero, and the intermediate values sorted into three equal-sized groups. The Statistical Divisions in the Australian Capital Territory were combined to calculate rates for the territory as a whole.

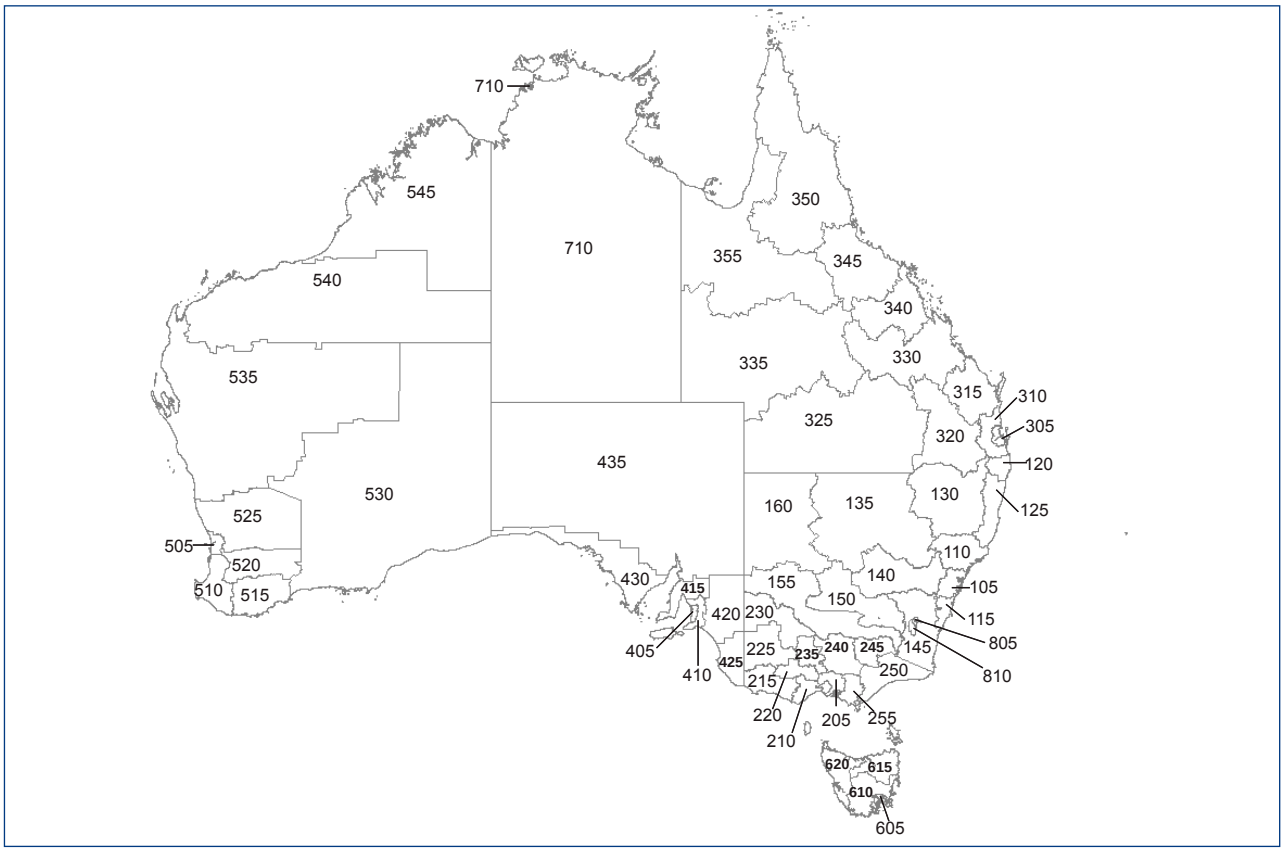
Information from communicable disease surveillance is disseminated through several avenues of communication. At the fortnightly teleconferences of the Communicable Diseases Network Australia the most up-to-date information on topics of interest to the network is provided. The *Communicable Diseases Intelligence* (*CDI*) quarterly journal publishes surveillance data and reports of research studies on the epidemiology and control of various communicable diseases. The Communicable Diseases Australia website publishes disease surveillance summaries from the NNDSS.

Notes on interpretation

The present report is based on 2005 'finalised' data from each state and territory. States and territories transmitted data to NNDSS on average every other day, and the final dataset for the year was agreed upon in June 2006. The finalised annual dataset represents a snap shot of the year after duplicate records and incorrect or incomplete data have been removed. Therefore, totals in this report may vary slightly from the totals reported in *CDI* quarterly publications.

Analyses in this report were based on the date of disease onset in an attempt to estimate disease activ-

Map 1. Australian Bureau of Statistics Statistical Divisions, and population by Statistical Division, 2005



Statistical Division	Population	Statistical Division	Population	Statistical Division	Population
Australian Capital Territory		*Queensland continued*		*Victoria*	
805 Canberra*	325,536	320 Darling Downs	222,478	205 Melbourne	3,634,233
New South Wales		325 South West	26,938	210 Barwon	269,752
105 Sydney	4,254,894	330 Fitzroy	189,838	215 Western District	101,441
110 Hunter	610,526	335 Central West	12,174	220 Central Highlands	148,294
115 Illawarra	414,168	340 Mackay	147,374	225 Wimmera	50,884
120 Richmond-Tweed	225,886	345 Northern	205,628	230 Mallee	92,087
125 Mid-North Coast	295,144	350 Far North	238,454	235 Loddon	175,406
130 Northern	179,103	355 North West	34,167	240 Goulburn	203,989
135 North Western	118,885	*South Australia*		245 Ovens-Murray	96,642
140 Central West	180,064	405 Adelaide	1,129,269	250 East Gippsland	83,126
145 South Eastern	202,757	410 Outer Adelaide	123,924	255 Gippsland	166,492
150 Murrumbidgee	153,871	415 Yorke and Lower North	44,907	*Western Australia*	
155 Murray	115,523	420 Murray Lands	68,756	505 Perth	1,477,815
160 Far West	23,428	425 South East	63,499	510 South West	219,812
Northern Territory		430 Eyre	34,661	515 Lower Great Southern	53,738
705 Darwin	111,300	435 Northern	77,017	520 Upper Great Southern	17,760
710 NT - balance	91,493	*Tasmania*		525 Midlands	52,372
Queensland		605 Greater Hobart	203,638	530 South Eastern	53,661
305 Brisbane	1,810,943	610 Southern	35,806	535 Central	59,925
310 Moreton	818,981	615 Northern	137,936	540 Pilbara	39,282
315 Wide Bay-Burnett	256,993	620 Mersey-Lyell	107,883	545 Kimberley	35,748
		910 *Other Territories*	2,683	Total Australia	20,328,984

* Includes Statistical Division 810 'ACT – balance'.

ity within the reporting period. Where the date of onset was not known however, the date of specimen collection or date of notification, whichever was earliest, was used. As considerable time may have lapsed between onset and diagnosis dates for hepatitis B (unspecified) and hepatitis C (unspecified), for these conditions the date of diagnosis, which is the earliest of specimen, notification or notification received dates supplied, was used.

Notified cases can only represent a proportion (the 'notified fraction') of the total incidence (Figure 1) and this has to be taken into account when interpreting NNDSS data. Moreover, the notified fraction varies by disease, by jurisdiction and by time.

Methods of surveillance vary between states and territories, each having different requirements for notification by medical practitioners, laboratories and hospitals. Although there is a list of national notifiable diseases, some diseases are not yet notifiable in some jurisdictions (Table 1).

Changes in surveillance practices introduced in some jurisdictions and not in others are additional factors that make comparison of data across jurisdictions difficult. In this report, information obtained from states and territories on any changes in surveillance practices including screening practices, laboratory practices, and major disease control or prevention initiatives undertaken in 2005, was used to interpret data.

Postcode information usually reflects the residential location of the case, but this does not necessarily represent the place where the disease was acquired. As no personal identifiers are collected in NNDSS, duplication in reporting may occur if patients move from one jurisdiction to another and were notified in both.

The completeness* of data in this report is summarised in Appendix 3. The case's sex was complete in 99.9% of notifications and date of birth in 99.8% of notifications. In 2005, indigenous status† was complete in 50% of notifications, but varied by jurisdiction. Indigenous status was complete for 100% of data reported in Western Australia, 92.3% in the Northern Territory, 89.2% in South Australia, and 52.4% in Victoria. In the remaining jurisdictions, less than 50% of data were complete for indigenous status.

Data completeness on indigenous status also varied by disease; in notifications of typhoid, syphilis, *Haemophilus influenzae* type B, tuberculosis (TB) and meningococcal infections was more than 90%

* Data completeness = (Total – unknown or missing)/total x 100.

† 'Indigenous status' is a variable defined by the following values:

1. Indigenous – (Aboriginal but not Torres Strait Islander origin);
2. Indigenous – (Torres Strait Islander but not Aboriginal origin);
3. Indigenous – (Aboriginal and Torres Strait Islander origin);
4. Not Indigenous – (not Aboriginal or Torres Strait Islander origin);
9. Not stated

Blank/missing/null =No information provided

Figure 1. Communicable diseases notification fraction

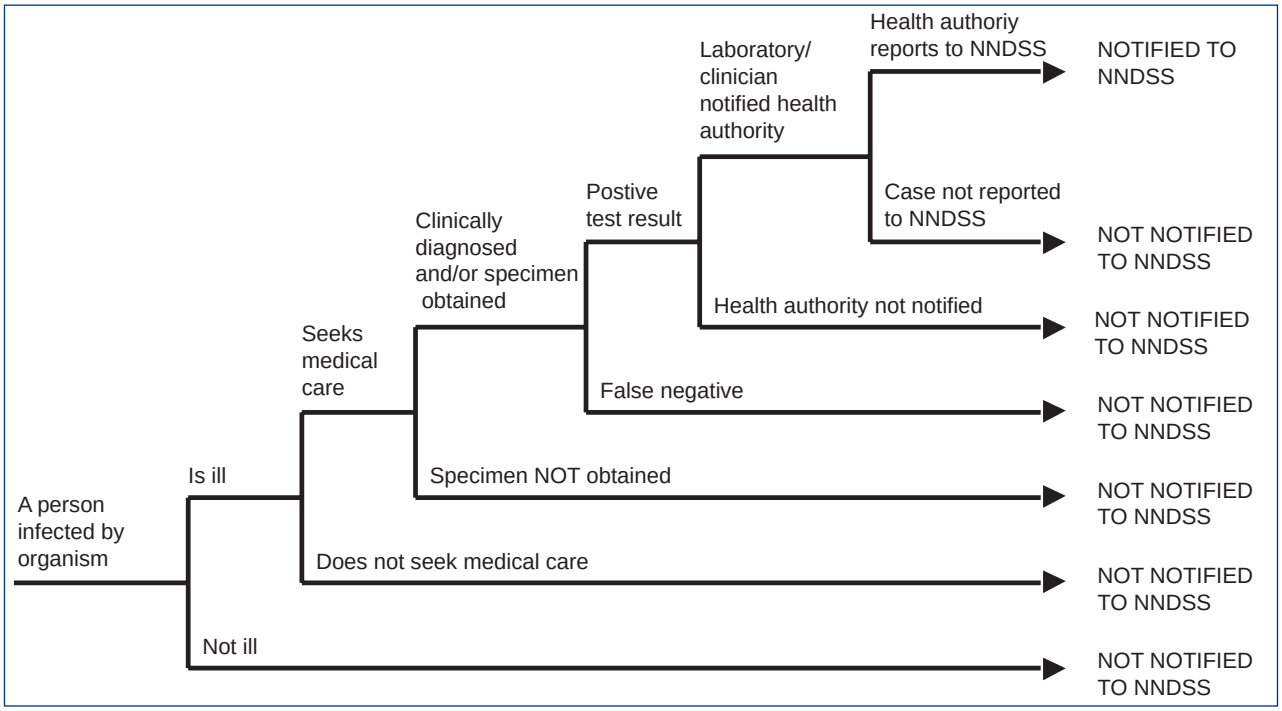


Table 1. Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2005

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 (NEC)	All jurisdictions
Shigellosis	All jurisdictions
SLTEC, VTEC§	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Tularaemia	All jurisdictions except ACT
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infection (NEC)‖	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis (all)¶	All jurisdictions
Syphilis – infectious	All jurisdictions
Syphilis – More than 2 years or unknown duration	All jurisdictions
Syphilis – congenital	All jurisdictions
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

Table 1. Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2005, *continued*

Disease	Data received from
Vectorborne diseases	
Barmah Forest virus infection	All jurisdictions
Dengue	All jurisdictions
Flavivirus infection (NEC)††	All jurisdictions except ACT
Japanese encephalitis virus	All jurisdictions
Kunjin virus‡‡	All jurisdictions except ACT
Malaria	All jurisdictions
Murray Valley encephalitis	All jurisdictions except ACT
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssavirus 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

* Unspecified hepatitis includes cases in whom the duration of infection could not be determined.

† In Queensland, includes incident hepatitis cases.

‡ Notified as 'foodborne disease' or 'gastroenteritis in an institution' in New South Wales.

§ Infection 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; the Northern Territory which excludes ocular specimens; and Western Australia which excludes ocular and perinatal infections.

¶ Does not include congenital syphilis.

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

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

‡‡ In the Australian Capital Territory, Murray Valley encephalitis virus and Kunjin virus are combined under Murray Valley encephalitis virus.

§§ In the Australian Capital Territory, ornithosis is reported as chlamydia not elsewhere classified.

|||| 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.

complete for indigenous status, while in notifications of other diseases such as Barmah Forest virus infection, influenza (laboratory-confirmed), and hepatitis C (unspecified) infections, data completeness was below 40%.

Notes on case definitions

In this report each notifiable disease is introduced with a case definition, the 'CDNA case definition'. These case definitions were agreed upon by CDNA to be implemented nationally by January 2004.

CDNA case definitions are only intended for reporting to NNDSS. In 2005 they were used by all jurisdictions for the first time. States and territories may also have case definitions which reflect their local public health needs. These may be the same as or more comprehensive than the CDNA case definitions.

Results

Summary of 2005 data

There were 125,461 communicable disease notifications received by NNDSS in 2005 (Table 2). Notification rates per 100,000 population for each disease by state or territory are shown in Table 3. Trends in notifications and rates per 100,000 population for the period 2001 to 2005 are shown in Table 4.

Table 2. Notifications of communicable diseases, Australia, 2005, by state or territory

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis B (incident)	5	56	5	59	8	3	78	31	245
Hepatitis B (unspecified)*	88	2,711	199	945	325	54	1,679	395	6,396
Hepatitis C (incident)	11	41	3	NN	50	26	122	104	357
Hepatitis C (unspecified)*,†	163	4,424	254	2,790	559	215	2,861	984	12,250
Hepatitis D	0	15	0	11	0	0	2	2	30
Gastrointestinal diseases									
Botulism	0	0	0	2	0	0	1	0	3
Campylobacteriosis‡	402	NN	248	4,416	2,089	760	6,109	2,444	16,468
Cryptosporidiosis	27	851	82	1,360	167	22	518	182	3,209
Haemolytic uraemic syndrome	0	11	0	2	1	2	3	1	20
Hepatitis A	3	83	64	50	10	2	59	54	325
Hepatitis E	2	7	0	8	0	0	12	2	31
Listeriosis	3	25	0	7	6	0	9	4	54
Salmonellosis (NEC)	95	2,179	393	2,613	577	302	1,481	801	8,441
Shigellosis	7	135	196	80	48	5	105	156	732
SLTEC, VTEC§	0	16	0	9	40	2	8	12	87
Typhoid	0	28	0	3	2	0	11	8	52
Quarantinable diseases									
Cholera	0	0	0	0	0	0	2	1	3
Plague	0	0	0	0	0	0	0	0	0
Rabies	0	0	0	0	0	0	0	0	0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0
Smallpox	0	0	0	0	0	0	0	0	0
Tularaemia	NN	0	0	0	0	0	0	0	0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0
Yellow fever	0	0	0	0	0	0	0	0	0
Sexually transmitted infections									
Chlamydial infection (NEC)‖	700	11,283	1,583	9,721	2,706	871	9,004	5,443	41,311
Donovanosis	0	0	4	8	0	0	0	1	13
Gonococcal infection	33	1,577	1,738	1,444	399	35	1,208	1,581	8,015
Syphilis (all)¶	14	845	229	380	18	30	496	191	2,203
Syphilis < 2 years duration	4	244	93	128	7	6	120	19	621
Syphilis > 2 years or unknown duration	10	601	136	252	11	24	376	172	1,582
Syphilis – congenital	0	8	5	2	0	0	0	0	15

Table 2. Notifications of communicable diseases, Australia, 2005, by state or territory, *continued*

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Vaccine preventable diseases									
Diphtheria	0	0	0	0	0	0	0	0	0
Haemophilus influenzae type b	0	7	1	4	0	0	3	2	17
Influenza (laboratory confirmed)**	39	1,414	61	1,698	275	19	595	466	4,567
Measles	0	5	0	1	0	1	2	1	10
Mumps	1	111	7	71	8	0	20	23	241
Pertussis	315	5,802	92	1,775	1,507	33	1,163	513	11,200
Pneumococcal disease (invasive)	30	641	71	325	134	44	300	139	1,684
Poliomyelitis	0	0	0	0	0	0	0		0
Rubella	0	10	0	9	0	0	6	6	31
Rubella – congenital	0	0	0	0	0	0	1	0	1
Tetanus	0	1	0	0	0	1	0	0	2
Vectorborne diseases									
Barmah Forest virus infection	0	448	51	680	40	1	16	83	1,319
Dengue	2	48	14	115	5	0	16	18	218
Flavivirus infection (NEC)††	NN	6	0	20	0	0	3	0	29
Japanese encephalitis virus	0	0	0	0	0	0	0	0	0
Kunjin virus‡‡	NN	0	0	1	0	0	0	0	1
Malaria	12	204	47	297	43	24	110	85	822
Murray Valley encephalitis virus	NN	0	1	1	0	0	0	0	2
Ross River virus infection	6	585	209	1,179	153	5	96	311	2,544
Zoonoses									
Anthrax	0	0	0	0	0	0	0	0	0
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0
Brucellosis	0	3	0	37	0	0	1	0	41
Leptospirosis	0	35	5	72	3	0	10	5	130
Ornithosis§§	0	121	0	2	1	0	34	3	161
Lyssavirus unspecified	0	0	0	0	0	0	0	0	0
Q fever	0	142	3	157	17	0	30	6	355
Other bacterial infections									
Legionellosis	0	89	3	49	58	3	63	70	335
Leprosy	0	1	3	3	0	0	0	3	10
Meningococcal infection‖‖	8	140	11	62	26	10	90	47	394
Tuberculosis	0	453	27	114	46	13	367	67	1,087
Total	1,966	34,561	5,609	30,582	9,321	2,483	26,694	14,245	125,461

* Unspecified hepatitis includes cases in whom the duration of infection could not be determined.

† In Queensland, includes incident hepatitis cases.

‡ Notified as 'foodborne disease' or 'gastroenteritis in an institution' in New South Wales.

§ Infection 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; the Northern Territory which excludes ocular specimens; and Western Australia which excludes ocular and perinatal infections.

¶ Does not include congenital syphilis.

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

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

‡‡ In the Australian Capital Territory, Murray Valley encephalitis virus and Kunjin virus are combined under Murray Valley encephalitis virus.

§§ In the Australian Capital Territory, ornithosis is reported as chlamydia not elsewhere classified.

‖‖ 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.

Table 3. Notification rate for communicable diseases, Australia, 2005, by state and territory (per 100,000 population)

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis B (incident)	1.5	0.8	2.5	1.5	0.5	0.6	1.6	1.5	1.2
Hepatitis B (unspecified)*	2.5	40.0	98.1	23.8	21.1	11.1	33.4	19.7	31.5
Hepatitis C (incident)	3.4	0.6	1.5	NN	3.2	5.4	2.4	5.2	1.8
Hepatitis C (unspecified)*,†	50.1	65.3	125.3	70.4	36.3	44.3	57.0	49.0	60.3
Hepatitis D	0.0	0.2	0.0	0.3	0.0	0.0	0.0	0.1	0.1
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis‡	123.6	NN	122.3	111.4	135.5	156.6	121.6	121.6	121.5
Cryptosporidiosis	8.3	12.6	40.4	34.3	10.8	4.5	10.3	9.1	15.8
Haemolytic uraemic syndrome	0.0	0.2	0.0	0.1	0.1	0.4	0.1	0.0	0.1
Hepatitis A	0.9	1.2	31.6	1.3	0.6	0.4	1.2	2.7	1.6
Hepatitis E	0.6	0.1	0.0	0.2	0.0	0.0	0.2	0.1	0.2
Listeriosis	0.9	0.4	0.0	0.2	0.4	0.0	0.2	0.2	0.3
Salmonellosis (NEC)	29.2	32.2	193.8	65.9	37.4	62.2	29.5	39.8	41.5
Shigellosis	2.2	2.0	96.7	2.0	3.1	1.0	2.1	7.8	3.6
SLTEC,VTEC§	0.0	0.2	0.0	0.2	2.6	0.4	0.2	0.6	0.4
Typhoid	0.0	0.4	0.0	0.1	0.1	0.0	0.2	0.4	0.3
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tularaemia	NN	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 transmitted infections									
Chlamydial infections (NEC)‖	215.3	166.6	780.6	245.2	156.0	179.5	179.3	270.8	203.2
Donovanosis	0.0	0.0	2.0	0.2	0.0	0.0	0.0	0.0	0.1
Gonococcal infection	10.1	23.3	857.0	36.4	25.9	82.2	24.1	78.7	39.4
Syphilis (all)¶	4.3	12.5	112.9	9.6	1.2	6.2	9.9	9.5	10.8
Syphilis < 2 years duration	1.2	3.6	45.9	3.2	0.5	1.2	2.4	0.9	3.1
Syphilis > 2 years or unknown duration	3.1	8.9	67.1	6.4	0.7	4.9	7.5	8.6	7.8
Syphilis – congenital	0.0	0.1	2.5	0.1	0.0	0.0	0.0	0.0	0.1
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	0.5	0.1	0.0	0.0	0.1	0.1	0.1
Influenza (laboratory confirmed)**	12.0	20.9	30.1	42.8	17.8	3.9	11.8	23.2	22.5
Measles	0.0	0.1	0.0	0.0	0.0	0.2	0.0	0.0	0.0
Mumps	0.3	1.6	3.5	1.8	0.5	0.0	0.4	1.1	1.2
Pertussis	96.9	85.6	45.4	44.8	97.7	6.8	23.2	25.5	55.1
Pneumococcal disease (invasive)	9.2	9.5	35.0	8.2	8.7	9.1	6.0	6.9	8.3
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.1	0.0	0.2	0.0	0.0	0.1	0.3	0.2
Rubella – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0

Table 3. Notification rate for communicable diseases, Australia, 2005, by state and territory (per 100,000 population), *continued*

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Vectorborne diseases									
Barmah Forest virus infection	0.0	6.6	25.1	17.2	2.6	0.2	0.3	4.1	6.5
Dengue	0.6	0.7	6.9	2.9	0.3	0.0	0.3	0.9	1.1
Flavivirus infection (NEC)††	NN	0.1	0.0	0.5	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‡‡	NN	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	3.7	3.0	23.2	7.5	2.8	4.9	2.2	4.2	4.0
Murray Valley encephalitis virus	NN	0.0	0.5	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	1.8	8.6	103.1	29.7	9.9	1.0	1.9	15.5	12.5
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	0.9	0.0	0.0	0.0	0.0	0.2
Leptospirosis	0.0	0.5	2.5	1.8	0.2	0.0	0.2	0.2	0.6
Lyssavirus unspecified	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis§§	0.0	1.8	0.0	0.1	0.1	0.0	0.7	0.1	0.8
Q fever	0.0	2.1	1.5	4.0	1.1	0.0	0.6	0.3	1.7
Other bacterial infections									
Legionellosis	0.0	1.3	1.5	1.2	3.8	0.6	1.3	3.5	1.6
Leprosy	0.0	0.0	1.5	0.1	0.0	0.0	0.0	0.1	0.0
Meningococcal infection‖‖	2.5	2.1	5.4	1.6	1.7	2.1	1.8	2.3	1.9
Tuberculosis	0.0	6.7	13.3	2.9	3.0	2.7	7.3	3.3	5.3

* Unspecified hepatitis includes cases in whom the duration of infection could not be determined.

† In Queensland, includes incident hepatitis cases.

‡ Notified as 'foodborne disease' or 'gastroenteritis in an institution' in New South Wales.

§ Infection 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; the Northern Territory which excludes ocular specimens; and Western Australia which excludes ocular and perinatal infections.

¶ Does not include congenital syphilis.

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

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

‡‡ In the Australian Capital Territory, Murray Valley encephalitis virus and Kunjin virus are combined under Murray Valley encephalitis virus.

§§ In the Australian Capital Territory, ornithosis is reported as chlamydia not elsewhere classified.

‖‖ 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.

Table 4. Notifications and notification rate for communicable diseases, Australia, 2001 to 2005, (per 100,000 population)

Disease	Notifications					Rate per 100,000 population				
	2001	2002	2003	2004	2005	2001	2002	2003	2004	2005
Bloodborne diseases										
Hepatitis B (incident)	422	383	345	282	245	2.2	2.0	1.7	1.4	1.2
Hepatitis B (unspecified)*	8,025	6,353	5,824	5,829	6,396	41.2	32.3	29.3	29.0	31.5
Hepatitis C (incident)	694	438	518	453	357	3.6	2.2	2.6	2.3	1.8
Hepatitis C (unspecified)*,†	19,370	14,462	13,716	12,993	12,250	99.4	73.6	69.0	64.6	60.3
Hepatitis D	20	20	27	28	30	0.1	0.1	0.1	0.1	0.1
Gastrointestinal diseases										
Botulism	2	0	1	1	3	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis‡	16,134	14,740	15,357	15,579	16,468	125.3	113.3	116.4	116.4	121.5
Cryptosporidiosis	1,629	3,266	1,223	1,684	3,209	8.4	16.6	6.2	8.4	15.8
Haemolytic uraemic syndrome	3	12	15	16	20	0.0	0.1	0.1	0.1	0.1
Hepatitis A	539	388	431	319	325	2.8	2.0	2.2	1.6	1.6
Hepatitis E	14	12	12	28	31	0.1	0.1	0.1	0.1	0.2
Listeriosis	64	62	69	67	54	0.3	0.3	0.3	0.3	0.3
Salmonellosis (NEC)	7,050	7,699	7,008	7,834	8,441	36.2	39.2	35.2	39.0	41.5
Shigellosis	567	504	442	520	732	2.9	2.6	2.2	2.6	3.6
SLTEC, VTEC§	46	58	52	49	87	0.2	0.3	0.3	0.2	0.4
Typhoid	77	68	51	76	52	0.4	0.3	0.3	0.4	0.3
Quarantinable diseases										
Cholera	4	5	1	5	3	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
Rabies	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Smallpox	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Tularaemia	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
Yellow fever	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Sexually transmitted infections										
Chlamydial infections (NEC)‖	20,330	24,043	30,439	36,227	41,311	104.3	122.4	153.1	180.1	203.2
Donovanosis	32	17	16	10	13	0.2	0.1	0.1	0.0	0.1
Gonococcal infection	6,291	6,279	6,792	7,187	8,015	32.3	32.0	34.2	35.7	39.4
Syphilis (all)¶	1,851	1,958	2,007	2,332	2,203	9.5	10.0	10.1	11.6	10.8
Syphilis < 2 years duration	0	0	0	615	621	0.0	0.0	0.0	3.1	3.1
Syphilis > 2 years or unknown duration	1,851	1,958	2,007	1,717	1,582	9.5	10.0	10.1	8.5	7.8
Syphilis – congenital	21	18	13	12	15	0.1	0.1	0.1	0.1	0.1
Vaccine preventable diseases										
Diphtheria	1	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	20	30	19	15	17	0.1	0.2	0.1	0.1	0.1
Influenza (laboratory confirmed)**	1,294	3,652	3,483	2,133	4,567	6.6	18.6	17.5	10.6	22.5
Measles	141	32	93	45	10	0.7	0.2	0.5	0.2	0.0
Mumps	116	67	77	102	241	0.6	0.3	0.4	0.5	1.2
Pertussis	9,506	5,407	5,096	8,752	11,200	48.8	27.5	25.6	43.5	55.1
Poliomyelitis	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Pneumococcal disease (invasive)	1,761	2,432	2,238	2,296	1,684	9.0	12.4	11.3	11.4	8.3
Rubella	264	254	54	31	31	1.4	1.3	0.3	0.2	0.2
Rubella – congenital	0	1	3	1	1	0.0	0.0	0.0	0.0	0.0
Tetanus	3	4	4	5	2	0.0	0.0	0.0	0.0	0.0

Table 4. Notifications and notification rate for communicable diseases, Australia, 2001 to 2005, (per 100,000 population), *continued*

Disease	Notifications					Rate per 100,000 population				
	2001	2002	2003	2004	2005	2001	2002	2003	2004	2005
Vectorborne diseases										
Barmah Forest virus infection	1,143	867	1,369	1,106	1,319	5.9	4.4	6.9	5.5	6.5
Dengue	131	165	860	351	218	0.7	0.8	4.3	1.7	1.1
Flavivirus infection (NEC)††	88	72	60	61	29	0.5	0.4	0.3	0.3	0.1
Japanese encephalitis virus	0	0	1	1	0	0.0	0.0	0.0	0.0	0.0
Kunjin virus‡‡	5	0	18	12	1	0.0	0.0	0.1	0.1	0.0
Malaria	719	462	595	558	822	3.7	2.4	3.0	2.8	4.0
Murray Valley encephalitis virus	6	2	0	1	2	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	3,226	1,451	3,850	4,210	2,544	16.6	7.4	19.4	20.9	12.5
Zoonoses										
Anthrax	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
Brucellosis	21	40	20	39	41	0.1	0.2	0.1	0.2	0.2
Leptospirosis	250	159	127	177	130	1.3	0.8	0.6	0.9	0.6
Ornithosis§§	137	199	199	237	161	0.7	1.0	1.0	1.2	0.8
Lyssavirus unspecified	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Q fever	693	762	562	463	355	3.6	3.9	2.8	2.3	1.7
Other bacterial infections										
Legionellosis	310	313	333	312	335	1.6	1.6	1.7	1.6	1.6
Leprosy	10	6	5	7	10	0.1	0.0	0.0	0.0	0.0
Meningococcal infection‖‖	686	681	558	405	394	3.5	3.5	2.8	2.0	1.9
Tuberculosis	932	1,041	959	1,061	1,087	4.8	5.3	4.8	5.3	5.3
Total	104,648	98,884	104,942	113,912	125,461					

* Unspecified hepatitis includes cases in whom the duration of infection could not be determined.

† In Queensland, includes incident hepatitis cases.

‡ Notified as 'foodborne disease' or 'gastroenteritis in an institution' in New South Wales.

§ Infection 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; the Northern Territory which excludes ocular specimens; and Western Australia which excludes ocular and perinatal infections.

¶ Does not include congenital syphilis.

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

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

‡‡ In the Australian Capital Territory, Murray Valley encephalitis virus and Kunjin virus are combined under Murray Valley encephalitis virus.

§§ In the Australian Capital Territory, ornithosis is reported as chlamydia not elsewhere classified.

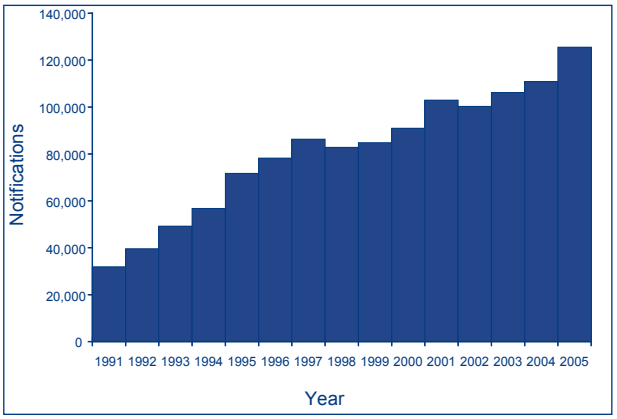
‖‖ Only invasive meningococcal disease is nationally notifiable. However, New South Wales, the Australian Capital Territory and South Australia also report conjunctival cases.

NN Not notifiable.

NEC Not elsewhere classified.

In 2005, the total number of notifications was the highest recorded in NNDSS since the system began in 1991. There was an increase of 10% compared to the total number of notifications in 2004 (Figure 2).

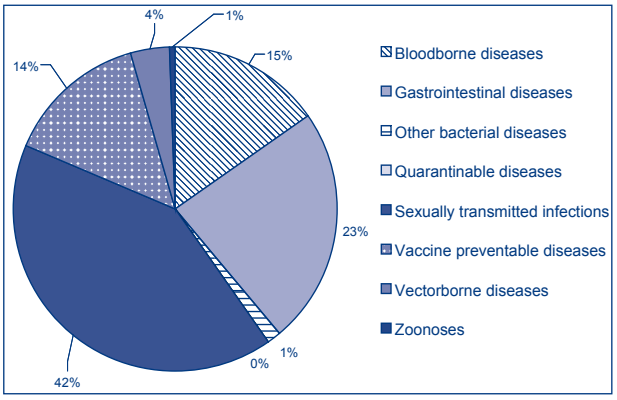
Figure 2. Trends in notifications received by the National Notifiable Diseases Surveillance System, Australia, 1991 to 2005



In 2005, the most frequently notified diseases were sexually transmissible infections (51,557 notifications, 41% of total notifications), gastrointestinal diseases (29,422 notifications, 23%) and bloodborne diseases (19,278 notifications, 15%).

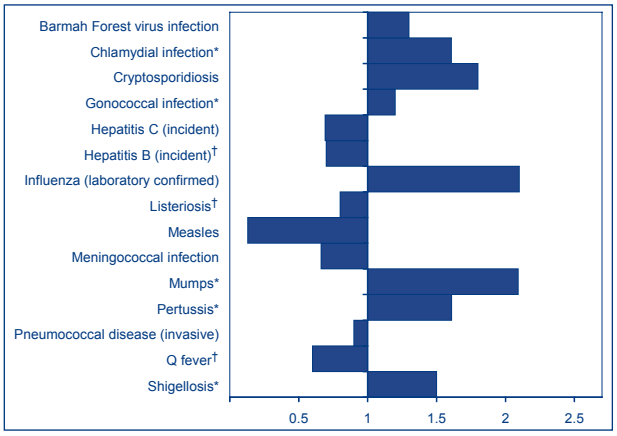
There were 17,753 notifications of vaccine preventable diseases; 4,935 notifications of vectorborne diseases; 1,826 notification of other bacterial infections and 687 notifications of zoonotic diseases (Figure 3).

Figure 3. Notifications to the National Notifiable Diseases Surveillance System, Australia, 2005, by disease category



The major changes in communicable disease notifications in 2005 are shown in Figure 4 as the ratio of notifications in 2005 to the mean number of notifications for the previous 5 years. The number of notifications of chlamydial, gonococcal, shigellosis, mumps, pertussis, SLTEC/VTEC and hepatitis E infections surpassed the expected range (5-year mean plus 2 standard deviations). Notifications of hepatitis B (incident), Q fever, flavivirus and listeriosis infections were below the expected range (5-year mean minus 2 standard deviations). Notifications for the remaining diseases were within the historical range.

Figure 4. Comparison of total notifications of selected diseases reported to the National Notifiable Diseases Surveillance System in 2005, with the previous 5-year mean



* Number of notifications surpassed the expected range (i.e. 5-year mean +2 standard deviations).

† Number of notifications was less than the expected range (i.e. 5-year mean –2 standard deviations).

In the financial year 2004–05, there were 87,520 hospital separations in Australian hospitals with a primary diagnosis of infectious diseases (International Classification of Diseases, version 10, Australian Modification (ICD10–AM) codes A01–B99, Australian Institute of Health and Welfare). This represents 1.2% of all hospital separations in that period. A further 65,494 separations were recorded with a principal diagnosis of influenza or pneumonia (ICD10–AM J10–J18).[1]

Bloodborne diseases

Bloodborne viruses reported to the NNDSS include hepatitis B, C, and D. HIV and AIDS diagnoses are reported directly to the National Centre in HIV Epidemiology and Clinical Research (NCHECR). Information on national HIV/AIDS surveillance can be obtained through the NCHECR website at www.med.unsw.edu.au/nchecr

Hepatitis B

Incident hepatitis B notifications

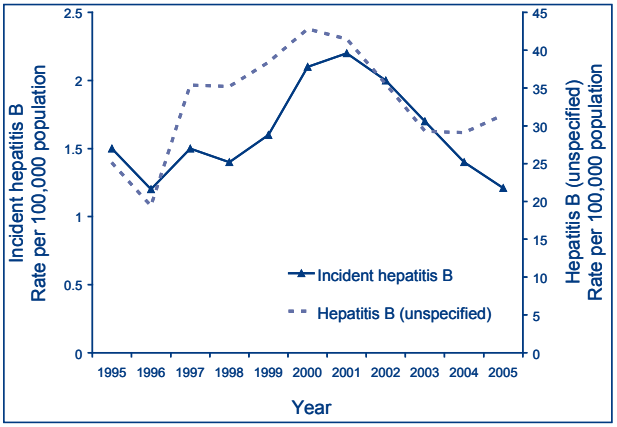
Case definition – Hepatitis B (incident)

*Only **confirmed cases** are reported.*

***Confirmed case:** Detection of hepatitis B surface antigen (HBsAg) in a case shown to be negative within the last 24 months, OR detection of hepatitis HBsAg and IgM to hepatitis B core antigen in the absence of prior evidence of hepatitis B infection OR detection of hepatitis B virus by nucleic acid testing and IgM to hepatitis B core antigen in the absence of evidence of prior hepatitis B infection.*

In 2005, 245 cases of incident hepatitis B infection were reported to NNDSS, giving a national notification rate of 1.2 cases per 100,000 population. The Northern Territory recorded the highest notification rate in 2005 with 2.5 cases per 100,000 population. Over the past 10 years, the rate of notification of incident hepatitis B infection increased from 1.5 cases per 100,000 population in 1996 to 2.2 cases per 100,000 population in 2002 and then declined to 1.2 cases per 100,000 population in 2005 (Figure 5).

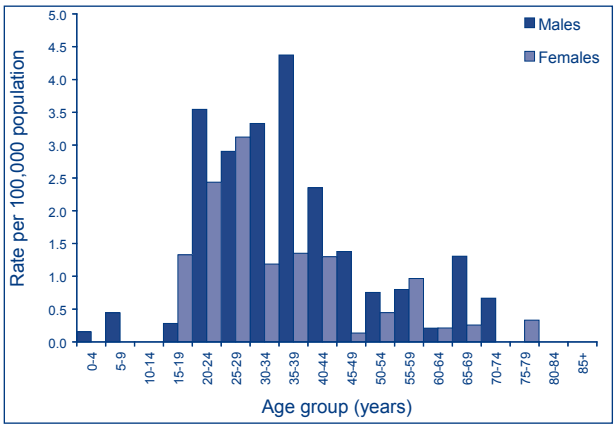
Figure 5. Notification rate of incident hepatitis B and hepatitis B (unspecified), Australia, 1995–2005, by year*



* Year of onset for incident hepatitis B and year of report for hepatitis B (unspecified) notifications.

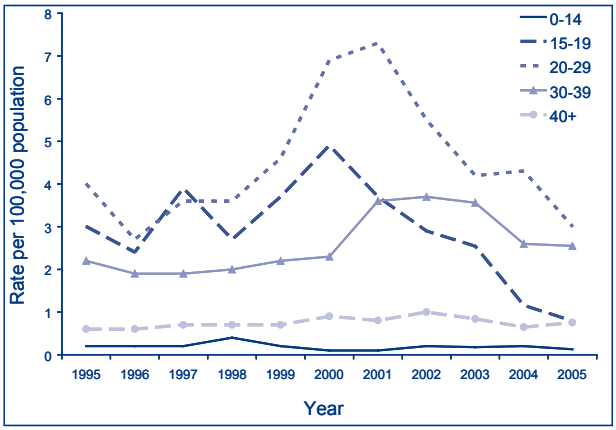
In 2005, the 35–39 year age group among males (4.4 cases per 100,000 population) had the highest rate of incident hepatitis B infection whereas the 25–29 year age group had the highest notification rate (3.1 cases per 100,000 population) (Figure 6) among females. Notifications of incident hepatitis B infection in males exceeded those in females, with a male to female ratio of 1.8:1 in 2005.

Figure 6. Notification rate of incident hepatitis B infections, Australia, 2005, by age group and sex



Trends in incident hepatitis B infection by year and age group are shown in Figure 7. In 2001–2005, the notification rate of incident hepatitis B fell by 81% among cases in the 15–19 year age group, and by 58% among cases in the 20–29 year age group. Increased adolescent vaccine coverage may have played a role in this reduction.

Figure 7. Notification rate of incident hepatitis B infections, Australia, 1995 to 2005, by year and age group



The source of exposure for cases of incident hepatitis B infection in 2005 was reported from South Australia, Victoria and the Australian Capital Territory (Table 5). In 2002–2005, the proportion of notifications of newly-acquired hepatitis B infection associated with injecting drug use, or heterosexual contact only, remained relatively stable at around 45%–50% and 21%–22%, respectively. The proportion of notifications of newly-acquired hepatitis B infections with an undetermined source of exposure to hepatitis B virus (HBV) declined from 23% in 2002 to 15% in 2005.

Table 5. Incident hepatitis B infection, Australia,* 2005, by exposure category

Exposure category	Number	Percentage
Injecting drug use	45	46
Sexual contact	33	34
Male homosexual contact	7	21
Heterosexual contact	22	67
Not specified	4	12
Blood/tissue recipient	0	0
Skin penetration procedure	1	1
Healthcare exposure	0	0
Household contact	3	3
Other	1	1
Undetermined	15	15
Total	98	100

Source: National Centre in HIV Epidemiology and Clinical Research 2006.[2]

* Data from South Australia, Victoria and the Australian Capital Territory only.

Hepatitis B (unspecified) notifications

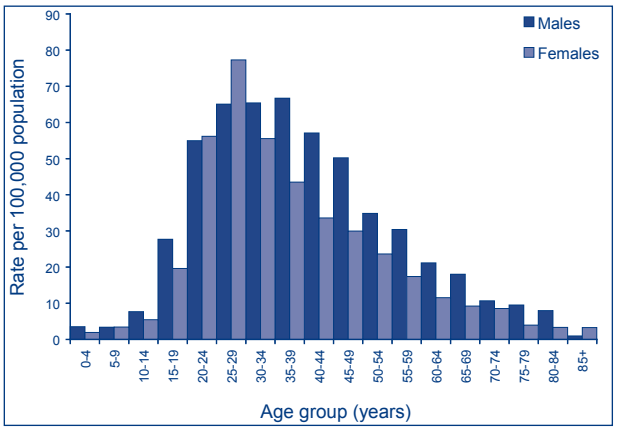
Case definition – Hepatitis B – unspecified

*Only **confirmed cases** are reported.*

***Confirmed case:** Detection of hepatitis B surface antigen or hepatitis B virus by nucleic acid testing in a case who does not meet any of the criteria for a newly acquired case.*

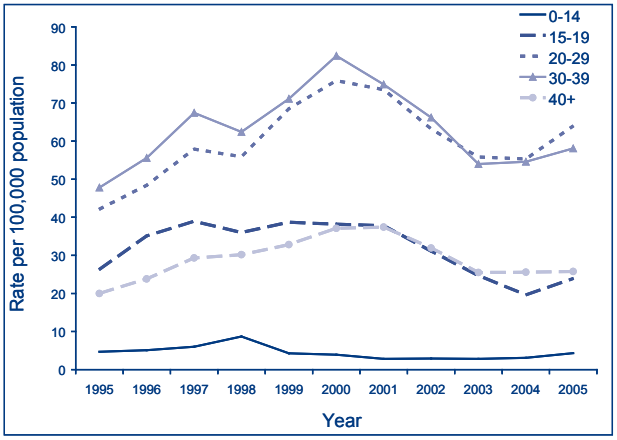
In 2005, a total of 6,396 cases of hepatitis B (unspecified) infection were notified to NNDSS, giving a rate of 31.5 cases per 100,000 population. The Northern Territory (98.1 cases per 100,000 population), New South Wales (40.0 cases per 100,000 population) and Victoria (33.4 cases per 100,000 population) recorded the highest notification rates. The male to female ratio was 1.7:1. Among males, the highest notification rate was in the 25–29, 30–34 and the 35 –39 year age groups (65.0 cases per 100,000 population), whereas among females, the highest notification rate was in the 25–29 year age group (77.0 cases per 100,000 population, Figure 8).

Figure 8. Notification rate of hepatitis B (unspecified) infections, Australia, 2005, by age group and sex



Notifications of hepatitis B infection (unspecified) increased from 19.4 in 1996 to 42.8 in 2000 and then declined to around 29 cases per 100,000 population in 2003–2005 (Figure 5). Trends in hepatitis B (unspecified) infection by age group and year are shown in Figure 9. In 2005, rates of hepatitis B (unspecified) notifications remained stable compared to 2003 and 2004 rates. There were marginal increases in the 15–19 and 20–29 year age groups by 22% and 17%, respectively.

Figure 9. Notification rate of hepatitis B (unspecified) infections, Australia, 1995 to 2005, by year and age group



In 2005, 36 cases of HBV (1 incident and 35 unspecified) infection in children in the 0–4 year age group were reported. Approximately 95% of infants born in Australia in 2005 received hepatitis B vaccination (http://www.ncirs.usyd.edu.au, 2006).

Hepatitis C

Incident hepatitis C notifications

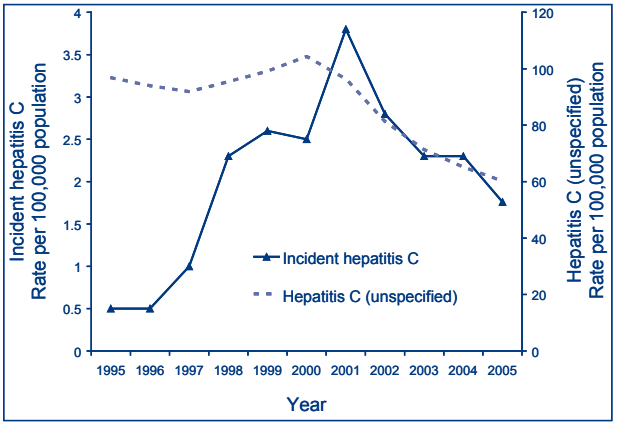
Case definition – Hepatitis C (newly acquired - incident)

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires detection of anti-hepatitis C antibody or detection of hepatitis C virus in a case with a negative test recorded in the last 24 months OR Detection of anti-hepatitis C antibody in a case aged 18 to 24 months or detection of hepatitis C virus in a case aged 1 to 24 months OR detection of anti-hepatitis C antibody or hepatitis C virus AND clinical hepatitis within the last 24 months (defined as jaundice, urine bilirubin or ALT seven times the upper limit of normal) where other causes of acute hepatitis have been excluded.*

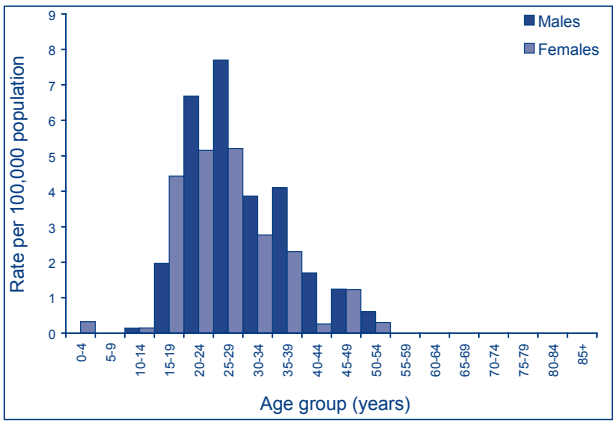
A total of 357 incident cases of hepatitis C with an onset date in 2005 were notified, giving a notification rate of 1.8 cases per 100,000 population (Figure 10). The proportion of all hepatitis C notifications in 2005 that were documented as incident cases was 3%. The highest rate of incident hepatitis C infection was reported from Tasmania and Western Australia (5.2 cases per 100,000 population).

Figure 10. Notification rates for hepatitis C infections (incident and unspecified), Australia, 1995 to 2005



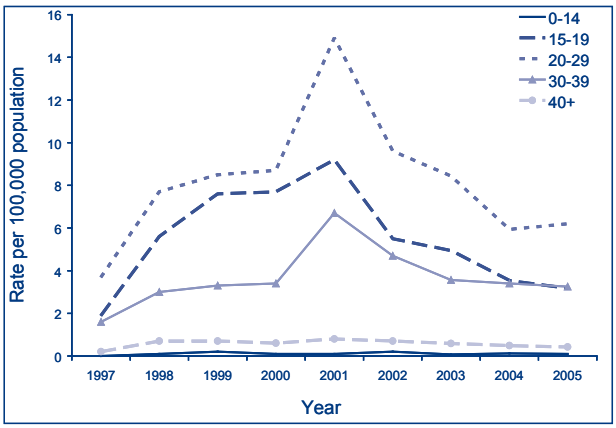
In 2005, the highest rate of incident hepatitis C notification was in the 25–29 age group in males (7.7 cases per 100,000 population) and in the 20–24 and 25–29 age groups in females (5.2 cases per 100,000 population) (Figure 11).

Figure 11. Notification rate of incident hepatitis C infections, Australia, 2005, by age group and sex



Trends in the age distribution of incident hepatitis C infection are shown in Figure 12. In 2001–2005, notification rates declined by 56% in the 15–19 year age group, by 51% in the 20–29 year age range and by 43% in the 30–39 year age range.

Figure 12. Notification rate of incident hepatitis C infections, Australia, 1997 to 2005, by year and age group



The exposure history of cases of incident hepatitis C was collected in the Australian Capital Territory, South Australia, Victoria and Western Australia in 2005 (Table 6). At least 65% of incident hepatitis C infections were among people with a history of injecting drug use.

A total of 9,700 cases (range 6,600–13,200 cases) of incident hepatitis C infection were estimated to have occurred in Australia in 2005.[3] This means that one in 27 incident cases (range 1 in 18 to 1 in 37 cases) had been notified.

Table 6. Incident hepatitis C infection, Australia,* 2005, by exposure category

Exposure category	Number	Percentage
Injecting drug use	261	62.3
Sexual contact	11	2.6
Blood/tissue recipient	1	0.2
Skin penetration procedure	7	1.7
Healthcare exposure	1	0.2
Household contact	3	0.7
Other	18	4.3
Undetermined	117	27.9
Total	419	100.0

* Data from the Australian Capital Territory, South Australia, Tasmania, Victoria and Western Australia only, (NCHECR, 2006[2]).

Hepatitis C (unspecified) notifications

Case definition – Hepatitis C (unspecified)

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires detection of anti-hepatitis C antibody or detection of hepatitis C virus in a case who does not meet any of the criteria for a newly acquired case and is aged more than 24 months.*

In 2005, 12,250 hepatitis C (unspecified) infections were notified to NNDSS, giving a notification rate of 64.6 cases per 100,000 population. The national notification rate for hepatitis C (unspecified) infection declined from 104 cases per 100,000 population in 2000 to 64.6 cases per 100,000 population in 2005 (Figure 10). Improved surveillance practice, such as better classification of incident cases and increased duplicate checking, may account for some of the decrease in hepatitis C (unspecified) notifications.

In 2005, the Northern Territory continued to have the highest notification rate (125.3 cases per 100,000 population). Nationally, the male to female ratio was 1.7:1. The highest notification rates occurred in the age groups 25–29, 30–34 and 35–39 year age groups (166.3 cases per 100,000 population) among males and in the 25–29 year age group (103.9 cases per 100,000 population) among females (Figure 13).

Trends in the age distribution of hepatitis C (unspecified) infection are shown in Figure 14. Between 2000 and 2005, the notification rates of hepatitis C

Figure 13. Notification rate of hepatitis C (unspecified) infections, Australia, 2005, by age group and sex

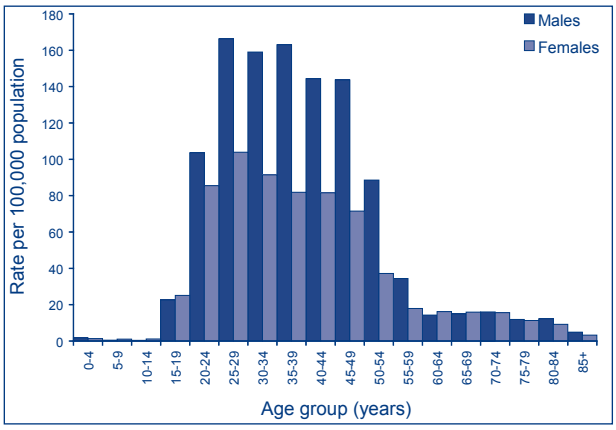
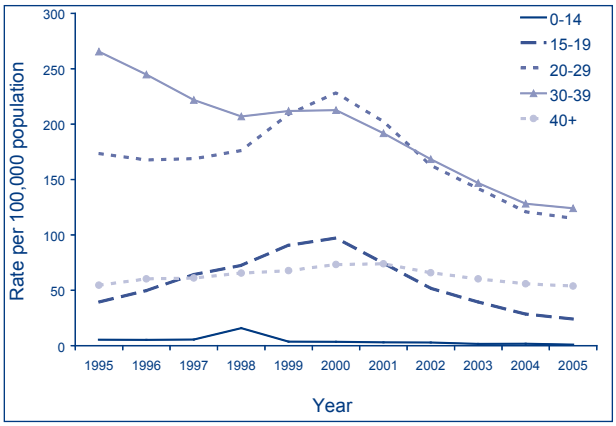


Figure 14. Notification rate of hepatitis C (unspecified) infection, Australia, 1995 to 2005, by age group



(unspecified) among the 15–19 year age group decreased on average by 19% per year, and in 2005 it decreased by 38%. Notification rates also fell by 4% per year in the same period (2000 to 2005) among cases in the 20–29 year age group and by 32% in 2005. Rates in the other age groups remained relatively stable during this period. The decline in the rate of notification of hepatitis C infection may be attributable to a reduction in risk behaviour related to drug injecting among young people, but changes in the rates of testing may also have contributed to the decline.

In 2005, an estimated 197,300 people were living in Australia with chronic hepatitis C infection, of which 153,900 had early liver disease (Stage 0/1); 38,100 had moderate liver disease (Stage 2/3); and 5,300 were living with hepatitis C related cirrhosis.[3]

Hepatitis D

Case definition – Hepatitis D

*Only **confirmed cases** are reported.*

***Confirmed case:** Detection of IgM or IgG antibodies to hepatitis D virus or detection of hepatitis D on liver biopsy in a case known to be hepatitis B surface antigen positive.*

Hepatitis D is a defective single-stranded RNA virus that requires the hepatitis B virus to replicate. Hepatitis D infection can be acquired either as a co-infection with hepatitis B or as a super-infection with chronic hepatitis B infection. People co-infected with hepatitis B and hepatitis D may have more severe acute disease and a higher risk of fulminant hepatitis compared with those with hepatitis B alone. The modes of hepatitis D transmission are similar to those for hepatitis B, and in countries with low hepatitis B prevalence, injecting drug users are the main risk group for hepatitis D.

There were 30 notifications of hepatitis D to the NNDSS in 2005 giving a notification rate of 0.2 cases per 100,000 population. The male to female ratio was 2.4:1. Of the 30 notifications, 15 were reported from New South Wales, 11 from Queensland and 2 each from Victoria and Western Australia.

Gastrointestinal diseases

In 2005, gastrointestinal diseases that were notified to NNDSS were: botulism, campylobacteriosis, cryptosporidiosis, haemolytic uraemic syndrome (HUS), hepatitis A, hepatitis E, listeriosis, salmonellosis, shigellosis, Shiga-like toxin-producing *Escherichia coli*/verotoxigenic *E. coli* (SLTEC/VTEC) infections and typhoid.

Notifications of gastrointestinal diseases increased by 12%; from 26,173 in 2004 to 29,422 in 2005 (Table 4). Compared with 2004, there was a decrease in the number of notifications of listeriosis (13 notifications; 19%) and typhoid (24 notifications; 31%) in 2005. Variable increases were reported for all other gastrointestinal disease; botulism (200%), campylobacteriosis (6%), cryptosporidiosis (91%), haemolytic uraemic syndrome (25%), hepatitis A (2%), hepatitis E (11%), salmonellosis (8%), shigellosis (41%) and SLTEC/VTEC (78%). The number of notifications were within the historical range (i.e. within the 5-year mean and 2 standard deviations) except for hepatitis E which had an excess of 1 case, shigellosis which had an excess of 136 cases, and SLTEC/VTEC, which had an excess of 26 cases above the upper historical range. Listeriosis notifications were 6 cases below the lower historical range.

Botulism

Case definition – Botulism

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of* Clostridium botulinum *OR detection of* Clostridium botulinum *toxin in blood or faeces AND a clinically compatible illness (e.g. diplopia, blurred vision, muscle weakness, paralysis, death).*

Three cases of infant botulism in 2 males and a female were reported to NNDSS in 2005. All were aged less than 12 months. There have been 9 cases of infant botulism reported, but no classic foodborne botulism reported in Australia since botulism surveillance commenced in 1992.

Campylobacteriosis

Case definition – Campylobacteriosis

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation or detection of* Campylobacter *species.*

There were 16,468 notifications of campylobacteriosis in Australia in 2005. Campylobacteriosis is notifiable in all jurisdictions except New South Wales. The national rate of notifications in 2005 was 121 cases per 100,000 population; an increase of 4% compared with the rate reported in 2004 (116 cases per 100,000 population). All jurisdictions with the exception of Victoria reported increases in notifications, with Western Australia and Tasmania reporting the largest increases (25% and 23%). Victoria reported a 5% decrease in notifications after a 12% increase in 2004. Tasmania had the highest notification rate in 2005 (157 cases per 100,000 population) and Queensland had the lowest notification rate (111 cases per 100,000 population) (Table 3).

Monthly notifications of campylobacteriosis in 2005, consistent with previous years (2000 to 2004), peaked in the fourth quarter of the year in early summer (Figure 15). In 2005, 12 *Campylobacter* related outbreaks were identified of which 9 were suspected to be foodborne.[4]

Children aged 0–4 years had the highest notification rate of *Campylobacter* infection (Figure 16). In this age group, notification rates were higher in males (260 cases per 100,000 population) than in females (187 cases per 100,000 population). The overall male to female ratio, as in previous years, was 1.2:1.

Figure 15. Trends in notifications of campylobacteriosis, Australia, 2000 to 2005, by month of onset

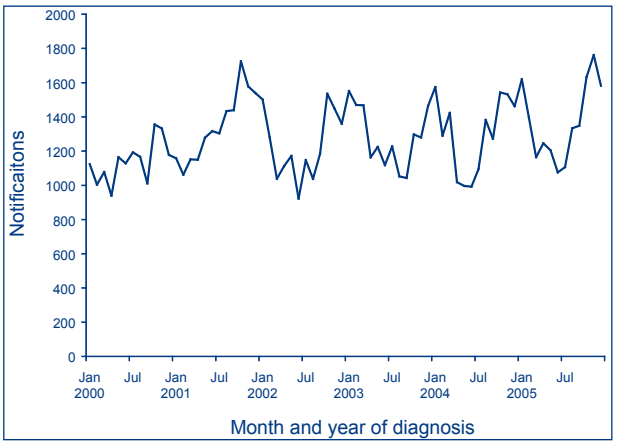
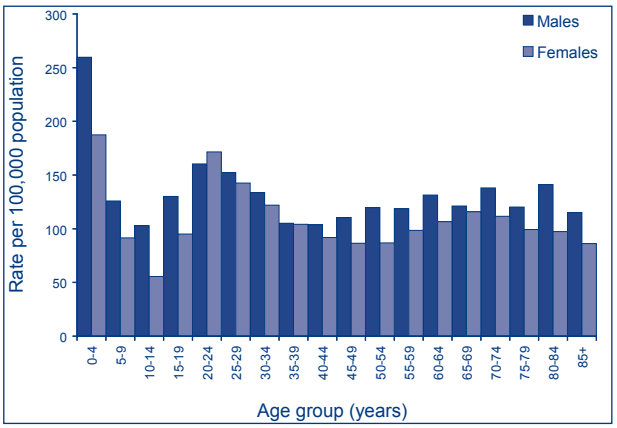


Figure 16. Notification rate for campylobacteriosis, Australia, 2005, by age group and sex



Cryptosporidiosis

Case definitions – Cryptosporidiosis

*Only **confirmed cases** are reported.*

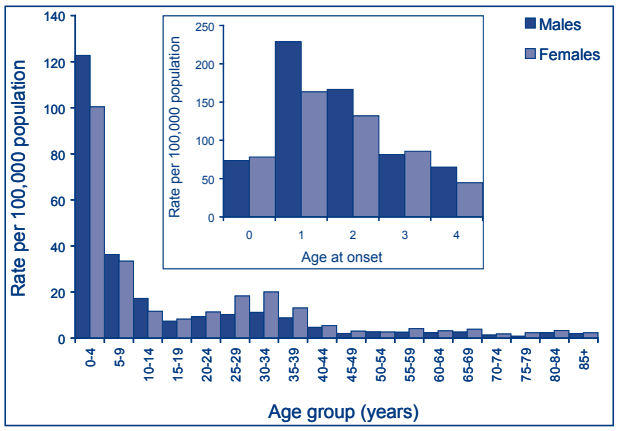
***Confirmed case:** Requires detection of* Cryptosporidium oocytes.

In 2005, a total of 3,209 cases of cryptosporidiosis were reported to NNDSS; an increase of 91% on the 1,684 cases reported in 2004. The national notification rate of 15.8 cases per 100,000 population represents an increase of 73% on the average notification rate for the previous 5 years.

All jurisdictions except the Northern Territory reported increases in cryptosporidiosis notifications, with increases ranging from 22% in Tasmania to 350% in the Australian Capital Territory. The Northern Territory and Queensland had a notification rate above the national average at 40 and 34 cases per 100,000 population, respectively.

Forty-four per cent of cryptosporidiosis cases notified in 2005 were under the age of 5 years. Compared to 2004, the notification rate in this age group increased by 72% in 2005. With a notification rate of 112 cases per 100,000 population, children under the age of 5 years continue to have the highest notification rate of cryptosporidiosis. Within this age group males aged 1 year had the highest notification rate at 229 cases per 100,000 population (Figure 17).

Figure 17. Notification rate for cryptosporidiosis, Australia, 2005, by age group and sex



Hepatitis A

Case definition – Hepatitis A

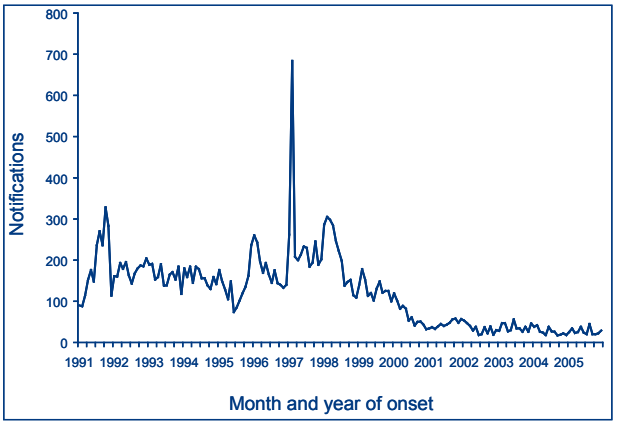
*Both **confirmed cases** and **probable cases** are reported.*

***Confirmed case:** Requires detection of anti-hepatitis A IgM, in the absence of recent vaccination, OR detection of hepatitis A virus by nucleic acid testing.*

***Probable case:** Requires clinical hepatitis (jaundice and/or bilirubin in urine) without a non-infectious cause AND contact between two people involving a plausible mode of transmission at a time when: (a) one of them is likely to be infectious (from two weeks before the onset of jaundice to a week after onset of jaundice), AND (b) the other has an illness that starts within 15 to 50 (average 28–30) days after this contact, AND at least one case in the chain of epidemiologically-linked cases (which may involve many cases) is laboratory confirmed.*

There were 325 cases of hepatitis A reported to NNDSS in 2005; a notification rate of 2 cases per 100,000 population. The notifications of hepatitis A have steadily decreased for the last decade, but remained stable in the period 2004 to 2005 (Figure 18).

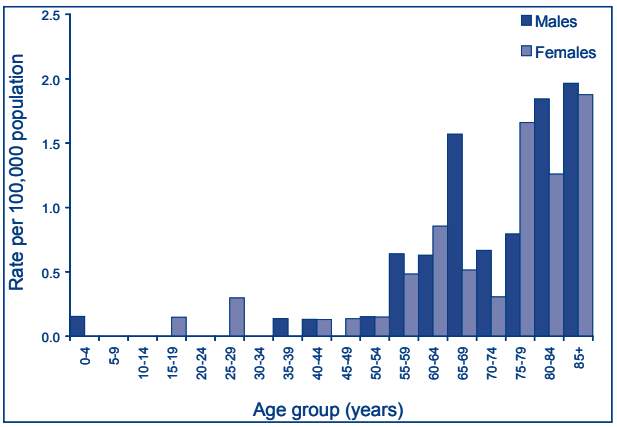
Figure 18. Trends in notifications of hepatitis A, Australia, 1991 to 2005, by month of notification



Compared to 2004, hepatitis A notification rates increased in 4 jurisdictions (ranging from 81% in Queensland to 351% in the Northern Territory) and decreased in 4 jurisdictions (ranging from 7% in Western Australia to 40% in New South Wales). The Northern Territory had the highest notification rate (32 cases per 100,000 population) followed by Western Australia (3 cases per 100,000 population).

The highest age-specific rate of hepatitis A notifications for both males and females was in the 5–9 year age group (3.2 cases and 3.4 cases per 100,000 population, respectively) (Figure 19). The overall male to female notification rate was 1:0.9.

Figure 19. Notification rate for hepatitis A, Australia, 2005, by age group and sex



Indigenous Australians had the highest burden of hepatitis A infection in 2005 with a rate of 9.9 cases per 100,000 population, compared with 0.6 cases per 100,000 population in the non-Indigenous population. In 2005 the indigenous status of 86% of cases of hepatitis A was complete and 15% of cases were identified as Indigenous people compared with 11% in 2004.

Hepatitis A is commonly spread from person to person or from contaminated food or water. Information on risk factors was known in 67% of all notifications. Overseas travel and household contact with another case were the main risk factors for hepatitis A infection (Table 7).

Table 7. Risk exposure associated with hepatitis A virus infection, Australia, 2005

Total number of cases	325
Number of cases with known risk factors*	
Injecting/recreational drug use	3
Household/close contact of case	52
Overseas travel	74
Childcare	9
Homosexual contact	9
Sex worker†	0
Other‡	2

* Exposures are not mutually exclusive hence more than one exposure per person is possible.

† Not available in New South Wales or Queensland.

‡ Includes association with persons from a country where hepatitis A is endemic and, living in an area where hepatitis A is endemic.

Hepatitis E

Case definition – Hepatitis E

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires detection of hepatitis E virus by nucleic acid testing OR, detection of hepatitis E virus in faeces by electron microscopy OR, detection of IgM or IgG to hepatitis E virus. If the person has not travelled outside Australia in the preceding 3 months, the antibody result must be confirmed by specific immunoblot.*

There were 31 cases of hepatitis E reported to NNDSS in 2005, an increase of 11% on the number of cases reported in 2004. Twelve cases were reported in Victoria, 8 in Queensland, seven in New South Wales, 2 in Western Australia and 2 in the Australian

Capital Territory. The male to female ratio was 2.1:1. Cases were aged between 10 and 74 years. Twenty-nine cases acquired their infections overseas: 17 had travelled to India, 3 to Vietnam, 3 throughout South East Asia and the remaining cases to other countries: mostly in Asia and South East Asia. One case in Victoria and 1 in Queensland were reported as locally acquired.

The Victorian Infectious Diseases Reference Laboratory detected a large increase in hepatitis E positive samples in the first quarter of 2005, which coincided with outbreaks of hepatitis E in India.[5]

Listeriosis

Case definitions – Listeriosis

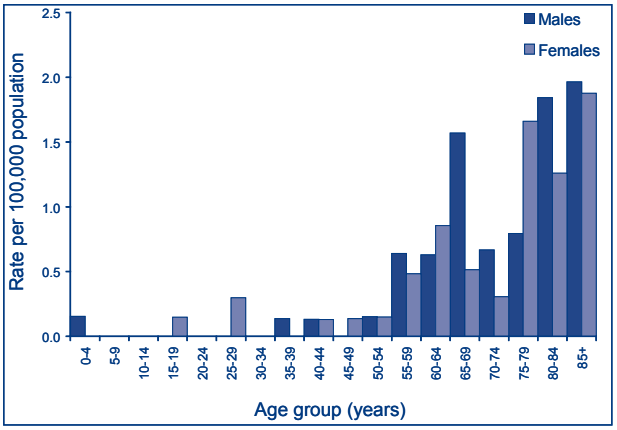
*Only **confirmed cases** are reported. Where a mother and foetus/neonate are both confirmed, both cases are reported.*

***Confirmed case:** Requires isolation or detection of* Listeria monocytogenes *from a site that is normally sterile, including foetal gastrointestinal contents.*

In 2005, 54 cases of listeriosis were reported to NNDSS, a notification rate of 0.3 cases per 100,000 population. This represents a decrease of 20% compared to the 5-year average. Eighty-five per cent of listeriosis cases were aged over 50 years, with the highest notification rate in the over 85 year age group in both males and females (Figure 20). Of 19 cases where the outcome of the infection was known, 3 cases died.

In 2005, there were 4 listeriosis cases of materno-foetal origin and 1 foetal death reported. An outbreak of listeriosis linked to the consumption of cold meats in South Australia, occurred in 2005. The Australian Capital Territory also reported a cluster of 3 cases but no common source was identified.[4]

Figure 20. Notification rate for listeriosis, Australia, 2005, by age group and sex



Salmonellosis (NEC)

Case definitions: – Salmonellosis

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation or detection of* Salmonella *species (excluding* S. typhi *which is notified separately under typhoid).*

A total of 8,441 salmonellosis cases were reported to NNDSS in 2005, a rate of 41.5 cases per 100,000 population and a 6.6% increase from the rate reported in 2004 (39.0 cases per 100,000 population). The national notification rate for 2005 showed an increase of 14.1% over the mean rate for the previous 5 years.

The Northern Territory, Queensland and Tasmania had notification rates 4.7, 1.6 and 1.5 times the national notification rate, respectively (Table 3). The highest rates of notification of salmonellosis were reported in the northern part of the country (Map 2). In 2005, the Northern Territory, excluding Darwin, had the highest notification rate at 275 cases per 100,000 population. This Statistical Division had a notification rate of 288 cases per 100,000 population in 2004.

Traditionally, the incidence of *Salmonella* infections fluctuates seasonally, peaking in March. In 2005, several outbreaks caused *Salmonella* notifications to peak in January (Figure 21). Thirty-three per cent of salmonellosis cases in 2005 had dates of onset during the summer months.

As in 2004, the highest rate of notification was in children aged between 0–4 years (195 cases per 100,000 population): 29% of salmonellosis notifications were in this age group (Figure 22).

The National Enteric Pathogens Surveillance Scheme reported serovars for 8,241 isolates in 2005.[6] The 10 most frequently isolated serovars and phage types of *Salmonella,* which accounted for 45% of all isolates, are shown in Table 8. *Salmonella* Typhimurium 135, *Salmonella* Typhimurium 197 and *Salmonella* Typhimurium 170 were the 3 most frequently isolated serovars/phage types. Several outbreaks were associated with these 3 phage types, the largest, which affected 268 people in Victoria, was caused by phage type 197. *Salmonella* Typhimurium 44 appeared in the top 10 serovars for the first time in 2005.

Map 2. Notification rate for salmonellosis, Australia, 2005, by Statistical Division of residence

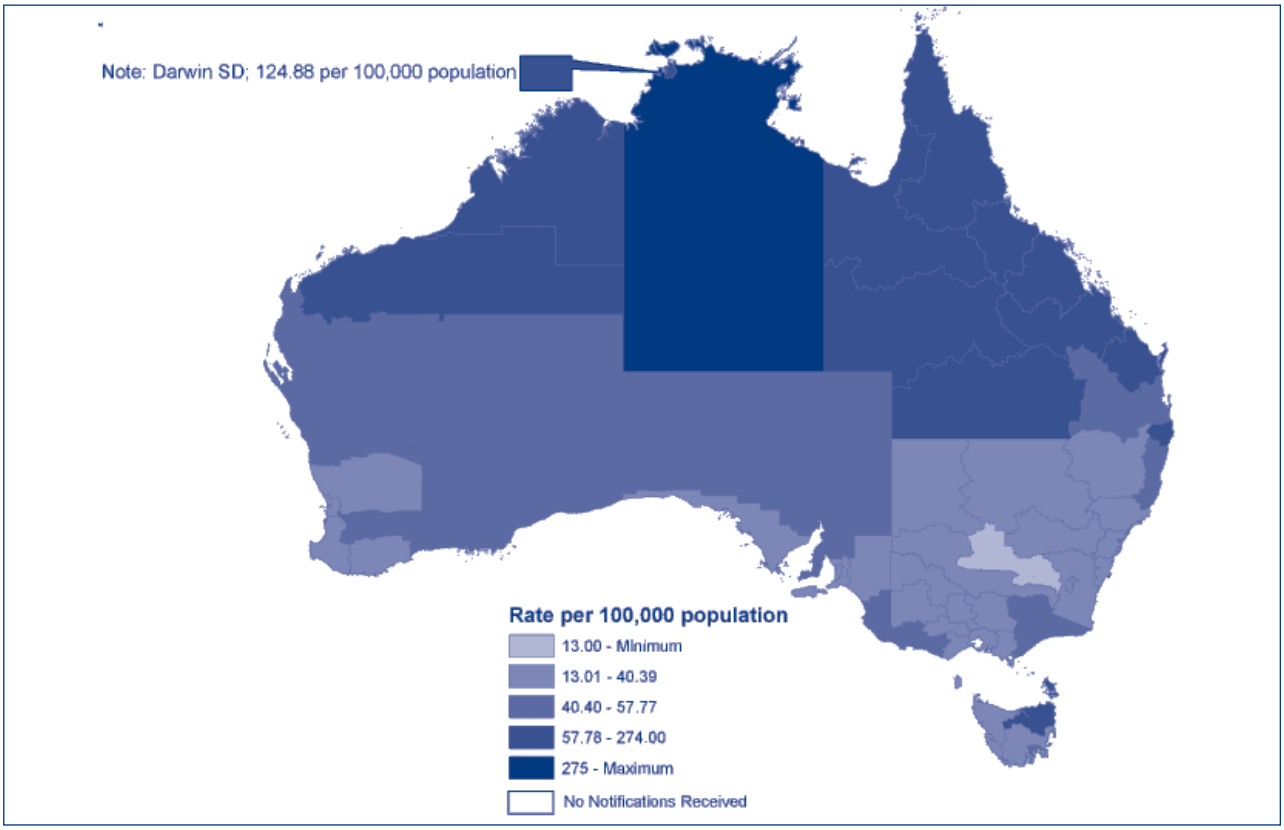


Figure 21. Trends in notifications of salmonellosis, Australia, 2000 to 2005, by month of onset

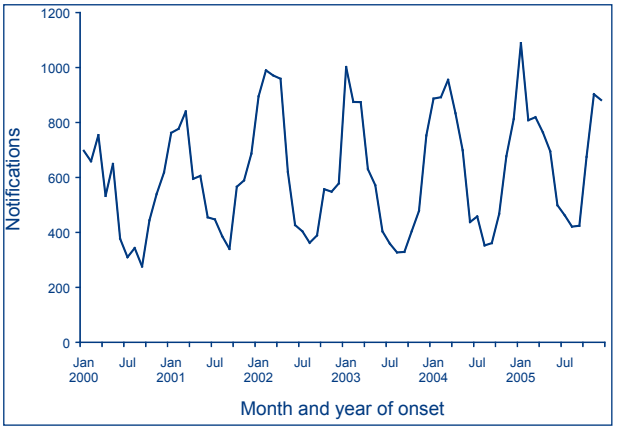
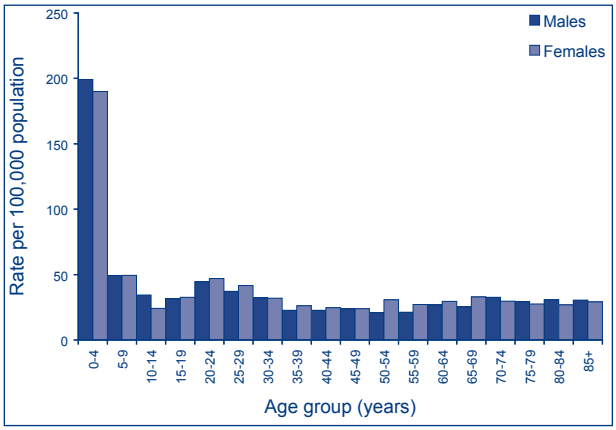


Figure 22. Notification rate for salmonellosis, Australia, 2005, by age group and sex



Salmonella Saintpaul was the most commonly reported serovar in Queensland and in the Northern Territory (11% and 12% of salmonellosis notifications). In all other jurisdictions *Salmonella* Typhimurium was the most commonly reported serovar. *Salmonella* Typhimurium 135 accounted for 59% of cases in Tasmania, 13% in the Australian Capital Territory and 9% in Western Australia. *Salmonella* Typhimurium 170 was the most commonly notified phage type in New South Wales and the Australian Capital Territory making up 15% and 13% of salmonellosis notifications respectively. In Victoria, *Salmonella* Typhimurium 197 was the most common phage type (19%) and in South Australia *Salmonella* Typhimurium 9 accounted for 10% of notifications (Table 8).

Outbreaks and clusters of salmonellosis

In 2005, OzFoodNet reported 104 clusters and outbreaks of salmonellosis of which 61% (63/104) were attributable to *S.* Typhimurium infection. Thirty-

Table 8. Top 10 isolates of *Salmonella*, Australia, 2005, by state or territory

Organism	State or territory									Total %
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust	
S. Typhimurium 135	14	188	1	135	23	175	198	68	802	16.6
S. Typhimurium 197	1	113	0	140	5	2	280	4	545	11.3
S. Typhimurium 170*	14	328	0	48	3	6	64	9	472	9.8
S. Saintpaul	3	42	48	271	13	2	24	33	436	9.0
S. Typhimurium 9	11	155	5	33	57	10	124	11	406	8.4
S. Virchow 8	2	28	10	182	6	1	7	12	248	5.1
S. Typhimurium 44	6	67	0	59	28	6	53	9	228	4.7
S. Birkenhead	0	85	0	128	0	0	6	1	220	4.5
S. Chester	1	30	14	87	14	1	10	29	186	3.8
S. Hvittingfoss	5	23	5	129	1	0	19	3	185	3.8
Sub-total	57	1,059	83	1,212	150	203	785	179	3,728	77.0
Other isolates	6	217	35	370	90	63	134	197	1,112	23.0

Source: National Enteric Pathogenic Surveillance System.

* Reported as *Salmonella* Typhimurium phage type 108 in some states and territories.

three foodborne outbreaks of salmonellosis were reported. These outbreaks affected 1,200 persons and resulted in 150 hospitalisations and 4 deaths.

Of the 5 significant foodborne outbreaks (affecting 50 or more persons each) in 2005, 4 were due to *Salmonella* Typhimurium: 1 outbreak of STM197 in Victoria; 2 of STM135 in Tasmania and one outbreak of STM64 in South Australia. The STM197 outbreak in Victoria was associated with dips served at a Turkish restaurant. The 2 STM135 outbreaks in Tasmania were associated with cakes prepared at a bakery and raw egg sauces in a restaurant. A single egg-farm supplied eggs to both premises. The STM64 outbreak in South Australia was associated with consumption of bread rolls from a restaurant. The fifth significant *Salmonella* outbreak occurred in Western Australia and was due to *Salmonella* Oranienburg associated with the consumption of alfalfa sprouts.[4]

Shigellosis

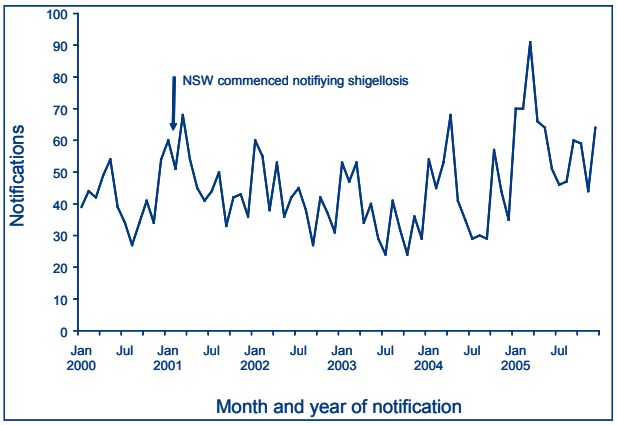
Case definitions – Shigellosis

Only ***confirmed cases*** *are reported.*

Confirmed case: *Isolation or detection of* Shigella *species.*

In 2005, a total of 732 cases of shigellosis were reported to NNDSS, a notification rate of 3.6 cases per 100,000 population. This rate was 39% higher than the rate reported in 2004 (2.6 cases per 100,000 population), and 40% higher than the 5-year average (Table 4). Notification rates for 2005 increased compared to 2004 in all jurisdictions except South Australia. The Northern Territory continued to have the highest notification rate at 96.7 cases per 100,000 population, an increase by 66.6% in notification rates compared to 2004. Nationally, notification rates of the disease had been declining for the period 1999 to 2003, then increased in 2004 and again in 2005. (Figure 23).

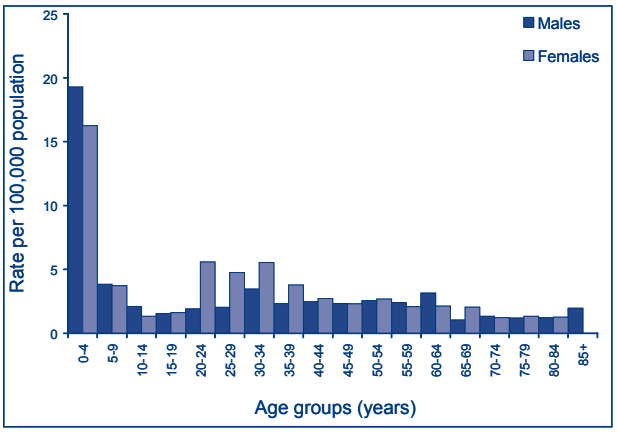
Figure 23. Trends in notifications of shigellosis, Australia, 2000 to 2005, by month of onset



The male to female rate ratio remained at 0.9:1. Children under the age of 4 years represented 31% of shigellosis notifications (Figure 24). This age group had a notification rate of 17.8 cases per 100,000

population, which was an increase of 40% compared to the rate reported in 2004 (12.7 cases per 100,000 population).

Figure 24. Notification rate for shigellosis, Australia, 2005, by age group and sex



The highest rate of shigellosis continues to be in Indigenous populations with a rate of 64 cases per 100,000 population compared to 0.5 cases per 100,000 population in the non-Indigenous population. In 2005, of the notifications of shigellosis where indigenous status of cases was complete (73% of all cases) 59% were identified as Indigenous. In the Northern Territory (where indigenous status was complete for 100% of notifications) 82% of shigellosis cases were Indigenous.

Shigella flexneri and *Shigella sonnei* infections accounted for 44% and 52% of shigellosis, respectively in 2005 (Table 9). Eighty-nine per cent of *Shigella flexneri* infections were further typed, of which 27% were type 4a mannitol negative and 27% were type 2a. Eighty-three per cent of *Shigella sonnei* infections were further typed, of which 54% were type A.

Shiga-like toxin-producing/verotoxigenic *Escherichia coli*

Case definitions – Shiga-like toxin-producing/verotoxin-producing Escherichia coli *(SLTEC/VTEC)*

Only ***confirmed cases*** *are reported.*

Confirmed case: *Requires isolation of Shiga-toxigenic/verotoxigenic* Escherichia coli *from faeces, OR, isolation of Shiga toxin or verotoxin from a clinical isolate of* E. coli *OR, identification of the gene associated with the production of Shiga toxin or vero toxin in* E. coli *by nucleic acid testing on isolate or raw bloody diarrhoea.*

Note: Where SLTEC/VTEC is isolated in the context of haemolytic uraemic syndrome (HUS), it should be notified as SLTEC/VTEC and HUS.

There were 87 cases of SLTEC/VTEC reported to NNDSS in 2005 compared with 49 cases in 2004. With a notification rate of 0.4 cases per 100,000 population, the rate of SLTEC/VTEC notifications represented an increase of 70% compared to the average for the previous 5 years. The increase in notifications was due to an increase in screening for SLTEC/VTEC by Western Australia, Victoria and parts of New South Wales. As in previous years, South Australia continued to routinely test bloody stools by polymerase chain reaction for genes coding for Shiga-like toxin. Forty-six per cent of all cases were notified in South Australia (2.6 cases per 100,000 population). The Australian Capital Territory and the Northern Territory did not report any cases of SLTEC/VTEC. OzFoodNet reported that among typed *E. coli* (49% of all notifications) 39% were subtype O157, 26% were subtype O11 and 16% were O26.[4]

Table 9. *Shigella* infections, Australia, 2005, by serogroups and state or territory

Organism	State or territory								Total	Per cent
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
S. boydii	0	4	1	0	0	0	2	1	8	1.1
S. dysenteriae	0	2	0	1	0	0	0	0	3	0.4
S. flexneri	1	29	128	20	32	3	21	91	325	45.5
S. sonnei	6	97	64	55	16	2	77	61	378	52.9
Sub-total	7	132	193	76	48	5	100	153	714	100.0
Unknown	0	3	3	4	0	0	5	3	18	–
Total	7	135	196	80	48	5	105	156	732	–

Haemolytic uraemic syndrome

Case definitions – Haemolytic uraemic syndrome (HUS)

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires acute microangiopathic anaemia on peripheral blood smear (schistocytes, burr cells or helmet cells) AND AT LEAST ONE OF THE FOLLOWING: acute renal impairment (haematuria, proteinuria or elevated creatinine level), OR, thrombocytopaenia, particularly during the first seven days of illness.*

Note: Where SLTEC/VTEC is isolated in the context of HUS, it should be notified as both SLTEC/VTEC and HUS.

In 2005, 20 cases of HUS were reported to NNDSS; a rate of 0.1 cases per 100,000 population, an increase of 23% on the rate in 2004 (15 cases). Eleven cases occurred in New South Wales. No HUS cases were notified in the Australian Capital Territory or the Northern Territory. Among the 20 cases of HUS notified in 2005, 55% were males. The median age among males was 13 years (range 1–68 years) and among females was 25 years (range 2–81 years). SLTEC was isolated in 9 cases of HUS. Toxigenic *E. coli* was identified in 9 of the 20 cases. The serotypes of these 9 were O111 (2), O157:H (2), OR:H– (1), O111:H– (1), O49 (1), and unknown (2).

Typhoid

Case definitions – Typhoid fever

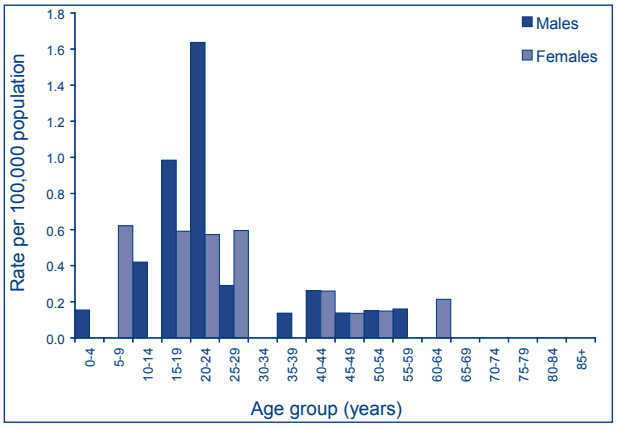
*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation or detection of* Salmonella typhi.

In 2005, there were 52 notifications of typhoid; a rate of 0.26 cases per 100,000 population, representing an decrease of 23% compared to the average notification rate for the previous 5 years. All jurisdictions reported a decrease in notification rates except Western Australia, which reported a 67% increase. Nationally, the male to female ratio was 1:0.7, with the highest notification rates in males aged 20–24 and 15–19 years (1.6 and 1.0 cases per 100,000 population respectively) and in females aged 5–9 and 15–29 years (0.6 cases per 100,000 population) (Figure 25).

The National Enteric Pathogen Surveillance Scheme identified 50 *Salmonella* Typhi isolates in 2005, 42 of which were from Australian residents. Of the 42 Australian residents, 9 had no travel history recorded, 1 had not travelled, 1 had carrier contact, 1 was a carrier and the remaining 30 cases had travelled outside Australia including in South East Asia, Africa, Europe, Pacific Islands, and South America.[6]

Figure 25. Notification rate for typhoid, Australia, 2005, by age group and sex



Quarantinable diseases

Human diseases covered by the *Quarantine Act 1908*, and notifiable in 2005 were cholera, plague, rabies, yellow fever, smallpox, highly pathogenic avian influenza in humans (HPAIH), severe acute respiratory syndrome (SARS) and 4 viral haemorrhagic fevers (Ebola, Marburg, Lassa and Crimean-Congo).

HPAIH was declared a quarantinable disease on 23 March 2004 and consequently became subject to the routine quarantine powers available under the *Quarantine Act 1908*. SARS was declared a quarantinable disease under the *Quarantine Act 1908* on 7 April 2003.

Cholera

Case definition – Cholera

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of toxigenic* Vibrio cholerae *O1 or O139.*

In 2005, there were 3 cases of cholera notified in Australia, 2 from Victoria, and 1 from Western Australia. All cases were female. All cases acquired their disease overseas: 2 of the Victorian cases acquired it from Tanzania and the Western Australian case acquired it from Indonesia.

Three notifications were toxin producing *Vibrio cholerae* serogroup O1 Ogawa.

Cholera, plague, rabies, yellow fever, SARS, HPAIH, tuleraemia and viral haemorrhagic fevers are of international public health importance and are notified to the World Health Organization. Although no local transmission had been reported in Australia, these diseases continue to occur around the world. Travellers are advised to seek information on the risk of contracting these diseases in their destinations and take appropriate measures. More information on quarantinable diseases and travel health can be found on DoHA's web site at: http://www.health.gov.au/internet/wcms/Publishing.nsf/Content/health-pubhlth-strateg-quaranti-index.htm

Sexually transmissible infections

In 2005, sexually transmissible infections (STIs) reported to NNDSS were chlamydial infections, donovanosis, gonococcal infections, and syphilis. Two categories of adult syphilis have been reported since 2004: syphilis of less than 2 years duration – infectious (primary, secondary and early latent); and syphilis of greater than 2 years or unknown duration. These 2 categories are combined under 'syphilis – all.' Congenital syphilis is also reported to NNDSS. These conditions were notified in all states and territories.

Other national surveillance systems that monitor STIs in Australia include the Australian Gonococcal Surveillance programme, which is a network of specialist laboratories, and the National Centre in HIV Epidemiology and Clinical Research.

The national trends in the number and rates of STI notifications reported to NNDSS between 2000 and 2005 are shown in Table 4. In interpreting these data it is important to note that changes in notifications over time may not solely reflect changes in disease prevalence. Increases in screening rates[7,8] more targeted screening, the use of more sensitive diagnostic tests, as well as periodic public awareness campaigns may contribute to changes in the number of notifications over time.

Age adjusted notification rates were calculated for Indigenous and non-Indigenous populations for jurisdictions that had indigenous status data completed in more than 50% of notifications. These data however, should be interpreted cautiously as STI screening occurs disproportionately among Indigenous populations. Similarly, rates of testing for STI also differ between sexes.

Chlamydial infection

Case definition – Chlamydial infection

*Only **confirmed cases** are reported.*

***Confirmed case:** Isolation of* Chlamydia trachomatis *or detection of* Chlamydia trachomatis *by nucleic acid testing or detection of* Chlamydia trachomatis *antigen.*

Chlamydial infection continued to be the most commonly notified condition in 2005. A total of 41,311 notifications of chlamydial infection were received; a rate of 203 cases per 100 000 population. This represents an increase of 13% on the rate reported in 2004 (180 cases per 100,000 population). The rate of chlamydia notifications has increased each year since surveillance of the condition commenced in 1991. Between 2001 and 2005, chlamydial infection notification rates increased from 104 to 203 cases per 100,000 population, an increase of 95% (Table 4). This increase provided the impetus for the launch of Australia's first National STI Strategy in July 2005.[9] The prevalence of chlamydia varies by age group and other demographic and behavioural factors, and most major sections of the population are unaffected. [10]

Chlamydial infection notification rates were higher than the national average in the Northern Territory (781 cases per 100,000 population), Western Australia (271 cases per 100,000 population), Queensland (245 cases per 100,000 population) and the Australian Capital Territory (215 cases per 100,000 population) (Table 3). At a regional level, the Northern Territory excluding Darwin had the highest chlamydial infection notification rate at 1,596 cases per 100,000 population (Map 3).

In 2005, notification rates of chlamydial infection in males and females were 166 and 240 cases per 100,000 population, respectively. In 2005, notification rates increased by 14% in males and by 13% in females compared to 2004. The male to female ratio in 2005 was 1:1.5, which is similar to previous years. Rates in females exceeded those in males in the 10–14, 15–19, and 20–24 year age groups with ratios of 1:7, 1:3 and 1:2, respectively (Figure 26). Sixty-six cases of chlamydia were identified as congenital chlamydia infections. These cases, while still included in the total number of chlamydial infections for 2005, were excluded from analyses.

Map 3. Notification rate for chlamydial infections, Australia, 2005, by Statistical Division of residence

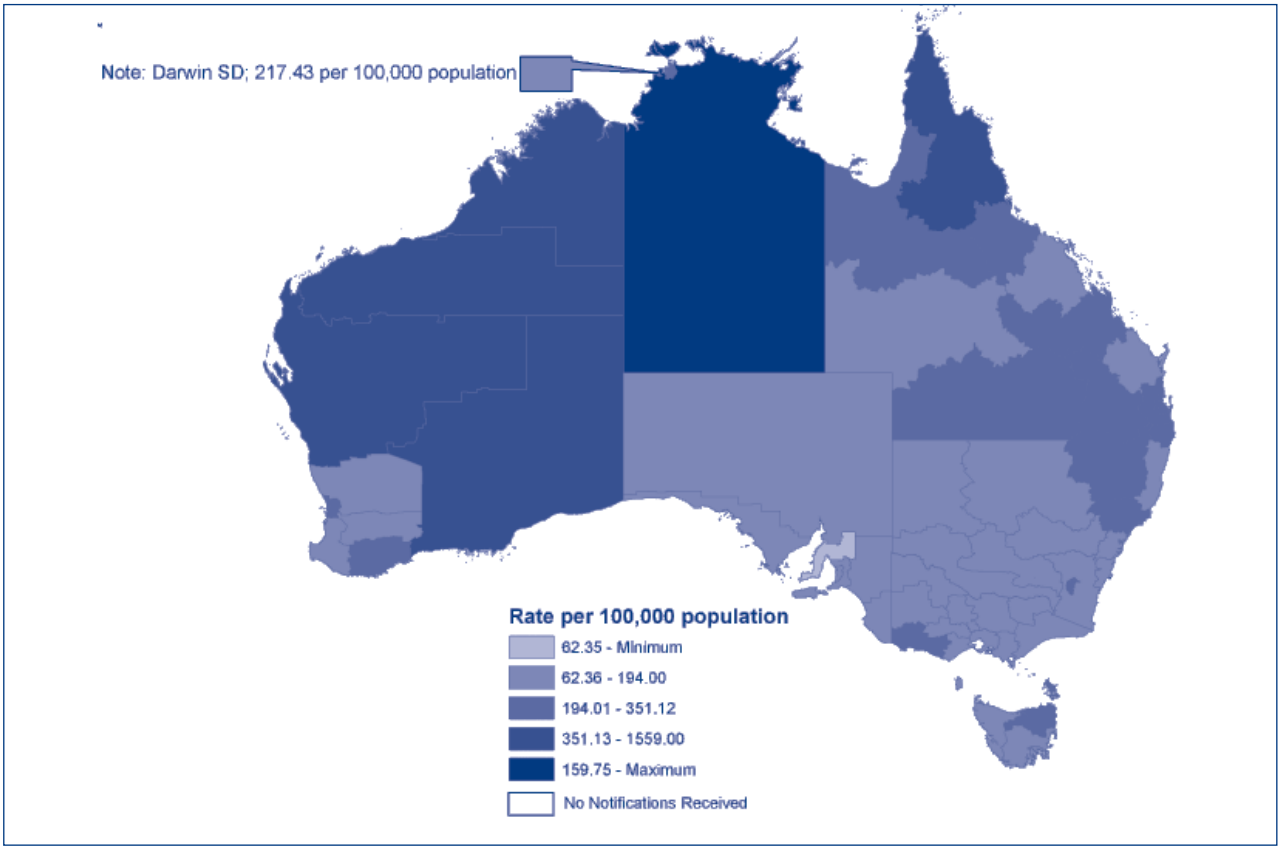


Figure 26. Notification rate for chlamydial infections, Australia, 2005, by age group and sex

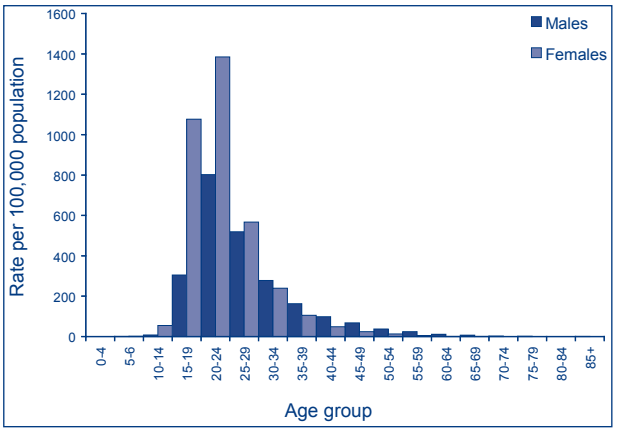
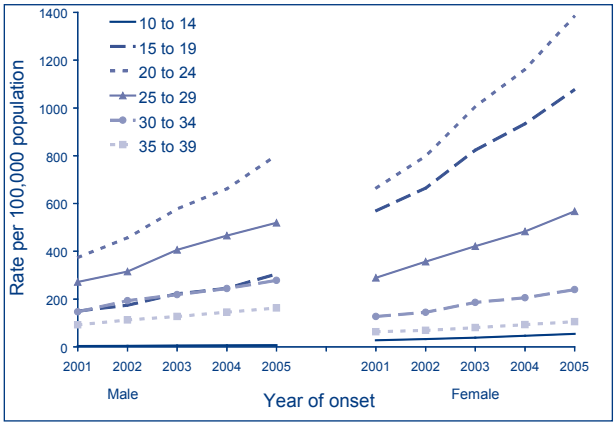


Figure 27. Trends in notification rate for chlamydia infection in persons aged 10–39 years, Australia, 2001 to 2005, by age group and sex



Age and sex notification rates between 2001 and 2005 show increases in all age groups between 10 and 39 years in both males and females (Figure 27). Since 2001, the highest average annual percentage increase occurred in the 20–24 year age group (21% in males and 20% in females).

In 2005, data on indigenous status was complete in 39% of cases of chlamydia infection; this is a decrease on the 59% reported in 2004 and the 43% notified in 2003. The combined chlamydial infection notifications in 4 jurisdictions with greater than 50% completeness of indigenous status (Northern Territory, South Australia, Victoria and Western Australia)

show that in 2005, the age adjusted notification rate was 989.9 cases per 100,000 Indigenous population, and 191.5 cases per 100,000 non-Indigenous population. The age adjusted ratio of Indigenous to non-Indigenous was 5.2:1.

Although surveillance data continues to show a substantial increase in chlamydia notifications nationally, it is important to note that changes in notifications over time may not solely reflect changes in disease prevalence. As a large proportion of cases with genital chlamydial infection are asymptomatic, notification rates for this disease are particularly susceptible to the overall rate of testing as well as the targeted testing of certain population sub-groups. In past years Medicare Australia data were utilised to determine if the number of chlamydia tests were also increasing.[10] With the changes to the Medicare item number, which occurred late for chlamydia testing in 2005, this is not currently possible.

Donovanosis

Case definition – Donovanosis

*Both **confirmed cases** and **probable cases** are reported.*

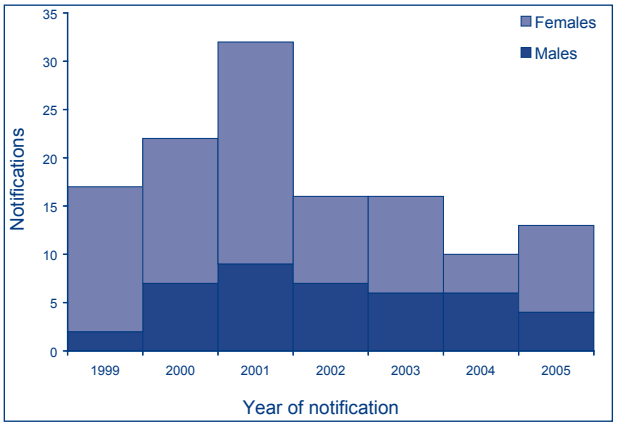
***Confirmed case:** Requires demonstration of intracellular Donovan bodies on smears or biopsy specimens taken from a lesion or detection of* Calymmatobacterium granulomatis *by nucleic acid testing of a specimen taken from a lesion AND clinically compatible illness involving genital ulceration.*

***Probable case:** Requires compatible sexual risk history in a person from an endemic area or a compatible sexual risk history involving sexual contact with someone from an endemic area.*

Donovanosis is a sexually transmissible infection characterised by a chronic ulcerative genital disease. Although uncommon, it is a disease of public health importance because it predominantly occurs in Indigenous communities; it has been identified as a potential co-factor in HIV transmission; and it is preventable.[11]

In 2005, 13 cases of donovanosis, 4 male and 9 female, were reported to NNDSS. Cases were reported from Northern Territory (4), Queensland (8) and Western Australia (1). Eleven cases of the total were among Indigenous people: 6 in Queensland, 4 in the Northern Territory and 1 in Western Australia. One non-Indigenous case and 1 case with unknown indigenous status were reported in 2005 (Figure 28). Cases in 2005 ranged in age from 12 to 53 years and the majority were aged 30–44 years.

Figure 28. Number of notifications of donovanosis, Australia, 1999 to 2005, by sex and year of notification



Gonococcal infections

Case definition – Gonococcal infection

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of* Neisseria gonorrhoeae, *or detection of* Neisseria gonorrhoeae *by nucleic acid testing or detection of typical Gram-negative intracellular diplococci in a smear from a genital tract specimen.*

In 2005, 8,015 notifications of gonococcal infection were received by NNDSS. This represents a rate of 39.4 cases per 100,000 population, an increase of 10% over the rate reported in 2004 (35.7 cases per 100,000 population). Nationally, there was an increase in the notification rates of females (by 13%), and males (by 9%) compared to 2004. The male to female ratio in 2005 was 2:1; unchanged in the previous 4 years and reflecting ongoing transmission among men who have sex with men.

The highest notification rate in 2005 was in the Northern Territory at 857 cases per 100,000 population (Table 3). Nationally, gonococcal notification rates for males and females were 54 and 25 cases per 100,000 population respectively. The exception to this pattern was the Northern Territory, where females had higher notification rates than males (820 versus 898 cases per 100,000 population). The geographical distribution of gonococcal notification rates

shows that the highest rate occurred in the Northern Territory (excluding Darwin) at 2,020 cases per 100,000 population (Map 4).

Notification rates of gonococcal infection in males exceeded those in females in all age groups except in the 10–14 and 15–19 year age groups (Figure 29). Trends in sex specific notification rates show that rates in males in the 15–19, 20–24 and 25–29 age groups continued to increase. Notification rates for males in the 30–44 age groups also increased in 2005. In females, increases occurred in the 15–19 and 20–24 age groups (Figure 30).

In 2005, the data completeness (68%) of indigenous status of gonococcal infection notifications was similar to that in 2004. The combined gonococcal infection notifications of 5 jurisdictions with indigenous status reported in more than 50% of notifications (the

Figure 29. Notification rate for gonococcal infections, Australia, 2005, by age group and sex

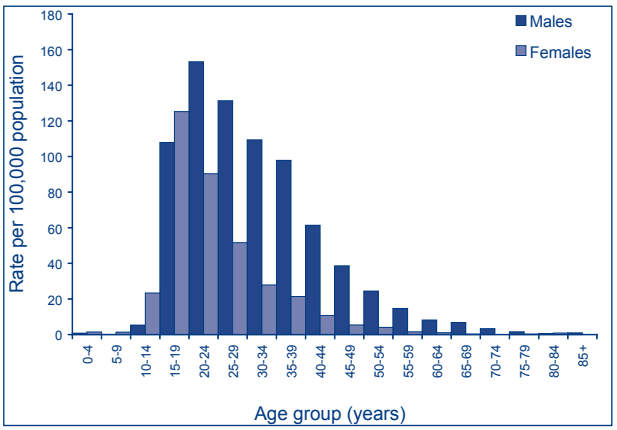
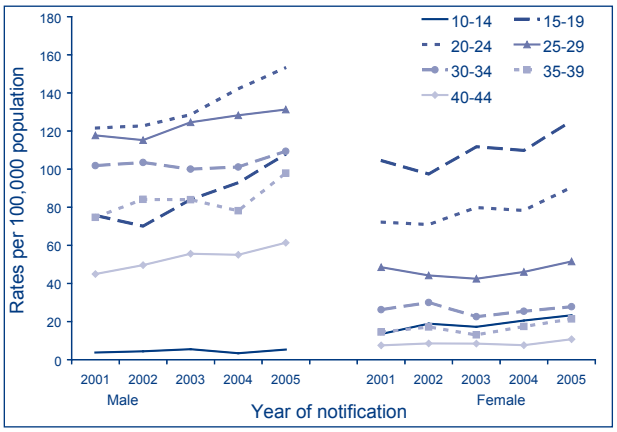
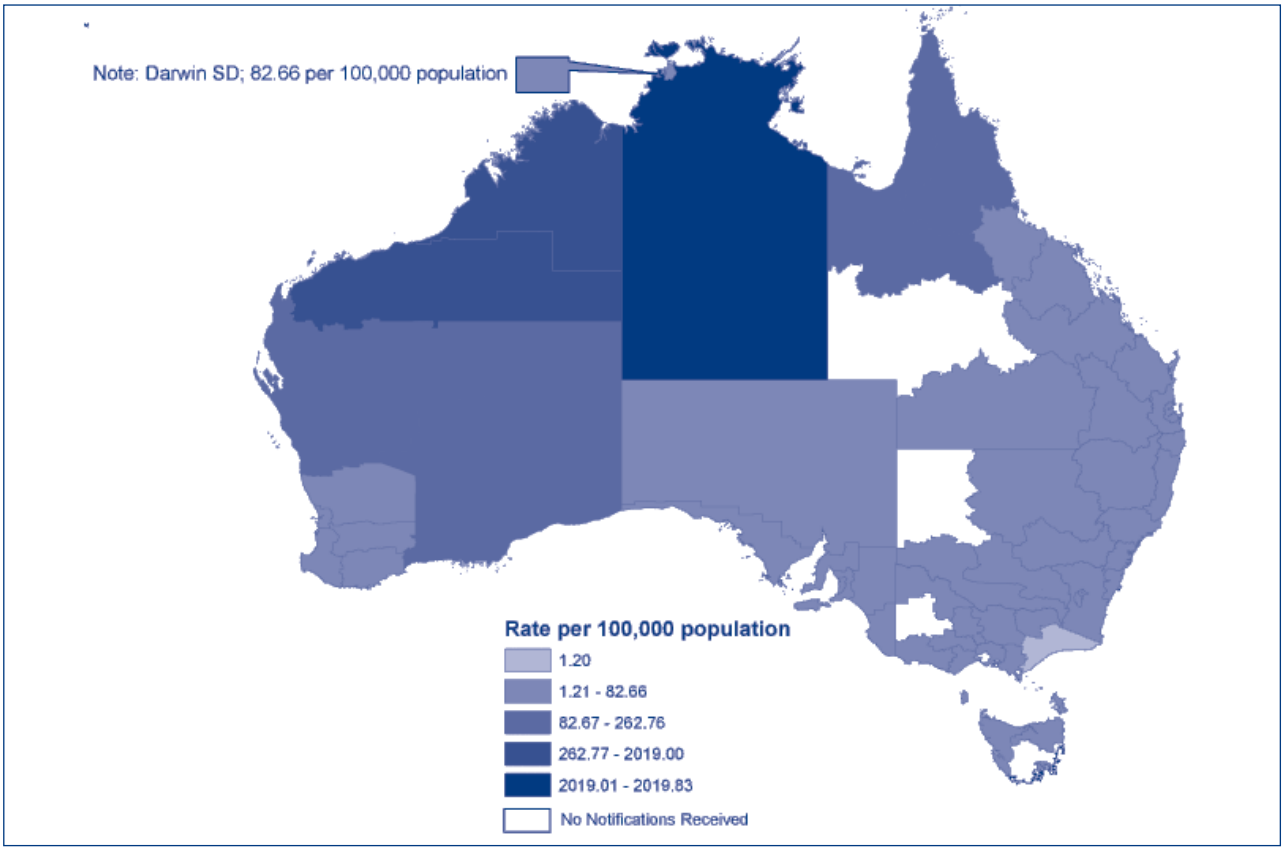


Figure 30. Trends in notification rate for gonococcal infections in persons aged 10–44 years, Australia, 2001 to 2005, by age group and sex



Map 4. Notification rate for gonococcal infections, Australia, 2005, by Statistical Division of residence



Northern Territory, Queensland, South Australia, Western Australia and Victoria) shows that the age adjusted notification rate in the Indigenous population was 1,590.9 cases per 100,000 population and 34.6 cases per 100,000 non-Indigenous population: a ratio of Indigenous to non-Indigenous of 46:1.

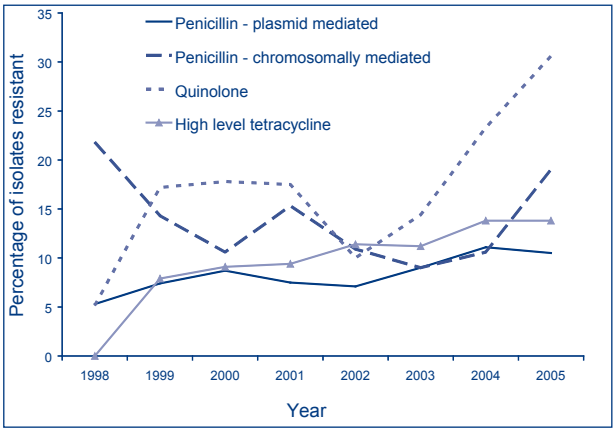
Other surveillance of gonococcal infections

The Australian Gonococcal Surveillance Programme (AGSP) is the national surveillance system of antibiotic susceptibility of gonococcal isolates. In each state and territory, a network of reference laboratories determines the susceptibility of isolates to a core group of antibiotics using a standard methodology. The following is the summary of their 2005 annual report.[12]

In 2005, a total of 3,980 isolates of gonococci were tested for antibiotic susceptibility. Eighty-three per cent of isolates were from men, of which 75% were obtained from the urethra, 13% from the rectum and 9% from the larynx. In females, 93% of isolates were obtained from the cervix. Proportions for site of infection were similar to those reported in 2004.

Trends in the proportion of isolates resistant to penicillin, quinolones and tetracycline are shown in Figure 31. In 2005, the proportion of isolates resistant to penicillin by plasmid-mediated resistance remained similar to 2004 (10.5%) while the proportion of isolates resistance to penicillin by chromosomally-mediated mechanisms increased to 19%. Quinolone resistance also increased to 30.6% from 23.3% in 2004. Ninety-three per cent of the quinolone resistant isolates were also resistant at a higher minimal inhibitory concentration (MIC) of 1 mg/L or more.

Figure 31. Proportion of gonococcal isolates showing antibiotic resistance, Australia, 1998 to 2005



Information on the country where resistant strains were acquired were available in 31% of infections for strains with plasmid-mediated resistance to penicillin, and 31% of infections for strains resistant to quinolone. This showed that 51% (66/128) of plasmid mediated resistance were locally required with the rest acquired from Western Pacific countries and South East Asia. Eight-four per cent of quinolone resistant strains were acquired locally and the remainder from overseas.

The distribution of infections with strains resistant to different antibiotic agents varies from jurisdiction to jurisdiction and urban to rural areas within each jurisdiction. The AGSP recommends that treatment regimes should be tailored to the local patterns of susceptibility. Nationally, the AGSP recommends the use of alternative treatments to quinolones for infections acquired.

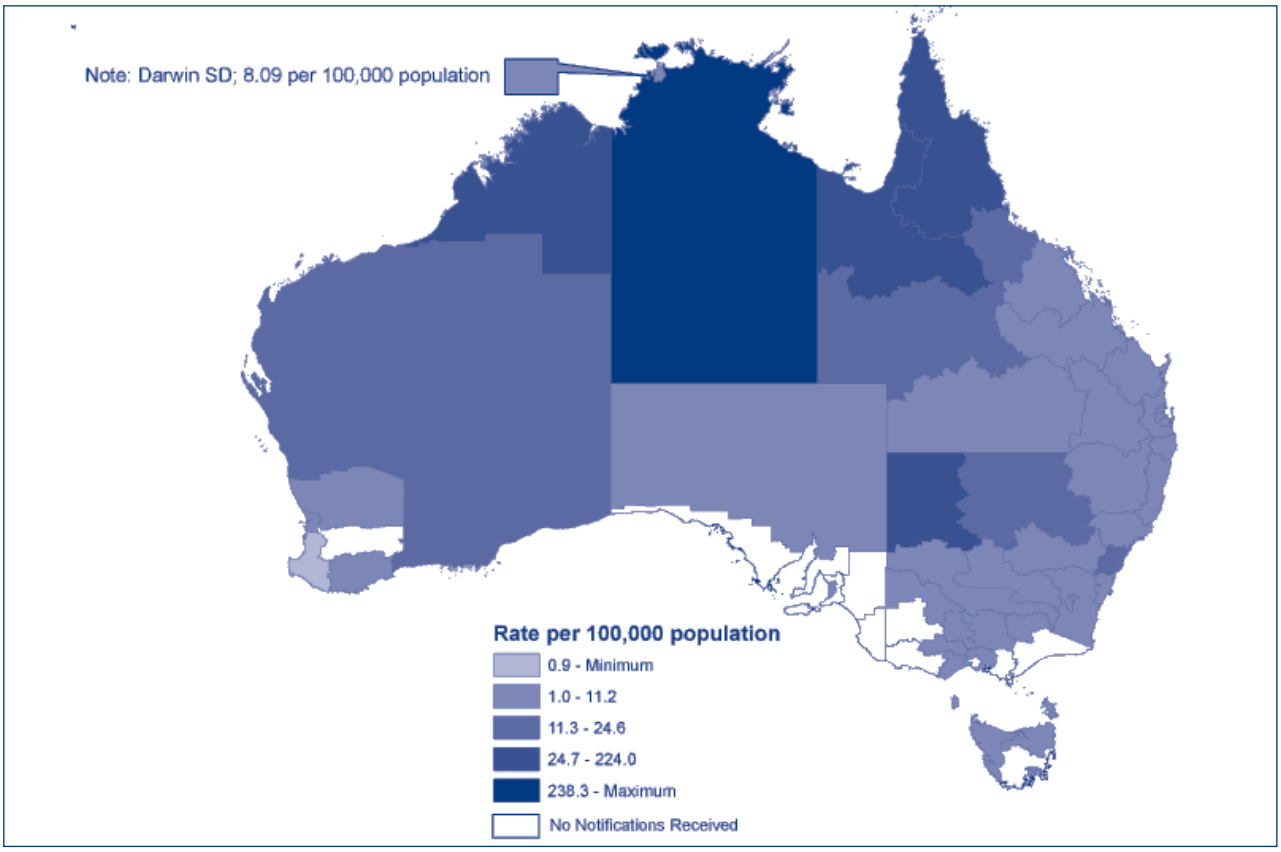
Syphilis (both categories)

In 2004, all jurisdictions began reporting adult syphilis infections to NNDSS categorised as: infectious syphilis of less than 2 years duration; and syphilis of more than 2 years or unknown duration; this continued in 2005. Detailed analysis is reported for the 2 categories, as well as for syphilis of both categories for the purpose of comparing rates to previous years.

In 2005, a total of 2,203 cases of syphilis infection of both categories were reported, representing a notification rate of 10.8 cases per 100,000 population, a decrease of 7% on the 11.6 cases per 100,000 population reported in 2004 (Table 4). The Northern Territory continued to have the highest notification rate of syphilis (113 cases per 100,000 population), although in 2005 the rate was 20% lower than the previous year. In 2005, there were increases in notification rates in the Australian Capital Territory (16%), Queensland (29%), and Victoria (15%). Recent outbreaks among men who have sex with men in Melbourne and Sydney[13,14] may have peaked. At the regional level, the highest notification rate was in the Northern Territory (excluding Darwin) at 238 cases per 100,000 population (Map 5).

Tasmania reported an increase of 114% but this was most likely in syphilis of unknown duration and due to screening practices.

Map 5. Notification rate for syphilis infections, Australia, 2005, by Statistical Division of residence



Syphilis – less than 2 years duration

Case definition – Syphilis – infectious (primary, secondary and early latent), less than 2 years duration

Only ***confirmed cases*** *are reported.*

Confirmed case: *Requires seroconversion in past two years (specific treponemal test (e.g. IgG enzyme immunoassay,* Treponema pallidum *haemagglutination assay,* Treponema palladium *particle agglutination,* Treponema pallidum *immobilisation assay), or fluorescent treponemal antibody absorption reactive when previous treponemal test non-reactive within past two years*

OR a fourfold or greater rise in non-specific treponemal antibody titre (e.g. Venereal Diseases Research Laboratory, Rapid Plasma Reagin) in the past two years, and a reactive specific treponemal test (e.g. IgG enzyme immunoassay, Treponema pallidum *haemagglutination assay,* Treponema pallidum *particle agglutination,* Treponema pallidum *immobilisation assay, or fluorescent treponemal antibody absorption)*

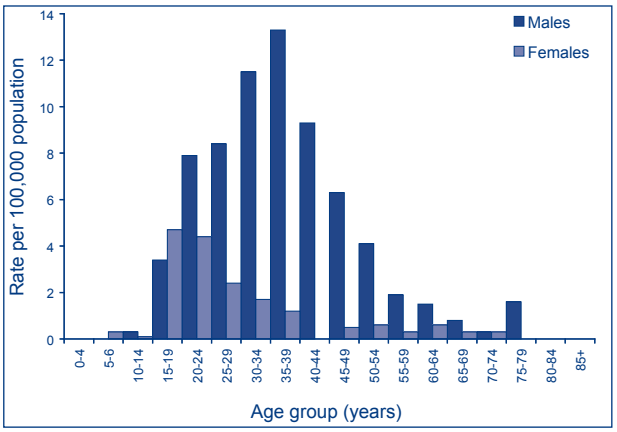
OR demonstration of Treponema pallidum *by darkfield microscopy (not oral lesions), direct fluorescent antibody tests, equivalent microscopic methods (e.g. silver stains), or nucleic acid testing or non-specific treponemal test (e.g. Venereal Diseases Research Laboratory, Rapid Plasma Reagin) reagin titre of greater than or equal to 1:8 AND presence of a primary chancre (or ulcer) or clinical signs of secondary syphilis.*

In 2005, a total of 621 cases of syphilis of less than 2 years duration were reported. This represents a notification rate of 3.1 cases per 100,000 population. The Northern Territory had the highest notification rate at 46 cases per 100,000 population in 2005.

The notification rates of syphilis of less than 2 years duration for males and females were 4.9 and 1.2 cases per 100,000 population, respectively. Notification rates were higher in males than in females in most jurisdictions. Nationally, the male to female ratio

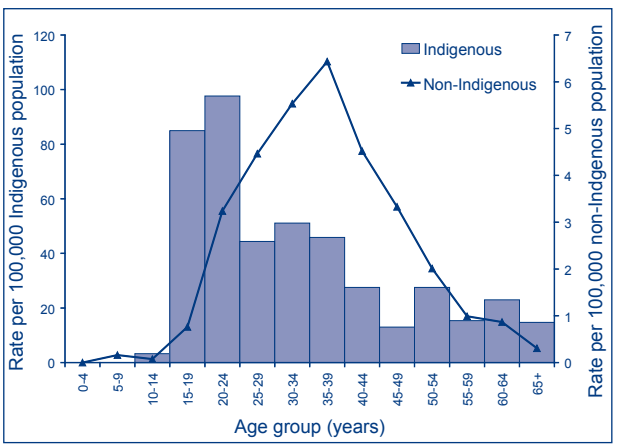
was 4:1, which was similar to 2004. Notification rates in males peaked in the 35–39 year age group (13 cases per 100,000 population) and in females in the 15–19 year age group (5 cases per 100,000 population) (Figure 32).

Figure 32. Notification rate for syphilis of less than two years duration, Australia, 2005, by age group and sex



Data on indigenous status was complete in 93% of cases of syphilis of less than 2 years duration. The age adjusted notification rate was 33.5 cases per 100,000 Indigenous population, and 2.3 cases per 100,000 non-Indigenous population: a ratio of Indigenous to non-Indigenous of 14:1. Age-specific notification rates showed that, compared to the non-Indigenous population, rates of syphilis of less than 2 years duration in the Indigenous population are an order of magnitude higher and peak in a younger age group (Figure 33).

Figure 33. Notification rate for syphilis of less than two years duration, Australia, 2005, by indigenous status



Syphilis of more than two years or unknown duration

Case definition – Syphilis of more than 2 years or unknown duration

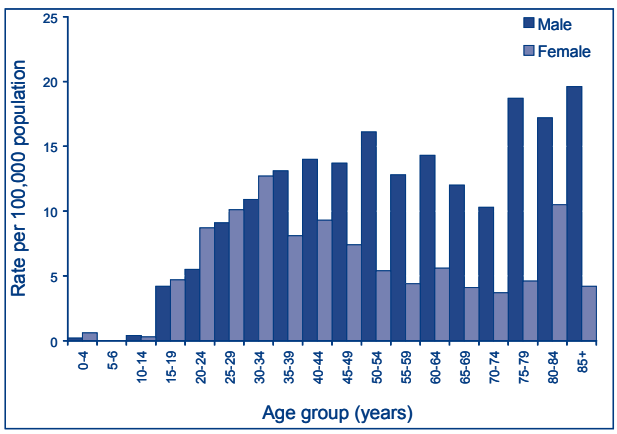
*Only **confirmed cases** are reported.*

***Confirmed case:** Does not meet the criteria for a case of less than 2 years duration AND either a reactive specific treponemal test (e.g. IgG enzyme immunoassay,* Treponema pallidum *haemagglutination assay,* Treponema pallidum *particle agglutination,* Treponema pallidum *immobilisation assay, or fluorescent treponemal antibody absorption) which is confirmed either by a reactive non-specific treponemal test (e.g. Venereal Diseases Research Laboratory, Rapid Plasma Reagin) OR a different specific treponemal test if the non-specific treponemal test is non-reactive AND the absence of a history of documented previous adequate treatment of syphilis, or endemic treponemal disease (e.g. Yaws).*

In 2005, a total of 1,582 cases of syphilis of more than 2 years or unknown duration were reported: a notification rate of 7.8 cases per 100,000 population. The Northern Territory had the highest notification rate at 67 cases per 100,000 population (Table 3).

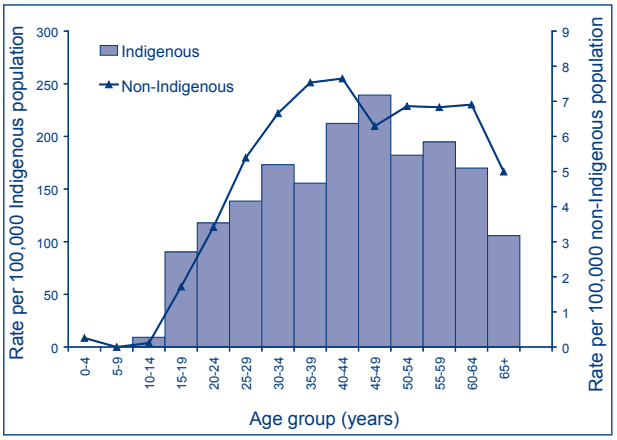
In 2005, notification rates of syphilis of more than two years or unknown duration in males and females were 9.4 and 6.1 cases per 100,000 populations, respectively. Notification rates were higher in males in all jurisdictions. Nationally, the male to female ratio was 1.5:1. Notification rates in males and females were similar in the younger age groups up to 30–34 years. In females, the rate peaked in the 30–34 year age group (13 cases per 100,000 population) while in males it remained high from 35 years (Figure 34).

Figure 34. Notification rate of syphilis of more than two years or unknown duration, Australia, 2005, by age group and sex



Data on indigenous status was complete in 67% of cases of syphilis of more than two years or unknown duration. The combined age adjusted rate for the jurisdictions with greater than 50% data completeness of indigenous status (all jurisdictions except New South Wales and the Australian Capital Territory) was 121 cases per 100,000 Indigenous population, and 5 cases per 100,000 non-Indigenous population: a ratio of Indigenous to non-Indigenous of 24:1. Age specific notification rates showed a similar pattern with age and no single distinct peak for either Indigenous and non-Indigenous groups. Overall, rates in the Indigenous population were an order of magnitude higher than those in the non-Indigenous (Figure 35).

Figure 35. Notification rate for syphilis of more than two years or unknown duration, Australia, 2005, by indigenous status



Congenital syphilis

Case definition – Congenital syphilis

*Both **confirmed cases** and **probable cases** are reported.*

***Confirmed case:** Requires treponemal-specific antibody titres (e.g.* Treponema pallidum *haemagglutination assay, pallidum particle agglutination, fluorescent treponemal antibody absorption in infant serum greater than fourfold higher than in maternal serum OR treponemal specific antibody titres in infant serum comparable with those in maternal serum and specific treponemal IgM enzyme-linked immunosorbent assay or immunofluorescence assay positive OR* T. pallidum *DNA in normally sterile specimen from infant (CSF, tissue) by nucleic acid testing.*

OR Dark field microscopy of infant lesion exudate or node aspirate smears (not oral lesions) to demonstrate characteristic morphology and motility of T. pallidum *OR demonstration of* T. pallidum *in infant tissues by special (e.g. silver) stains OR detection of* T. pallidum *DNA from an infant non-sterile site by nucleic acid testing OR reactive fluorescent treponemal absorbed-19S-IgM antibody test or IgM enzyme linked immunosorbent assay and treponemal-non specific antibody titre (e.g. RPR) in infant serum greater than fourfold higher than in maternal serum AND asymptomatic infection (in the infant of an infected mother) OR foetal death in utero OR stillbirth, which is a foetal death that occurs after a 20-week gestation or in which the foetus weighs greater than 500 g and the mother is untreated or inadequately treated for syphilis at delivery. Inadequate treatment is a non-penicillin regimen or penicillin treatment given less than 30 days prior to delivery OR clinical evidence of congenital syphilis on examination on:*

a. Age <2years: Hepatosplenomegaly, rash, condyloma lata, snuffles, jaundice (non-viral hepatitis), pseudoparalysis, anaemia, oedema

b. Age >2 years: Interstitial keratitis, nerve deafness, anterior bowing of shins, frontal bossing, mulberry molar, Hutchinson teeth, saddle nose, rhagades or Clutton joints

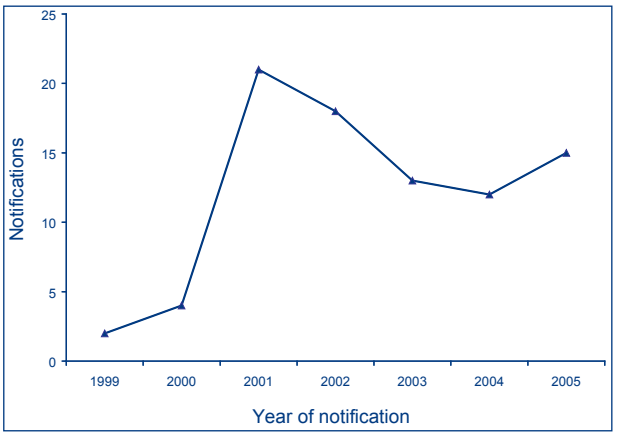
c. Evidence of congenital syphilis on long bone X-ray

d. Evidence of congenital syphilis on cerebrospinal fluid (CSF) examination

***Probable case:** An infant (regardless of clinical signs) whose mother has been inadequately treated for syphilis during pregnancy or an infant or child who has a reactive treponemal antibody test for syphilis and any one of the following: (1) any evidence of congenital syphilis on physical examination, (2) any evidence of congenital syphilis on radiographs of long bones, (3) a reactive cerebrospinal fluid Venereal Disease Research Laboratory Titre, (4) an elevated CSF cell count or protein (without other cause), (5) reactive fluorescent treponemal antibody absorbed assay –19S-IgM antibody test or IgM enzyme-linked immunosorbent assay.*

There were 15 cases of congenital syphilis notified in 2005, 8 males, 6 female and 1 of unknown sex. Eight of the cases were reported in New South Wales, 5 in the Northern Territory and 2 in Queensland. Eight were Indigenous, 4 non-Indigenous and 3 were unknown. There has been a gradual decline in the number of congenital syphilis notified in the Indigenous population since 2001 (Figure 36).

Figure 36. Trends in notifications of congenital syphilis, Australia, 2000 to 2005, by indigenous status* and year of notification



* Notifications with unknown indigenous status are recorded as non-Indigenous.

Vaccine preventable diseases

Introduction

This section summarises the national notification data for influenza and diseases targeted by the National Immunisation Program (NIP) in 2005. These include diphtheria, *Haemophilus influenzae* type b infection, measles, mumps, pertussis, invasive pneumococcal disease, poliomyelitis, rubella and tetanus. Data on hepatitis B and meningococcal disease, which are also targeted by the NIP, can be found in this report under 'bloodborne diseases' and 'other bacterial infections' respectively. Other vaccine preventable diseases presented in this report include hepatitis A and Q fever.

Two significant changes to the NIP occurred during this reporting period. In January 2005, free universal immunisation with the 7-valent pneumococcal conjugate vaccine (7vPCV) for children in the first year of life replaced the previous targeted immunisation program and free universal 23-valent pneumococcal polysaccharide vaccine (23vPPV) immunisation for adults over 65 years replaced a previous subsidised immunisation program. In November 2005, universal childhood immunisation against varicella at 18 months was introduced, with a catch-up program for children up to 12 years of age who had not had varicella vaccine, or a history of varicella infection. Inactivated polio vaccine (IPV) replaced oral polio vaccine (OPV) in various combination vaccines in 2005.

There were 17,775 notifications of vaccine preventable diseases (VPDs) with onset dates in 2005; 14% of the total notifications to NNDSS. Pertussis was the most commonly notified VPD (11,200 or 63% of all VPD notifications). Numbers of notifications and notification rates for VPDs in Australia are shown in Tables 2 and 3.

Diphtheria

Case definition – Diphtheria

*Both **confirmed cases** and **probable cases** are reported.*

***Confirmed case:** Requires isolations of toxigenic* Corynebacterium diphtheriae *or toxigenic* C. ulcerans.

***Probable case:** Requires isolation of* Corynebacterium diphtheriae *or* C. ulcerans *(toxin production unknown) and pharyngitis/laryngitis or toxic symptoms OR clinical symptoms and epidemiological links with laboratory confirmed case.*

There were no cases of diphtheria reported in 2005. The last case of diphtheria reported in Australia was a case of cutaneous diphtheria in 2001.

Haemophilus influenzae type b disease

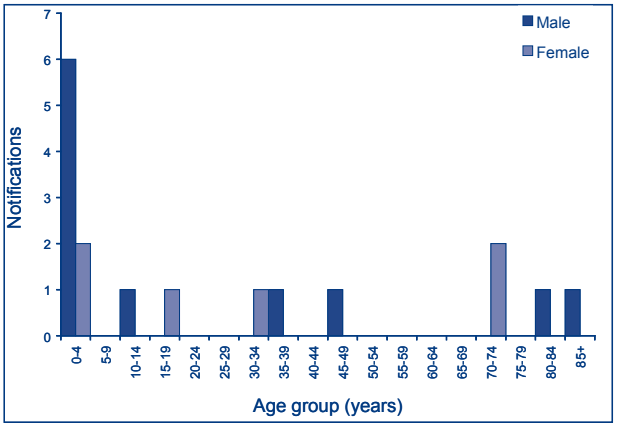
Case definition – Haemophilus influenzae *type b*

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of* Haemophilus influenzae *type b (Hib) from a sterile site OR detection of Hib antigen in cerebrospinal fluid consistent with meningitis.*

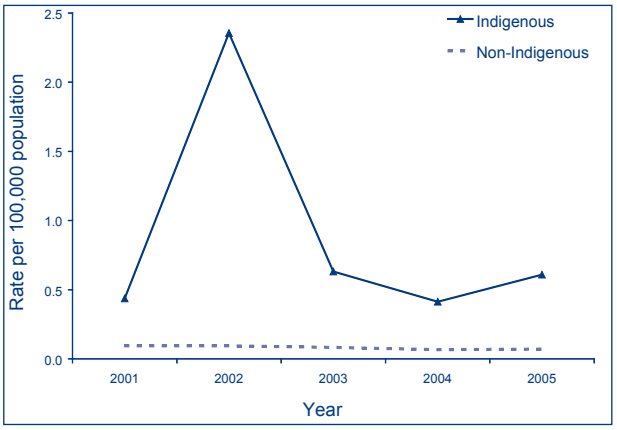
There were 17 notifications of Hib disease in 2005, a rate of 0.1 cases per 100,000 population. This was 2 more cases than reported in 2004. Eight cases (47% of total) were in children aged less than 5 years and 2 were infants aged less than 1 year. There were 11 cases in males and 6 in females, (male:female ratio 1.8:1) (Figure 37).

Figure 37. Notifications of *Haemophilus influenzae* type b infection, Australia, 2005, by age group and sex



Indigenous status was recorded for 16 of the 17 cases; 3 were Indigenous and 13 were non-Indigenous. The Hib notification rate was 0.6 cases per 100,000 population in Indigenous people and 0.07 cases per 100,000 population in non-Indigenous people; a ratio of 8.6:1. Between 2001 and 2005, Hib notification rates in Indigenous people have been between 4.6 and 8.6 times the rates in non-Indigenous people except in 2002 when the Indigenous rate was 25 times that of the non-Indigenous rate (Figure 38).

Figure 38. Notification rate for *Haemophilus influenzae* type b infections, Australia, 2001 to 2005, by indigenous status.



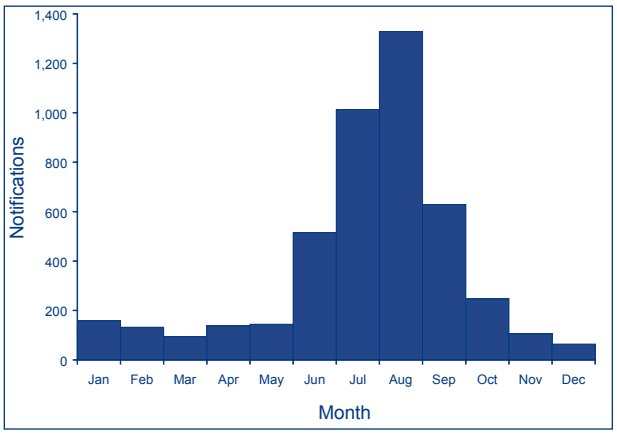
Cases under the age of 15 years were eligible for Hib vaccination. Of these 9 cases, 3 were unvaccinated, 2 partially vaccinated and 4 were fully vaccinated. The 4 fully vaccinated cases were all aged less than 5 years and met the case definition for vaccine failure, having received at least 2 doses of vaccine.

Influenza

> *Case definition – Influenza*
>
> *Only **confirmed cases** are notified.*
>
> ***Confirmed case:** Requires isolation of influenza virus by culture OR detection of influenza virus by nucleic acid testing OR detection of influenza virus antigen from an appropriate respiratory tract specimen OR a significant increase in antibody levels, or IgG seroconversion or fourfold or greater rise in antibody titre or a single high titre antibody.*

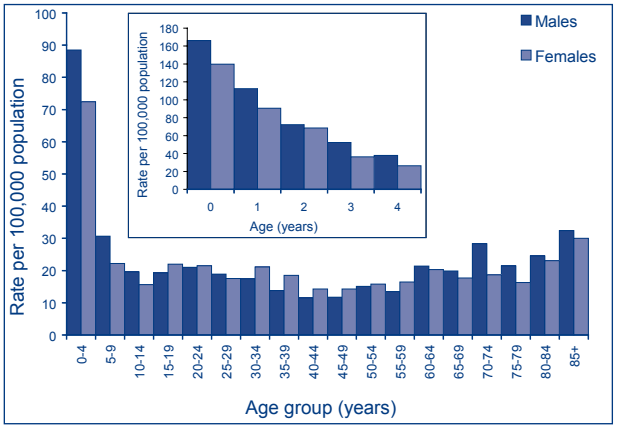
There were 4,567 reports of laboratory-confirmed influenza in 2005, a rate of 10.5 cases per 100,000 population. Notifications of influenza showed a peak in August (Figure 39).

Figure 39. Notifications of laboratory-confirmed influenza, Australia, 2005, by month of onset



Children aged less than 5 years made up 22% of all notifications and had a notification rate of 80.7 cases per 100,000 population (Figure 40) Children aged less than 1 year had the highest rate (153 cases per 100,000 population). The overall male to female ratio was 1:1.

Figure 40. Notification rate of laboratory-confirmed influenza, Australia, 2005, by age group and sex



In 2005, 4,379 (96%) influenza notifications had viral serotype data. Of these, 76% (3,338) were influenza A and 24% (1,041) were influenza B.

Of 1,174 influenza virus isolates analysed at the WHO Collaborating Centre for Reference and Research on Influenza in 2005, 689 were A(H3N2), 210 were A(H1N1) strains and 275 were influenza B. The majority of A(H3N2) viruses were antigenically similar to the 2005 vaccine strain A/Wellington /1/2004, but a quarter of isolates were more closely matched to the A/California/7/2004 viruses.[15]

There were a number of outbreaks of influenza in 2005, including an outbreak in New South Wales in a nursing home. Outbreaks of influenza B were reported in school-age children New Zealand in 2005 which resulted in 3 deaths.[16]

Measles

Case definition – Measles

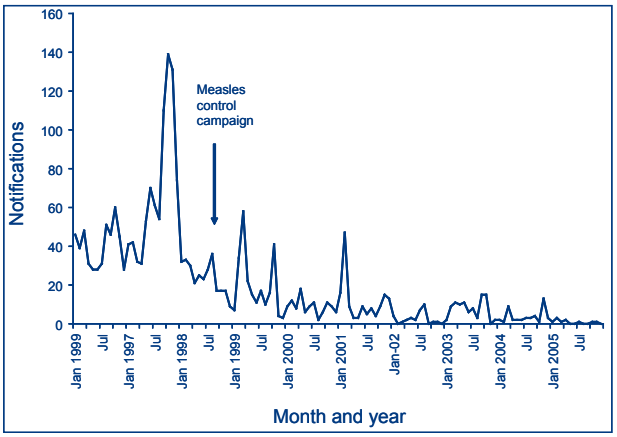
*Both **confirmed cases** and **probable cases** are notified.*

***Confirmed case:** Requires isolation of measles virus or detection of measles virus by nucleic acid testing OR detection of measles virus antigen OR IgG seroconversion or significant increase in antibody level or fourfold or greater rise in titre or detection of measles specific IgM antibody in a reference laboratory (except when vaccinated 8 days to 8 weeks prior to testing) OR clinical illness characterised by a maculopapular rash and fever and cough, coryza, conjunctivitis or koplik spots and epidemiological link to a laboratory confirmed case.*

***Probable case:** Requires detection of measles IgM antibody in other than an approved reference laboratory and clinical illness.*

There were 10 notified measles cases in 2005: 8 confirmed and 2 probable. This is the lowest annual rate for Australia since national surveillance began in 1991 (Figure 41). Five cases were reported from New South Wales, 2 from Victoria and single cases in Queensland, Tasmania and Western Australia. In 2005, there were no cases reported from the Australian Capital Territory, Northern Territory or South Australia (Tables 2 and 3).

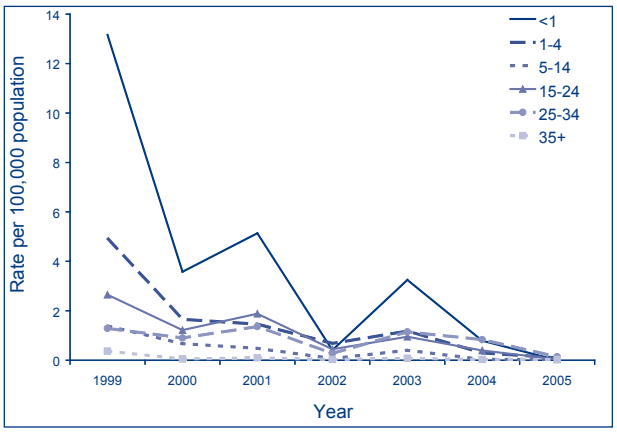
Figure 41. Notifications of measles, Australia, 1996 to 2005, by month of onset



There was only a single case of measles in children aged less than 5 years. The remaining 9 cases were aged between 11 and 42 years. Five cases were unvaccinated and 3 (including the child aged less than 5 years) were classified as fully vaccinated for age; however data on the number of doses received was missing in 2 of these cases. The vaccination status of the other 2 cases (aged 25 and 36 years) was unknown.

Figure 42 shows trends in measles notification rates by age group. In 2005, the largest proportion of measles cases occurred in adults, which reflects the success of measles vaccination programs in children and adolescents.

Figure 42. Trends in notification rate for measles, Australia, 1999 to 2005, by age group



Of the 10 measles cases reported in 2005, three cases were known to have acquired their infection outside Australia.

Mumps

Case definition – Mumps

*Only **confirmed cases** are notified.*

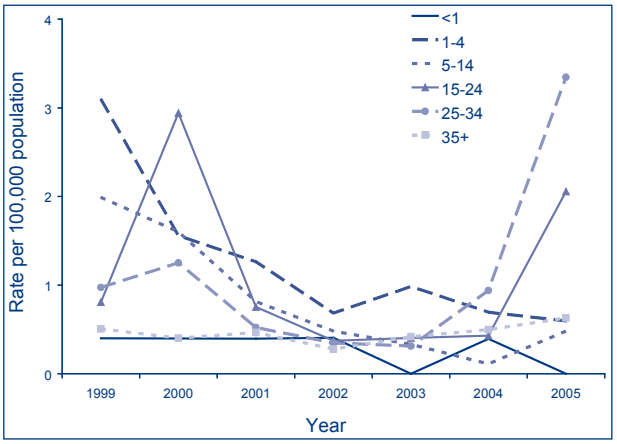
***Confirmed case:** Requires isolation of mumps virus or detection of mumps virus by nucleic acid testing or IgG seroconversion or significant increase in antibodies or a significant increase in antibody level, or a fourfold or greater rise in titre to mumps virus (except where there has been recent mumps vaccination) OR detection of mumps specific IgM antibody (in the absence of recent mumps vaccination) AND a clinically compatible illness characterised by swelling of the parotid or other salivary glands lasting two days or more without other apparent cause OR a clinically compatible illness AND an epidemiological link to a laboratory confirmed case.*

In 2005, there were 241 notifications of mumps (1.2 cases per 100,000 population), which was a 2.3-fold increase on the 102 cases reported in 2004. Cases were reported from all jurisdictions except Tasmania, with the largest number of cases (111) in New South Wales.

The highest rates were in males in the 25–29 year age group (6.2 cases per 100,000 population). The rate for the 0–4 year age group (0.6 cases per 100,000 population) was the same as in 2004. Unlike 2004 when the male to female ratio was 1:1, in 2005 there was a preponderance of male cases with a male to female ratio of 1.4:1.

Trends in age group notification rates for mumps show a sharp increase in the rates in the 25–34 year age and the 15–24 year age groups in 2005 (Figure 43).

Figure 43. Trends in notification rate of mumps, Australia 2005, by age group



The high rate of mumps in these age groups 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.

Eight cases were recorded as fully vaccinated; 9 as partially vaccinated; 108 as unvaccinated and there was no information on the vaccination status of the remaining 115 cases. Clusters of mumps cases were reported in 2005, one cluster of 5 cases occurred in an unvaccinated family of refugees in Queensland.

Pertussis

Case definition – Pertussis

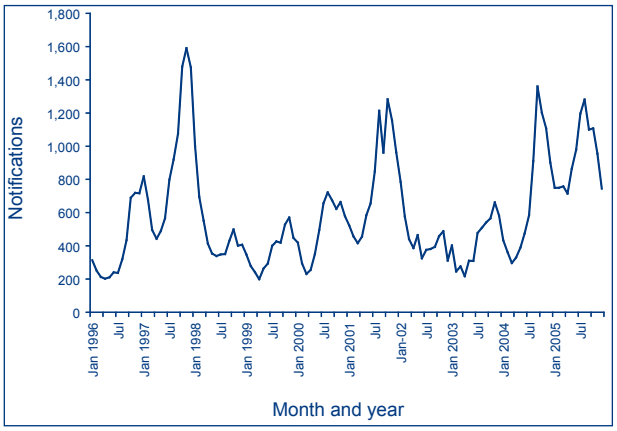
*Both **confirmed cases** and **probable cases** are notified.*

***Confirmed case:** Requires isolation of* Bordetella pertussis *or detection of* B. pertussis *by nucleic acid testing OR seroconversion or significant increase in antibody level or fourfold or greater rise in titre (in the absence of pertussis vaccination) or a single high-titre IgA to whole cells or detection of B. pertussis by immunofluorescence AND **clinical evidence** (a coughing illness lasting 2 weeks or more or paroxysms of coughing or inspiratory whoop or post-tussive vomiting) OR **clinical evidence** AND epidemiological link to a confirmed case.*

***Probable case:** Requires clinically compatible illness.*

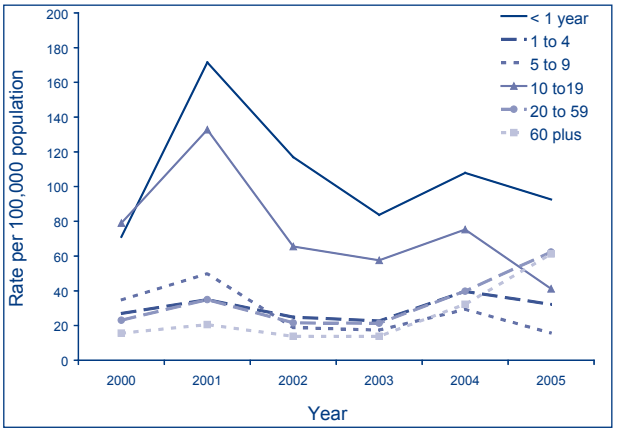
Pertussis continues to be the most common vaccine preventable illness in Australia, with periodic epidemics occurring at intervals of 3 to 5 years on a background of endemic circulation (Figure 44). In 2005 there were 11,200 cases notified to NNDSS (55.1 cases per 100,000 population). Of these, 10,744 were confirmed and 454 were probable, while the status of the remaining 2 cases was unknown.

Figure 44. Notifications of pertussis, Australia, 1996 to 2005, by month of onset



The highest notification rate was among children aged less than 1 year (237 cases, 92.2 cases per 100,000 population). The notification rate in persons aged 20–59 years and 60 years and over continued to increase in 2005 to 63.1 and 61.2 cases per 100,000 population, respectively (Figure 45). In 2005, 83% of pertussis cases were aged 20 years and over compared to 59% in 1999. Although severe morbidity and mortality are less likely in these age groups, they are an important pertussis reservoir,

Figure 45. Trends in notification rate of pertussis, Australia, 1999 to 2005, by age group

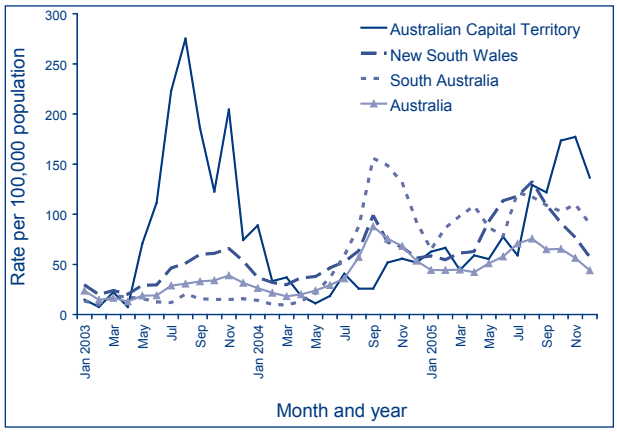


facilitating transmission to children too young to be fully vaccinated. In 2005, pertussis incidence in adolescents aged 10–19 years fell substantially from an average rate of 75.7 cases per 100,000 population between 1999–2004, to 41.5 cases per 100,000 population in 2005. School-based adolescent pertussis vaccination programs (including 2 whole of high school programs in New South Wales and Western Australia) began in a number of states in 2004, and the decrease in incidence in the targeted age group in 2005 may be the first evidence of the impact of this vaccine. Pertussis notifications were more common among women with a male to female ratio of 0.7:1.

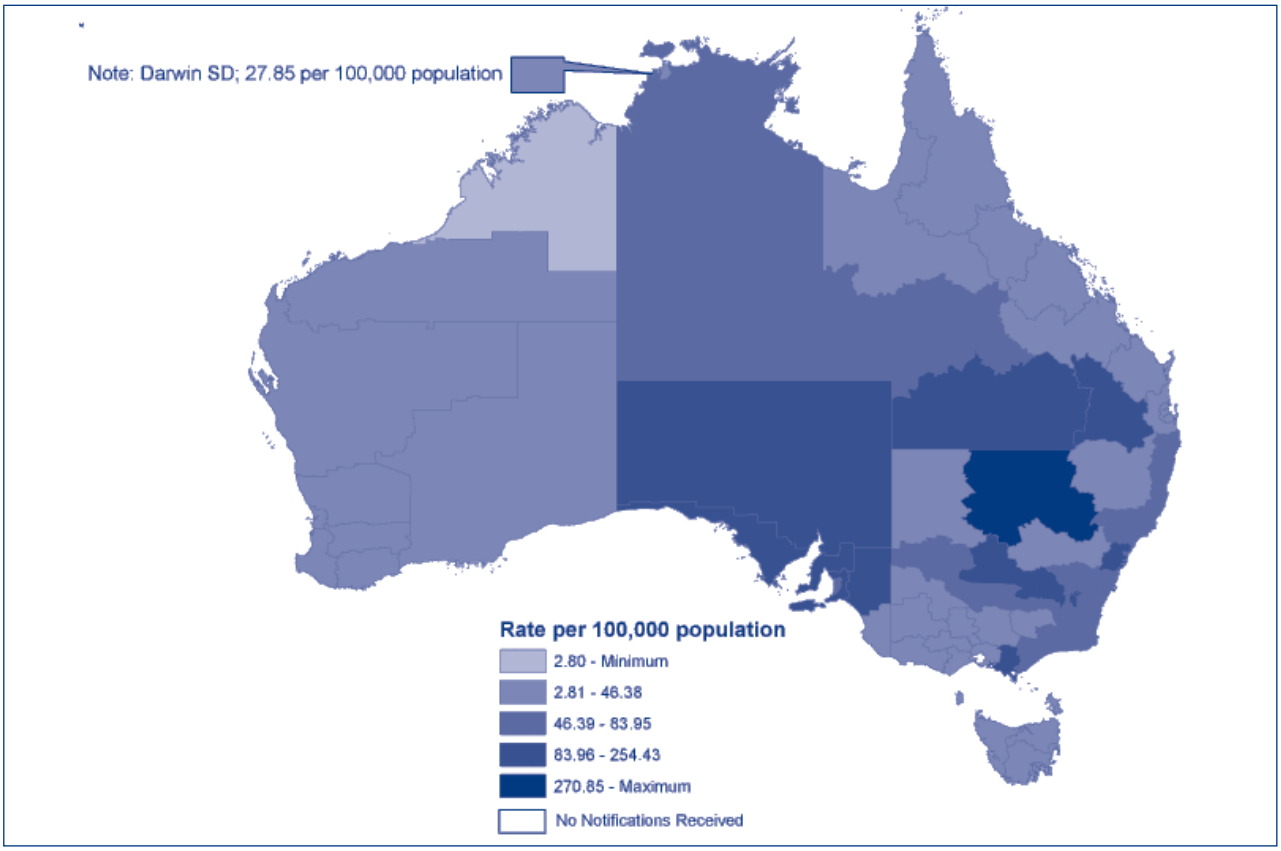
Notification rates of pertussis varied considerably by geographic location (Map 6).

The highest rates were reported from South Australia, New South Wales and the Australian Capital Territory. The trends in pertussis notification rates by month of diagnosis are shown for these 3 states and for Australia in Figure 46.

Figure 46. Notification rate for pertussis, Australian Capital Territory, New South Wales, South Australia, and Australia, 2003 to 2005, by month of notification



Map 6. Notification rate for pertussis, Australia, 2005, by Statistical Division of residence



Invasive pneumococcal disease

Case definition – Invasive pneumococcal disease

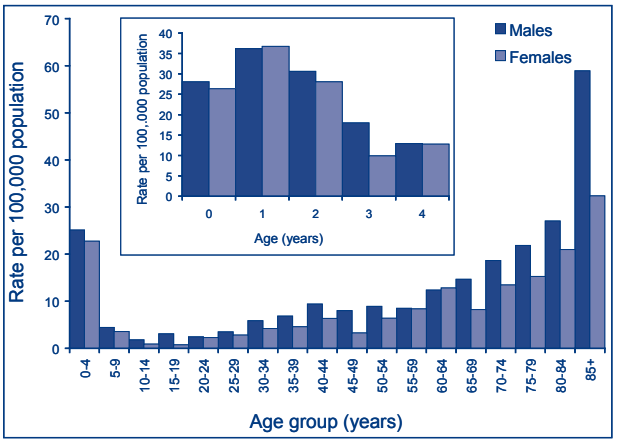
*Only **confirmed cases** are notified.*

***Confirmed case:** Requires isolation of* Streptococcus pneumoniae *from a normally sterile site by culture or detection by nucleic acid testing.*

There were 1,684 notifications of invasive pneumococcal disease (IPD) in Australia in 2005 giving a rate of 8.3 cases per 100,000 population. Notification rates declined in 2005 by 30% nationally with the declines in all jurisdictions of between 21 and 46%. The Northern Territory continued to have the highest notification rate (35 cases per 100,000 population) while Victoria had the lowest (6 cases per 100,000 population). The geographical distribution of IPD varied within states and territories, with the highest rates in central and northern Australia.

In 2005, rates of IPD fell in all age groups, particularly in children aged less than 5 years (20.4 cases per 100,000 population compared with 54.3 cases per 100,000 population in 2004). The rates in 1-year-olds also fell from 114 cases per 100,000 population in 2004 to 36.5 cases per 100,000 population. The highest rates in 2005 were in adults aged more than 85 years (40.9 cases per 100,000 population, Figure 47). The male to female ratio of IPD cases was 1.3:1.

Figure 47. Notification rate of invasive pneumococcal disease, Australia, 2005, by age group and sex



There were 164 cases of IPD among Indigenous people (9.7% of all cases). This represents a rate of 66 cases per 100,000 population compared with a rate of 7.6 cases per 100,000 population in non-Indigenous people.

Additional data were collected on cases of invasive pneumococcal disease in all Australian jurisdictions during 2005. Analyses of these data are reported separately.[17]

Poliomyelitis

Case definition – Poliomyelitis

*Both **confirmed cases** and **probable cases** are notified.*

***Confirmed case:** Requires isolation of wild-type poliovirus or detection of wild-type poliovirus by nucleic acid testing (confirmed in reference laboratory) and acute flaccid paralysis.*

***Probable case:** Requires acute flaccid paralysis not due to other causes as determined by the Polio Expert Committee.*

No cases of poliomyelitis were reported in Australia in 2005.

There were 36 notifications of acute flaccid paralysis (AFP) reported in 2005. Of these 30 occurred in children aged less than 15 years. This represents an AFP notification rate of 0.9 cases per 100,000 children aged less than 15 years which almost reaches the WHO indicator target for adequate AFP reporting of 1 case per 100,000 children. Three AFP cases, 1 aged more than 15 years, had poliovirus isolated from stool samples. The Polio Expert Committee reviewed the 3 cases and classified them as a non-polio AFP, diagnosed as transverse myelitis with the incidental isolation of a Sabin-like virus in 2 cases, while in the third, a type B/E toxin-producing *Clostridium botulinum* was detected and the case was classified as infant botulism.[18]

Rubella

Case definition – Rubella

*Both **confirmed cases** and **probable cases** are notified.*

***Confirmed case:** Requires isolation of rubella virus OR detection of rubella virus by nucleic acid testing OR IgG seroconversion or significant increase in antibody level or fourfold or greater rise in titre to rubella virus in the absence of recent rubella vaccination, OR detection of rubella specific IgM in the absence of recent rubella vaccination and confirmed in a reference laboratory.*

Probable case:** Requires **clinical evidence** AND **laboratory suggestive evidence** OR **epidemiological evidence.

***Laboratory suggestive evidence:** In a pregnant patient, detection of rubella-specific IgM that has not been confirmed in a reference laboratory, in the absence of recent rubella vaccination.*

Clinical evidence: A generalised maculopapular rash AND fever AND arthralgia/arthritis OR lymphadenopathy OR conjunctivitis

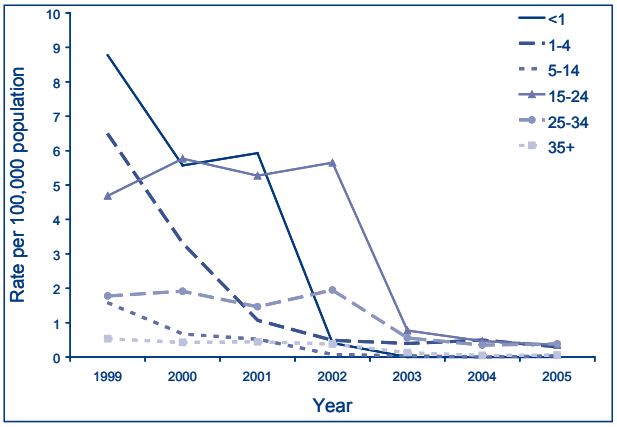
***Epidemiological evidence:** An epidemiological link is established when there is: 1. Contact between two people involving a plausible mode of transmission at a time when: a) one of them is likely to be infectious (about one week before to at least four days after appearance of rash) AND b) the other has an illness which starts within 14 and 23 days after this contact AND 2. At least one case in the chain of epidemiologically linked cases (which may involve many cases) is laboratory confirmed.*

In 2005, there were 31 notifications of rubella; 30 confirmed and 1 probable case, which represents a notification rate of 0.2 cases per 100,000 population. This is the lowest rate on record and a 6% reduction on 2004 (33 notifications, 0.2 cases per 100,000 population). In 2005, rubella cases were reported from NSW (10 cases), Queensland (9 cases), and 6 cases each from Victoria and Western Australia. No cases were reported from other jurisdictions.

The male to female ratio of notified cases in 2005 was 1:1, as in 2004. A predominance of male cases was seen in 1999 (M:F ratio: 1.4:1), 2002 (M:F ratio: 3.0:1) and 2003 (M:F ratio: 1.6:1).

Figure 48 shows trends in rubella notification rates in different age groups. The rates in all age groups remained stable in 2005. This pattern of declining rates by age group over time is similar to that for measles, with the exception of higher rubella notification rates in the 15–24 year age group, which persisted until 2002. Rubella cases in this age group were predominantly in males, and this may be related to the schoolgirl measles-mumps-rubella (MMR) vaccination program, prior to the inclusion of boys in 1993.

Figure 48. Trends in notification rate of rubella, Australia, 2005, by age group and sex



There was a single case of congenital rubella reported from Victoria in 2005: born to an unvaccinated overseas-born woman. Altogether there were 13 cases of rubella notified from women of child bearing age (15–49 years) in 2005.

Tetanus

Case definition – Tetanus

*Only **confirmed cases** are notified.*

***Confirmed case:** Requires isolation of* Clostridium tetani *from a wound in a compatible clinical setting and prevention of positive tetanospasm in mouse test using a specific tetanus antitoxin OR a clinically compatible illness without other apparent cause.*

In 2005, there were 2 notifications of tetanus. One was an 84 year old female and one was a 74-year old-male.

Childhood vaccination coverage reports

Estimates of vaccination coverage both overall and for individual vaccines for children at 12 months, 24 months and 6 years of age in 2005 are shown in Table 10, Table 11 and Table 12, respectively. During 2005, there were no significant changes in coverage for 'fully immunised' and individual vaccines for all 3 milestone ages. It is notable that the estimates for 'fully immunised' and diphtheria-tetanus-pertussis (DTP) vaccine at 24 months of age are higher than the 12 months coverage estimates since the 18 months DTPa booster was no longer required from September 2003. Estimates at 6 years of age for all vaccines still remain significantly lower than estimates at the 12 and 24 month milestones.

Vectorborne diseases

Notifications

During 2005, there were 4,935 notifications of mosquito-borne diseases reported to NNDSS. The notifiable mosquito-borne diseases include those caused by the alphaviruses (Barmah Forest virus and Ross River virus), flaviviruses (the viruses causing dengue, Murray Valley encephalitis, Kunjin and Japanese encephalitis) and malaria.

Alphaviruses

Alphaviruses are RNA viruses which cause disease epidemics characterised by fever, rash and polyarthritis. In Australia, Barmah Forest virus and Ross River virus are the alphaviruses of major public health significance. There are a variety of mosquito

Table 10. Percentage of Australian children born in 2004 immunised according to data available on the Australian Childhood Immunisation Register, estimate at one year of age

Birth date Vaccine	1 Jan–31 Mar 2004 % vaccinated	1 Apr–30 Jun 2004 % vaccinated	1 Jul–30 Sep 2004 % vaccinated	1 Oct–31 Dec 2004 % vaccinated
DTP	92.3	92.4	92.4	91.7
Polio	92.2	92.3	92.3	91.6
Hib	94.3	94.3	94.4	93.8
Hepatitis B	94.6	94.7	94.8	94.3
Fully immunised	91.0	91.0	91.0	90.2

Table 11. Percentage of Australian children born in 2003 immunised according to data available on the Australian Childhood Immunisation Register, estimate at two years of age

Birth date Vaccine	1 Jan–31 Mar 2003 % vaccinated	1 Apr–30 Jun 2003 % vaccinated	1 Jul–30 Sep 2003 % vaccinated	1 Oct–31 Dec 2003 % vaccinated
DTP	95.5	95.3	95.2	95.1
Polio	94.9	95.2	95.2	95.0
Hib	93.3	93.5	93.6	93.5
MMR	93.4	93.7	93.8	93.8
Hepatitis B	95.7	95.9	95.9	95.9
Fully immunised	91.8	92.1	92.1	92.1

Table 12. Percentage of Australian children born in 1999 immunised according to data available on the Australian Childhood Immunisation Register, estimate at six years of age

Birth date Vaccine	1 Jan–31 Mar 1999 % vaccinated	1 Apr–30 Jun 1999 % vaccinated	1 Jul–30 Sep 1999 % vaccinated	1 Oct–31 Dec 1999 % vaccinated
DTP	84.4	84.8	85.1	84.9
Polio	84.5	85.1	85.2	84.8
MMR	84.4	84.9	85.2	84.9
Fully immunised	83.2	83.8	84.0	83.8

vectors for Barmah Forest virus and Ross River virus that facilitate the transmission of these viruses in diverse environments (freshwater habitats, coastal regions, salt marshes, floodwaters, established wetlands and urban areas).[19]

Barmah Forest virus infection

Case definition – Barmah Forest virus infection

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of Barmah Forest virus, OR detection of Barmah Forest virus by nucleic acid testing, OR IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to Barmah Forest virus, OR detection of Barmah Forest virus-specific IgM.*

There were 1,319 notifications of Barmah Forest virus (BFV) infection notified to NNDSS in 2005, which accounts for 27% of total mosquito-borne disease notifications for the reporting period. Fifty-two per cent of BFV notifications were reported from Queensland (n=680) and 34% from New South Wales (n=448). BFV notifications during 2005 were 1.3 times the mean for the previous 5 years.

The highest rates of BFV notifications were reported by the Northern Territory (25.1 cases per 100,000 population), Queensland (17.2 cases per 100,000 population), and New South Wales (6.6 cases per 100,000 population). The national BFV notification rate was 6.5 cases per 100,000 population, which was the second highest since 1999.

There was a peak in the BFV notification rate in the Northern Territory (82.8 cases per 100,000 population) during April 2005 and this was almost 4 times the peak notification rate observed in May 2004 (Figure 49). Queensland reported a peak BFV notification rate in May 2005 (32.1 cases per 100,000 population), whereas New South Wales reported a peak BFV notification rate in April 2005 (10.6 cases per 100,000 population). These were slight increases over the peak notification rates in the previous season.

The highest rate of BFV infection in 2005, was reported in the Mid-North Coast area of New South Wales (67.5 cases per 100,000 population, Map 7).

Map 7. Notification rate of Barmah Forest virus infections, Australia, 2005, by Statistical Division of residence

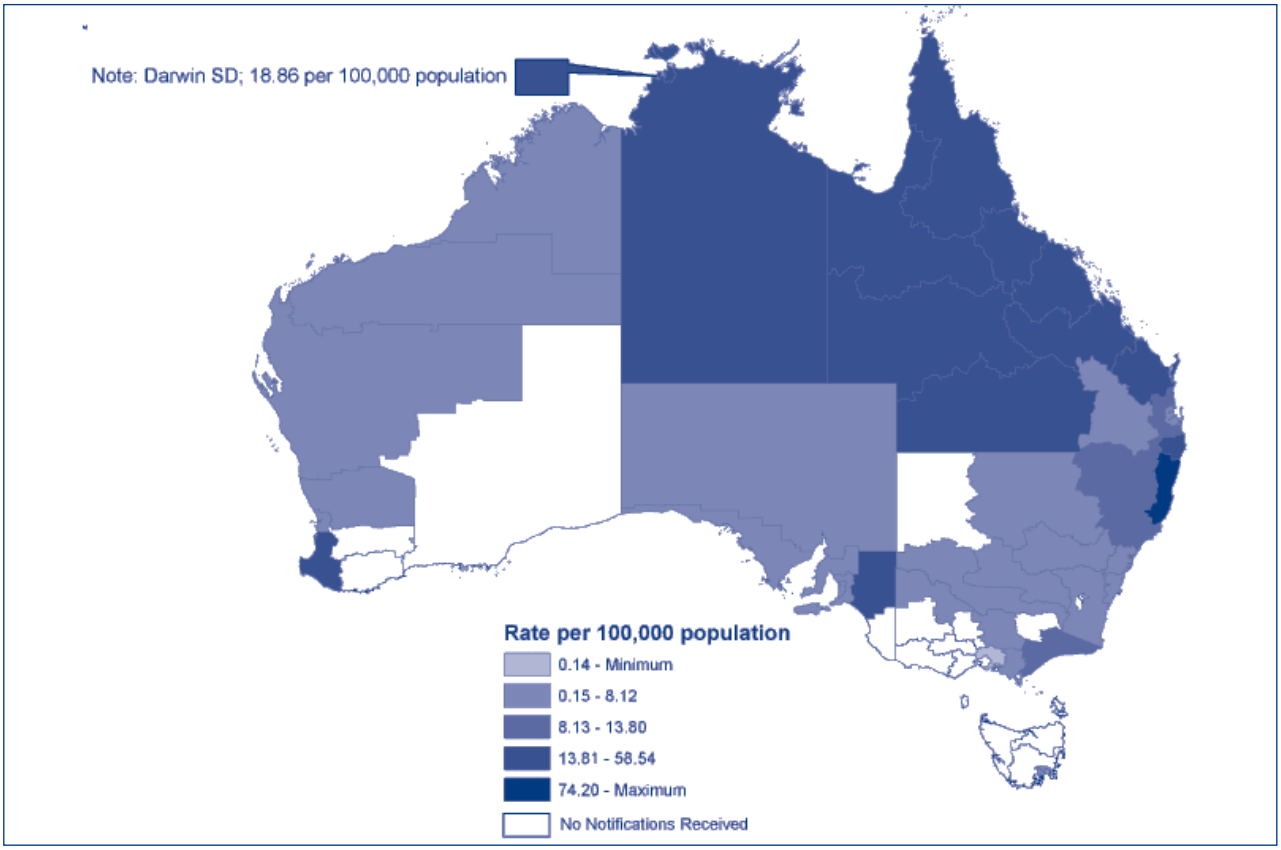


Figure 49. Notification rate of Barmah Forest virus infections, select jurisdictions, 1999 to 2005, by month and year of onset

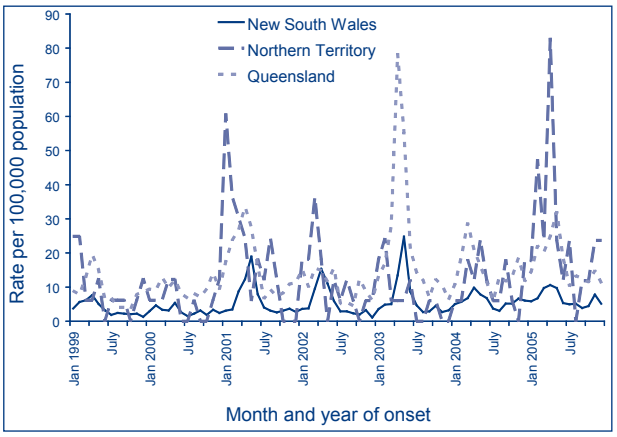
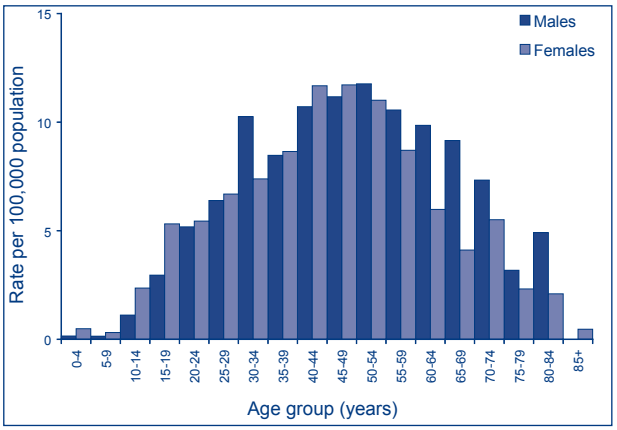


Figure 50 shows the age and sex distribution of BFV notifications. The BFV notification rate was highest amongst the 45–54 year age range (11.4 cases per 100,000 population), and the male to female ratio was 1:1. Males in the 50–54 year age group had the highest age-specific rate (11.8 cases per 100,000 population). The highest age-specific BFV notification rate in females was in the 40–44 and 45–49 year age groups (11.7 cases per 100,000 population).

Figure 50. Notification rate of Barmah Forest virus infections, Australia, 2005, by age group and sex



Ross River virus infection

Case definition – Ross River virus infection

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of Ross River virus, OR detection of Ross River virus by nucleic acid testing, OR IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to Ross River virus, OR detection of Ross River virus-specific IgM.*

There were 2,544 notifications of Ross River virus (RRV) infection reported to NNDSS in 2005, which accounts for over one half (52%) of the total mosquito-borne disease notifications received in 2005.

The largest contributors to RRV notifications in 2005 were Queensland (46%, n=1,179) and New South Wales (23%, n=585). The highest rates of infection were reported by the Northern Territory (103.1 cases per 100,000 population), Queensland (29.7 cases per 100,000 population), and Western Australia (15.5 cases per 100,000 population). The national RRV notification rate for 2005 was 12.5 cases per 100,000 population.

Map 8 shows that the highest rate of RRV infection in 2005 was in the Northern Territory (122.4 cases per 100,000 population) and Mackay in Queensland (107.9 cases per 100,000 population).

RRV infection notifications in the Northern Territory peaked in February 2005 at 319.5 cases per 100,000 population (Figure 51). This was a 52% reduction from the peak notification rate from January 2004. Queensland reported a peak notification rate for RRV in March 2005 at 99.6 cases per 100,000 population, which was almost a 40% reduction from the peak notification rate in March 2004 (251.6 cases per 100,000 population).

The age and sex distribution of RRV notifications are shown in Figure 52. The national notification rate was highest in the 45–49 year age group (22.8 cases per 100,000 population) and the highest BFV notification rate in males (21.4 cases per 100,000 population) was also observed in this age group. The highest notification rate in females was recorded in the 45–49 year age range (25.1 cases per 100,000 population).

Map 8. Notification rate of Ross River virus infections, Australia, 2005, by Statistical Division of residence

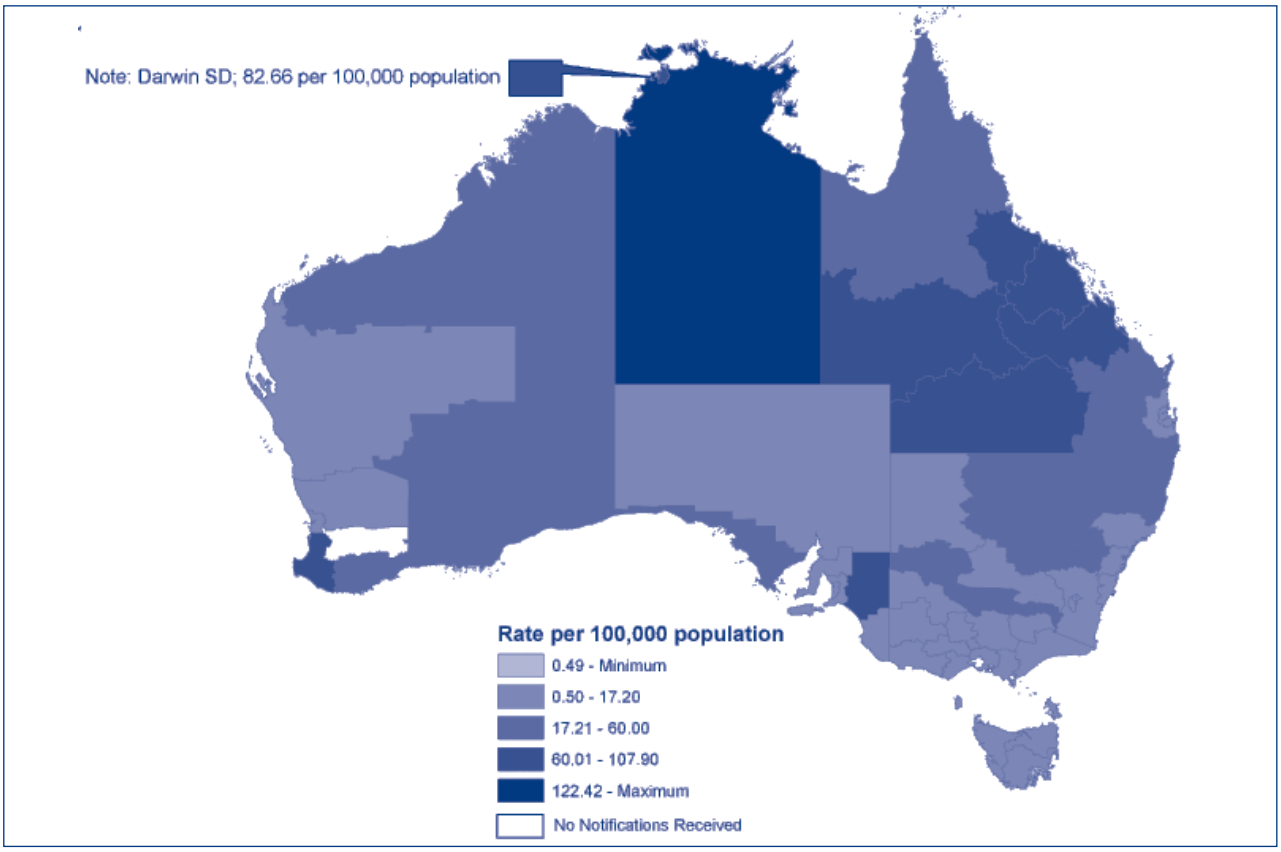


Figure 51. Notification rate of Ross River virus infections, select jurisdictions, 1999 to 2005, by month and season of onset

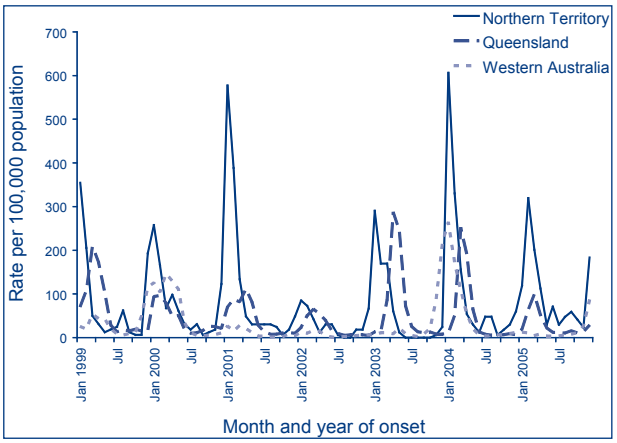
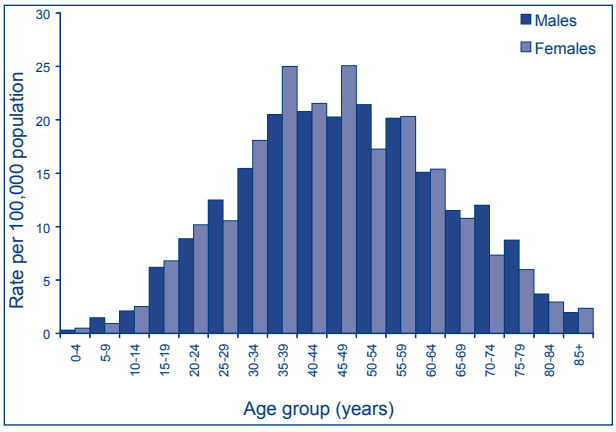


Figure 52. Notification rate of Ross River virus infections, Australia, 2005, by age group and sex



Flaviviruses

Flaviviruses are single-stranded RNA viruses, some of which are associated with epidemic encephalitis in various regions of the world. In Australia, the flaviviruses of public health importance are Murray Valley encephalitis (MVEV), Kunjin (KUNV), Japanese encephalitis virus (JEV) and dengue viruses (DENV).

The Sentinel Chicken Programme is a surveillance network involving New South Wales, the Northern Territory Victoria and Western Australia, and is designed to provide early warning of increased flavivirus activity.[20] Antibodies to MVEV and KUNV are detected in sentinel flocks located in four Australian states. Sentinel chicken surveillance reports from previous seasons have been

published,[21,22,23] and the latest report has been published as part of the National Arbovirus and Malaria Advisory Committee Annual Report 2005–06.[24]

Murray Valley encephalitis virus infection

Case definition – Murray Valley encephalitis virus

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of Murray Valley encephalitis virus, OR detection of Murray Valley encephalitis virus by nucleic acid testing, OR IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to Murray Valley encephalitis virus, OR detection of Murray Valley encephalitis virus-specific IgM in cerebrospinal fluid in the absence of IgM to Kunjin, Japanese encephalitis or dengue viruses, OR detection of Murray Valley encephalitis virus-specific IgM in serum in the absence of IgM to Kunjin, Japanese encephalitis or dengue viruses. This is only accepted as laboratory evidence for encephalitic illnesses.*

AND Non-encephalitic disease: acute febrile illness with headache, myalgia and/or rash, OR encephalitic disease: acute febrile meningoencephalitis characterised by one or more of the following: 1. focal neurological disease or clearly impaired level of consciousness, 2. an abnormal computerised tomograph or magnetic resonance image or electrocardiograph, 3. presence of pleocytosis in cerebrospinal fluid, OR asymptomatic disease: Case detected as part of a serosurvey should not be notified.

Confirmation of laboratory result by a second arbovirus reference laboratory is required if the case occurs in areas of Australia not known to have established enzootic/endemic activity or regular epidemic activity.

There were 2 cases of MVEV infection reported in 2005. In March 2005, a case of MVEV was reported in a 30-year-old male from Normanton, Queensland. The second case of MVEV disease was also reported in March 2005 in a 3-year-old boy from a community in Arnhem Land who was transferred to Royal Darwin Hospital for treatment. The boy had a relatively mild illness and made a complete recovery. The boy's community was located near an extensive freshwater wetland with numerous water birds and frequent high numbers of common banded mosquitoes *Culex annulirostris* and *Culex palpalis*: 2 vectors of MVEV.

Kunjin virus infection

Case definition – Kunjin virus

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of Kunjin virus, OR detection of Kunjin virus by nucleic acid testing, OR IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to Kunjin virus, OR detection of Kunjin virus-specific IgM in cerebrospinal fluid, OR detection of Kunjin virus-specific IgM in serum in the absence of IgM to Murray Valley encephalitis, Japanese encephalitis or dengue viruses. This is only accepted as laboratory evidence for encephalitic illnesses.*

AND Non-encephalitic disease: acute febrile illness with headache, myalgia and/or rash, OR encephalitic disease: acute febrile meningoencephalitis characterised by one or more of the following: 1. focal neurological disease or clearly impaired level of consciousness, 2. an abnormal computerised tomograph or magnetic resonance image or electrocardiograph, 3. presence of pleocytosis in cerebrospinal fluid, OR asymptomatic disease: case detected as part of a serosurvey should not be notified.

Confirmation of laboratory result by a second arbovirus reference laboratory is required if the case occurs in areas of Australia not known to have established enzootic/endemic activity or regular epidemic activity.

There was one notification of KUNV from Queensland during 2005, in a 48-year-old female with an onset in February 2005.

Dengue virus infection

Case definition – Dengue virus

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of dengue virus, OR detection of dengue virus by nucleic acid testing, OR IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to dengue virus, proven by neutralisation or another specific test, OR detection of dengue virus-specific IgM in cerebrospinal fluid, in the absence of IgM to Murray Valley encephalitis, Kunjin, or Japanese encephalitis viruses, OR detection of dengue virus-specific IgM in serum, except in North Queensland. In North Queensland, dengue virus-specific IgM in serum is acceptable evidence ONLY when this occurs during a proven outbreak.*

AND A clinically compatible illness (e.g. fever, headache, arthralgia, myalgia, rash, nausea, and vomiting, with a possible progression to dengue haemorrhagic fever, dengue shock syndrome or meningoencephalitis).

Confirmation of laboratory result by a second arbovirus reference laboratory is required if the case occurs in previously unaffected areas of Australia. Currently North Queensland is the only area with the potential for indigenous (epidemic) dengue virus in Australia.

During 2005, there were 218 notifications of dengue virus infection reported to NNDSS, of which Queensland reported 115 notifications (53%). The only locally acquired notifications were reported by Queensland (n=74), while other jurisdictions reported imported cases from overseas or from unknown sources. Queensland reported a peak in DENV notifications in March 2005 (n=49). This was much lower than in the previous 2 years (Figure 53).

The Queensland notifications resulted from outbreaks that peaked in March in the Torres Strait Islands, and in May in Townsville. A summary of identified outbreaks of locally-acquired cases is shown in Table 13.

Dengue serotype 4 was the major serogroup circulating in Queensland during these outbreaks.

Figure 53. Notifications of dengue (locally-acquired and imported cases), select jurisdictions, January 1999 to December 2005, by month and year of onset

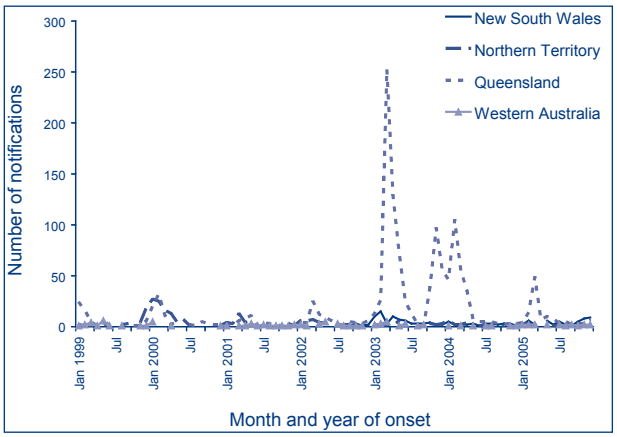


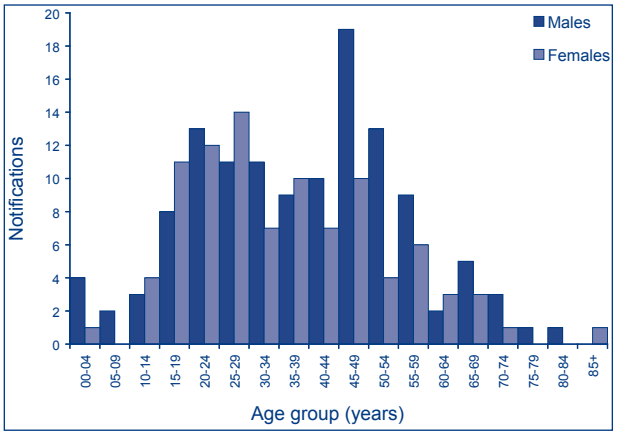
Table 13. Outbreaks of locally acquired cases of dengue, Queensland, 2005

Location	Reported cases	Duration	Type
Torres	56	10 weeks	Dengue 4
Townsville	18	16 weeks	Dengue 4

Data provided by Dr Jeffrey Hanna, Tropical Public Health Unit, Cairns, 2005.

The age and sex distribution of DENV notifications is shown in Figure 54. The highest rates occurred in the 45–49 year age group (19 cases) for males, and in females in the 25–29 year age range (14 cases).

Figure 54. Notifications of dengue (locally-acquired and imported cases), Australia, 2005, by age group and sex



Japanese encephalitis virus infections

Case definition – Japanese encephalitis virus

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of Japanese encephalitis virus, OR detection of Japanese encephalitis virus by nucleic acid testing, OR IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre of Japanese encephalitis virus-specific IgG proven by neutralisation or another specific test, with no history of recent Japanese encephalitis or yellow fever vaccination, OR detection of Japanese encephalitis virus-specific IgM in cerebrospinal fluid, in the absence of IgM to Murray Valley encephalitis, Kunjin and dengue viruses, OR detection of Japanese encephalitis virus-specific IgM in serum in the absence of IgM to Murray Valley encephalitis, Kunjin and dengue viruses, with no history of recent Japanese encephalitis or yellow fever vaccination.*

AND A clinically compatible febrile illness of variable severity associated with neurological symptoms ranging from headache to meningitis or encephalitis. Symptoms may include headache, fever, meningeal signs, stupor, disorientation, coma, tremors, generalised paresis, hypertonia, and loss of coordination. The encephalitis cannot be distinguished clinically from other central nervous system infections.

Confirmation of laboratory result by a second arbovirus reference laboratory is required if the case appears to have been acquired in Australia.

There were no human cases of JEV notified in 2005. The last JEV notification was reported by Queensland in February 2004 when a 66-year-old male acquired JEV from Papua New Guinea. There have been 9 other cases of JEV reported to NNDSS since 1995, although JEV was not nationally notifiable until 2001. Four of these 9 notifications were reported in Torres Strait Islanders from the Badu Island community. The other locally acquired JEV case was reported in a resident from the Cape York Peninsula, Queensland. The remaining 4 cases were reported as acquired overseas.

Flavivirus infections (NEC)

Case definition – Flavivirus infection (not elsewhere specified)

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of a flavivirus that cannot be identified in Australian reference laboratories or which is identified as one of the flaviviruses not otherwise classified, OR detection of a flavivirus, by nucleic acid testing, that cannot be identified in Australian reference laboratories or which is identified as one of the flaviviruses not otherwise classified, OR IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre of flavivirus specific IgG that cannot be identified or which is identified as being specific for one of the flaviviruses not otherwise classified. There must be no history of recent Japanese encephalitis or yellow fever vaccination, OR detection of flavivirus IgM in cerebrospinal fluid, with reactivity to more than one flavivirus antigen (Murray Valley encephalitis, Kunjin, Japanese encephalitis and/or dengue) or with reactivity only to one or more of the flaviviruses not otherwise classified, OR detection of flavivirus IgM in the serum, with reactivity to more than one flavivirus antigen (Murray Valley encephalitis, Kunjin, Japanese Encephalitis and/or dengue) or with reactivity only to one or more of the flaviviruses not otherwise classified. This is only accepted as laboratory evidence for encephalitic illnesses. There must be no history of recent Japanese encephalitis or yellow fever vaccination.*

AND Non-encephalitic disease: acute febrile illness with headache, myalgia and/or rash, OR encephalitic disease: acute febrile meningoencephalitis characterised by one or more of the following: 1. focal neurological disease or clearly impaired level of consciousness, 2. an abnormal computerised tomograph or magnetic resonance image or electrocardiograph, 3. presence of pleocytosis in cerebrospinal fluid.

Confirmation by a second arbovirus reference laboratory is required if the case cannot be attributed to known flaviviruses.

There were 29 flavivirus infection (not elsewhere classified or NEC) notifications during 2005; notified by Queensland (n=20), New South Wales (n=6) and Victoria (n=9).

There were 5 Kokobera notifications and 1 KUNV from Queensland in this category. Eight notifications of the alphavirus Sindbis were included under flavivirus infections (NEC).

Malaria

Case definition – Malaria

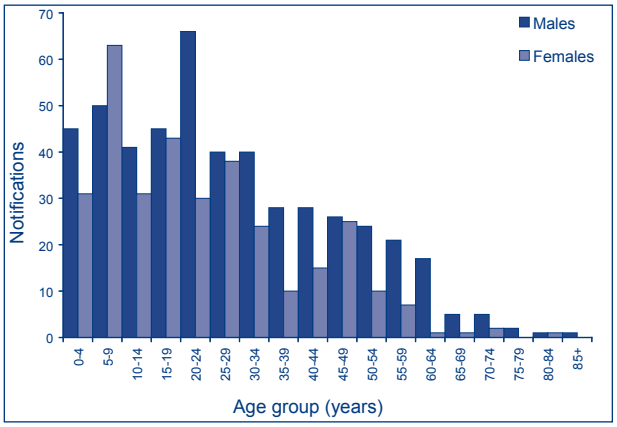
*Only **confirmed cases** are reported.*

***Confirmed case:** Requires detection and specific identification of malaria parasites by microscopy on blood films with confirmation of species in a laboratory with appropriate expertise, OR detection of* Plasmodium *species by nucleic acid testing.*

There were 822 notifications of malaria in Australia in 2005. The majority of cases were reported by Queensland (36%, n=297), New South Wales (25%, n=204), Victoria (13%, n=110) and Western Australia (10%, n=85). There were no reports of locally-acquired malaria during the reporting period.

The largest number (n=113) of malaria notifications was reported in refugee children,[25] in the 5–9 year age group (Figure 55). The male to female ratio was 1:0.7.

Figure 55. Notifications of malaria, Australia, 2005, by age group and sex



The infecting *Plasmodium* species was reported for 97% of malaria notifications in 2005 (Table 14). Of these 822 notifications, *P. falciparum* (56%, n=460) and *P. vivax* (35%, n=285) were the predominant species while untyped *Plasmodium* species accounted for 2% (n=13). The remaining cases were *P. ovale* (3%, n=24), *P. malariae* (1%, n=10) and mixed *Plasmodium* species infections (4%, n=30).

Zoonoses

Zoonoses are diseases and infections naturally transmitted between non-human vertebrate animals and humans.[26] Animal hosts play an essential role in maintaining the infection in nature, and humans are only accidental hosts.[27] Animals are thought to be the origin of approximately 75% of emerging human infectious diseases[28] and wildlife contribute

Table 14. Malaria notifications in Australia, 2005 by parasite type and jurisdiction

Parasite type	State or territory									
	Type (%)	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Plasmodium falciparum	56	6	119	29	149	33	18	39	67	460
Plasmodium malariae	1	0	2	0	4	0	0	2	2	10
Plasmodium ovale	3	0	6	2	8	0	0	6	2	24
Plasmodium vivax	35	6	64	16	133	5	6	50	5	285
Plasmodium species	2	0	2	0	3	3	0	2	3	13
Mixed P. falciparum and *P. vivax**	0.2	–	1	–	–	1	0	0	0	2
Mixed *P. falciparum* and other species*	3	–	9	–	–	1	0	11	0	21
Mixed *P. vivax* and other species*	0.1	–	1	–	–	0	0	0	0	1
Mixed infection (unspecified)*	0.7	–	0	–	–	0	0	0	6	6
Total	100	12	204	47	297	43	24	110	85	822

* New South Wales, South Australia, Tasmania, Victoria and Western Australia report mixed species infections per notified case. Queensland, the Northern Territory and the Australian Capital Territory report one notification for each species in a mixed infection.

† Unknown.

significantly to this threat. In Australia, the Federal Government, through the animal and human agencies, is proactively addressing this threat by strengthening the link between animal and human health systems. In 2005, zoonotic diseases notifiable to the NNDSS were anthrax, Australian bat lyssaviral or lyssaviral (unspecified) infection, brucellosis, leptospirosis, ornithosis and Q fever. During 2005, a total of 687 notifications of zoonotic disease (0.5% of total notifications) were made to the NNDSS.

Anthrax

Case definition – Anthrax

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of* Bacillus anthracis*-like organisms or spores confirmed by a reference laboratory*

OR Detection of Bacillus anthracis *by microscopic examination of stained smears, OR detection of* Bacillus anthracis *by nucleic acid testing AND Cutaneous: skin lesion evolving over 1–6 days from a papular through a vesicular stage, to a depressed black eschar invariably accompanied by oedema that may be mild to extensive, OR gastrointestinal: abdominal distress characterised by nausea, vomiting, anorexia and followed by fever, OR rapid onset of hypoxia, dyspnoea and high temperature, with radiological evidence of mediastinal widening, OR meningeal: acute onset of high fever, convulsions, loss of consciousness and meningeal signs and symptoms.*

No cases of anthrax were notified to NNDSS in 2005. The last reported human cases of anthrax in Australia (both cutaneous anthrax) occurred in July 1998 and February 1997.

Anthrax is a notifiable animal disease subject to compulsory government control strategies including: vaccination of susceptible livestock located on sites with a known history of anthrax; epidemiological investigation of outbreaks; quarantine and decontamination of affected premises; and safe disposal of carcases. Certain rural areas in central New South Wales and northern and north-eastern Victoria are associated with recurring cases of anthrax in cattle and sheep. In these endemic areas, anthrax has a low and decreasing prevalence and cases only occur sporadically.

In 2005, 9 confirmed anthrax incidents occurred. All except one occurred in the known anthrax endemic areas; the exception was in an area where anthrax had been reported in a neighbouring district in 1973. Cases involved sheep, cattle or both. In all cases, properties were subject to the recommended protocol of quarantine, carcass incineration or burial, site disinfection and vaccination of in-contact animals. All movements from affected properties were traced, and there was no risk of further spread of disease.[31]

Australian bat lyssaviral and lyssaviral (unspecified) infections

Case definition – Australian bat lyssavirus

*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of Australian bat lyssavirus confirmed by sequence analysis, OR detection of Australian bat lyssavirus by nucleic acid testing.*

Case definition – Lyssavirus (unspecified)

*Only **confirmed cases** are reported AND only where there is insufficient evidence to meet a case definition for Australian bat lyssavirus or rabies.*

***Confirmed case:** Requires positive fluorescent antibody test result for lyssaviral antigen on fresh brain smears, OR specific immunostaining for lyssaviral antigen on formalin fixed paraffin sections of central nervous system tissue, OR presence of antibody to serotype 1 lyssavirus in the cerebrospinal fluid, OR detection of lyssavirus-specific RNA (other than to Australian bat lyssavirus or rabies).*

AND Acute encephalomyelitis with or without altered sensorium or focal neurological signs.

No new cases of either Australian bat lyssaviral or lyssaviral (unspecified) infections were notified during 2005. The 2 known cases of human infection with Australian bat lyssavirus were fatal and occurred in 1996 and 1998 following close contact between bat-handlers and infected bats.

There are 2 strains of Australian bat lyssavirus known: one circulates in frugivorous bats, sub-order Megachiroptera, and the other circulates in the smaller, mainly insectivorous bats, sub-order Microchiroptera. Each strain has been associated with one human fatality. Surveillance indicates infected bats are widespread at a low frequency on the Australian mainland.[29] Research suggests that the virus has been associated with bats in Australia for more than 1,500 years[30] and that its recent 'emergence' is in all likelihood due to changes in human behaviour and in bat ecology due to habitat loss and changes in feed availability.

Brucellosis

In 2005, 41 cases of brucellosis were reported to the NNDSS, giving a national notification rate of 0.2 cases per 100,000 population. Cases were from Queensland (37 cases), New South Wales (3 cases) and Victoria (1 case). The highest notification rate (90 cases per 100,000 population) was from the Central West region of Queensland (Map 9). There is little evidence of change in the national or Queensland notification rates of brucellosis over the last 13 years (Figure 56). Most cases were male (n=35, male to female ratio 6:1), and of these, 80% were aged between 15 and 39 years.

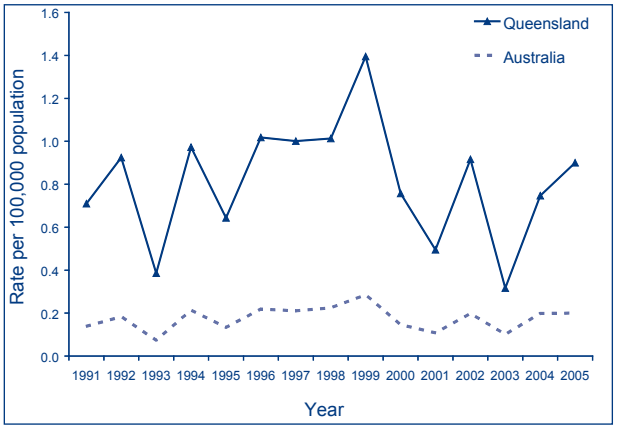
Case definition – Brucellosis

*Only **confirmed cases** are reported.*

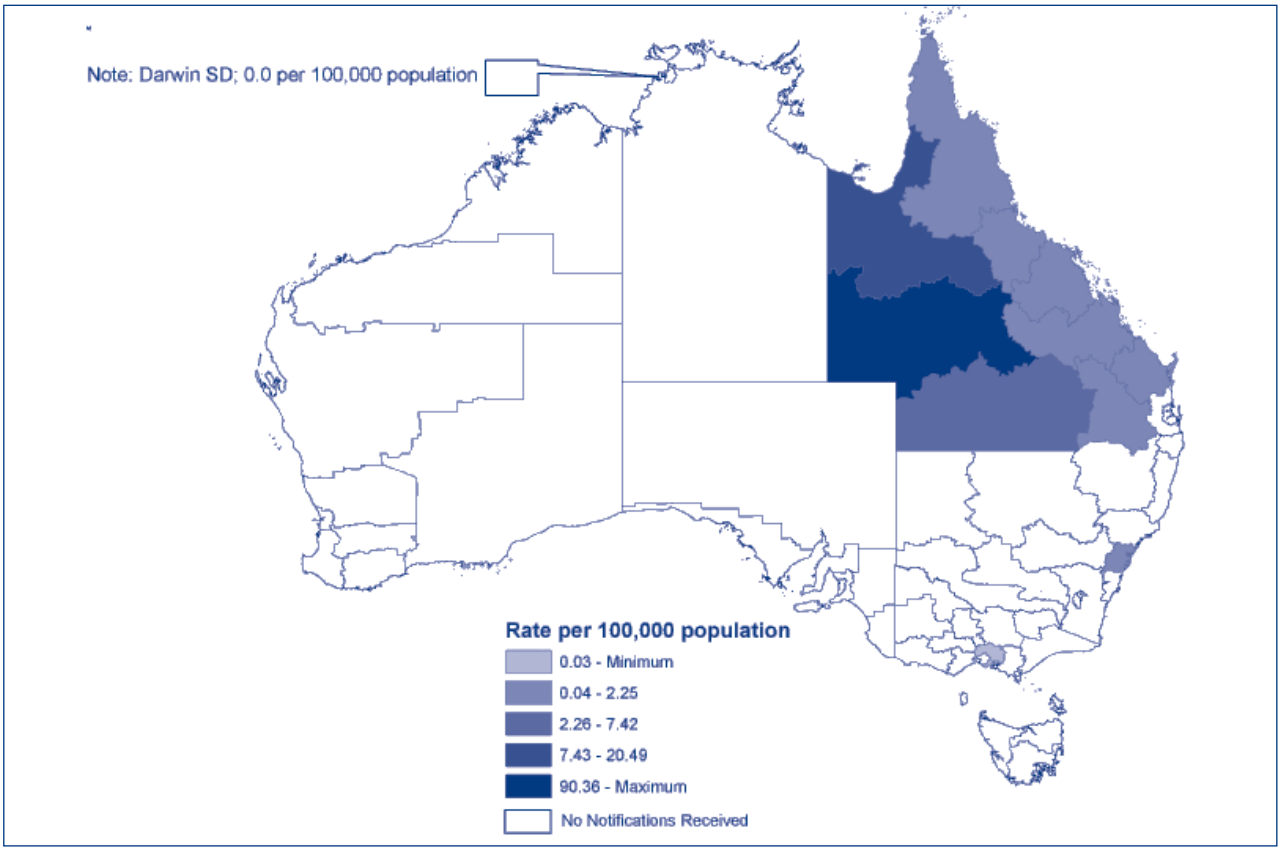
***Confirmed case:** Requires isolation of* Brucella *species, OR IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre in* Brucella *agglutination titres or complement fixation titres between acute and convalescent phase serum samples. (Where possible both tests should be conducted at the same laboratory), OR a single high* Brucella *agglutination titre.*

Species data was available for 31% of notifications (n= 13). Of these 10 were *Brucella suis*, (all acquired in Queensland) and 2 cases from New South Wales and a case from Victoria were *Br. melitensis* (all overseas acquired).

Figure 56. Trends in notification rate for brucellosis, Australia and Queensland, 1991 to 2005



Map 9. Notification rate for brucellosis, Australia 2005, by Statistical Division of residence



Bovine brucellosis (*Brucella abortus*) was eradicated from the Australian cattle herd in 1989[31] and is presently considered an exotic animal disease in Australia. Caprine and ovine brucellosis (caused by *Brucella melitensis*) has never been reported in Australian sheep or goats. Swine brucellosis (caused by *B. suis*) is confined to small areas of northern Australia where it occurs in feral pigs and occasionally spills over into domestic pigs. *B. suis* was not detected in domestic piggeries during 2005.[31]

Leptospirosis

Case definition – Leptospirosis

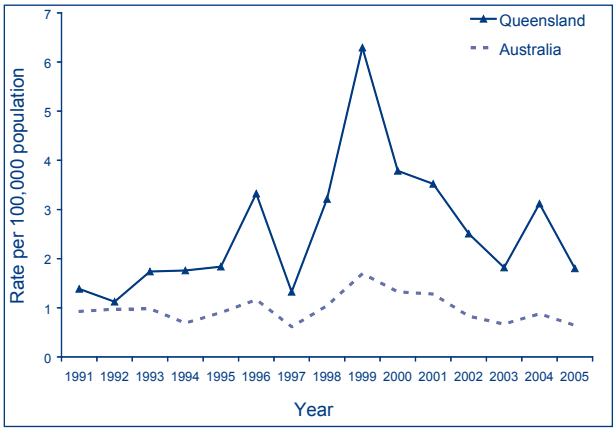
*Only **confirmed cases** are reported.*

***Confirmed case:** Requires isolation of pathogenic* Leptospira *species, OR a fourfold or greater rise in* Leptospira *agglutination titre between acute and convalescent phase sera obtained at least two weeks apart and preferably conducted at the same laboratory, OR a single* Leptospira *micro agglutination titre greater than or equal to 400 supported by a positive enzyme-linked immunosorbent assay IgM result.*

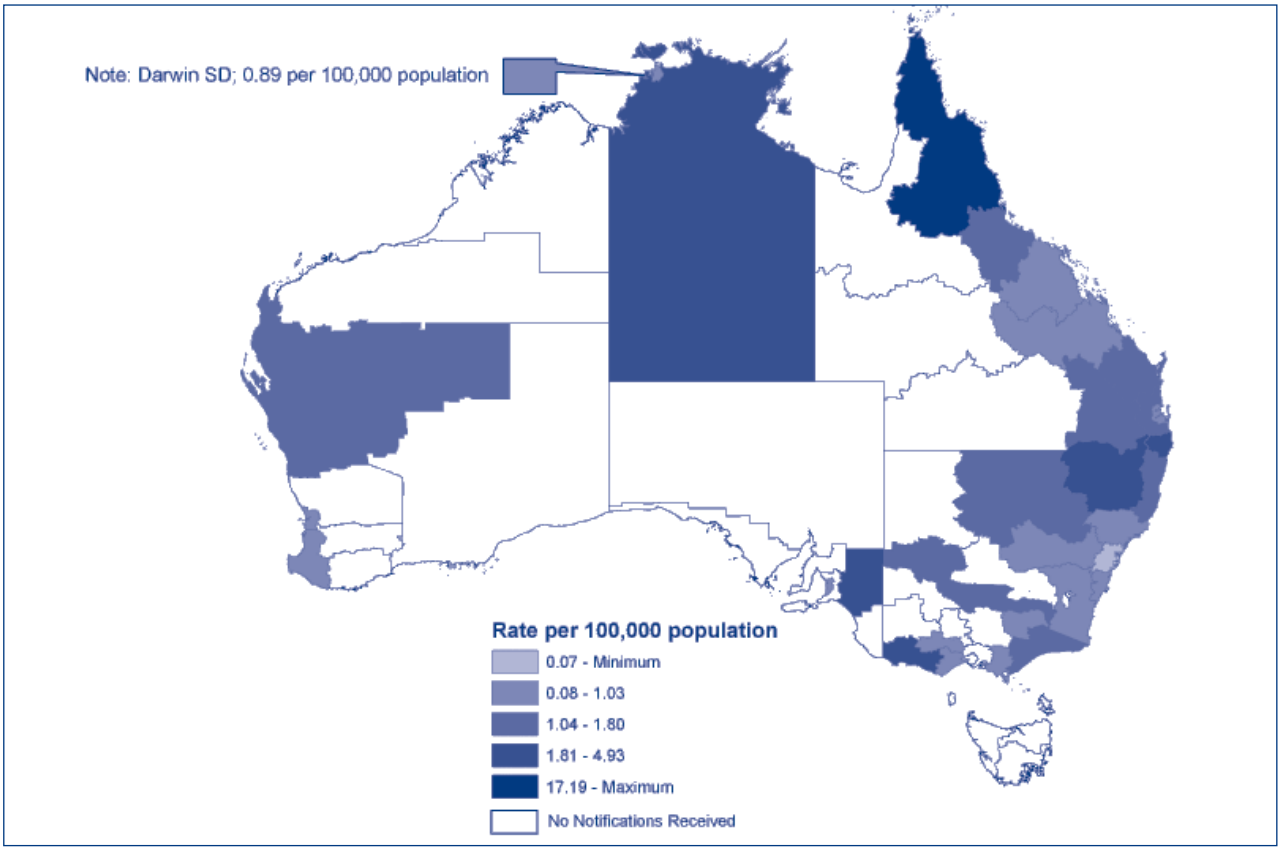
Leptospirosis is caused by spirochaetes of the genus, *Leptospira*. Nationally, 130 notifications of leptospirosis were received during 2005 (0.6 cases per 100,000 population). This rate is lower than in 2004. During the last 13 years, notification rates peaked in 1999 and declined from 2000 onwards (Figure 57).

In 2005, the highest notification rates were in Northern Territory (5 notifications, 2.5 cases per 100,000 population), Queensland (72 notifications, 1.8 cases per 100,000 population), and New South Wales (35 notifications, 0.5 cases per 100,000 population). Thirty-two per cent of all notifications were

Figure 57. Trends in notification rate for leptospirosis, Australia and Queensland, 1991 to 2005



Map 10. Notification rate for leptospirosis, Australia 2005, by Statistical Division of residence



from Far North Queensland (Map 10); the notification rate in this region was 18 cases per 100,000 population.

Most leptospirosis cases were male (n=109, male to female ratio 5:1), and the 30–34 year age group had the highest notification rate (2.8 cases per 100,000 population).

Ornithosis

Case definition – Ornithosis

*Both **confirmed cases** AND **probable cases** are reported.*

***Confirmed case:** Requires A fourfold rise or greater in antibody titre against* Chlamydia psittaci *as demonstrated by micro-immunofluorescence (MIF) on acute and convalescent sera (collected at least two weeks later) tested in parallel, OR detection of* C. psittaci *by nucleic acid testing or culture.*

AND Pneumonia, OR AT LEAST TWO of the following: fever, headache, myalgia, rigors, dry cough or dyspnoea.

AND Exposure to birds or bird products, or proximity to an outbreak of ornithosis.

***Probable case:** Requires a single high total antibody level or detection of IgM antibody to* C. psittaci *by MIF, OR a single high total antibody titre to* Chlamydia *species demonstrated by complement fixation (CF) in at least one sample obtained at least two weeks after onset of symptoms, OR a fourfold or greater rise in antibody titre against* Chlamydia *species as demonstrated by CF.*

AND Pneumonia, OR AT LEAST TWO of the following: fever, headache, myalgia, rigors, dry cough or dyspnoea.

AND Exposure to birds or bird products, or proximity to an outbreak of ornithosis.

In 2005, there were 161 ornithosis infections notified to NNDSS, giving a national rate of 0.8 cases per 100,000 population; representing a decrease on the 1.2 cases per 100,000 population reported in 2004. The national notification rate increased from 1997 to 2004, but in 2005 slightly decreased to equal that reported in 2001 (Figure 58).

New South Wales had the highest number of notifications (121 notifications, 1.8 cases per 100,000 population). Notifications were also received from Victoria (34 cases), Queensland (2 cases), Western Australia (3 cases) and South Australia (1 case). The majority of cases were male (n=89, male to female ratio 1.2:1). Eighty per cent of cases were aged 40 years and over, with the highest notification rate in males in the 80–84 year age group (6 notifications, 3.5 cases per 100,000 population) and in females in the 55–59 year age group (12 notifications, 1.9 cases per 100,000 population) (Figure 59).

Figure 58. Trends in notification rate for ornithosis, Australia, 1991 to 2005

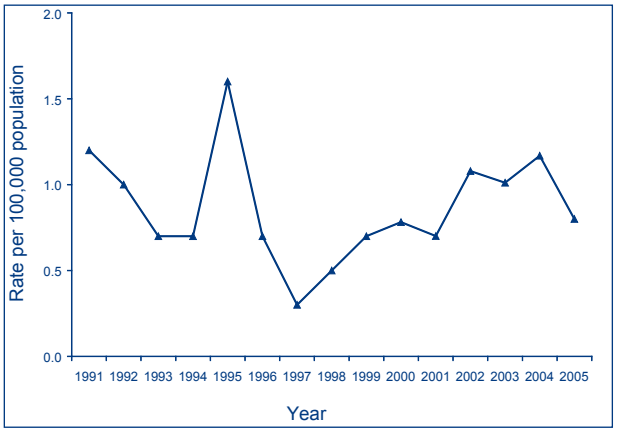
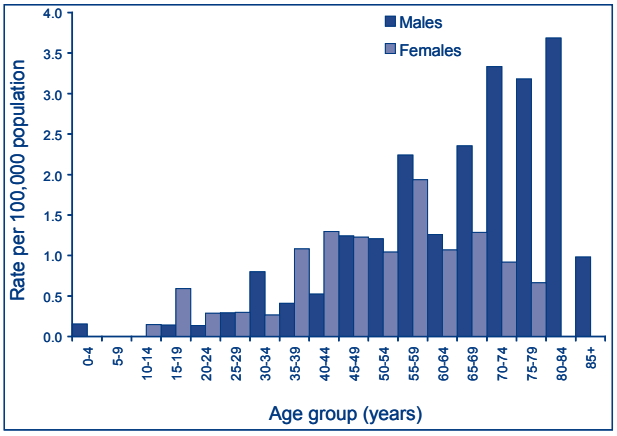


Figure 59. Notification rate for ornithosis, Australia, 2005, by age group and sex



Notification rates of ornithosis continued to be highest in the older age groups, which may reflect increased investigation and laboratory testing for atypical community-acquired pneumonia in this group. Previously reported outbreaks have been associated with aviaries, pet shops and poultry processing plants. An outbreak investigation in rural Victoria in 1995 showed an association with lawn mowing and gardening in areas with high numbers of native birds.[32] Shedding of *Chlamydia psittaci* into the environment by native birds and subsequent inhalation of aerosolised dust and bird excreta was postulated as the mechanism of human

infection. Sub-clinical infection with *C. psittaci* is common in numerous wild and domesticated bird species in Australia.[33, 34]

Q fever

Case definition – Q fever

*Only **confirmed cases** are reported.*

Confirmed case: *Requires detection of* Coxiella burnetii *by nucleic acid testing, OR seroconversion or significant increase in antibody level to Phase II antigen in paired sera tested in parallel in absence of recent Q fever vaccination, OR detection of* C. burnetii *by culture (note this practice should be strongly discouraged except where appropriate facilities and training exist).*

OR Detection of specific IgM in the absence of recent Q fever vaccination.

AND A clinically compatible disease.

In 2005, 355 cases of Q fever were notified to the NNDSS, a decrease of 23% on 2004. At 1.7 cases per 100,000 population, the Q fever notification rate in 2005 was lowest since 1991 (Figure 60). The highest rates of notifications were from Queensland (157 notifications, 4 cases per 100,000 population) and New South Wales (142 notifications, 2 cases per 100,000 population). The highest age-specific rates were in the 40–44 and 50–54 year age groups for males (5.7 cases per 100,000 population), and in the 45–49 and 50–54 year age groups for females (1.6 cases per 100,000 population). Few cases were reported from children or the elderly. The male to female ratio was 4:1.

Q fever has long been associated with work in the Australian livestock industry and abattoir workers are at high risk of infection. Since October 2000, abattoir workers and shearers have been eligible for free vaccination under the National Q Fever Management Program (Figure 61). The second phase of the Q fever vaccination program began in October 2001 to include workers in the beef, sheep and dairy industries and was completed on 30 June 2004. However, Victoria and South Australia have extended the Program until 30 June 2006 and Queensland has extended it until 30 June 2007.

Figure 60. Trends in notification rate for Q fever, Australia, 1991 to 2005

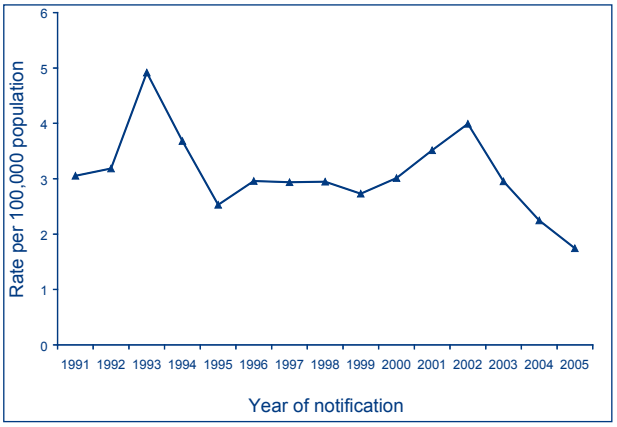
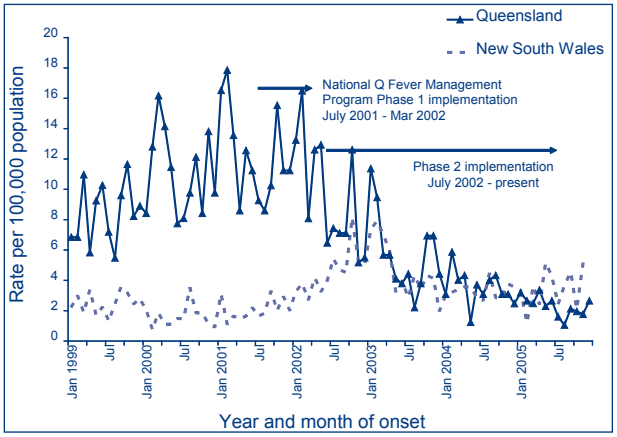


Figure 61. Notification rate for Q fever, Queensland and New South Wales, 1999 to 2005, by month of onset*



Other bacterial infections

Legionellosis, leprosy, meningococcal infection and tuberculosis were notifiable in all states and territories in 2005 and classified as 'other bacterial infections' in NNDSS. A total of 1,826 notifications were included in this group in 2005, which accounted for 1.4% of all the notifications to NNDSS, a similar total and proportion as in 2004 (1,719 notifications and 1.6% of total).

Legionellosis

Case definition – Legionellosis

*Both **confirmed cases** and **probable cases** are notified.*

Confirmed case: *Requires isolation of* Legionella, *OR the presence of* Legionella *urinary antigen OR seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to* Legionella.

AND fever or cough or pneumonia.

Probable case: *Single high titre antibody titre to* Legionella, *OR detection of* Legionella *by nucleic acid testing, OR detection of* Legionella *by direct fluorescence assay.*

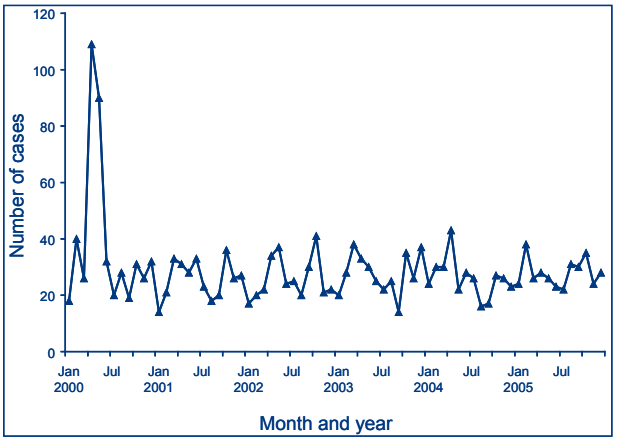
AND fever or cough or pneumonia.

Legionellosis includes notifications of infections caused by all *Legionella* species. There were 335 notifications of legionellosis reported in 2005, giving a national rate of 1.65 cases per 100,000 population. Of these, 264 cases (78.8%) were confirmed and 71 (21.2%) had a probable diagnosis.

In 2005, the highest rates of legionellosis were reported in South Australia (1.9 cases per 100,000 population, 58 cases) and Western Australia (1.7 cases per 100,000 population, 70 cases). Overall, the rate of notification was 1.65 cases per 100,000 population.

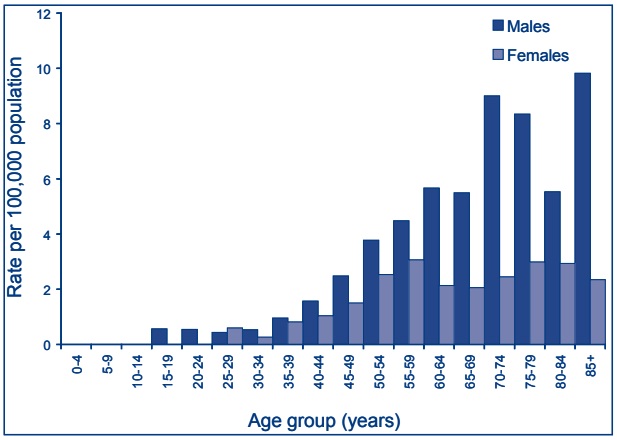
Legionellosis notifications showed a peak in autumn and spring, as in previous years (Figure 62). Rates of legionellosis have ranged between 0.8 and 2.6 cases per 100,000 population between 1999 and 2005; except in 2000, when rates reached 6.9 cases per 100,000 population as a result of the Melbourne aquarium outbreak with 125 cases.[34]

Figure 62. Trends in notification rate of legionellosis, Australia, 2000 to 2005, by month of onset



In 2005, men accounted for 220 of the 335 notified cases of legionellosis resulting in a male to female ratio of 1.9:1. There were no cases in children aged under 15. Overall, the highest rate of infection was 5.6 cases per 100,000 population in the 70–74 year age group. The highest rate in men occurred in the over 85 year age group (9.8 cases per 100,000, n=10) and in women the highest rate was in the 75–79 year age group (3 cases per 100,000 population, n=21) (Figure 63).

Figure 63. Notification rate for legionellosis, Australia, 2005, by age group and sex



Data on the causative species were available for 315 of 335 (94%) legionellosis cases. Of these, 159 (50.5%) cases were identified as *L. pneumophilia*, 153 (48.6%) were *L. longbeachae* and 3 (1%) cases were *L. micdadei* or *L. bozemanii* (Table 15).

Data on the deaths in legionellosis cases was available for 195 (58.2%) notifications. There were 16 deaths due to legionellosis in Australia in 2005, giving a case fatality rate of 4.8%. The break down of deaths by jurisdiction and infecting *Legionella* species is shown in Table 16. The case fatality rate for infections with *L. longbeachae* infections (4.6%) was higher than for *L. pneumophila* (3.8%) though this difference did not reach statistical significance. Case fatality rates may be overestimated given the large proportion of cases without details of outcomes.

Table 15. Notifications of legionellosis, Australia, 2005, by species and state or territory

Species	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Legionella longbeachae	0	24	1	15	45	1	10	57	153
Legionella pneumophila	0	64	1	27	12	1	44	10	159
*Other Legionella**	0	0	0	1	1	0	1	0	3
Unknown species	0	1	1	6	0	1	8	3	20
Total	0	89	3	49	58	3	63	70	335

* *Legionella micdadei* or *Legionella bozemanii.*

Table 16. Deaths due to legionellosis, Australia, 2005, by species and state or territory

Species	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Legionella longbeachae	0	0	0	1	1	1	0	4	7
Legionella pneumophila	0	0	0	1	2	0	1	2	6
Legionella micdadei	0	0	0	0	0	0	0	0	0
Unknown species	0	0	0	0	0	0	3	0	3
Total	0	0	0	2	3	1	4	6	16

An outbreak of 14 cases of Legionnaires' disease was reported from southern Sydney, New South Wales. Of these, 12 were initially diagnosed by detection of urinary antigen and 2 by a fourfold rise in antibody titres to *Legionella pneumophila* serogroup 1. Nine people were hospitalised and there were no fatalities. A notable factor in this outbreak was the mild nature of the symptoms. The people affected were aged from 18 to 88 years, and 86% were male.

Leprosy

Case definition – Leprosy

*Only **confirmed cases** are notified.*

***Confirmed case:** Requires demonstration of acid fast bacilli in split skin smears and biopsies prepared from ear lobe or other relevant sites or histopathological report from skin or nerve biopsy compatible with leprosy (Hansen's disease) examined by an anatomical pathologist or specialist microbiologist AND compatible nerve conduction studies or peripheral nerve enlargement or loss of neurological function not attributable to trauma or other disease process, or hypopigmented or reddish skin lesions with definite loss of sensation.*

Leprosy is a chronic infection of the skin and peripheral nerves with the bacterium *Mycobacterium leprae*. Leprosy is a rare disease in Australia, with the majority of cases occurring among Indigenous communities and migrants to Australia from leprosy-endemic countries.

In 2005, 10 leprosy cases were notified to NNDSS compared to 5 cases in 2004. There were 3 cases each in Western Australia, the Northern Territory and Queensland and 1 in New South Wales. Four cases occurred in men and 6 in women. Fifty per cent of cases were Indigenous Australians (2 in Western Australia and 3 in the Northern Territory). One case was reported to have been imported from overseas. The youngest case notified in 2005 was aged 19 years, and the oldest was aged 85 years.

Invasive meningococcal disease

Case definition – Invasive meningococcal disease

*Both **confirmed cases** and **probable cases** are notified.*

***Confirmed case:** Defined as isolation of* Neisseria meningitidis *from a normally sterile site. Alternatively, detection of meningococcus by nucleic acid testing, or Gram negative diplococci in Gram stain in specimens from a normally sterile site or from a suspicious skin lesion, OR high titre IgM or a significant rise in IgM or IgG titres to outer membrane protein antigens, OR positive polysaccharide antigen test in cerebrospinal fluid AND disease compatible with invasive meningococcal disease.*

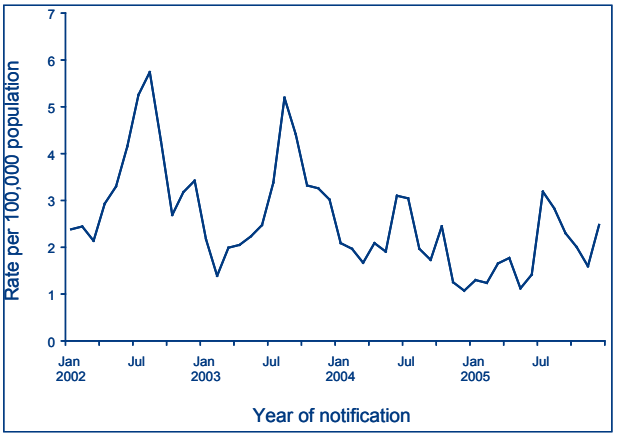
***Probable case:** Defined as the absence of evidence for other causes of clinical symptoms AND EITHER clinically compatible disease including haemorrhagic rash OR clinically compatible disease and close contact with a confirmed case within the previous 60 days.*

Historically, in Australia, serogroups B and C have been the major cause of invasive meningococcal disease; however in 2005 serogroup B caused more disease than serogroup C as a result of the National Meningococcal C Vaccination Program, which commenced in January 2003.[35]

In 2005, there were 393 notifications of invasive meningococcal disease in Australia, a decrease from 408 in 2004, and the lowest notifications since 1996. The national notification rate in 2005 was 1.9 cases per 100,000 population. The highest rate was reported from the Northern Territory (5.4 cases per 100,000, 11 cases).

Fifty-five per cent of cases (n=218) occurred in males, giving a male to female ratio of 1.2:1. As in previous years, the largest number of cases occurred in winter and spring (Figure 64). The majority of cases (n=352, 89.3%) were confirmed, and 42 (10.7%) had a probable diagnosis.

Figure 64. Trends in notification rate for meningococcal infections, Australia, 2002 to 2005, by month of onset

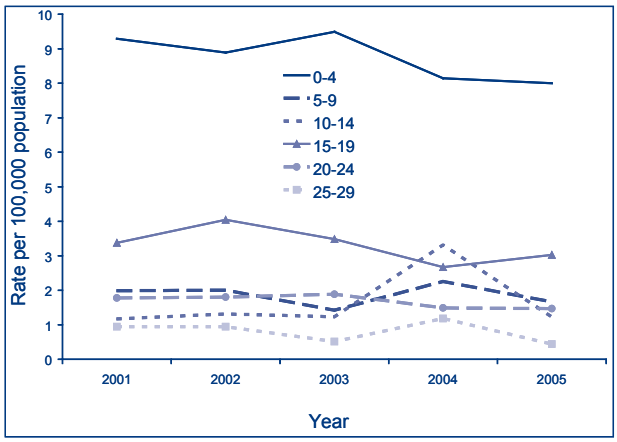


Of the 393 meningococcal notifications in 2005, 326 (82.7%) were serogrouped. Of these, 256 (78.5%) were serogroup B, 46 (14.1%) were serogroup C and 24 (7.4%) were infections with serogroup Y, serogroup W135 or serogroup A or serogroup 29–E (Table 17). In comparison, in 2004 81% (332/408) of notified cases were serogrouped; 240 (72.2%) were serogroup B and 74 (22%) were serogroup C.

The highest age specific meningococcal notification rate was in children aged 0–4 years with a rate of 10.4 cases per 100,000 population (131 cases). Seventy-seven per cent (101/131) of cases were serogroup B infection, which is the highest age-specific rate for serogroup B infection, with 8 cases per 100,000 population (Figure 65). In the 15–19 year age group, the overall rate of meningococcal infection was 3.9 cases per 100,000 population (54 cases), 42 (78%) of which were serogroup B. There were decreases in notification rates for the 25–29, 10–14 and 5–9 year age groups (Figure 65).

In the 25–29 year age group, there was a significant decrease in the number of serogroup B infections between 2004 and 2005 (OR=2.72, 1.51–4.92, p<0.001). There were 16 cases (1.2 cases per 100,000 population) in 2004, which was the highest reported number in the previous 5 years. In 2005, there were 6 notified cases (rate of 0.4 cases per 100,000 population), which is comparable to the number of notifications in 2003 (7 cases, 0.5 cases per 100,000 population). Decreases in the age-specific rates in the 10–14 and 5–9 years age groups were not significant (Figure 65).

Figure 65. Notification rate for meningococcal group B infections, Australia, 2001 to 2005, by age group



There was a marked decrease in meningococcal C infection rates during 2003, the year the National Meningococcal C Vaccination Program was introduced. In 2005, the decrease in rates of serogroup C infection was greatest in the 15–19 year age group (Figure 66). In 2002, the serogroup C infection rate in this age group was 4.6 cases per 100,000 population (63 cases). Since then the rate in this age

Table 17. Notifications of meningococcal infection, Australia, 2005, by serogroup and state or territory

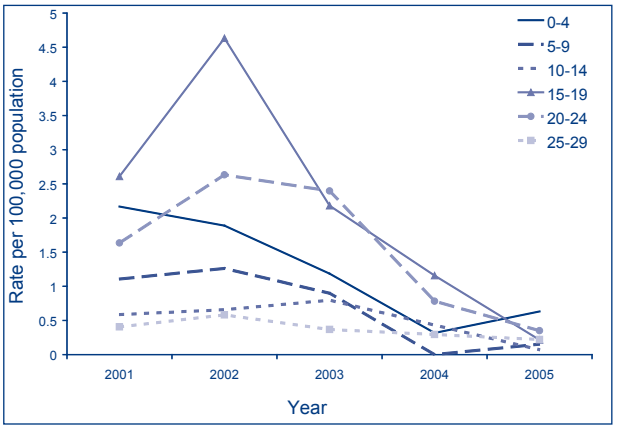
Species	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Serogroup B	4	72	6	43	18	10	62	41	256
Serogroup C	3	17	2	12	4	0	7	1	46
Other serogroups*	1	12	0	1	1	0	1	0	24
Unknown serogroup	0	39	3	6	3	0	13	3	67
Total	8	140	11	62	26	10	89	47	393

* Other includes serogroups A, 29-E, Y and W135.

group has decreased steadily to 0.2 cases per 100,000 population (3 cases) in 2005. There was a significant decrease in the number of infections between 2004 and 2005 (OR=5.35, 1.48–23.06, p<0.005).

There were decreases in rates of serogroup C infections in all age groups except 0–4 years. The rate in the 20–24 year age group fell from 0.8 cases per 100,000 population (11 cases) in 2004 to 0.3 cases per 100,000 (5 cases) in 2005. In the 0–4 year age group, 8 cases (0.3 cases per 100,000 population) of meningococcal C infection were reported compared to 4 cases in 2004 (0.6 cases per 100,000 population). This is the first increase in the notification rate since 2001 (Figure 66).

Figure 66. Notification rate for meningococcal group C infections, Australia, 2000 to 2005, by age group



The proportion of notified meningococcal samples that were not typed has decreased in recent years in all age groups. The proportion of untypeable samples in each age group is similar.

Data on death outcomes of meningococcal cases were available for 195 (49.5%) cases of meningococcal infection. There were 21 deaths recorded in 2005 giving a crude case fatality rate of 10.8%. The breakdown of deaths by jurisdiction and serogroup are shown in Table 18. The case fatality rate for group C meningococcal infections was 8.7%. For meningococcal group B infections it was 5.1%.

Laboratory-based meningococcal surveillance

The Australian Meningococcal Surveillance Programme was established in 1994 to monitor and analyse isolates of *Neisseria meningitidis* from cases of invasive meningococcal disease in Australia. The program is undertaken by a network of reference laboratories in each state and territory, using agreed standard methodology to determine the phenotype (serogroup, serotype and serosubtype) and the susceptibility of *N. meningitidis* to a core group of antibiotics. The results of the surveillance in 2005 have recently been published.[36]

In 2005, a total of 345 isolates of *N. meningitidis* were analysed by the program. Consistent with routine surveillance data, serogroup B continued to be the predominant strain (251 isolates, 72.8%) nationally, followed by serogroup C (50 isolates, 14.5%). Serogroup B strains predominated in all jurisdictions.

The pattern of age distribution for meningococcal infection varied by phenotype. The highest proportion of serogroup B infections, occurred in the 0–4 year age group (99 cases, 90%). The largest proportions of serogroup C occurred in the 20–24 year (62%), and over 25 years (27%) age groups. This represents a shift in the age distribution of serogroup C infections, which have previously been reported most frequently in the 15–19 year age group.

In 2005, 206 of the 345 (59.7%) isolates were tested for susceptibility to the penicillin group of antibiotics. While 65 (31.5%) specimens were fully sensitive to penicillin (MIC 0.03 mg/L or less), 140 (68%) were less sensitive (MIC 0.06–0.5 mg/L). All isolates tested

Table 18. Deaths due to meningococcal infection, Australia, 2005, by serogroup and state or territory

Species	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Serogroup B	0	4	0	1	1	0	4	3	13
Serogroup C	0	2	0	2	0	0	0	0	4
Other serogroups*	0	2	0	0	0	0	1	0	3
Unknown serogroup	0	0	0	0	0	0	1	0	1
Total	0	8	0	3	1	0	6	3	21

* Other includes serogroups A, Y and W135. (2 deaths were W135 infections, 1 was Y).

were susceptible to third generation cephalosporins and the prophylactic antibiotics, ciprofloxacin and rifampicin.

Tuberculosis (TB)

Case definition – Tuberculosis

*Only **confirmed cases** are notified.*

***Confirmed case:** Defined as of* Mycobacterium tuberculosis *complex by culture, OR detection of* M. tuberculosis *complex by nucleic acid testing except which it is likely to be due to previously treated or inactive disease OR clinical diagnosis of tuberculosis including clinical follow-up assessment to ensure a consistent clinical course.*

While Australia has one of the lowest rates of tuberculosis in the world, the disease remains a public health problem in the overseas-born and Indigenous communities. In 2005, 1,087 TB notifications were received by NNDSS; a rate of 5.4 cases per 100,000 population. In 2004, there were 1,076 cases notified nationally. The notification rate of TB was higher than the national average in the Northern Territory (6.7 cases per 100, 000 population), while the lowest rate occurred in Tasmania (1.3 cases per 100, 000 population).

The highest incidence was reported in people born overseas (20.6 cases per 100,000 population) and Indigenous Australians (5.2 cases per 100,000 population). The rate in the non-Indigenous Australian-born population was 0.8 cases per 100,000 population.

Further details can be found in the report published in this journal, 'Tuberculosis notifications in Australia, 2005'.[37]

Other communicable disease surveillance

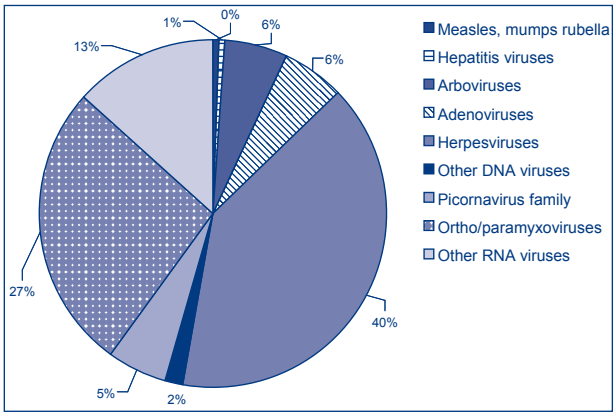
Laboratory Virology and Serology Reporting Scheme

The Laboratory Virology and Serology Reporting Scheme (LabVISE) is a passive surveillance scheme based on voluntary reports of infectious agents from sentinel virology and serology laboratories around Australia. LabVISE provides data on diagnoses of a number of infectious viruses, parasites and fungi. Interpretation of data from LabVISE is limited by uncertainties regarding its representativeness, lack of denominator data to calculate positivity rates, variable reporting coverage over time and lack of consistent case definitions. However, LabVISE has an important role in supplementing information of diseases under surveillance in NNDSS and in monitoring infectious agents that are not reported by other surveillance systems.

In 2005, a total of 12 laboratories reported 22,316 infectious agents to LabVISE. This represents a 15% decrease in the number of reports received in 2004 (Table 19). Most of the reports were from South Australia (30%), Queensland (27%) and Western Australia (16%) (Table 19).

Fifty three per cent (11,747) of all reports received by LabVISE were viral infectious agents, and the remaining 47% (10,569) were bacterial or other infectious agents. Among viruses, herpes viruses (40%; 4,691) were the most commonly reported followed by ortho/paramyxoviruses (27%; 3,158), which includes influenza, parainfluenza and respiratory syncytial viruses (Figure 67). Among non-viral infectious agents, *Chlamydia trachomatis* (48%; 5,049), *Bordetella pertussis* (15%; 1,573) and *Mycoplasma pneumoniae* (12%; 1,309) were the most commonly reported pathogens.

Figure 67. Reports of viral infections to the Laboratory Virology and Serology Reporting Scheme, 2005, by viral group



Australian Sentinel Practice Research Network

The Research and Health Promotion Unit of the Royal Australian College of General Practitioners operates the Australian Sentinel Practice Research Network (ASPREN). ASPREN is a national network of general practitioners that report each week on a number of conditions selected annually. Sentinel general practices contributing to the ASPREN scheme are mostly located in capital cities and larger regional centres on the east coast of Australia. The data provide an indicator of the burden of disease in the primary care setting and allows trends in consultation rates to be detected.

Table 19. Infectious agents reported to the Laboratory Virology and Serology Reporting Scheme, 2005, by state or territory

Organism	State and Territory								Total 2005	Total 2004
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Measles virus	–	1	–	4	2	–	1	–	8	35
Mumps virus	–	1	–	10	11	–	15	1	38	6
Rubella virus	–	–	–	6	–	–	4	2	12	20
Hepatitis A virus	–	3	2	17	29	–	–	2	53	51
Hepatitis D virus	–	–	–	1	9	–	4	–	14	8
Hepatitis E virus	–	–	–	2	1	–	9	–	12	14
Ross River virus	–	7	45	269	93	2	15	21	452	743
Barmah Forest virus	–	8	1	144	32	–	–	–	185	195
Flavivirus (unspecified)	–	1	3	30	–	–	3	–	37	102
Adenovirus type 1	–	1	–	–	–	–	6	–	7	–
Adenovirus not typed/pending	–	167	1	84	308	1	118	1	680	1,052
Herpes virus type 6	–	–	–	–	–	–	2	–	2	6
Cytomegalovirus	13	295	11	90	524	10	96	3	1,042	834
Varicella-zoster virus	7	152	11	882	386	11	48	2	1,499	2,061
Epstein-Barr virus	–	93	87	812	729	4	66	357	2,148	2,367
Poxvirus group not typed	–	1	–	–	–	–	1	–	2	2
Parvovirus	1	16	–	93	61	1	30	–	202	413
Coxsackievirus A9	–	3	–	–	–	–	–	–	3	1
Coxsackievirus A16	1	5	–	–	–	–	–	–	6	5
Echovirus type 5	–	2	–	–	–	–	–	–	2	–
Echovirus type 6	–	2	–	–	–	–	–	–	2	–
Echovirus type 7	–	8	–	–	–	–	–	–	8	12
Echovirus type 9	–	2	–	–	–	–	–	–	2	10
Echovirus type 11	–	4	–	–	–	–	–	–	4	20
Echovirus type 13	–	1	–	–	–	–	–	–	1	–
Echovirus type 18	1	13	–	–	–	–	–	–	14	19
Echovirus type 22	–	1	–	–	–	–	–	–	1	2
Echovirus type 30	1	34	–	–	1	–	–	–	36	7
Poliovirus type 1 (uncharacterised)	–	21	–	–	–	–	–	–	21	18
Poliovirus type 2 (uncharacterised)	–	19	–	–	–	–	–	–	19	21
Poliovirus type 3 (uncharacterised)	–	6	–	–	–	–	–	–	6	9
Rhinovirus (all types)	3	265	–	–	58	1	2	–	329	617
Enterovirus type 71 (BCR)	1	2	–	–	–	–	–	–	3	3
Enterovirus not typed/pending	5	126	–	25	13	1	18	–	188	205
Picornavirus not typed	–	–	–	–	–	1	–	–	1	4
Influenza A virus	–	159	3	97	356	–	93	–	708	492
Influenza A virus H3N2	–	1	–	–	–	–	1	–	2	–
Influenza B virus	–	46	–	25	146	1	39	–	257	219
Parainfluenza virus type 1	–	25	–	2	17	–	20	–	64	143
Parainfluenza virus type 2	–	22	–	4	18	–	5	–	49	15
Parainfluenza virus type 3	–	129	–	13	201	2	45	–	390	655
Respiratory syncytial virus	2	750	–	262	338	57	267	3	1,679	2,599
Paramyxovirus (unspecified)	–	–	–	–	–	–	9	–	9	–
HTLV-1	–	–	–	–	8	–	1	–	9	15
Rotavirus	2	484	1	1	588	12	182	–	1,270	1,247
Astrovirus	–	–	–	–	–	–	4	–	4	–

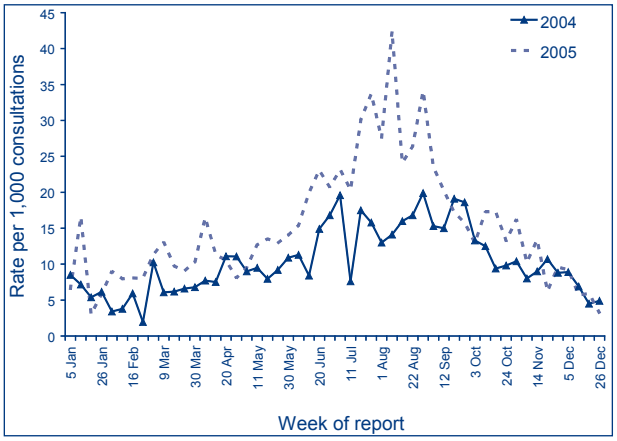
Table 19. Infectious agents reported to the Laboratory Virology and Serology Reporting Scheme, 2005, by state or territory, *continued*

Organism	State and Territory								Total 2005	Total 2004
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Norwalk agent	–	–	–	–	–	–	267	–	267	659
Chlamydia trachomatis not typed	10	773	10	2,324	1,809	59	61	3	5,049	5,257
Chlamydia pneumoniae	–	4	–	–	–	–	4	–	8	9
Chlamydia psittaci	–	2	–	–	1	–	50	–	53	173
Chlamydia species	–	–	–	–	–	–	1	–	1	3
Mycoplasma pneumoniae	–	111	20	458	342	45	244	89	1,309	1,374
Mycoplasma hominis	–	7	–	–	–	–	–	–	7	5
Coxiella burnetii (Q fever)	1	10	–	39	87	–	25	–	162	173
Rickettsia prowazeki	–	–	–	–	161	–	–	–	161	105
Rickettsia australis	–	–	–	–	–	–	1	–	1	–
Rickettsia tsutsugamushi	–	–	–	–	71	–	–	–	71	67
Rickettsia - Spotted fever group	–	–	–	–	232	4	–	–	236	139
Streptococcus group A	–	11	–	441	–	1	156	–	609	467
Yersinia enterocolitica	–	6	–	–	–	–	–	–	6	8
Brucella abortus	–	1	–	–	1	–	1	–	3	6
Brucella species	–	5	–	9	–	–	–	–	14	9
Bordetella pertussis	1	87	5	224	992	1	263	–	1,573	1,358
Bordetella parapertussis	–	–	–	–	–	–	2	–	2	1
Legionella pneumophila	–	5	–	–	10	–	8	–	23	77
Legionella longbeachae	–	2	–	–	41	–	8	–	51	76
Legionella species	–	–	–	1	–	–	–	–	1	15
Cryptococcus species	–	2	–	8	31	–	–	–	41	38
Leptospira species	–	1	–	19	13	–	–	–	33	23
Treponema pallidum	2	180	4	489	410	–	1	–	1,086	1,154
Entamoeba histolytica	–	–	–	7	–	–	7	–	14	14
Toxoplasma gondii	–	16	–	10	13	1	5	–	45	41
Echinococcus granulosus	–	1	–	–	9	–	–	–	10	15
Total	51	4,100	204	6,902	8,152	215	2,208	484	22,316	25,513

In 2005, influenza-like illnesses (ILI), gastroenteritis, and varicella infections (chickenpox and shingles) were the communicable diseases reported to ASPREN. Each week an average of 29 general practitioners (range 15–36) provided information from an average of 2,996 (range 1,081–3,698) consultations per week.

During 2005, a rise in reports of Influenza-like illness (ILI) to ASPREN was evident from mid-June (week 24), one week earlier than in 2004 (Figure 68). In 2005, the peak ILI rate was observed in early August (week 32) at 42 cases per 1,000 consultations, which was over twice the peak rate in 2004.

Figure 68. Consultation rate of influenza-like illness, ASPREN, 2005 compared with 2004, by week of report



Consultations for gastroenteritis fluctuated between 9 to 24 cases per 1,000 consultations. Rates reported for 2005, appeared to be similar to 2004 (Figure 69).

Reports of varicella infections were available only from week 13 in 2005. Rates of shingles exceeded those for chickenpox in most weeks but there was no recognisable seasonal pattern (Figure 70).

Figure 69. Consultation rate of gastroenteritis, ASPREN, 2005 compared with 2004, by week of report

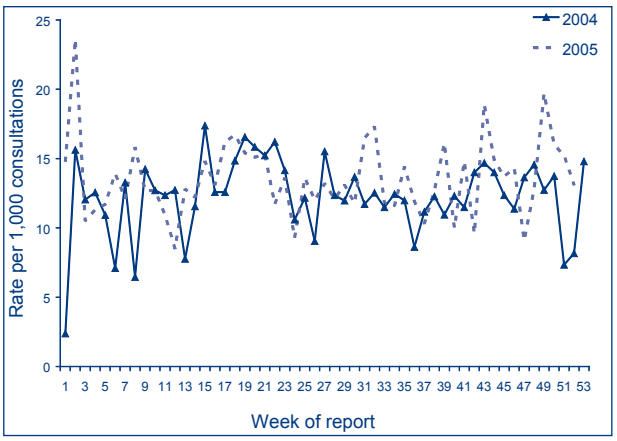
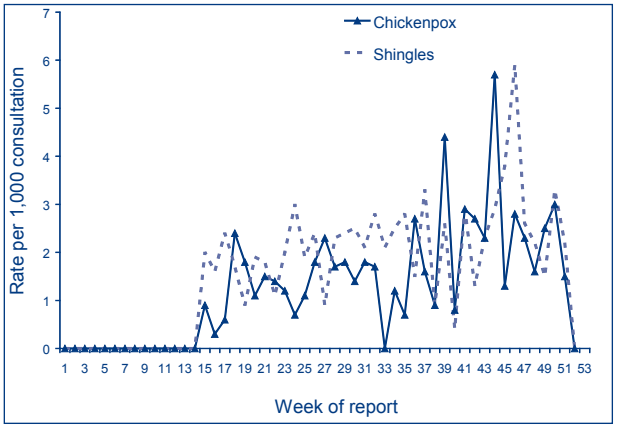


Figure 70. Consultation rate for varicella infections, ASPREN, 2005, by week of report



Appendices

Appendix 1. Mid-year estimate of Australian population, 2005, by state or territory

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Male	160,939	3,369,591	106,695	1,981,864	764,238	239,448	2,478,879	1,007,798	10,110,836
Female	164,222	3,404,658	96,098	1,982,104	777,795	245,815	2,543,467	1,002,315	10,217,773
Total	325,161	6,774,249	202,793	3,963,968	1,542,033	485,263	5,022,346	2,010,113	20,328,609

Appendix 2. Mid-year estimate of Australian population, 2005, by state or territory and age group

Age	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
0–4	20,185	424,073	17,499	253,957	87,820	30,072	306,350	124,313	1,264,507
5–9	20,453	438,016	16,527	268,698	94,737	31,905	317,482	133,407	1,321,465
10–14	21,810	457,361	16,495	284,410	101,053	34,539	334,764	141,554	1,392,249
15–19	23,604	454,452	14,771	277,675	103,078	34,076	335,465	145,108	1,388,471
20–24	28,286	464,323	16,299	285,703	104,614	30,988	356,918	144,095	1,431,363
25–29	25,530	456,598	16,816	263,517	94,057	26,614	343,837	134,164	1,361,259
30–34	25,473	510,560	18,294	293,071	103,565	30,345	380,007	147,174	1,508,671
35–39	24,087	482,143	16,916	284,852	107,820	32,239	374,844	148,621	1,471,707
40–44	24,538	510,760	16,274	299,080	115,971	36,313	378,221	155,070	1,536,470
45–49	23,823	481,819	14,184	282,153	113,184	36,261	358,481	149,107	1,459,226
50–54	22,452	439,663	12,741	258,771	104,974	33,954	325,527	136,630	1,334,942
55–59	20,276	410,855	10,027	245,445	100,585	32,292	302,834	122,881	1,245,336
60–64	13,341	317,556	6,547	186,500	75,820	25,240	229,389	90,449	944,942
65–69	9,858	262,815	3,971	146,142	63,414	20,833	191,996	72,702	771,804
70–74	7,365	217,394	2,274	113,434	53,425	16,681	159,522	56,896	627,027
75–79	6,204	193,608	1,589	96,885	50,420	14,316	142,325	47,626	552,987
80–84	4,587	141,475	882	69,274	37,591	10,480	103,364	33,499	401,156
85+	3,289	110,778	687	54,401	29,905	8,115	81,020	26,817	315,027
Total	325,161	6,774,249	202,793	3,963,968	1,542,033	485,263	5,022,346	2,010,113	20,328,609

Appendix 3. Completeness of National Notifiable Diseases Surveillance System data received, Australia, 2005, by state or territory

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total notifications	1,677	31,016	5,132	27,534	7,992	1,970	24,246	14,356	113,923
Sex									
Unknown/missing	1	0	0	7	0	1	151	7	167
Per cent complete*	99.9	100	100	99.9	100	100	98.9	100	99.9
Age									
Unknown/missing	1	24	9	0	2	7	106	12	161
Per cent complete*	99.9	99.9	99.8	100	100	99.6	99.6	99.9	99.8
Indigenous status†									
Not stated/missing	1,619	22,886	394	17,725	861	1,465	11,540	3	56,493
Per cent complete*	3.5	26.2	92.3	35.6	89.2	25.6	52.4	100	50.4

* Data completeness = (Total – unknown or missing)/total x 100.

† 'Indigenous status' is a variable defined by the following values:

1. Indigenous – (Aboriginal but not Torres Strait Islander origin);
2. Indigenous – (Torres Strait Islander but not Aboriginal origin);
3. Indigenous – (Aboriginal and Torres Strait Islander origin);
4. Not Indigenous – (not Aboriginal or Torres Strait Islander origin);
9. Not stated

Blank/missing/null =No information provided

Author details

Rhonda Owen[1]
Paul W Roche[1]
Kirsty Hope[1]
Keflemariam Yohannes[1]
April Roberts[1]
Conan Liu[1]
Stefan Stirzaker[1]
Fiona Kong[1]
Mark Bartlett[2]
Basil Donovan[3]
Iain East[4]
Gerard Fitzsimmons[5]
Ann McDonald[3]
Peter B McIntyre[6]
Robert I Menzies[6]

1. Surveillance Policy and Systems Section, Office of Health Protection, Australian Government Department of Health and Ageing, Canberra, Australian Capital Territory
2. Manager, Surveillance, Communicable Diseases Branch, NSW Health Department, North Sydney, New South Wales
3. National Centre in HIV Epidemiology and Clinical Research, University of New South Wales, Sydney, New South Wales
4. Epidemiology and Modelling Section, Office of the Chief Veterinary Officer, Australian Government Department of Agriculture, Fisheries and Forestry, Canberra, Australian Capital Territory
5. Epidemiologist, OzFoodNet, Food Safety and Surveillance Section, Office of Health Protection, Australian Government Department of Health and Ageing, Canberra, Australian Capital Territory
6. National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, Westmead, New South Wales

Corresponding author: Ms Rhonda Owen, Surveillance Policy and Systems Section, Office of Health Protection, Australian Government Department of Health and Ageing, PO Box 9848 (MDP 14), CANBERRA ACT 2601. Telephone: +61 2 6289 9040. Facsimile: +61 2 6289 7791. Email: rhonda.owen@health.gov.au

References

1. Australian Institute of Health and Welfare (AIHW). Australian Hospital Statistics 2003-04. 2005. Available from: http://www.aihw.gov.au/publications/hse/ahs03-04/Supplementary_Chapter_9.xls Accessed on 3 November 2005.
2. National Centre in HIV Epidemiology and Clinical Research HIV/AIDS. viral hepatitis and sexually transmissible infection in Australia, Annual Surveillance Report 2006. Sydney: The University of New South Wales,; 2006.
3. Ministerial Advisory Committee on AIDS SHaH, Hepatitis C Sub-Committee, Hepatitis C Virus Projections Working Group. Estimates and projections of the hepatitis C virus epidemic in Australia. Sydney: NCHECR, the University of New South Wales; 2006.
4. OzFoodNet Working Group. Burden and causes of foodborne disease in Australia: annual report of the OzFoodNet network, 2005. *Commun Dis Intell* 2006;30:278-300.
5. Cowie C, Breschkin A, Kelly H. Hepatitis E virus: overseas epidemics and Victorian travellers. *Med J Aust* 2005;183:491-493.

6. National Enteric Pathogens Surveillance System. NEPPS human annual report, 2005. Melbourne: Microbiological Diagnostic Unit, University of Melbourne; 2006.
7. Hocking J, Fairley C, Counahan M, Crofts N. The pattern of notification and testing for genital *Chlamydia trachomatis* infection in Victoria, 1998-2000: an ecological analysis. *Aust N Z J Public Health* 2003;27:405-408.
8. Chen M, Fairley C, Donovan B. Nowhere near the point of diminishing returns: correlations between chlamdyia testing and notification rates in New South Wales. *Aust N Z J Public Health* 2005;29:249-253.
9. Australian Government Department of Health and Ageing. National Sexually Transmissible Infections Strategy: 2005-2008. Canberra: Commonwealth of Australia; 2005.
10. Chen M, Donovan B. Genital *Chlamydia trachomatis* infection in Australia: epidemiology and clinical implications. *Sex Health* 2004;1:189-196.
11. Bowden F, on behalf of the National Donovanosis Eradication Advisory Committee. Donovanosis in Australia: going, going... *Sex Transm Infect* 2005;81:365-366.
12. Australian Gonococcal Surveillance Programme. Annual report of the Australian Gonococcal Surveillance Programme, 2005. *Commun Dis Intell* 2006;30:205-210.
13. Jin F, Prestage G, Kippax S, Pell C, Donovan B, Kaldor J, et al. Epidemic syphilis among homosexually active men in Sydney. *Med J Aust* 2005;183:179-183.
14. Fairley C, Hocking J, Medland N. Syphilis: back on the rise, but not unstoppable. *Med J Aust* 2005;183:172-173.
15. Firestone SM, Barr IG, Roche PW, Walker JC. Annual report of the National Influenza Surveillance Scheme. *Commun Dis Intell* 2006;30:189-200.
16. Annual NZ Notifiable Disease Report, 2005. Available from: ://www.surv.esr.cri.nz/surveillance/annual_surveillance.php?we_objectID=1016 Accessed on.
17. Roche P, Krause V, Cook H, Bartlett M, Coleman D, Davis C, et al. Invasive pneumococcal disease in Australia, 2005. *Commun Dis Intell* 2007;31:86-100.
18. Brussen KA, Roberts J, Ibrahim A, Stambos V, Thorley BR. Annual report of the Australian National Poliovirus Reference Laboratory, 2005. *Commun Dis Intell* 2006;30:334-340.
19. Russell RC, Dwyer DE. Arboviruses associated with human disease in Australia. *Microbes Infect* 2000;2:1693-1704.
20. Broom AK, Azuolas J, Hueston L, Mackenzie JS, Melville L, Smith DW, *et al.* Australian encephalitis: Sentinel Chicken Surveillance Programme. *Commun Dis Intell* 2001;25:157-160.
21. Broom AK, Whelan PI, Azuolas J, Dwyer D, Hueston L, Mackenzie JS, *et al.* Sentinel Chicken Surveillance Programme in Australia, 1 July 2001 to 30 June 2002. *Commun Dis Intell* 2002;26:428-429.
22. Broom AK. Sentinel Chicken Surveillance Program in Australia, July 2002 to June 2003. *Commun Dis Intell* 2003;27:367-369.
23. Broom AK, Whelan PI. Sentinel Chicken Surveillance Program in Australia, July 2003 to June 2004. *Commun Dis Intell* 2005;29:65-70.
24. Liu C, Johansen C, Kurucz N, Whelan P. Communicable Diseases Network Australia National Arbovirus and Malaria Advisory Committee annual report 2005-06. *Commun Dis Intell* 2006;30:411-429.
25. Liu C, Broom AK, Kurucz N, Whelan PI. Communicable Diseases Network Australia: National Arbovirus and Malaria Advisory Committee annual report 2004-05. *Commun Dis Intell* 2005;29:341-357.
26. World Health Organization. Zoonoses. Technical report series no. 169. Geneva; 1959.
27. World Health Organization. Report of the WHO/FAO/OIE joint consultation on emerging zoonotic diseases. Geneva; 2004.
28. Taylor LH, Latham SM, Woolhouse ME. Risk factors for human disease emergence. *Philos Trans R Soc Lond B Biol Sci* 2001;356:983-989.
29. Warrilow D. Australian bat lyssavirus: a recently discovered new rhabdovirus. *Curr Top Microbiol Immunol* 2005;292:25-44.
30. Warrilow D, Smith IL, Harrower B, Smith GA. Sequence analysis of an isolate from a fatal human infection of Australian bat lyssavirus. *Virology* 2002;297:109-119.
31. Animal Health Australia. Animal Health in Australia, 2004. Canberra, Australia: AHA, Department of Agriculture, Forestry and Fisheries (NAHIS); 2005.
32. Williams J, Tallis G, Dalton C, Ng S, Beaton S, Catton M, et al. Community outbreak of psittacosis in a rural Australian town. *Lancet* 1998;351:1697-1699.
33. Hughes KL. Chlamydial Infections in Birds and Mammals and their Zoonotic Potential. In: *Zoonoses: Australian Veterinarians in Public Health (Proceedings 194)*: Post Graduate Committee in Veterinary Science University of Sydney; 1992.
34. Li J, O'Brien ED, Guest C. A review of national legionellosis surveillance in Australia, 1991 to 2000. *Commun Dis Intell* 2002;26:462-470.
35. Cohen J. Introduction of the National Meningococcal C Vaccination Program. *Commun Dis Intell* 2003;27.
36. Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 2005. *Commun Dis Intell* 2006;30:211-221.
37. Roche P, Bastian I, Krause V, Antic R, Brown L, Christensen A, et al. Tuberculosis notifications in Australia, 2005. *Commun Dis Intell* 2007;31:71-80.

TUBERCULOSIS NOTIFICATIONS IN AUSTRALIA, 2005

Paul Roche, Ivan Bastian, Vicki Krause, with input from, Ral Antic, Lynne Brown, Amanda Christensen, Christina Drummond, Sandra Gebbie, Mark Hurwitz, Anastasios Konstantinos, Avner Misrachi, as members of the National Tuberculosis Advisory Committee, for the Communicable Diseases Network Australia

Abstract

The National Notifiable Disease Surveillance System received 1,072 tuberculosis (TB) notifications in 2005, of which 1,022 were new cases and 50 were relapses. The incidence of TB in Australia was 5.3 cases per 100,000 population in 2005 and has remained at a stable rate since 1985. The high-incidence groups remain people born overseas and Indigenous Australians at 20.6 and 5.9 cases per 100,000 population, respectively. By contrast, the incidence of TB in the non-Indigenous Australian-born population was 0.8 cases per 100,000 population. Rates in the Australian-born, both Indigenous and non-Indigenous have been declining since 1991, while rates in the overseas-born have been increasing. TB control in Australia relies on pre-migration screening and provision of free and effective treatment. *Commun Dis Intell* 2007;31:71–80.

Keywords: disease surveillance, tuberculosis

Introduction

There were reportedly 9 million cases of tuberculosis (TB) globally in 2005 with more than 50% of cases occurring in Australia's neighbouring countries in South East Asia and the Western Pacific.[1] Global TB control is evolving from the Directly Observed Therapy Short-course (DOTS) strategy to the recently launched Stop TB Plan.[2] The key components of this plan are to pursue high quality DOTS expansion and enhancement; address TB and HIV co-infection, multi-drug resistant TB and other challenges (e.g. TB in prisoners); contribute to health system strengthening; engage all care providers; empower people with TB and communities; and enable and promote research. The Stop TB Plan is particularly relevant to high-burden countries and many elements are applicable in low-incidence countries.

Australia has one of the lowest rates of TB in the world with rates between 5 and 6 cases per 100,000 population for the last 10 years. During the same period, people born outside Australia have made up between 69% and 83% of Australia's annual TB notifications. Australia and other low-incidence countries must confront additional challenges such as: maintaining treatment services (including specially-trained staff, drug supplies and funding) for patients with active TB disease, and providing screening and preventative treatment programs for latent tuberculosis infection (LTBI) among high-risk groups.[3]

A crucial component of effective TB control in a low-incidence country is the collection of accurate comprehensive and timely statistics. These data must be compared against performance indicators to ensure that strategic directions are identified, that outcomes are achieved and that Australia's enviable record of TB control is maintained. This paper presents the TB notification data from the National Notifiable Diseases Surveillance System (NNDSS) in 2005. The data are compared against the National Tuberculosis Performance Indicators set by the National TB Advisory Committee (NTAC) in the *National Strategic Plan for TB Control in Australia Beyond 2000*.[4]

Methods

Data collection

TB is a notifiable disease in Australia. Medical practitioners, public health laboratories and other health professionals are legally required to report cases of TB to the state and territory health authorities. Information on notified cases for 2005 was collated by jurisdictions and sent electronically to the National Notifiable Diseases Surveillance System managed by the Australian Government Department of Health and Ageing. Records were dispatched in a de-identified format to ensure confidentiality. The National Tuberculosis Advisory Committee, as a sub-committee of Communicable Diseases Australia Network, was responsible for determining the dataset collected in 2005 and for its transmission to NNDSS. Key data fields in the enhanced TB dataset that are analysed in this report are listed in Table 1, with a brief description of each variable. While some TB drug susceptibility data on bacteriologically-confirmed cases is collected in NNDSS, the definitive dataset is collected, analysed and reported by the Australian Mycobacterial Reference Laboratory Network in the accompanying report.[5]

Data processing and quality control

Data on all TB notifications reported in 2005 were received by September 2006. Updated information on the outcomes of treatment of patients notified in 2004 was received by December 2006. Data received from the jurisdictions were examined for completeness and accuracy. Any invalid or missing entries were returned to the jurisdictions for review and correction.

Table 1. Description of key data fields in the enhanced tuberculosis dataset of the National Notifiable Diseases Surveillance System used in this report*

Data field	Description
TB outcomes	Options are: • cured (bacteriologically confirmed pulmonary cases only); • completed treatment (80% of standard regimen completed); • interrupted treatment for less than 2 months (but still completed); • died of TB during treatment phase; • died of other cause during treatment phase; • defaulter (failed to complete treatment); • treatment failure (completed treatment but failed to be cured); and • transferred out of Australia during treatment phase.
Indigenous status	Whether notified case is Indigenous (Aboriginal and/or Torres Strait Islander) Australian by descent, community acceptance or self-identification
Selected risk factors	Options are: • household member or close contact with a TB patient; • currently or recently residing in a correctional facility within last 5 years; • currently or recently residing in an aged care facility within last 5 years; • currently or previously employed in an institution within last 5 years; • currently or previously employed in the health industry within last 5 years; • HIV status (positive or negative); and • past residence (3 months or more) in a high risk country (as defined by the Department of Immigration and Citizenship).

* Other data collected on each case included country of birth, length of residence in Australia (for overseas-born cases), and site of tuberculosis disease.

Almost all cases of TB in Australia are reported to the surveillance system. Reasons for the high level of reporting include the presence of effective TB screening programs, a high standard of health care, and specialised and multi-disciplinary TB services in each jurisdiction. The terms 'notification rate' and 'incidence' are therefore used interchangeably in this report.

Case definitions

TB cases were classified as new or relapsed. A new case required a diagnosis accepted by the Director of TB Control (or equivalent) in the relevant jurisdiction, based on laboratory or clinical evidence, and in the absence of any previous treated or untreated TB diagnosis. Laboratory evidence includes either the isolation of *Mycobacterium tuberculosis* complex (*M. tuberculosis, M. bovis or M. africanum)* from a clinical specimen by culture; or nucleic acid amplification testing (NAAT) indicating *M. tuberculosis* complex, except where it is likely to be due to previously treated or inactive disease. The inclusion of NAAT in this definition is to ensure full case ascertainment and does not endorse the use of NAAT for TB diagnosis. Microscopy and culture remain mainstays of TB laboratory diagnosis and provide the capacity for assessing the level of risk for transmission and drug susceptibility testing.

Clinical evidence is a diagnosis made by a clinician experienced in tuberculosis and includes clinical follow-up assessment, with or without supporting radiology.

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

Population estimates for 2005

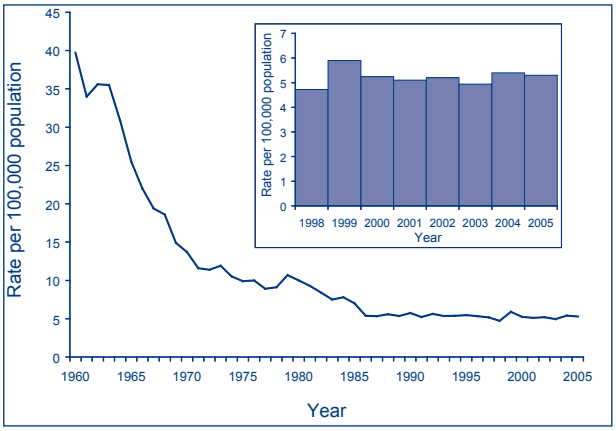
The rates presented in this report were calculated using population data produced by the Australian Bureau of Statistics. The estimated resident population as at 30 June 2005, in each state and territory and in Australia as a whole, was used as the denominator in crude rate calculations. Estimates of the Indigenous Australian population were based on projections from the 2001 census estimate of the Indigenous population in Australia. The 2001 census data were also used to calculate incidence rates of TB in people born overseas.[6]

Results

Tuberculosis notification rates

The total number of cases reported across Australia in 2005 was 1,072 (5.3 cases per 100,000 population). This is similar to that reported in 2004 (1,076 and 5.4 cases per 100,000 population, Figure 1). In 2005, there were 1,022 new cases and 50 relapses. Of the 50 relapsed cases, 15 relapsed after full treatment in Australia, 3 following partial treatment in Australia, 8 following full treatment overseas and 14 following partial treatment overseas. There was no information on the previous treatment given to the remaining 10 relapse cases.

Figure 1. Incidence rates for tuberculosis notifications, Australia, 1960 to 2005

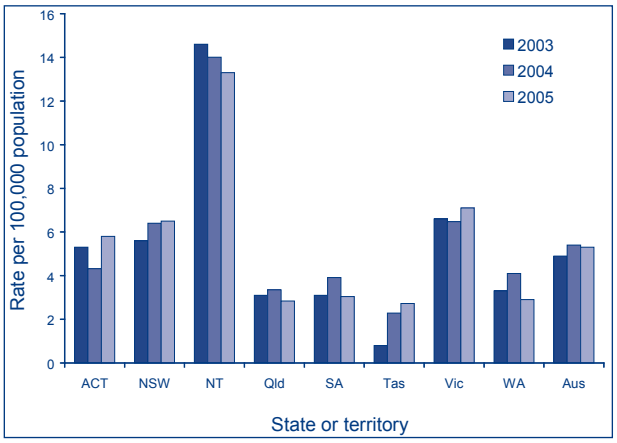


Tuberculosis notifications by state or territory

New South Wales reported the largest number of TB cases (442), however the highest rate was recorded in the Northern Territory (13.3 cases per 100,000 population, Table 2).

Figure 2 presents the TB notifications rates by state or territory for 2003 to 2005. The small increases and decreases over time are often difficult to interpret due to the small number of cases within jurisdictions.

Figure 2. Tuberculosis notification rates, Australia, 2003 to 2005, by state or territory



Tuberculosis in the non-Indigenous Australian-born population

Indigenous status was reported for 148 of 149 (99%) Australian-born patients. The incidence of TB in non-Indigenous Australians for 2005 was 0.8 cases per 100,000 population, which is the lowest rate reported for this population since 1991 (Figure 3 and Table 3).

Tuberculosis in Indigenous Australians

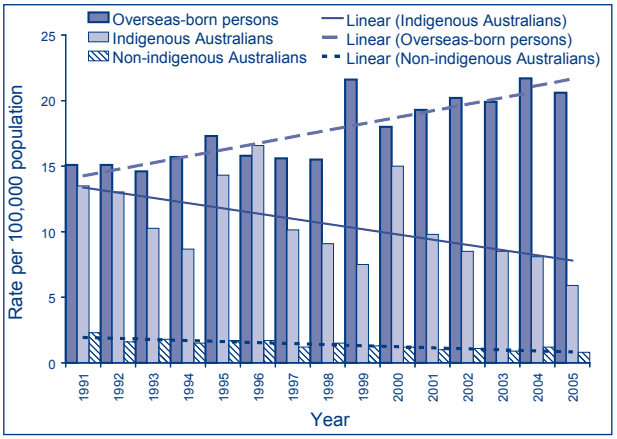
The TB incidence rate in the Indigenous Australian-born population (5.9 cases per 100,000 population) continued the decline in the incidence of TB among Indigenous people from 15 per 100,000 in the year

Table 2. New and relapsed cases and rates per 100,000 population, Australia, 2005, by state or territory

	New cases	New cases rate	Relapse cases	Relapse case rate	Total notifications	Total rate
Australian Capital Territory	19	5.8	0	0.0	19	5.8
New South Wales	420	6.2	22	0.3	442	6.5
Northern Territory	26	12.8	1	0.5	27	13.3
Queensland	104	2.6	6	0.2	110	2.8
South Australia	44	2.9	2	0.1	46	3.0
Tasmania	12	2.5	1	0.2	13	2.7
Victoria	343	6.8	13	0.3	356	7.1
Western Australia	54	2.7	5	0.2	59	2.9
Australia	1,022	5.0	50	0.2	1,072	5.3

2000. The TB incidence in Indigenous Australians in 2005 was 7.4 times the rate in non-Indigenous Australian-born people.

Figure 3. Tuberculosis incidence rates, Australia 1991 to 2005, by indigenous status and country of birth

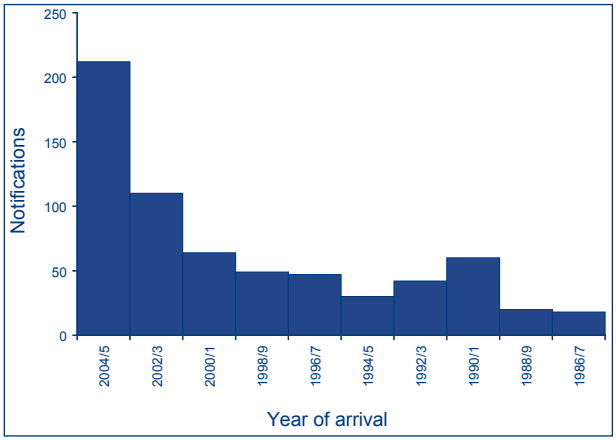


Tuberculosis notifications in the overseas-born population

In 2005, the country of birth was reported for 1,070 of the 1,072 cases. Of these, 923 (86%) cases were overseas-born. The rate of notification, 20.6 cases per 100,000 population, was similar to rates in this population in the previous 2 years (21.7 and 19.1 cases per 100,000 population in 2004 and 2003 respectively, Figure 3). Rates of TB in the overseas-born have shown an increase since 1991. Amongst people born overseas in the Australian population, the largest numbers of TB cases were in those born in India, Vietnam, the Philippines and China as in previous years (Table 4). TB rates were highest among those born in Somalia, Sudan and Ethiopia, although these represent a relatively small number of cases in a small resident population.

Data on the year of arrival was available for 791 of the 923 overseas-born cases in 2005. Two hundred and twelve (26%) of the 2005 cases presented within 2 years of arrival in Australia and 652 (82%) within 20 years of arrival (Figure 4).

Figure 4. Notifications of tuberculosis in the overseas-born population, Australia, 2005, by year of arrival in Australia



Tuberculosis notifications by age and sex

Information on the age of TB cases was available for all cases in 2005 and sex was identified in all but one case (Figure 5). The male to female ratio in TB notifications was 1.5:1 in non-Indigenous Australian-born TB cases, 1.4:1 in Indigenous cases and 0.9:1 in overseas-born cases.

One of the most important measures of TB control is the incidence in children less than 15 years of age because these cases represent recent TB infection. TB was notified in 65 children aged less than 15 years. These were 25 Australian-born non-Indigenous children, 39 children born overseas and one Indigenous child. The overall notification rate for the less than 15 year age group was 1.6 cases per 100,000 popula-

Table 3. Tuberculosis notifications and incidence rates in all Australian-born, Australia, 2005, by state or territory

	Indigenous	Indigenous rate	Non-Indigenous	Non-Indigenous rate	Total Australian-born	Total rate
ACT	0	0.0	3	1.2	3	1.2
NSW	4	3.1	50	1.0	54	1.0
NT	11	18.4	0	0.0	11	6.4
Qld	8	6.1	14	0.4	22	0.7
SA	1	3.8	10	0.8	11	0.9
Tas	1	5.6	5	1.2	6	1.4
Vic	1	3.8	37	1.0	38	1.0
WA	1	1.5	3	0.2	4	0.3
Australia	27	5.9	122	0.8	149	0.9

Table 4. Notification of tuberculosis and estimated rate per 100,000 population for selected countries of birth, Australia, 2005

Country of birth	New	Relapse	Total cases	ERP x COB 2005	Rate per 100,000 population in Australia 2005*	WHO incidence rate per 100,000 2004†
India	137	4	141	138,662	101.7	168
Vietnam	108	9	117	177,728	65.8	176
Philippines	75	4	79	129,401	61.1	293
China‡	58	2	60	191,194	31.4	101
Indonesia	43	1	44	65,914	66.8	245
Sudan	36	2	38	23,787	159.8	411
PNG	26	1	27	26,212	103.0	233
Somalia	24	2	26	5,431	478.7	220
Cambodia	21	0	21	27,490	76.4	510
Bangladesh	19	0	19	12,577	151.1	229
Pakistan	18	1	19	18,083	105.1	181
Hong Kong SAR	18	1	18	76,218	23.6	75
Greece	15	0	15	127,226	11.8	19
Thailand	12	3	15	30,885	48.6	142
Ethiopia	12	1	13	6,925	187.7	353
Other overseas-born	257	11	271	3,771,768	7.1	
Total overseas-born	879	42	923	4,829,501	19.1	
Australia†	141	8	149	20,328,609	0.7	
Total	1,020	50	1,072			

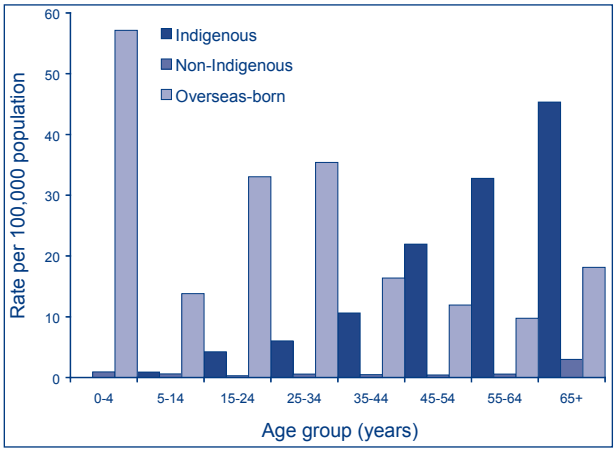
* The denominator for the rate calculation is the estimated resident population (ERP) from the 2001 census.

† Rates from the World Health Organization 2005 Global tuberculosis report.

‡ China excludes Hong Kong SAR and Taiwan.

tion (target of less than 0.1 cases per 100,000 population for all groups). The rate was highest in overseas-born children (18 cases per 100,000 population) and remained low in the non-Indigenous (0.7 cases per 100,000 population) and Indigenous Australian-born children (0.6 cases per 100,000 population, Table 5). Of the 65 children, 41 had bacteriological investigations performed [1/1 Indigenous, 11/25 (44%) non-Indigenous Australian-born and 29/39 (74%) overseas-born children]. Other cases were diagnosed on the basis of positive non-bacteriological laboratory tests (such as histology or NAAT), an abnormal chest X-ray or positive tuberculin skin testing in children with exposure to TB or clinical symptoms. Among the 41 children with bacteriological investigations there were 2 sputum smear positive cases, 8 sputum culture positive cases, 4 cases positive by microscopy of body fluid other than sputum, and 17 cases culture positive in body fluids other than sputum.

Figure 5. Tuberculosis incidence in Australian-born and overseas-born, Australia, 2005, by age and sex



The age group incidence rates for TB in overseas-born, Indigenous Australian-born, and non-Indigenous Australian-born populations are shown in Figure 5 and Table 5. As in previous years, TB incidence in the overseas-born population showed 2 peaks: 1 among infants aged less than 5 years and a second among young adults (15 to 34 years). TB rates among Indigenous and non-Indigenous Australians showed increasing rates throughout adult life with the highest TB rates in those aged 65 years or more.

Table 5. Tuberculosis notifications and estimated incidence rate, Australia, 2005, by age group, indigenous status and country of birth

	Indigenous Australian-born		Non-Indigenous Australian-born		Overseas-born	
Age group	n	Rate	n	Rate	n	Rate
0–4	0	0.0	11	0.9	12	57.1
5–14	1	0.9	14	0.6	27	13.8
Subtotal < 15 years	1	0.6	25	0.7	39	18.0
15–24	4	4.2	7	0.3	148	33.0
25–34	4	6.0	12	0.6	241	35.4
35–44	5	10.6	10	0.5	145	16.4
45–54	6	22.0	8	0.4	106	11.9
55–64	4	32.8	8	0.6	77	9.8
65+	3	45.3	52	3.0	167	18.1

Tuberculosis and selected risk factors

Information on risk factors for TB disease excluding HIV was reported of the 1,072 cases (Table 6). Household contact and residence in a TB high risk endemic country (incidence greater than 12.5 cases per 100,000 population) for more than 3 months were the most common risk factors in all 3 populations. Two non-Indigenous Australian-born and 9 overseas-born TB cases were working or had been employed as a health care worker in the past 5 years.

Tuberculosis and HIV status

Information on HIV status was reported in 393 cases (37%). Nine people were identified with HIV infection at the time of diagnosis with TB; 1 was Indigenous; 2 were non-Indigenous Australians; and 6 were overseas-born. The *National Strategic Plan for TB Control in Australian Beyond 2000*[4] recommends that HIV status of all TB cases be reported. In 2005, the proportion of cases with HIV status reported was similar to that in 2004.

Anatomical site of disease

The anatomical site of TB infection was recorded in 989 cases. Of these, 465 (47%) cases had pulmonary disease only, a further 86 (8.7%) cases had pulmonary disease and disease at an extrapulmonary site. Pulmonary TB was reported in 70% of the Australian-born cases and 52% of the overseas-born cases. Four hundred and thirty-eight (44%) cases had extrapulmonary disease only. The sites of disease in new and relapse cases are shown in Table 7.

Treatment outcomes of 2004 tuberculosis patient cohort

Treatment outcomes for TB cases reported in 2004 were reported by December 2006 for 1,056 of the 1,076 (98%) cases. Treatment success,s including those with bacteriologically confirmed cure and those who completed treatment without bacteriological evidence of cure, were reported for 920 (96.9%) of 949 cases with assessable outcomes (Table 8).

There was no treatment failure recorded. Seventeen (1.8%) cases were reported as defaulting treatment. The proportion of cases cured or who completed treatment was 94% among Indigenous Australians, 95% among non-Indigenous Australian-born, and 97% among overseas-born. In the 2004 patient cohort there were 11 deaths due to TB reported and the case fatality rate was 1.2% of assessable outcomes.

Table 6. Selected risk factors* in tuberculosis notifications, Australia, 2005, by indigenous status

Risk factor	Indigenous	Non-Indigenous	Overseas-born
Household contact	9	23	51
Currently or recently resident in correctional facility	–	–	4
Currently or recently resident in aged care facility	–	2	–
Currently or recently employed in an institution	–	1	3
Currently or previously employed in health industry	–	2	9
Past residence in high risk country	2	12	864

* Excludes HIV status (see below); includes multiple risk factors.

Table 7. New and relapsed tuberculosis cases, Australia, 2005, by site of disease

Site	New	Relapse	Total	Per cent of cases
Total pulmonary disease	528	23	551	55.7
Pulmonary only	446	19	465	47.0
Pulmonary plus other sites	82	4	86	8.7
Extrapulmonary only	423	15	438	44.3
Pleural	50	1	51	5.2
Lymph nodes	151	7	158	16.0
Bone/joint	31	2	33	3.3
Genito/urinary	23		23	2.3
Milliary	10	1	11	1.1
Meningeal	20		20	2.0
Peritoneal	8		8	0.8
Other	54	3	57	5.8

Table 8. Tuberculosis treatment outcomes, Australia, 2004, by population group

Outcomes	Indigenous		Non-Indigenous Australian-born		Overseas-born		Total cases	
Assessable outcomes	**n**	**% assessable**	**n**	**% assessable**	**n**	**% assessable**	**n**	**% assessable**
Treatment success	34	91.9	121	92.4	765	95.5	920	94.9
Cured* (bacteriologically confirmed)	9	24.3	15	11.5	51	6.4	75	7.7
Completed treatment	25	67.6	106	80.9	714	89.1	845	87.2
Interrupted treatment†	0	0.0	0	0.0	1	0.1	1	0.1
Died of TB	0	0.0	3	2.3	8	1.0	11	1.1
Defaulted‡	2	5.4	3	2.3	12	1.5	17	1.8
Failure§	0	0.0	0	0.0	0	0.0	0	0.0
Not followed up, outcome unknown	1	2.7	4	3.1	15	1.9	20	2.1
Total assessable	37	100.0	131	100.0	801	100.0	969	100.0
Non-assessable outcomes	**n**	**% total**	**n**	**% total**	**n**	**% total**	**n**	**% total**
Transferred out of Australia	0	0.0	2	1.3	42	4.7	44	4.1
Died of other causes	2	5.1	19	12.5	38	4.3	59	5.5
Still under treatment	0	0.0	0	0.0	4	0.5	4	0.4
Total	39		152		885		1,076	

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

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

‡ Defaulted means failed to complete treatment.

§ Failed means treatment completed but failed to be cured.

National Performance Indicators

The performance criteria for the National Performance Indicators were set by NTAC in 2002 and reviewed in 2003 (Table 9). In previous TB annual reports, the performance criteria for people born overseas applied to people who have been living in Australia for more than 5 years. In this report, the criteria has been applied to all cases regardless of length of residence.

In 2005, Australia met the key performance indicators of maintaining the incidence of TB in the non-Indigenous Australian-born below 1 case per 100,000 population. Key performance indicators for treatment outcome measures in the 2004 patient cohort were also met.

Discussion

In 2005, rates of TB in Australia continued to remain low with the largest proportion of cases in people born outside Australia. While overall rates in Australia have remained between 5 and 6 cases

Table 9. National tuberculosis performance indicators, performance criteria and the current status of tuberculosis in Australia, 2004 and 2005

National TB performance indicator	Performance criteria	2004	2005
Annual incidence of TB (per 100,000 population)			
Crude incidence			
Indigenous Australians	<1	8.1	5.9
Non-Indigenous Australian-born	<1	1.2	0.8
Overseas-born persons	*	21.7	20.6
Relapse cases initially treated in Australia	<2% of total treated cases	1.0	TBA
Incidence in children <15 years, by risk group			
Indigenous Australian children	<0.1	0	0.6‡
Non-Indigenous Australian-born children	<0.1	0.4	0.7
Overseas-born children	*	11.4	18.0
Collection of HIV status in TB cases (% of cases with data collected)	100% over next 3 years	34	37
Treatment outcome measures (%)			
Cases evaluated for outcomes†	100	98	TBA
Cases that have treatment completed and are cured	>90	96.9	TBA
Cases recorded as treatment failures	<2	0	TBA

* Performance criteria currently under review.

† Evaluation of outcomes of 2004 patient cohort re-assessed in December 2006.

‡ A single case of TB in Indigenous children <15 y in 2005.

per 100,000 population since 1991, rates in the Australian-born show a decline, while rates in the overseas-born have increased.

Although rates vary year by year, the overall rates of tuberculosis in Indigenous Australians are declining (from 13.5 cases per 100,000 population in 1991 to 5.9 cases per 100,000 population in 2005). In 2005, there was only 1 case of TB in an Indigenous child aged less than 15 years and the highest rates were in adults aged 65 years and above – an age distribution similar to that seen in non-Indigenous Australians. Two five-year audits of tuberculosis cases in Far North Queensland in 1993–1997 and 1998–2002 showed significant declines in the number of new cases and relapse cases of TB in Indigenous Australians.[7]

Notification rates of TB in the non-Indigenous Australian-born have also declined in the same period (from 2.3 cases per 100,000 population to 0.8 cases per 100,000 population). Although rates of TB generally increase with age in this group, the number of cases reported in non-Indigenous children aged less than 15 years increased from 15 in 2004 to 25 in 2005. This may reflect cases of TB in the Australian-born children of overseas-born parents: of these 25 children, 15 were reported to have household contact with a TB case.

As Australia's migrant intake changes to include a larger proportion of entrants from TB-endemic countries, the rate of TB in overseas-born Australians has increased (from 15.1 to 20.6 cases per 100,000 population between 1991 and 2005). In 2005, 864 (94%) of the 923 cases reported in overseas-born Australians had resided for more than 3 months in a country with an incidence above 12.5 cases per 100,000 population, defined by the Australian Government Department of Immigration and Citizenship as a high risk country.

Migrants to Australia from TB-endemic countries are required to undergo health checks, including a chest X-ray depending on the TB incidence in their country of residence, their intended length of stay and their occupation. If active untreated TB is found, migrants are asked to undergo a course of treatment. If TB has been successfully treated or if there is evidence of previous but now inactive TB infection, migrants are asked to sign an undertaking whereby they agree to contact health authorities on arrival for follow-up and monitoring.

The effectiveness of Australia's migrant screening is supported by the lower rate of TB among the overseas-born population relative to the United Kingdom (152 cases per 100,000 population) and the United States of America (40.2 cases per 100,000 population).[8] The United Kingdom has recently proposed to screen visa applicants from high risk countries for tuberculosis before entry.[9] The rate of TB diagnosed in migration applicants exceeds the published rates of some countries[10] and rates in resident populations in Australia of people born in

certain countries may also exceed the World Health Organization (WHO) estimates of TB incidence in those countries. For example, in this report (Table 4) the rate of TB in Somali-born Australian residents is more than double that estimated for Somalia by the WHO.

In 2005, 27 cases of TB in people born in Papua New Guinea (PNG) were reported in Australia. This is nearly a 10-fold increase on the number (3) reported in 2001. Many of these cases are reported from Far North Queensland, where the number of patients from PNG increased from 7 (8%) of the 87 cases notified between 1993 and 1997 to 44 (48%) of the 92 cases reported in the following 5 years (1998–2002).[7] In 2005, 6 patients with multi-drug resistant tuberculosis from the PNG/Torres Strait Islands cross-border region, who accessed health services in Queensland, were reported.[5] The increase in TB patients from PNG, including a substantial number with multi-drug resistance, accessing health services in Queensland is a significant emerging problem for local public health, and potentially for national TB control.

Household contact with another TB case was the most significant risk factor in Indigenous and non-Indigenous Australian-born and the second most common risk factor in overseas-born cases, after residence in a high-risk country. Higher incidence rates for tuberculosis may persist within ethnic communities in low TB prevalence countries due to ongoing local transmission within the community and reactivation of latent TB.

Internationally, the greatest risk factor for TB infection is underlying infection with HIV. In 2005, only 37% of TB patients had an HIV test recorded and of these 9 were HIV positive. The proportion of TB patients who have been tested for HIV has not increased. The National Tuberculosis Advisory Committee aims to increase the proportion of Australian TB patients who are tested for HIV by alerting treating physicians to the importance of testing their TB patients for HIV.

The outcomes of the 2004 patient cohort, shown in this report, demonstrate treatment success (defined as bacteriological cure or completion of therapy) in more than 90% of patients. Outcomes were equivalent in the Australian and overseas-born. There were no treatment failures recorded and less than 2% of patients defaulted. There were 20 patients (1.9%) on whom an outcome could not be ascertained. The case fatality rate of 1.2% compared favourably with 5.3% reported in New Zealand (2000 to 2004) and 5.8% reported in Canada in 2001.

Nine overseas-born cases and 2 non-Indigenous Australian-born cases were, or had been employed in the health care industry. Transmission of tuberculosis from health care workers is an emerging issue of concern in the USA and other countries, as the proportion of health care workers born in TB-endemic regions increases.[11] In 2003, a nurse from the Philippines worked in a hospital nursery in New York with undiagnosed pulmonary tuberculosis for 2 months. Fifteen hundred people had contact with the nurse, but only one-third of these could be traced for follow-up. Among those followed-up, at least 4 infants were infected with TB.[12] Latent TB had been detected in the nurse 11 years earlier but not treated. The National TB Advisory Committee is reviewing the Australian data to document the risk of TB in health care settings and is developing strategies to best protect both workers and patients in this setting.

Author details

Paul Roche[1]
Ivan Bastian[2]
Vicki Krause[3]
Ral Antic,[2]
Lynne Brown, Program Manager[4]
Amanda Christensen, Manager[5]
Christina Drummond, Head[6]
Sandra Gebbie, Director[7]
Mark Hurwitz, Director[8]
Anastasios Konstantinos, Director[9]
Avner Misrachi,[10]

1. Surveillance Policy and Systems Section, Australian Government Department of Health and Ageing, Canberra, Australian Capital Territory
2. Infectious Diseases Laboratories, Institute of Medical and Veterinary Science, Adelaide, South Australia
3. Centre for Disease Control, Department of Health and Community Services, Casuarina, Northern Territory
4. TB Control, Department of Human Services, Melbourne, Victoria
5. NSW TB Program, Communicable Diseases Branch, NSW Health Department, Sydney, New South Wales
6. Tuberculosis Services, Perth Chest Clinic, Perth, Western Australia
7. Communicable Disease and Health Risk Policy Section, Australian Government Department of Health and Ageing, Canberra, Australian Capital Territory
8. Thoracic Medicine, The Canberra Hospital, Garran, Australian Capital Territory
9. Queensland TB Control Centre, Queensland Health, Coorparoo, Queensland
10. Communicable Disease Prevention Unit, Department of Health and Human Services, Hobart, Tasmania.

Corresponding author: Dr Paul Roche, Surveillance Policy and Systems Section, Office of Health Protection, Department of Health and Ageing, GPO Box 9848 (MDP14), Canberra ACT 2601. Telephone +61 2 6289 8152. Facsimile +61 2 6289 7100. Email paul.roche@health.gov.au

References

1. World Health Organization. Global tuberculosis control: surveillance, planning, financing. WHO report 2006. Geneva: World Health Organization; 2006. Report No.: WHO/HTM/TB2006.362.
2. Stop TB Partnership, World Health Organization. Global Plan to Stop TB 2006 – 2015. Geneva: World Health Organization; 2006.
3. Broekmans JF, Migliori GB, Rieder HL, Lees J, Ruutu P, Loddenkemper R, et al. European framework for tuberculosis control and elimination in countries with a low incidence. Recommendations of the World Health Organization (WHO), International Union Against Tuberculosis and Lung Disease (IUATLD) and Royal Netherlands Tuberculosis Association (KNCV) Working Group. *Eur Respir J* 2002;19:765-775. [Review].
4. Communicable Diseases Network Australia. *National Strategic Plan for TB Control in Australia Beyond 2000.* Australian Government Department of Health and Ageing, Canberra; 2002. Available from: http://www.cda.gov.au/pubs/other/tb_plan.htm
5. Lumb R, Bastian I, Gilpin C, Jelfs P, Keehner T, Sievers A. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2005. *Commun Dis Intell* 2007;31:80–86.
6. Roche PW, Antic R, Bastian I, Brown L, Christensen A, Hurwitz M, *et al.* Tuberculosis in Australia, annual report, 2004. *Commun Dis Intell* 2006;30:93–101.
7. Simpson G, Clark P, Knight T. Changing patterns of tuberculosis in Far North Queensland. *Med J Aust* 2006;184:252.
8. Department of Immigration and Multicultural Affairs. Annual report 2005–06. Canberra; 2006.
9. Welshman J, Bashford A. Tuberculosis, migration and medical examination: lessons from history. *J Epidemiol Community Health* 2006;60:282–284.
10. Plant AJ, Watkins RE, Motus N, Jones W, O'Rourke T, Streeton J, *et al.* Results of tuberculosis screening in applicants for migration in Vietnam and Cambodia. *Int J Tuberc Lung Dis* 2005;9:157–163.
11. Sterling TR, Haas DW. Transmission of *Mycobacterium tuberculosis* from health care workers. *N Engl J Med* 2006;355:118–121.
12. Centers for Disease Control and Prevention. *Mycobacterium tuberculosis* transmission in a newborn nursery and maternity ward – New York City, 2003. *MMWR Morb Mortal Wkly Rep* 2005;54:1280–1283.

TUBERCULOSIS IN AUSTRALIA: BACTERIOLOGICALLY CONFIRMED CASES AND DRUG RESISTANCE, 2005

A report of the Australian Mycobacterium Reference Laboratory Network

Richard Lumb, Ivan Bastian, Chris Gilpin, Peter Jelfs, Terillee Keehner, Aina Sievers

Abstract

The Australian Mycobacterium Reference Laboratory Network (AMRLN) collects and analyses laboratory data on new cases of disease caused by the *Mycobacterium tuberculosis* complex. In 2005, a total of 810 cases were identified by bacteriology; an annual reporting rate of 4.0 cases per 100,000 population. Isolates were identified as *M. tuberculosis* (n=806), *Mycobacterium africanum* (n=2) and *Mycobacterium bovis* (n=2). Fifteen children aged under 10 years had bacteriologically-confirmed tuberculosis. Results of *in vitro* drug susceptibility testing were available for all 810 isolates for isoniazid (H), rifampicin (R), ethambutol (E), and pyrazinamide (Z). A total of 74 (9.1%) isolates of *M. tuberculosis* were resistant to at least one of these anti-tuberculosis agents. Resistance to at least H and R (defined as multi-drug resistance, MDR) was detected in 12 (1.5%) isolates; nine were from the respiratory tract (sputum n=8, bronchoscopy n=1). Of the 74 *M. tuberculosis* isolates resistant to at least one of the standard drugs, 67 (90.5%) were from new cases, 6 from previously treated cases, and no information was available on the remaining case. Eight were Australian-born, 65 were overseas-born, and the country of birth of one was unknown. Of the 65 overseas-born persons with drug resistant disease, 41 (63.1%) were from 4 countries; Vietnam (n=16), Papua New Guinea (n=10), the Philippines (n=9), and India (n=6). A retrospective review of AMRLN data on isolates collected between 2000 and 2005 found that none of 70 MDR-TB isolates met the new definition for extensively drug resistant TB (XDR-TB, i.e. MDR-TB with additional resistance to quinolones and second-line injectable agents). *Commun Dis Intell* 2007;31:80–86.

Keywords: *Mycobacterium tuberculosis, Mycobacterium bovis,* laboratory diagnosis, tuberculosis, drug resistance, nucleic acid amplification test

Introduction

Australia continues to record one of the lowest notification rates (5–6 cases per 100,000 population) of tuberculosis (TB) in the world.[1] New Zealand experienced a long-term decline in TB notification rates until the mid-1980s when annual rates stabilised at around 10 cases per 100,000 population.[2] In contrast, TB has a large impact on the health of our regional neighbours in the World Health Organization (WHO) Regions of South East Asia (SEAR) and the Western Pacific (WPR).[3,4] The SEAR, comprising 11 countries with a combined population of over 1.5 billion, has one-third of all annual cases worldwide. In 2005, there were an estimated 5.7 million prevalent cases of TB in the SEAR (351 cases per 100,000 population), of which almost three million were new cases (incidence rate of 190 cases per 100,000 population). Five countries, namely Bangladesh; India; Indonesia; Myanmar; and Thailand, accounted for almost 95% of all new cases.[3] The WPR includes 37 countries with a combined population of over 1.7 billion. In 2004, there were an estimated 3.8 million prevalent cases of TB (216 cases per 100,000 population), of which almost 2 million were new cases (111 cases per 100,000 population). Three countries (China, the Philippines and Vietnam) accounted for approximately 90% of all estimated cases in the region.[4]

There are two sources of TB-related data for Australia. Since 1991, the National Notifiable Diseases Surveillance System (NNDSS) has provided statistics on TB notifications reported to public health authorities in Australia's states and territories. The Australian Tuberculosis Reporting Scheme has been conducted by the Australian Mycobacterium Reference Laboratory Network (AMRLN) since 1986. Statistics compiled by the AMRLN relate to cases of bacteriologically-confirmed tuberculosis whereas NNDSS data also includes cases that are identified on the basis of clinical and epidemiological information, or on non-bacteriological laboratory investigations. This report describes the bacteriologically-confirmed TB diagnoses for the year 2005.

Methods

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

For each new bacteriologically-confirmed case, the following information was collected where available:

- demography: patient identifier, age, sex, HIV status and state of residence;
- specimen: type, site of collection, date of collection and microscopy result;
- isolate: *Mycobacterium* species and results of drug susceptibility testing;
- nucleic acid amplification testing results; and
- for drug resistant isolates: patient country of origin, and history of previous TB treatment to determine whether resistance was initial or acquired.

Data from contributing laboratories were submitted in standard format to the AMRLN coordinator for collation and analysis. Duplicate entries (indicated by identical patient identifier and date of birth) were deleted prior to analysis. Rates were calculated using mid-year estimates of the population for 2005 supplied by the Australian Bureau of Statistics.[6]

For each case, the nature of the first clinical specimen that yielded an isolate of MTBC was used to record the nominal site of disease. Culture-positive specimens collected at bronchoscopy or by gastric lavage were counted as pulmonary disease. Patients with isolates recovered from multiple sites were counted as pulmonary disease (the most important category for public health purposes) if a sputum, bronchoscopy, or lung biopsy specimen was culture-positive.

Drug resistance among new cases (proxy for primary resistance) was defined as the presence of resistant isolates of *M. tuberculosis* in patients who, in response to direct questioning, denied having received any prior anti-TB treatment (for more than one month) and, in countries where adequate documentation is available, for whom there is no evidence of such a history. Drug resistance among previously treated cases (proxy for acquired resistance) is defined as the presence of resistant isolates of *M. tuberculosis* in cases who, in response to direct questioning, admit having been treated for one month or more or, in countries where adequate documentation is available, for whom there is evidence of such a history.[7]

The participating laboratories were also asked to review their laboratory records for 2000–2005 to identify MDR-TB isolates that fulfilled the current definition for extensively drug resistant TB (XDR-TB, i.e. MDR-TB with additional resistance to a quinolone and to at least one of the second-line injectable agents: kanamycin, amikacin, capreomycin).

Results

There were 810 bacteriologically-confirmed cases of tuberculosis in 2005, representing an annual rate of 4.0 cases per 100,000 population. State-specific reporting rates varied from 2.1 (Tasmania and Western Australia) to 11.9 (Northern Territory) cases per 100,000 population (Table 1).

Causative organism

Almost all isolates were identified as *M. tuberculosis* (n=806), the remaining isolates being *Mycobacterium africanum* (n=2) and *Mycobacterium bovis* (n=2).

Distribution by gender, age and site of disease

Complete information for gender and age was available for 806 (99.5%) patients. Of the 810 MTBC isolates, 379 (47.0%) were from females, 427 (53.0%) were from males, and gender was unknown for 4 cases. Fifteen children aged under 10 years (male n=8, female n=7) had bacteriologically-confirmed tuberculosis (gastric aspirate n=8, lymph node n=3, pleural n=2, sputum n=1, nasopharyngeal aspirate n=1).

The site of disease was dependent upon age and gender. The overall male:female ratio was 1.1:1. For respiratory isolates, the male:female percentage was 1.5:1. For TB lymphadenitis, the female:male percentage was 2.1:1. For males, there were two distinct peak age groups in bacteriologically-confirmed rates: a rise to 8.0 cases of TB per 100,000 population at 20–24 years and a second peak in elderly males aged more than 75 years (>9.9 cases of TB per 100,000 population). The age distribution of female cases was similar with 7.5 and 11.3 bacteriologically-confirmed TB cases per 100,000 population at the 25–29 and >84 year age groups, respectively. The median age group for patients with bacteriologically-confirmed disease was 30–34 years for males and 35–39 years for females.

The predominant culture-positive specimen type was sputum (n=354, 43.7%); a further 103 (12.7%) were obtained from bronchoscopy, and 5 were from lung biopsies (Table 2). Forty-seven pleural specimens (29 fluid, 18 biopsy/tissue) were culture-positive. Of these 47 pleural specimens, only a single biopsy was smear-positive. The most commonly encountered extrapulmonary culture-positive specimen was lymph tissue (n=173, 21.4%) followed by pleural (n=47, 5.8%), peritoneal (n=24, 3.0%), bone/joint (n=24, 3.0%), and genitourinary tract (n=19, 2.3%).

Association with HIV

The AMRLN database recorded the HIV status of only 42 (5.2%) patients. No patient was identified as HIV-seropositive.

Microscopy

Results of microscopy were available for 790 of 810 (97.5%) of specimens; microscopy was not performed on 19 specimens and no result was provided for the remaining one specimen. Smears were positive in 189 of 354 (54.3%) sputum and 35 of 103 (34.3%) bronchoscopy specimens respectively (Table 2). Of 47 pleural specimens (18 biopsy and 29 fluids) that

Table 1. Bacteriologically-confirmed cases of tuberculosis in Australia, 1995 and 2003 to 2005, cases and rate per 100,000 population, by state or territory

State or territory	2005		2004*		2003*		1995*	
	n	Rate	n	Rate	n	Rate	n	Rate
New South Wales†	346	4.9	308	4.4	325	4.6	305	4.8
Northern Territory	24	11.9	21	10.5	20	10.1	37	21.3
Queensland	91	2.3	88	2.3	91	2.4	86	2.6
South Australia	36	2.3	43	2.8	36	2.4	33	2.2
Tasmania	10	2.1	8	1.7	4	0.8	2	0
Victoria	261	5.2	262	5.3	254	5.2	186	4.1
Western Australia	42	2.1	57	2.9	54	2.8	56	3.2
Total	810	4.0	787	3.9	784	3.9	705	3.9

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

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

were culture-positive for *M. tuberculosis*, only one biopsy was smear-positive. Lymph node specimens were smear-positive in only 29 of 173 (17.3%) cases.

Table 2. Site of specimens smear– and culture-positive for *Mycobacterium tuberculosis* complex, Australia, 2005

	n	Smear positive (%)*
Sputum	354	189 (54.3)
Bronchoscopy	103	35 (34.3)
Lymph node	173	29 (17.3)
Pleural	47	1 (2.2)
Genitourinary	19	ND†
Bone/joint	24	ND†
Peritoneal	24	ND†
Skin	9	ND†
Cerebrospinal fluid	4	ND†

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

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

Drug susceptibility testing

Results of *in vitro* drug susceptibility testing were available for all 810 isolates for isoniazid (H), rifampicin (R), ethambutol (E), and pyrazinamide (Z). A total of 74 (9.1%) isolates of *M. tuberculosis* were resistant to at least one of these anti-tuberculosis agents. Results of testing for streptomycin (S) were available for 200 of 810 (24.7%) isolates with 35 demonstrating resistance to at least S; 9 had mono-resistance, 14 were resistant to S and H, 10 MDR-TB strains were also S-resistant, and there was a single S/R resistance, and a single S/H/Z resistance. Resistance to at least H and R (defined as MDR) was detected in 12 (1.5%) isolates. All of the MDR isolates were *M. tuberculosis* (Table 3). Of the 12 MDR-TB isolates, 10 were from the respiratory tract (sputum n=8, bronchoscopy n=1, nasopharyngeal aspirate n=1) and 1 each from a knee synovium biopsy and a lymph node. Four of the MDR-TB-positive sputum specimens were smear-positive, as were samples from lymph node, nasopharyngeal neck abscess and bronchoscopy specimens.

Six patients with MDR-TB were from the Papua New Guinea (PNG) – Torres Strait Islands (TSI) cross-border region who access health services in outer TSI and are eligible to receive treatment in Australia.[8] MDR-TB was also isolated from patients born in Vietnam (n=2) and Australia (n=2), with a single case each from India and the Sudan. Of the two Australian-born MDR-TB cases, one had travelled extensively in South East Asia. There was no additional information on the second case.

Mono-resistance to isoniazid (H) was detected in 42 isolates. One isolate was resistant to rifampicin (R) alone and another isolate was resistant to pyrazinamide (Z) alone. No ethambutol mono-resistance was observed. Seventy-one isolates demonstrated resistance to H at a concentration of 0.1 mg/L. Of these, 49 (69.0%) demonstrated resistance to H at the higher level of 0.4 mg/L. Among MDR-TB strains, 10/12 (83.3%) demonstrated H resistance at the higher concentration (0.4 mg/L). Twenty-seven of 74 (36.5%) specimens culture-positive for drug resistant strains, including 21 of 51 (41.2%) sputum or bronchoscopy specimens, were smear-positive for acid-fast bacilli The two *M. bovis* isolates, which are inherently resistant to pyrazinamide, were not included in the above results.

AMRLN isolate susceptibility results between 2000 and 2005 were reviewed for isolates that might meet the definition of 'extensive drug resistance (XDR-TB). None of the 70 MDR-TB strains met the case definition for XDR-TB. Several MDR-TB isolates were also resistant to fluoroquinolones, including a patient from South Africa, but none of these isolates was also resistant to the second-line injectable agents.

Table 3. Drug resistance patterns in multi-drug resistant strains, Australia, 1995 to 2005

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

* The streptomycin result was not considered for this table.

† H = isoniazid, R = rifampicin, E = ethambutol, Z = pyrazinamide

New or previously treated cases, and country of birth

Of the 74 *M. tuberculosis* isolates resistant to at least one of the standard drugs (H,R,E,Z) 67 (90.5%) were from new cases, 6 were from previously treated cases, and treatment information was not available for the remaining case. The country of birth was known for 73 (98.6%) cases; 8 were born in Australia and 65 were overseas-born. Of the 65 overseas-born cases with drug resistant disease, 41 (63.1%) were from four countries: Vietnam (n=16), Papua New Guinea (n=10), the Philippines (n=9), and India (n=6). The remaining 24 came from 14 other countries.

Discussion

In 2005, there were 810 cases of bacteriologically-confirmed tuberculosis representing 4.0 cases per 100,000 population, a similar rate to that found in 2004 and consistent with the results dating back to 1986.[9–21] *Mycobacterium tuberculosis* was the predominant species reported with only two isolates each of *M. bovis* and *M. africanum* identified in 2005.

The level of drug resistance in *M. tuberculosis* isolates remains at a relatively constant level; excluding resistance to streptomycin, only 9.1% (74 of 810) of strains had resistance to one or more anti-tuberculosis drugs. Most cases with drug-resistant strains occurred in the overseas-born as observed in previous years.[9–18,22,23] The rates of resistance in these cases who most likely acquired their infections outside Australia, reflect the prevalence of drug resistant TB in their countries of birth. These findings reflect the performance of the TB program from their country of origin rather than the clinical management of these patients in Australia. Therefore, national drug resistance data has limited usefulness as a measure of performance of Australia's TB control program.

Resistance to isoniazid and rifampicin, defined as MDR-TB, remained at a constant low level in 2005 (Table 3). Australia's MDR-TB rate (1.5%) is lower than recent published estimates of MDR-TB globally (2.7%), and in the SEAR (2.2%) and WPR (4.2%) regions.[24]

The number of TB patients born in Papua New Guinea and diagnosed in Queensland, has increased in recent years. In the period 1993–1997, patients from PNG represented only 7 (8.0%) of 87 notified TB cases from Far North Queensland, but in the period 1998–2002, 44 (47.8%) of 92 notified TB cases were from PNG, including three MDR-TB cases.[8] In 2005, the Queensland Mycobacterium Reference Laboratory identified 6 patients with MDR-TB from the PNG–TSI cross-border region who accessed health services in outer TSI (Anastasios Konstantinos, Director, TB Services, Queensland, personal communication). This influx of TB patients from PNG-TSI represents a significant burden for the Queensland health services.

MDR-TB is recognised as a threat to global TB control. Management of MDR-TB cases is intensive, expensive, prolonged, and associated with a greater likelihood of treatment failure.[25] Unfortunately, a small percentage of MDR-TB strains have additional resistance to second-line drugs (SLD). Such strains are termed extensively drug-resistant. Until recently, XDR-TB was defined as a strain which was resistant to isoniazid and rifampicin, and at least three of the six main classes of SLD's (aminoglycosides, polypeptides, fluoroquinolones, thiomides, cycloserine, and para-amino salicylic acid).[26] A survey of a supranational network of TB laboratories determined that 17,690 TB isolates evaluated between 2000–2004 were tested for first–line drugs and at least three of the six SLD classes.[27] Overall, 20% were MDR-TB and 2% were XDR-TB. In addition, previously identified MDR-TB 'hot-spots' (Armenia, Azerbaijan, Czech Republic, Latvia, Republic of Georgia, Russia, South Korea) had higher levels of XDR-TB. South Korea provided data for 11,939 isolates of which 1,298 (11%) were MDR-TB, and of these, 200 (15%) were XDR-TB. The remaining supranational laboratories provided data from 5,751 isolates, of which 2,222 (38.6%) were MDR-TB, but of these, only 147 (6.6%) were XDR-TB. Although the data is likely biased as supranational laboratories are more likely to receive isolates from retreatment cases, treatment failures or other complex cases, it does provide clear evidence that XDR-TB has a global distribution. Areas identified as 'hot spots' for MDR-TB have higher levels of XDR-TB than non-'hot spot' areas.[28] Furthermore, it is likely that XDR-TB is associated with an even worse prognosis than for MDR-TB. XDR-TB gained international notoriety following an outbreak in an HIV hospital/outpatient setting in Kwazulu-Natal, South Africa. Of the 53 patients identified with XDR-TB, 52 had died within an average of 25 days of diagnosis.[27]

The WHO Global Task Force on XDR-TB met in October 2006 and developed a revised laboratory case definition: 'XDR-TB is TB showing resistance to at least isoniazid and rifampicin; which is the definition of MDR-TB, in addition to any fluoroquinolone, and to at least one of the following 3 injectable drugs used in anti-TB treatment: capreomycin, kanamycin, and amikacin'.[22] There are three rationales for the revised definition: (i) protocols for drug susceptibility testing of fluoroquinolones and injectable anti-TB agents are established and there is good inter-laboratory agreement; there is less agreement for the other SLD's, and none whatsoever for cycloserine, (ii) the fluoroquinolones and

injectable agents are the most potent SLD's, and form the cornerstones of most MDR-TB treatment regimens, and (iii) are often the only SLD's available in developing countries.[22]

No XDR-TB strains were identified in Australia between 2000–2005. The widespread use and the documented rapid development of fluoroquinolone resistance[29] has prompted the AMRLN to institute routine fluoroquinolone susceptibility testing of all isolates from the beginning of 2006.

Acknowledgements

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

- Institute of Medical and Veterinary Science, Adelaide, South Australia.
- Queensland Health Pathology Services, Herston Hospitals Complex, Herston, Queensland.
- Victorian Infectious Diseases Reference Laboratory, North Melbourne, Victoria.
- PathWest Laboratory Medicine WA – QEIIMC, Hospital Avenue, Nedlands, Western Australia
- Institute of Clinical Pathology and Medical Research, Westmead Hospital, Westmead, New South Wales.

Additional information and support from Ms Amanda Christensen, Dr Ral Antic, Dr Vicki Krause, Dr Graham Tallis, Dr Anastasios Konstantinos, and Dr Christine Drummond is gratefully acknowledged.

Author details

Richard Lumb[1,2]
Ivan Bastian[1,2]
Chris Gilpin[2]
Peter Jelfs[2]
Terillee Keehner[2]
Aina Sievers[2]

1. Infectious Diseases Laboratories, Institute of Medical and Veterinary Science, Adelaide, South Australia
2. Australian Mycobacterium Reference Laboratory Network

Corresponding author: Mr Richard Lumb, Chief Medical Scientist, Mycobacterium Reference Laboratory, Infectious Diseases Laboratories, Institute of Medical and Veterinary Science, PO Box 14, Rundle Mall, Adelaide, South Australia 5000. Telephone: +61 8 8222 3579. Facsimile: +61 8 8222 3543.
Email: richard.lumb@imvs.sa.gov.au

References

1. Li J, Roche P, Spencer J, Bastian I, Christensen A, Hurwitz M, et al. Tuberculosis notifications in Australia, 2003. *Commun Dis Intell* 2004;28:464–473.
2. Das D, Baker M, Calder L. Tuberculosis epidemiology in New Zealand: 1995–2004. *N Z Med J* 2006;119: U2249 Available from: http://www.nzma.org.nz/journal/119-1243/2249/
3. World Health Organization. Tuberculosis control in the South East Asian Region. 2005. (SEA-TB-282).
4. World Health Organization. Tuberculosis control in the Western Pacific Region. 2006. Available from: http://stoptb.wpro.who.int
5. Communicable Diseases Network Australia. *National Strategic Plan for TB Control in Australia Beyond 2000.* Commonwealth Department of Health and Ageing, Canberra, July 2002.
6. Australian Bureau of Statistics. Australian Demographic Statistics, June Quarter 2005.
7. World Health Organization. Anti-tuberculosis drug resistance in the world. Third global report. Available from: WHO/HTM/TB/2004.343.
8. Simpson G, Clark P, Knight T. Changing patterns of tuberculosis in Far North Queensland. *Med J Aust* 2006;184:252.
9. Dawson S, Anargyros P, Blacklock Z, Chew W, Dagnia H, Gow B, et al. Tuberculosis in Australia: an analysis of cases identified in reference laboratories in 1986–88. *Pathology* 1991;23:130–134.
10. Dawson D, Cheah DF, Chew F, Haverkort F, Lumb R, Sievers AS. Tuberculosis in Australia, 1989–1992. Bacteriologically-confirmed cases and drug resistance. *Med J Aust* 1995;162:287–290.
11. Curran M, Dawson D, Cheah D. Laboratory surveillance of *Mycobacterium tuberculosis* isolates in Australia, 1992. *Commun Dis Intell* 1994;18:337–339.
12. Curran M, Dawson D. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 1993. *Commun Dis Intell* 1995;19:343–345.
13. Dawson D. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 1994 and 1995. *Commun Dis Intell* 1997;21:245–249.
14. Dawson D. Tuberculosis in Australia; bacteriologically confirmed cases and drug resistance, 1996. *Commun Dis Intell* 1998;22:183–188.
15. Dawson D. Tuberculosis in Australia; bacteriologically confirmed cases and drug resistance, 1997. *Commun Dis Intell* 1999;23:349–353.
16. Dawson D. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 1998 and 1999. *Commun Dis Intell* 2001;25:261–265.
17. Lumb R, Bastian I, Dawson D, Gilpin C, Haverkort F, James G, Sievers A. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2000. *Commun Dis Intell* 2002;26:226–233.
18. Lumb R, Bastian I, Dawson D, Gilpin C, Haverkort F, James G, Sievers A. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2001. *Commun Dis Intell* 2003;27:173–180.
19. Lumb R, Bastian I, Chew W, Gilpin C, Haverkort F, Sievers A. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2002. *Commun Dis Intell* 2003;27:458–464.

20. Lumb R, Bastian I, Crighton T, Gilpin C, Haverkort F, Sievers A. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2003. *Commun Dis Intell* 2004;28:474–480.

21. Lumb R, Bastian I, Crighton T, Gilpin C, Haverkort F, Sievers A. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2004. *Commun Dis Intell* 2005;30:102–108.

22. World Health Organization. Extensively drug-resistant tuberculosis (XDR-TB): recommendations for prevention and control. *Wkly Epidemiol Rec* 2006;45:430–432. Available from: http://www.who.int/wer

23. National Tuberculosis Advisory Committee. Guidelines for Australian Mycobacteriology Laboratories. *Commun Dis Intell* 2006;30:116–128.

24. Zignol M, Hosseini MS, Wright A, Weezenbeek CL, Nunn P, Watt CJ, et al. Global incidence of multidrug-resistant tuberculosis. *J Infect Dis* 2006;194:479–485.

25. World Health Organization/International Union Against Tuberculosis and Lung Disease Global Project on Anti-Tuberculosis Drug Resistance Surveillance. *Anti-tuberculosis Drug Resistance in the World:* report number 3. Geneva, Switzerland: World Health Organization; 2004.

26. World Health Organization. Guidelines for the programmatic management of drug resistant tuberculosis. Geneva, Switzerland: World Health Organization; 2006. (WHO/HTM/TB/2006.361).

27. World Health Organization. Report from the Expert Consultation on Drug-Resistant Tuberculosis. Johannesburg, South Africa, 7–8 September 2006. Available from: http//: www.who.int/tb/kg1/en/index.html

28. Centers for Disease Control and Prevention. Emergence of *Mycobacterium tuberculosis* with extensive resistance to second-line drugs – worldwide, 2000–2004. *MMWR Morb Mortal Wkly Rep* 2006;55:301–305.

29. Ginsberg AS, Woolwine SC, Hooper, Benjamin WH Jr, Bishai WR, Dorman SE, et al. The rapid development of fluoroquinolone resistance in M*ycobacterium tuberculosis. N Engl J Med* 2003;349:1977–1978.

Invasive pneumococcal disease in Australia, 2005

Paul Roche, Vicki Krause, Heather Cook, with input from Mark Bartlett, David Coleman, Craig Davis, James Fielding, Carolien Giele, Robin Gilmour, Ros Holland, Riemke Kampen, as members of the Enhanced Invasive Pneumococcal Disease Surveillance Working Group and with laboratory data supplied by Mitchell Brown, Lyn Gilbert, Geoff Hogg, Denise Murphy, for the Pneumococcal Working party of the Communicable Diseases Network Australia.

Abstract

Enhanced surveillance for invasive pneumococcal disease (IPD) was carried out in all Australian states and territories in 2005 with comparative data available since 2001. There were 1,680 cases of IPD notified to the National Notifiable Diseases Surveillance System in Australia in 2005; a notification rate of 8.3 cases per 100,000 population. The rates varied between states and territories and by geographical region with the highest rates in the Northern Territory, the jurisdiction with the largest proportion of Indigenous people. Invasive pneumococcal disease was reported most frequently in those aged 85 years or over (41 cases per 100,000 population) and in 1-year-old children (36.5 cases per 100,000 population). Enhanced data provided additional information on 1,015 (60%) of all notified cases. The overall rate of IPD in Indigenous Australians was 8.6 times the rate in non-Indigenous Australians. There were 126 deaths attributed to IPD resulting in an overall case fatality rate of 7.5%. While the rate of IPD in the Indigenous under 2-year-old population decreased from 219 cases per 100,000 population since targeted introduction of the 7-valent conjugate pneumococcal vaccine (7vPCV) in 2001, the rate in 2005 (94 cases per 100,000 population) was significantly greater than in non-Indigenous children (20.4 cases per 100,000 population). Rates of disease in all children aged less than 2 years, caused by serotypes in the 7vPCV decreased by 75% between 2004 and 2005 as a result of the introduction of a universal childhood 7vPCV immunisation program. Significant decreases in IPD caused by 7vPCV serotypes also occurred in the 2–14 years and 65 years or over age groups. There is no evidence of replacement disease with non-vaccine serotypes. Serotypes were identified in 90% of all notified cases, with 61% of disease caused by serotypes in the 7vPCV and 88% caused by serotypes in the 23-valent polysaccharide pneumococcal vaccine (23vPPV). Reduced penicillin susceptibility remains low and reduced susceptibility to 3rd generation cephalosporins is rare. *Commun Dis Intell* 2007;31:86–100.

Keywords: disease surveillance, pneumococcal disease, *Streptococcus pneumoniae*

Introduction

While the 23-valent polysaccharide pneumococcal vaccine (23vPPV) was available for older children and adults for many years, it was the anticipation of a conjugate vaccine for infants and young children that prompted national notification and enhanced surveillance of invasive pneumococcal disease (IPD) in Australia. In 2000, the most comprehensive reporting of the nationwide burden of IPD to date was assembled[1] and the call for IPD to be nationally notified was realised in 2001. Since then, enhanced data has been collected and analysed from all states and territories and published annually. Australia is fortunate to be able to provide country-wide surveillance data for IPD that includes the vaccination status for all cases aged less than 5 years and the majority of adult cases. Serotype information has been available in past reports for up to 86% of all isolates notified.[2] The 2004 report, following 3½ years after the introduction of the 7-valent conjugate pneumococcal vaccine (7vPCV) for at-risk (including all Indigenous) children, showed the rate of IPD in Indigenous children was reduced to that of non-Indigenous children (91.5 and 93.6 cases per 100,000 population, respectively).[2] In January 2005, universal 7vPCV for infants and children was introduced. Ninety-one per cent of Australian children were reported as having received 3 doses of 7vPCV vaccine by 12 months of age between January and September 2005 (Australian Childhood Immunisation Register). The impact of this vaccine is reported in this report, as well as on-going surveillance of IPD in all ages. In addition, while some jurisdictions had funded the 23vPPV for older adults, 2005 was the first year it was federally funded for all those aged 65 years or older (Table 1).

Methods and materials

Case definition

A case of IPD was defined as the isolation from or the detection by nucleic acid test (NAT) in blood, cerebrospinal fluid (CSF) or other sterile site of *Streptococcus pneumoniae.*

Data collection

Invasive pneumococcal disease has been a notifiable disease in some Australian states and territories for several years. In 2001, IPD was made notifiable in all states and territories and data are forwarded to the

Table 1. Recommendations and funding initiatives for pneumococcal vaccination in Australia

Vaccine	23-valent polysaccharide vaccine	7-valent conjugate vaccine
Pneumococcal serotypes	1, 2, 3, 4, 5, 6B, 7F, 8, 9N, 9V, 10A, 11A, 12F, 14, 15B, 17F, 18C, 19A, 19F, 20, 22F, 23F, 33F	4, 6B, 9V, 14, 18C, 19F, 23F
Target populations	All individuals aged 65 years or over to receive a single dose of vaccine with a booster 5 years later* Aboriginal and Torres Strait Islander people aged 50 years or over to receive a single dose of vaccine with a booster 5 years later† Aboriginal and Torres Strait Islander people aged between 15 and 49 years at high risk to receive a single dose of vaccine and appropriate booster(s)‡ Children who have underlying chronic illnesses predisposing to IPD (including asplenia and immunocompromised)§,‖ Immunocompetent individuals with chronic illness including chronic cardiac, renal or pulmonary disease, diabetes and alcohol-related problems Individuals with cerebrospinal fluid leaks‖ Tobacco smokers¶ As a booster dose at 18 to 24 months of age following a primary course of 7vPCV in Aboriginal and Torres Strait Islander children in regions of high incidence.** As a booster dose at 4 to 5 years of age following a primary course of 7vPCV in children at risk because of predisposing medical conditions**	Children at 2, 4 and 6 months†† Children born between 1 January 2003 and 31 December 2004‡‡ Additional booster dose for children in specific high-risk groups**

* Funded in Victoria from 1998, Funded nationally from 2005.

† Targeted funded programs in north Western Australia, Far North Queensland and the Northern Territory from 1995. Funded nationally from 1999.

‡ Funded nationally from 1999. Funded for all persons aged15 years or over in the Northern Territory from 1999.

§ Targeted funded programs for high risk persons aged over 2 years in north Western Australia and the Northern Territory from 1986.

‖ Recommended nationally for persons aged over 2 years (pre-July 2001) and aged over 5 years from July 2001.

¶ Recommended nationally from 2003.

** Funded nationally from July 2001.

†† Funded nationally for Indigenous children from July 2001 and all children from 2005.

‡‡ Funded nationally as a catch-up program in 2005.

National Notifiable Diseases Surveillance System (NNDSS). Since this required changes to state and territory public health legislation, the data in 2001 were incomplete in some states and territories, but were complete for all jurisdictions from 2002.

NNDSS data in 2005 comprised core data, which is a set of data collected on all cases of all notifiable diseases as well as 'enhanced' data specific to IPD.

Clinical presentation

Clinical presentations were coded as pneumonia, meningitis, bacteraemia, other, or unknown. Pneumonia was defined as blood culture or NAT positive for *S. pneumoniae* with clinical and/or radiological signs of pneumonia. Meningitis was defined as the detection of *S. pneumoniae* in the CSF and/or blood with supportive clinical findings. Bacteraemia was defined as the detection of *S. pneumoniae* in blood with no localising signs. 'Other' presentations included detection of *S. pneumoniae* in pleural, peritoneal or joint fluid. More than one clinical presentation could be recorded for each case.

Risk factors

The national surveillance working party defined risk factor categories for IPD. They include prematurity, (<37 weeks gestation), congenital or chromosomal abnormality, anatomical or functional asplenia, being immunocompromised, chronic illness, childcare attendee, previous episode of IPD, other (e.g. a smoker), and unknown. Other risk factors defined by jurisdictions were also collected. More than one risk factor could be recorded for each case.

Antibiotic resistance

Antibiotic susceptibility results are reported from the patient's treating institutions and classified as sensitive, intermediate resistance, or resistant. In some cases the results are from referral laboratories. Reduced susceptibility includes intermediate and fully resistant results.

Vaccination

The definitions of vaccination status, vaccination confirmation and vaccine failure used in this report are described in Table 2.

Populations under surveillance

There were different populations under enhanced surveillance in jurisdictions in 2005 (Table 3).

Data were analysed by date of diagnosis which was the earliest of the dates recorded in NNDSS (date of onset, specimen date, notification date or notification received date).

Data analysis

The notification rates presented in this report were calculated using population data from the Australian Bureau of Statistics (ABS). The Estimated Resident Population (ABS 3201.0) in each State and Territory and in Australia as a whole, as at 30 June 2005, was used as the denominator in rate calculations. Estimates of the Indigenous Australian population were based on projections from the 2001 census. The ABS calculated projections based on assumptions about future births, deaths and migrations in the Indigenous population and a 'low' and 'high' estimate were reported. The 'low' estimate has been used in this report, consistent with the reporting of other national communicable diseases.

The significance of differences in rates and proportions was calculated using the Chi-square test with Yates correction.

Results

There were 1,680 notifications of IPD to NNDSS in 2005; a 30% decrease compared to the number of notifications in 2004 with declines in all jurisdictions of between 21 and 46%. The number of notifications and notification rate per 100,000 population are shown in Table 4. The Northern Territory continued to have the highest notification rate (35 cases per 100,000 population) while Victoria had the lowest (6 cases per 100,000 population).

When notification rates of IPD were examined by geographical distribution, variation within states was apparent (Map).

The number of notifications of IPD was greatest in winter months with the peak number of notifications in July (226 notifications). The effect of season was more evident in the distribution of cases aged 5 years or more, compared with younger children (Figure 1).

The highest rates of IPD disease in 2005 were among the elderly aged 85 years or over (41 cases per 100,000 population) and children aged 1 year (36.5 cases per 100,000 population, Figure 2). In all age groups there were more male than female cases (overall male to female ratio 1.3:1).

In 2005, the highest rates of IPD continue to be at the extremes of age (those 85 years or older and those aged 1 year (12–23 months). Between 2004 and 2005, the rate fell by 57% in the under 5 years age group (from 55.4 to 24 cases per 100,000 population) and by 69% specifically, in children aged 1 year (from 119 to 36.5 cases per 100,000 population) reflecting the impact of the introduction of universal 7vPCV at 2, 4 and 6 months with a catch-up program for

Table 2. Definitions of vaccination status and vaccine failure used in this report

Category	Definition
Fully vaccinated – aged <15 years	Those that have completed the primary course of the relevant vaccine(s) required for their age, indigenous status, geographical location and/or other risk factor(s) according to the most recent edition of the *Australian Immunisation Handbook,* at least 2 weeks prior to disease onset with at least 28 days between doses of vaccine. This includes the following: • a child that received a vaccine as 'catch up' and therefore does not require a full 3 dose primary schedule. Providing they have had the number of doses required for the age they were at first dose they should be considered fully vaccinated. • a child <15 years who received at least one 23vPPV vaccine aged at over 5 years and they are not yet due a subsequent dose of 23vPPV. NB: A young child who has had all the required doses for their age but is not old enough to have completed the primary course would not be assessed as fully vaccinated.
Fully vaccinated – aged ≥15 years	Those that have had the number of doses of 23vPPV required for their age, indigenous status, geographical location and/or other risk factor(s) according to the most recent edition of the *Australian Immunisation Handbook,* at least 2 weeks prior to disease onset with at least 28 days between doses of vaccine. NB: This is calculated on the age they were when they had their first dose of 23vPPV aged at least ≥15 years.
Partially vaccinated – aged <15 years	Those that have received at least one dose, but not *all* the recommended doses of the relevant vaccine(s) required for their age, indigenous status, geographical location and/or other risk factor(s) according to the most recent edition of the *Australian Immunisation Handbook,* at least 2 weeks prior to disease onset with at least 28 days between doses of vaccine. This includes the following; • a child who is too young to have completed their primary course; • a child that is overdue (>8 weeks) for a subsequent dose of their primary course; and • a child that is overdue for a booster dose of the relevant vaccine.
Partially vaccinated – aged ≥15 years	Those that have been vaccinated with at least one dose of 23vPPV but the time frame for a subsequent dose is outside the recommended schedule according to the *Australian Immunisation Handbook.*
Not vaccinated – all ages	Those that have never received a pneumococcal vaccine.
Vaccination validation	Written confirmation of vaccination through the Australian Childhood Immunisation Register, state or territory immunisation register or health record.
Vaccine failure	A fully vaccinated person (as defined above) with disease due to a serotype found in the corresponding vaccine.

Table 3. Enhanced IPD surveillance data collection, 2005, by state or territories

Age group	Jurisdictions
Under 5 years	Australian Capital Territory, New South Wales, Queensland, South Australia, Victoria
Over 50 years	New South Wales, Queensland
Over 64 years	South Australia, Victoria
All ages	Northern Territory, north Queensland, Tasmania, Western Australia

children up to 2 years of age. There was also a 24% reduction in the rate in the 65 year or over age group (from 25 to 19 cases per 100,000 population).

In 2005, the proportion of children aged 12 months of age immunised with 3 doses of 7vPCV was 91.1%. The proportion of children who are fully vaccinated against pneumococcal disease has increased steadily since 2001 (Figure 3).

An examination of trends in rates of IPD in different age groups from 2002 to 2005 is shown in Figure 4. The rates in children aged under 2 years declined by 68% ($p<0.0001$) and declined in adults aged 65 years or more by 30% ($p<0.0001$). Rates of IPD in other age groups not specifically targeted for pneumococcal immunisation also declined—there was a 52% ($p<0.0001$) reduction in the

Table 4. Notifications, rates and demographics of IPD cases, Australia, 2005, by state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Notifications	30	636	71	325	134	45	299	140	1,680
Rate per 100,000	9.2	9.4	35.0	8.2	8.7	9.3	6.0	7.0	8.3
Male:female ratio	0.9:1	1.3:1	1.3:1	1.3:1	1.1:1	1.8:1	1.3:1	1.1:1	1.3:1
Notifications by age									
<5 years	6	137	10	57	19	10	42	21	302
5 to 64 years	13	289	55	182	66	22	151	87	865
≥65 years	11	210	6	86	49	13	106	32	513
Notifications by indigenous status									
Indigenous	1	13	59	36	7	0	7	41	164
Non-Indigenous	10	438	12	218	125	34	280	99	1,216
Unknown	19	185	0	71	2	11	12	0	300

Figure 1. Notifications of invasive pneumococcal disease, Australia, 2005, by month of report and age group

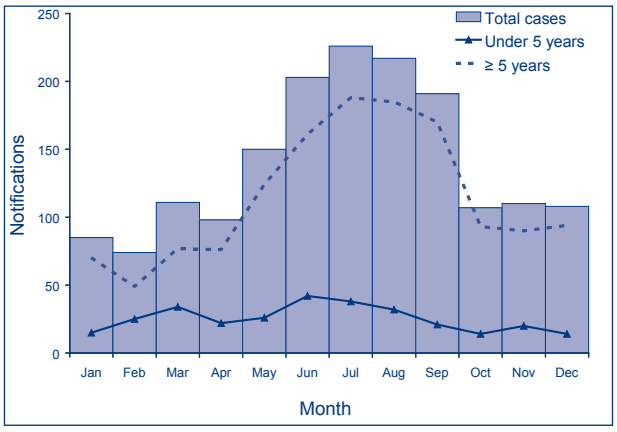


Figure 2. Notification rates of invasive pneumococcal disease, Australia, 2005, by age group and sex

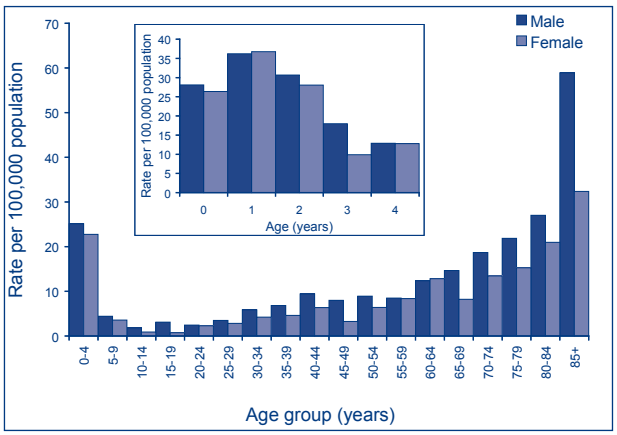


Figure 3. The proportion of children aged 12 months fully vaccinated with 7vPCV, Australia, 2001 to 2005, by indigenous status

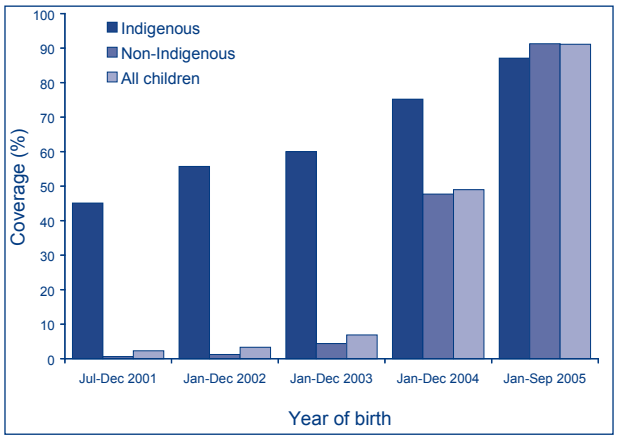
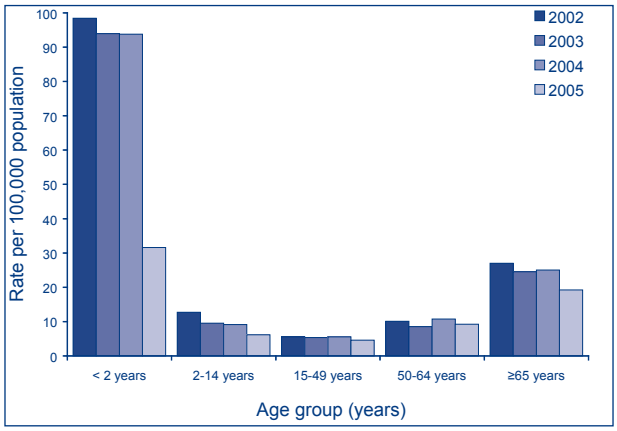
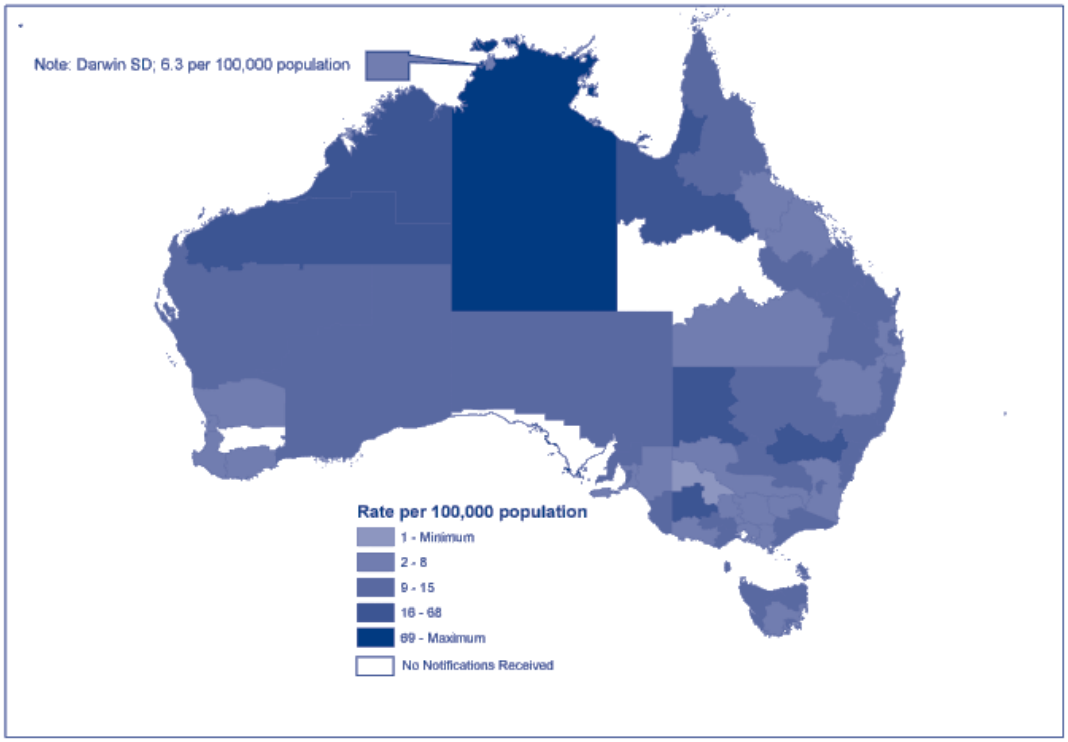


Figure 4. Pneumococcal disease notification rate, Australia 2002 to 2005, by age group



Map. Notification rates of invasive pneumococcal disease, Australia, 2005, by Statistical Division of residence



2–14 year age range; an 18% ($p<0.05$) decline in the 15–49 year age range and a 9% decline ($p=ns$) in the 50–64 year age range.

Rates in Indigenous people

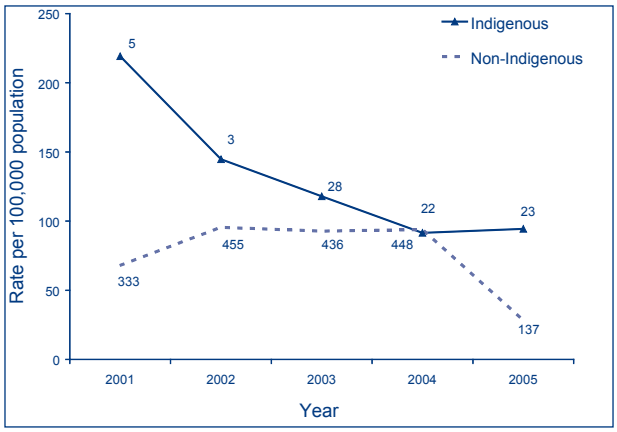
In 2005, indigenous status was reported for 1,380 (82%) notifications. There were 164 cases of IPD among Indigenous people (9.7% of all cases). This represents a rate of 66 cases per 100,000 in the Indigenous population—a rate 8.6 times that seen in the non-Indigenous population (7.6 cases per 100,000 population).

Rates in children

In 2005, rates remained similar to those in 2004 in Indigenous children, at 94 cases per 100,000 population, while a large decline to 28.7 cases per 100,000 population was recorded in non-Indigenous children in 2005 (Figure 5).

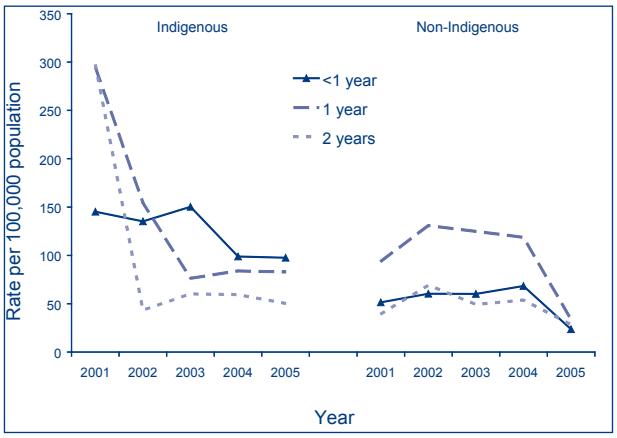
Figure 6 shows the annual rates in single year age groups in children aged less than 2 years. In 2005, the rates of IPD in Indigenous children aged less than 1 year (0–11 months) and in 1-year-olds (12–23 months) was similar to the rates in 2004 (97 versus 98) and (82 versus 84 cases per 100,000 population, respectively, both $p=ns$). Rates in Indigenous 2-year-old (24–35 months) children

Figure 5. Notification rates of invasive pneumococcal disease in Indigenous and non-Indigenous children aged less than 2 years, Australia, 2001 to 2005



fell from 59 to 50 cases per 100,000 population in 2005 ($p=ns$). Between 2004 and 2005, rates fell in non-Indigenous children aged less than 1 year, from 68 to 23 cases per 100,000 population, ($p<0.0001$); in the 1 year age group from 118 to 34 cases per 100,000 population ($p<0.0001$) and in the 2 years age group from 53 to 28 cases per 100,000 population ($p<0.0001$, Figure 6).

Figure 6. Rates of invasive pneumococcal disease 2001 to 2005 in children aged 2 years and under, by indigenous status and single year age group



Clinical presentations of invasive pneumococcal disease

Enhanced surveillance including data on clinical presentation and risk factors were available for 1,015 (60%) of all cases. Clinical presentation was reported in 783 (77%) of enhanced notifications of which 483 (61%) were pneumonia, 242 (31%) were bacteraemia and 46 (5.8%) were meningitis, with the remainder being other presentations (n=12).

The clinical presentations of IPD in Indigenous and non-Indigenous children aged less than 2 years were examined over the period 2001 and 2005. There was a decline in the proportion of Indigenous IPD cases presenting with pneumonia (from 22 cases in 2001 to 4 cases in 2005) making the proportions of cases presenting as pneumonia similar in Indigenous (27%) and non-Indigenous children (22%) in 2005 (Figure 7). Conversely, the proportion of Indigenous children with bacteraemia increased from 2001 to 2005, when the proportion (73%) was similar to that seen in non-Indigenous children (70%). Meningitis was a rare presentation in both groups and in all years, with the 'peak' in 2004 in Indigenous children representing only 2 cases.

Deaths in invasive pneumococcal disease cases

There were 128 deaths recorded among IPD cases in Australia in 2005, a case fatality rate of 7.6% (Table 5). The case fatality rate in those aged 65 years or older (16.4%) was significantly higher than in children aged less than 5 years (2.6 %, p <0.0001). The case fatality rate was lower but not significantly different in Indigenous (5.5%) compared to non-Indigenous cases (9.6%). Of the 8 children whose deaths were associated with

Figure 7. Changes in clinical presentations of invasive pneumococcal disease cases aged less than 2 years, 2001 to 2005, by indigenous status

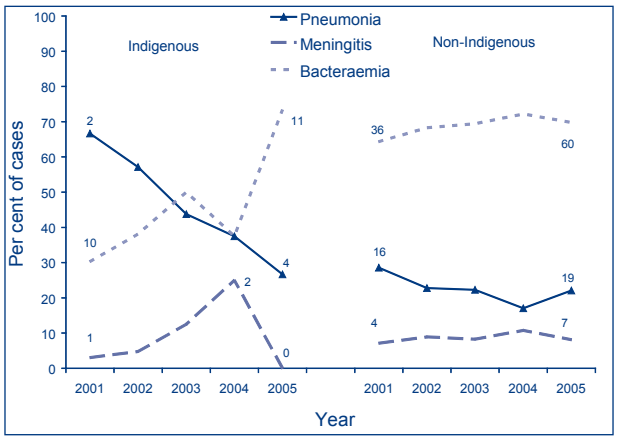


Table 5. Case fatality rates for invasive pneumococcal disease, Australia, 2005, by age, indigenous status and state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Cases	30	636	71	325	134	45	299	140	1,680
Deaths	2	64	4	4	7	2	31	14	128
Case fatality rate	6.7	10.1	5.6	1.2	5.2	4.4	10.4	10.0	7.6
Deaths in under 5 years	0	2	0	2	1	0	2	1	8
Case fatality rate <5 years	0.0	1.4	0.0	3.5	5.3	0.0	4.9	4.8	2.6
Deaths in ≥65 years	2	53	0	1	3	1	19	5	84
Case fatality rate ≥65 years	18.2	25.2	0.0	1.2	6.1	7.7	17.9	15.6	16.4
Deaths in Indigenous people	0	1	4	0	1	0	2	1	9
Case fatality rate – Indigenous	0.0	7.7	6.8	0.0	14.3	0.0	28.6	2.4	5.5
Deaths in non-Indigenous people	2	62	0	4	6	2	29	13	118
Case fatality rate – non-Indigenous	6.9	14.2	0.0	1.8	4.8	5.9	10.4	13.1	9.6

IPD, one was infected at birth, a second within the first month; three more within the first year; the remaining three were aged between 18 months and 2 years. Seven of the 8 cases had serotype information and of these seven, 3 were infected with 7vPCV serotypes (2 with 19F, 1 with 23F); 3 with serotypes in the 23vPPV (1, 10A, 19A) and the remaining case was infected with a non-vaccine strain (23B). Two of the 8 cases were recorded as fully vaccinated (Table 2) and both were infected with a non-7vPCV serotype (10A and 23B). Of the three 7vPCV serotype deaths, one was unvaccinated (serotype 23F), and the 2 cases due to 19F serotype disease were only partially vaccinated. One child was too young for the third dose of vaccine but was fully vaccinated for age while the other child was overdue for the third vaccine dose.

Risk factors for pneumococcal disease

Recognised risk factors were collected in 686 (67.5%) cases in the enhanced dataset. The most commonly reported risk factor was chronic disease (376 cases, 54.8%), which included chronic respiratory, cardiac and renal disease, and diabetes.

The frequency of risk factors for IPD in Indigenous and non-Indigenous people are shown in Table 6. While 29% of Indigenous children with IPD had predisposing risk factors, this was not significantly different when compared with 12% of non-Indigenous children. Chronic disease was a significantly more common risk factor for IPD in older Indigenous children and adults than in non-Indigenous cases in the same age range ($p<0.005$).

Pneumococcal serotypes causing disease in Australia

Pneumococcal serotypes were identified for isolates from 1,507 (89.7%) of all notified cases in 2005. Of these, 916 (60.8%) were serotypes in the 7vPCV and 1,331 (88.3%) were serotypes in the 23vPPV (Table 7).

The proportion of 7vPCV serotypes in cases of IPD in the Northern Territory (12.1%) and Western Australia (49.6%) were significantly lower than the proportion in the national total (60.8%). The propor-

Table 6. The frequency of risk factors for invasive pneumococcal disease, Australia, 2005, by age group and indigenous status

Risk factor	Cases aged less than 5 years			Cases aged 5 years or more		
	Indigenous n=34	Non-Indigenous n=269	Significance of difference	Indigenous n=130	Non-Indigenous n=1,247	Significance of difference
Premature birth	3	13	NS	NA	NA	–
Congenital abnormality	1	7	NS	NA	NA	–
Asplenia	1	0	NS	1	10	NS
Immuno-compromised	1	2	NS	14	105	NS
Chronic illness	4	10	NS	43	260	p<0.005

NS Not significant.

NA Not applicable.

Table 7. Number and proportion* of pneumococcal serotypes in cases of invasive pneumococcal disease covered by the 7-valent and 23-valent pneumococcal vaccines, Australia, 2005, by state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
7v serotypes	15	347	8†	182	90	22	186	66‡	916
(%)*	(62.5)	(63.7)	(12.1)	(62.1)	(68.7)	(73.3)	(65.3)	(49.6)	(60.8)
23V serotypes	22	488	41†	252	121	28	265	114	1,331
(%)*	(91.7)	(89.5)	(62.1)	(86.0)	(92.4)	(93.3)	(93.0)	(85.7)	(88.3)
Total serotyped	24	545	66	293	131	30	285	133	1,507

* As a proportion of serotyped isolates.

† Significantly lower proportion of 7vPCV and 23vPPV serotypes compared with the national total ($p<0.0001$).

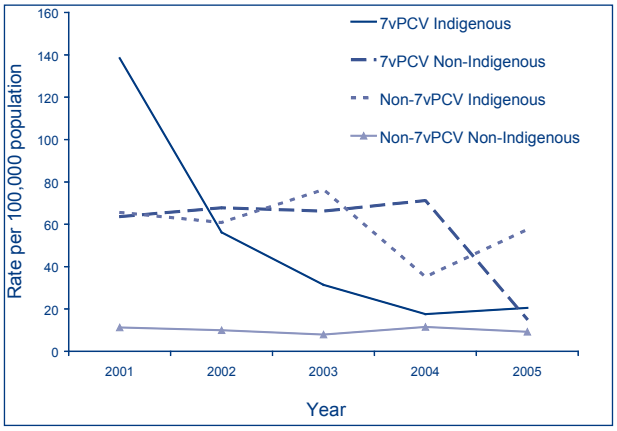
‡ Significantly lower proportion of 7vPCV serotypes compared with the national total ($p<0.05$).

tion of 23vPPV serotypes in the Northern Territory (62.1%) was also significantly lower than the proportion in the national total (88.3%, Table 7).

As in previous years, the proportion of 7vPCV serotypes causing disease in Indigenous children aged less than 2 years (23%) was significantly lower than in non-Indigenous children in the same age group (55%, $p<0.0001$). Similarly the proportion of 23vPPV serotypes causing disease in Indigenous cases aged 2 years or over was significantly lower (62%) than in non-Indigenous cases in the same age group (84%, $p< 0.0001$).

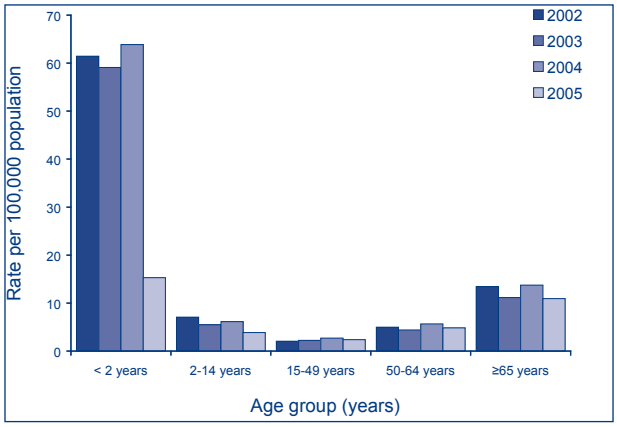
An examination of the rates of IPD disease caused by 7vPCV and non-7vPCV serotypes and indigenous status in children aged less than 2 years, showed a decline in rates in Indigenous children (from 138 to 20 cases per 100,000 population) between 2001 and 2005. Rates of disease caused by non-7vPCV serotypes also decreased in the same period but not significantly (65.5 in 2001 to 57.5 cases per 100,000 population in 2005). In non-Indigenous children, rates of 7vPCV serotype disease fell from 71 cases per 100,000 population to 15 cases per 100,000 population between 2004 and 2005, while rates of disease caused by non-vaccine serotypes in the same period were little changed (11.5 cases per 100,000 population in 2004 and 9.3 cases per 100,000 population in 2005, Figure 8).

Figure 8. Notification rates of 7-valent and non-7-valent serotypes causing cases of invasive pneumococcal disease in children aged less than 2 years, 2001 to 2005, by indigenous status



The total population rates of IPD caused by 7-valent vaccine serotypes fell 75% between 2002 and 2005 in children aged under 2 years (61.4 to 15.3 cases per 100,000 population), 45% in the 2–14 year age range (7.1 to 3.9 cases per 100,000 population) and 18% in the 65 years or over age group (13.4 to 10.9 cases per 100,000 population). All these declines were statistically significant. There was no significant change in the rates in the 15–49 years age range (2.1 to 2.4 cases per 100,000 population) or the 50–64 year age range (5 to 4.9 cases per 100,000 population, Figure 9).

Figure 9. Rates of invasive pneumococcal disease caused by 7-valent pneumococcal vaccine serotypes, 2002 to 2005, by age group



Rates of IPD due to 23vPPV serotypes in Indigenous adults aged 50 years or over declined from 71 to 28 cases per 100,000 population ($p<0.005$) between 2001 and 2005. In the same period, rates of disease caused by non-23vPPV serotypes increased from 3 to 21 cases per 100,000 population, but this increase was not statistically significant. Rates of 23vPPV serotype disease in non-Indigenous adults aged 65 years or over showed little change over the period 2001 and 2005 (Figure 10).

Figure 10. Notification rates of 23-valent and non-23-valent serotypes causing cases of invasive pneumococcal disease in Indigenous adults (aged more than 50 years) and non-Indigenous adults (aged 65 years or over), 2001 to 2005

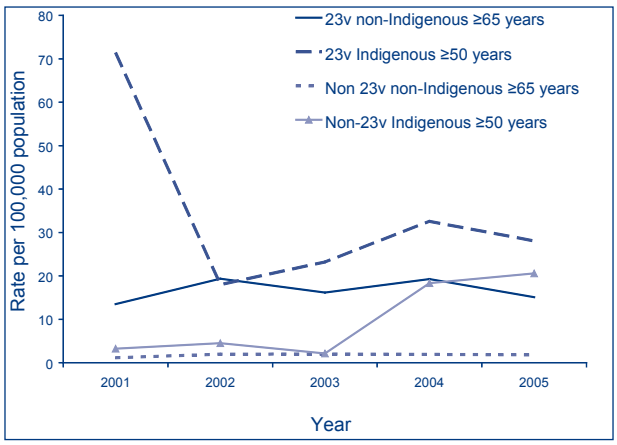


Table 8. *Streptococcus pneumoniae* susceptibility to penicillin and ceftriaxone/cefotaxime, Australia, 2005, by state or territory

Antibiotic	Description	State or territory								Australia
		ACT	NSW	NT	Qld	SA	Tas	Vic*	WA	
Penicillin	Resistant	0	32	1	18	2	1	6	3	63
	Intermediate	6	45	1	18	9	1	15	18	113
	Susceptible	24	507	68	220	105	42	228	111	1,305
	Total tested	30	584	70	256	116	44	249	132	1,481
	% reduced susceptibility	20.0	13.2	2.9	14.1	9.5	4.5	8.4	15.9	11.9
Ceftriaxone/cefotaxime	Resistant	0	7	0	1	0	1	1	0	10
	Intermediate	0	26	0	4	2	1	0	1	34
	Susceptible	25	397	52	251	27	42	188	131	1,113
	Total tested	25	430	52	256	29	44	189	132	1,157
	% reduced susceptibility	0.0	7.7	0.0	2.0	6.9	4.5	0.5	0.8	3.8

More complete data from Victoria found slightly higher prevalences of reduced susceptibility to penicillin (12.6%) and third-generation cephalosporins (1.7%).

Antibiotic resistance in invasive pneumococcal disease

The penicillin susceptibility was tested in 1,481 isolates and ceftriaxone/cefotaxime susceptibility was tested in 1,157 isolates (Table 8).

A total of 176 (11.9%) isolates had reduced susceptibility to penicillin, which was lower than the number and proportion of isolates with reduced penicillin susceptibility in 2004 (250 isolates, 13.5%). Forty-four (3.8%) isolates had reduced susceptibility to ceftriaxone/cefotaxime in 2005, which was similar to the number and proportion in 2004 (42 isolates, 3.7%).

Of the 176 isolates with reduced susceptibility to penicillin, 149 were serotyped. One hundred and eighteen (80%) isolates with reduced penicillin susceptibility were serotypes in the 7vPCV and 137 (92%) were serotypes in the 23vPPV. Of the penicillin insensitive isolates, 45 were serotype 9V, 24 were serotype 14 and 19 were serotype 19F, accounting for 75% of isolates with known serotypes. There was no significant difference in the prevalence of serotypes with reduced penicillin susceptibility between children less than 5 years and adults aged 65 years or over.

Vaccination status

Data on vaccination status was available for 1,127 (67%) of all IPD cases in 2005. Of the 1,127 cases with a vaccination history, the majority (781, 69%) were reported as unvaccinated. IPD due to 7vPCV serotype disease was reported in 7 cases who were fully vaccinated with the 7vPCV and IPD due to 23vPPV serotype disease was reported in 165 cases who were fully vaccinated with the 23vPPV.

Of the 7 fully vaccinated cases of IPD due to 7vPCV serotype disease, 3 had received 3 scheduled doses and 4 had received 1 or 2 doses as per the recommended catch up schedule (Table 9). All cases were aged less than 5 years and none of the cases were Indigenous. The serotypes causing disease in the 3 children who received 3 doses of conjugate vaccine were 19F, 6B and 23F. The serotypes in the 4 children that developed 7vPCV serotype disease following 7vPCV catch up vaccination (1 or 2 vaccine doses) were 9V, 14, and 19F (2 children).

Of the cases of IPD with 23vPPV serotype disease that were fully vaccinated with the 23vPPV, 21 were Indigenous, 142 non-Indigenous and the indigenous status of the remaining 2 cases was unknown. Risk factor information was available on 163 of the 165 (99%) 23vPPV failures. Seventy-nine cases had one risk factor recorded, 44 had 2 risk factors recorded and 15 had 3 or more risk factors recorded (Table 9).

Discussion

This report shows an overall 30% reduction of IPD from 2004 to 2005 with the highest rates still remaining in the extremes of age (those 85 years or older and those aged 1 year (12–23 months). The Northern Territory, the jurisdiction with an Indigenous population of 30% compared to 3% nationally, continued to have the highest notification rate. The major reduction over this time was in children under 5 years (57%) and more specifically in the 1-year-olds (69%) reflecting the impact of the introduction of universal 7vPCV at 2, 4 and 6 months with a catch-up program for children aged up to 2 years.

Table 9. Vaccination reporting and vaccine failure in cases of invasive pneumococcal disease, Australia, 2005, by state or territory

All cases	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Vaccination history									
Received at least one vaccine ever	0	128	43	37	24	10	78	26	346
Never vaccinated	11	317	28	78	80	32	173	62	781
No vaccine data collected	19	191	0	210	30	3	48	52	553
Vaccine failures 7-valent vaccine									
<5 years of age	0	4	0	0	1	0	2	0	7
Received 3 doses	0	2	0	0	0	0	1	0	3
Received 1 or 2 doses	0	2	0	0	1	0	1	0	4
23-valent failures									
≥5 years of age	0	64	13	18	16	5	38	11	165
1 risk factor recorded	0	43	2	6	7	4	12	5	79
2 risk factors recorded	0	10	6	7	2	1	18	0	44
3 or more risk factors recorded	0	3	5	1	0	0	6	0	15
No risk factor data collected	0	0	0	2	0	0	0	0	2

Immunisation of young children may have contributed to a decline in rates of IPD in other age groups. The rates of IPD in Australia overall declined from 12.6 per 100,000 to 8.3 per 100,000 between 2002 and 2005. In the same period, there was a 75% decrease in 7vPCV serotype disease (and 68% decrease in total IPD rates) in children aged less than 2 years, who were eligible for immunisation with the 7vPCV. Over the same period the 2-14 year age group (most of whom were not immunised) and those aged more than 65 years (who were eligible for23vPPV immunisation) also showed significant decreases in 7vPCV serotype IPD rates (45% and 18% respectively) as well as for all IPD serotypes (52% and 30% respectively). The 15–49 years age range showed a significant reduction in all IPD rates but not specifically when analysing the 7vPCV serotypes. These changes in the epidemiology of IPD in Australia for children and adults are similar to those seen in the United States of America (USA) after the introduction of the 7-valent vaccine there in 2000,[3,4] however the early reduction noted in the Australian 2–14 year age range was not initially seen in the USA and is more striking than that recently reported in the USA.[5] Poehling et al[6] have reported that in the years following the introduction of the 7vPCV in the USA, neonate and infants too young to receive 7vPCV or a full course are also benefiting from a herd immunity effect. This is encouraging and the 0–90 day age group in Australia will be a group to further evaluate in coming years.

Indigenous adults (50 years or over) and non-Indigenous adults (65 years or over) have been eligible for free immunisation with the 23vPPV since 1999 and 2005 respectively. The reduction in IPD in the 65 years or over age group from 2004 to 2005 was 24%—possibly reflecting some contribution from the universal 23vPPV funded program in this age group, as well as herd effect from the 7vPCV. However, the specific impact of each vaccine will be better assessed following a second year of the programs. Of note, comparing 2001 to 2005, disease due to 23vPPV serotypes has changed little in the non-Indigenous adults aged 65 years or over, the majority age group eligible for the now funded 23vPPV program (Figure 10).

Current recommendations for immunisation with the 23vPPV in Australia include 'at risk' groups of any age (Table 1) as well as all those 65 years or over (50 years or over for Indigenous adults). An assessment of targeted populations for 23vPPV immunisation in the USA compared the proportions of vaccine preventable disease in the 50–64 year age range with that preventable in persons of any age with current risk factors. Increasing coverage in eligible age groups and with recognised risk factors would prevent more disease than expanding eligible age groups or list of existing risk factors.[7] In 2004, only an estimated 51% of 65 years or older Australians had received 23vPPV vaccination, but whether the vaccine was within the last 5 years was not determined.[8] The development and provision of a whole-of-life immunisation register to inform strategies to improve uptake and achieve better vaccine coverage in Australian adults may be more beneficial.

A recent review of vaccine preventable diseases and vaccination policies in Indigenous people in Australia, New Zealand, Canada and the United States of America, noted that despite significant

reductions in pneumococcal disease, higher rates persist in Indigenous populations as a result of differences in circulating serotypes, heavy rates of nasopharyngeal colonisation and a higher prevalence of risk factors.[9]

The decline in rates of IPD in non-Indigenous children in 2005 as a result of the universal 7vPCV immunisation program has re-opened the gap between Indigenous and non-Indigenous children aged less than 2 years. Rates of IPD in the vaccine eligible Indigenous children in 2004 were similar to those in the vaccine non-eligible non-Indigenous children. In 2005, the rate in Indigenous children remained relatively unchanged, while the rate in non-Indigenous children fell from 94 to 28.7 cases per 100,000 population making the rate of IPD in Indigenous children 3-times that in non-Indigenous children aged less than 2 years. There were however, only 23 cases reported in Indigenous children in this age group in 2005. Of these children only 5 had disease caused by a 7vPCV serotype, suggesting as already reported from north Queensland,[10] that rates in this age group may not fall much further if relying on the current 7-valent vaccine. It was well recognised prior to the 7vPCV vaccine programs that Indigenous children under 2 years had a significantly smaller proportion of serotypes contained in the 7vPCV (55%) than non-indigenous children (86% $p<0.005$).[11] Of note, at least 40% of the Indigenous children had one or more pre-disposing risk factors for IPD with most being chronic or at risk conditions that are mainly preventable and therefore efforts for their control offer other strategies for IPD reduction while awaiting vaccines with broader serotype coverage.

The 7vPCV has been shown to protect against nasopharyngeal colonisation with vaccine serotypes. Two studies of the impact of the 7vPCV in high endemic Indigenous communities in North America demonstrated that paediatric vaccination was associated with a decline in colonisation with vaccine serotypes in vaccinees[12] and in unvaccinated adults.[13] However, the reduction in colonisation in 7vPCV vaccine recipients was offset by an increase in colonisation with non-vaccine pneumococcal serotypes. While recognising that not all serotypes are equally invasive, the potential of colonising serotypes to become invasive serotypes has been a concern, especially if there is a shift to non-7vPVC serotypes. Post 7vPVC introduction surveillance studies vary on the impact of serotype replacement on IPD. Reporting from the Intermountain Health Care region in the USA, Byington[14] found significant increases in the proportion of cases due to non-7vPCV serotypes. Other studies have found only modest increases in non-7vPCV serotypes.[3,4,5] In a matched case control study a small increase in IPD from non-7vPCV serotypes was observed, particularly from the vaccine-associated serotype 19A, as well as vaccinated children having a higher risk of disease from serotype 22F, a 23vPPV serotype.[15] A study from an large urban USA city found no statistical increase in strictly non-7vPCV serotypes but a significant increase in 7vPCV-associated serotypes, specifically again, 19A, as well as 6A.[16] Poehling et al,[6] in studies covering portions of 8 USA states, showed no evidence of non-7vPCV replacement. The impact of pneumococcal immunisation on nasopharyngeal colonisation in Australian children will be an important area for further research.

In 2005, the largest proportion (90%) of isolates from notified cases in Australia to date have serotype results providing extensive information to assess the possibility of serotype replacements following vaccine introduction, as well as other serotype linked associations. There is no evidence in children aged 2 or under of non-7vPCV serotype replacement causing IPD in either Indigenous children who have been eligible for 7vPCV since mid-2001 or non-Indigenous children who have been eligible since January 2005. The low rates of non-23vPPV serotypes causing IPD in non-Indigenous adults remained stable from 2001 to 2005, and rates in Indigenous adults aged 50 years or over have increased, but not significantly. Continued monitoring for serotype replacement is essential and the ability to look for emerging trends in incidence of disease, clinical presentations and drug resistance patterns associated with specific serotypes is valuable and may serve to shape future vaccines.

The change in clinical presentations seen among Indigenous children aged less than 2 years from a marked predominance of pneumonia to that of bacteremia following the introduction of the 7vPCV, is remarkable. The level of bacteremia is now of similar proportions to non-Indigenous same-age children. Whether this reflects the 7vPCV serotypes causing more pneumonia is unclear. The proportion of clinical presentations in non-Indigenous children one year following 7vPCV introduction remained unchanged.

Completeness of data for indigenous status continues to be problematic particularly in New South Wales, the Australian Capital Territory and parts of Queensland. Those with unknown indigenous status are mainly in the ages 5–49 years or 64 years, where enhanced data is not collected and reflects the capture of indigenous status in other notifiable diseases. A group working on the 'Improving Indigenous Identification in Communicable Disease Reporting Project' have recently finalised a report and a way forward to try to remove some of the barriers to more complete reporting.

As drug susceptibility results from this surveillance are uniformly not from reference laboratories, but from the treating institutions, these results are viewed as indicative of drug resistance trends. The number and proportion of pneumococci with reduced susceptibility to penicillin (11.8%) or ceftriaxone (3.8%) remained low in 2005. The reduced susceptibility for both drugs varied among the jurisdictions. The Australian Group on Antimicrobial Resistance (AGAR) also reported on antimicrobial resistance (AMR) in invasive and non-invasive isolates of *S. pneumoniae* in 2005.[17] AGAR has been monitoring AMR in pneumococcal infections since 1989 in all Australian states and territories except the Northern Territory and Tasmania. Penicillin resistance in pneumococcal infections in Australia increased rapidly between 1994 and 1999, and more slowly since, with lower rates than in many other countries. Rates of penicillin non-susceptibility in the 2005 AGAR survey for invasive isolates were slightly higher (16.5%) than in this study and, as expected, the AGAR non-invasive isolate rates were significantly higher (30.6%). Non-invasive isolate information is not gathered by this working party. The AGAR result of 14.9% cefotaxime/ceftriaxone non-susceptibility is only on penicillin non-susceptible isolates and therefore, though useful, is not comparable to this study.

Pneumococcal vaccination has been demonstrated to reduce the prevalence of antibiotic insensitive pneumococcal disease. In the USA, there was a 57% decline in penicillin non-susceptible strains of pneumococci between 1999 and 2004.[18] While large reductions were reported in children aged less than 2 years (81%), an increase in disease caused by a penicillin insensitive serotype 19A (a 23vPPV strain) was noted in the same age group. An increase in 19A serotypes has not been seen in Australia and to date there has been no emergence of a predominant penicillin insensitive non-vaccine pneumococcal serotype causing disease. Additionally, in contrast to previous years,[2,19] there was no significant difference in the prevalence of serotypes with reduced penicillin susceptibility in children aged between less than 5 years and adults aged 65 years or over. With 80% of isolates with reduced penicillin susceptibility covered by the 7vPCV and 92% covered by the 23vPPV the prospect of reducing penicillin resistance in the future is encouraging.

The initial year of the universal 7vPCV program has been remarkable for the reduction in IPD in those targeted for the vaccine as well as other age groups. There is no evidence to date to suggest any increase from non-7vPCV serotype disease is occurring. Continued surveillance of pneumococcal disease is essential to measure the effectiveness of the government funded universal pneumococcal immunisation programs and to guide other strategies for IPD control.

Acknowledgements

The following laboratories are gratefully acknowledged for their support of Pneumococcal Laboratory Surveillance:

ACT

ACT Pathology

New South Wales

The Children's Hospital, Westmead
Central Coast Pathology
Concord Hospital
Douglass Hanley Moir
Hunter Area Pathology
CIDM, ICPMR
Laverty Pathology
THE Pathology
Nepean Hospital
PaLMS
Royal Prince Alfred Hospital
SEALS
St George
St Vincents Hospital
SWAPS
Sydney Adventist Hospital
Wollongong Hospital

Northern Territory

Royal Darwin Hospital, Department of Microbiology
Private laboratories
Alice Springs Hospital Department of Microbiology
Katherine Hospital Department of Microbiology

Queensland

Queensland Health Pathology Laboratories and the Microbiology Discipline Working Party
Private Pathology Laboratories throughout Queensland
Tropical Public Health Unit, Cairns
Communicable Diseases Unit, Brisbane

South Australia

Women's and Children's Hospital, Adelaide
The Gribbles Group
South Path Microbiology and Infectious Diseases
Clinipath Laboratories
Institute for Medical and Veterinary Science laboratories

Tasmania

Royal Hobart Hospital
Launceston General Hospital (Northern Tasmanian Pathology Service)
North West Pathology

Victoria

MDU PHL is grateful to the following labs who have been identified as having contributed isolates to the reported data-set.

Box Hill Hospital Pathology Service
Royal Childrens Hospital (Parkville) Pathology Service
Dorevitch Pathology Mayne Health (Heidelberg)
Gippsland Pathology Service Sale (& Traralgon)
Alfred Hospital Pathology Service
Monash Medical Centre (Clayton) Pathology Service
Austin Hospital Pathology Service
Bendigo Health Pathology Service
Goulburn Valley Health (Shepparton) Pathology Service
Northern Hospital (Epping) Pathology Service
St John of God Health Care Ballarat Pathology Service
Geelong Hospital Pathology Service (Pathcare)
South West Healthcare (Warnambool) Pathology Service
Saint Frances Xavier Cabrini Hospital Pathology Service
North East Health Wangaratta Pathology Service
Royal Melbourne Hospital (Parkville) Pathology Service
St Vincents Hospital (Melbourne) Ltd Pathology Service
Ballarat Health Services (Base campus) Pathology Service
Forensicare – Victorian Institute of Forensic Medicine
Wimmera Base Hospital (Horsham) Pathology Service
Gribbles Pathology (Melbourne)
Echuca Hospital Pathology Service
Mildura Base Hospital Pathology Service
Melbourne Pathology
St John of God Health Care Mildura Pathology Service

From MDU PHL, Ms Janet Strachan and Ms Jo Stylianopoulos contributed to testing, Dr Mark Veitch and Ms Marion Easton to data management.

Western Australia

We would like to acknowledge the Vaccine Impact Surveillance Network, which is funded by the Meningitis Centre of Western Australia and The Telethon Institute for Child Health Research,

Princess Margaret and King Edward Memorial Hospitals Department Microbiology
Fremantle Hospital Department Microbiology
PathWest
Royal Perth Hospital Department Microbiology
St John of God Pathology Department Microbiology
Western Diagnostic Pathology Department Microbiology
Clinipath Department of Microbiology

Author details

Paul Roche[1]
Vicki Krause[2]
Heather Cook[2]
Mark Bartlett[3]
David Coleman[4]
Craig Davis[5]
James Fielding[6]
Carolien Giele[7]
Robin Gilmour[3]
Ros Holland[8]
Riemke Kampen[9]
Mitchell Brown[10]
Lyn Gilbert[10]
Geoff Hogg[11]
Denise Murphy[12]

1. Surveillance Policy and Systems Section, Department of Health and Ageing, Canberra, Australian Capital Territory
2. Centre for Disease Control, Department of Health and Community Services, Darwin, Northern Territory
3. Communicable Diseases Branch, Department of Health, Sydney, New South Wales
4. Communicable Disease Prevention Unit, Department of Health and Human Services, Hobart, Tasmania
5. Communicable Disease Unit, Queensland Health, Brisbane, Queensland
6. Communicable Diseases Control Unit, Department of Human Services, Melbourne, Victoria
7. Communicable Disease Control Directorate, Department of Health, Perth, Western Australia
8. Communicable Disease Control Branch, Department of Health, Adelaide, South Australia
9. Communicable Diseases Control Unit, ACT Health, Canberra, Australian Capital Territory
10. Centre for Infectious Diseases and Microbiology, Institute of Clinical Pathology and Medical Research, Westmead, New South Wales
11. Microbiological Diagnostic Unit, University of Melbourne, Melbourne, Victoria
12. Queensland Health Pathology and Scientific Services, Brisbane, Queensland

Corresponding author: Dr Paul Roche, Surveillance Policy and Systems Section, Office of Health Protection, Department of Health and Ageing, GPO Box 9848 (MDP14), Canberra ACT 2601. Telephone +61 2 6289 8152. Facsimile +61 2 6289 7100. Email paul.roche@health.gov.au

References

1. Pneumococcal disease in Australia. *Med J Aust* 2000;173: S17–S64.
2. Roche PW, Krause VL, Bartlett M, Coleman D, Cook H, Davis C, et al. Invasive pneumococcal disease in Australia, 2004. *Commun Dis Intell* 2006;30:80–92.
3. Whitney CG, Farley MM, Hadler J, Harrison LH, Bennett NM, Lynfield R, et al. Decline in invasive pneumococcal disease after the introduction of protein-polysaccharide conjugate vaccine. *N Engl J Med* 2003;348:1737–1746.

4. Lexau CA, Lynfield R, Danila R, Pilishvili T, Facklam RR, Farley M, et al. Changing epidemiology of invasive pneumococcal disease among older adults in the era of pediatric pneumococcal conjugate vaccine. *JAMA* 2005;294:2043–2051.
5. Centers for Disease Control and Prevention. Direct and indirect effects of routine vaccination of children with 7-valent pneumococcal conjugate vaccine on incidence of invasive pneumococcal disease – United States, 1998–2003. *MMWR Morb Mortal Wkly Rep* 2005;54:893–897.
6. Poehling KA, Talbot TR, Griffen MR, Craig AS, Whitney CG, Zell E, et al. Invasive pneumococcal disease among infants before and after introduction of pneumococcal conjugate vaccine. *JAMA* 2006;295:1668–1674.
7. Greene CM, Kyaw MH, Ray SM, Schaffner W, Lynfield R, Barrett NL, *et al. Prevent*ability of invasive pneumococcal disease and assessment of current polysaccharide vaccine recommendations for adults: United States, 2001–2003. *Clin Infect Dis* 2006;43:141–150.
8. Australian Institute of Health and Welfare. 2004 Adult vaccination survey: summary results. 2005. Available from: http://www.aihw.gov.au/publications/index.cfm/title/10109 Accessed on 2 February 2007.
9. Menzies R, McIntyre P. Vaccine preventable diseases and vaccination policy for Indigenous populations. *Epidemiol Rev* 2006.
10. Hanna J, Humphreys J, Murphy D. Invasive pneumococcal disease in Indigenous people in north Queensland, 1999–2004. *Med J Aust* 2006;184:118–121.
11. Roche P, Krause V. Invasive pneumococcal disease in Australia, 2001. *Commun Dis Intell* 2002;26:505–519.
12. Millar EV, O'Brien KL, Watt JP, Brondson MA, Dallas J, Whitney CG, et al. Effect of community-wide conjugate pneumococcal vaccine use in infancy on nasopharyngeal carriage through 3 years of age: a cross-sectional study in a high-risk population. *Clin Infect Dis* 2006;43:8–15.
13. Hammitt LL, Bruden DL, Butler JC, Baggett HC, Hurlbert DA, Reasonover A, et al. Indirect effect of conjugate vaccine on adult carriage of *Streptococcus pneumoniae*: an explanation of trends in invasive pneumococcal disease. *J Infect Dis* 2006;193:1487–1494.
14. Byington CL, Samore MH, Stoddard GJ, Barlow S, Daly J, Korgenski K, et al. Temporal trends of invasive disease due to *Streptococcus pneumoniae* among children in the Intermountain West: emergence of non-vaccine serogroups. *CID* 2005;41:21–29.
15. Whitney CG, Pilishvili T, Schaffner W, Craig AS, Lynfield R, Nyquist AC, et al. Effectiveness of seven-valent pneumococcal conjugate vaccine against invasive pneumococcal disease: a matched case-controol study. *Lancet* 2006;368:1495–1502.
16. Steenhof AP, Shah SS, Ratner AJ, Patil SM, McGowan KL. Emergence of vaccine-related pneumococcal serotypes as a cause of bacteremia. *CID* 2006;42:907–914.
17. Gottlieb T, Collignon P, Robson J, Pearson JMH, Bell J. *Streptococcus pneumoniae* survey: 2005 antimicrobial susceptibility report. Canberra: Australian Group for Antimicrobial Resistance; 2006.
18. Kyaw MH, Lynfield R, Schaffner W, Craig AS, Hadler J, Reingold A, et al. Effect of the introduction of the pneumococcal conjugate vaccine on drug-resistant *Streptococcus pneumoniae*. *N Engl J Med* 2006;354:1455–1463.
19. Watson M, Roche P, Bayley K, Bell JM, Collignon P, Gilbert GL, et al. Laboratory surveillance of invasive pneumococcal disease in Australia, 2003 – predicting the future impact of the universal childhood conjugate vaccine program. *Commun Dis Intell* 2004;28:455–464.

INFLUENZA SURVEILLANCE IN VICTORIA, 2006

James E Fielding, Emma R Miller, Josie Adams, Bronwyn Hawking, Kristina Grant, Heath A Kelly

Abstract

The Victorian influenza season in 2006 remained within normal seasonal activity thresholds and was relatively mild compared with recent years. The season peaked in mid-August, with influenza-like illness (ILI) rates from general practitioner sentinel surveillance and the Melbourne Medical Locum Service (MMLS), and cases of laboratory-confirmed influenza notified to the Department of Human Services, reaching their zeniths within one week of each other. A total of 74 general practitioners (GPs) participated in the sentinel surveillance in 2006, reporting a total of 136,732 consultations during the surveillance period from May to September inclusive. Participating GPs reported a total of 765 patients with an ILI; an average ILI rate of 5.6 cases per 1,000 consultations. The average ILI rate from the MMLS in the same period was 8.5 cases per 1,000 call-outs. Eighty-two per cent of laboratory-confirmed influenza notifications during the surveillance period were type A; the remainder were type B. Typing indicated circulation of two predominant strains during the season: A/Wisconsin/67/2005(H3N2)-like virus and B/Malaysia/2506/2004-like virus. The influenza vaccine for 2006 contained A/New Caledonia/20/99(H1N1)-like virus, A/California/7/2004(H3N2)-like virus and B/Malaysia/2506/2004-like virus. *Commun Dis Intell* 2007;31:100–106.

Keywords: surveillance, epidemiology, influenza

Introduction

Influenza surveillance in Victoria is comprised of three core elements. The Victorian Infectious Diseases Reference Laboratory (VIDRL) coordinates sentinel general practice (GP) surveillance for influenza-like illness (ILI) with laboratory testing of selected cases and surveillance of ILI through the Melbourne Medical Locum Service (MMLS). The Department of Human Services (DHS) coordinates the surveillance of laboratory-confirmed influenza, which is legislated under the *Health (Infectious Diseases) Regulations 2001*.

The objectives of the Victorian influenza surveillance are to:

- monitor the epidemiology of laboratory-confirmed influenza in Victoria;
- identify the onset, duration and magnitude of annual influenza seasons in Victoria; and
- characterise the circulating influenza strains in the community to assist in the evaluation of the current season's, and formulation of the following season's vaccine.

Additionally, the World Health Organization (WHO) Collaborating Centre for Reference and Research on Influenza, conducts and provides data on strain typing of influenza isolates or influenza-positive specimens forwarded by VIDRL and two Melbourne hospital laboratories. This report summarises the results from Victorian influenza surveillance in 2006 and provides a comparison with previous influenza seasons.

Methods

General Practice Sentinel Surveillance

Geographic representativeness of the sentinel GP influenza surveillance scheme was sought by recruiting general practitioners to achieve an approximate coverage of one practice per 200,000 population in metropolitan areas and one practice per 100,000 population in rural areas, as recommended in the *Framework for an Australian Influenza Pandemic Plan*.[1] In 2006, 54 GPs were recruited from metropolitan Melbourne and 20 from rural and regional Victoria (Figures 1a and 1b), corresponding to rates in these areas of 1.5 and 1.4 GPs per 100,000 population, respectively.

Continuing Professional Development points from the Royal Australian College of General Practitioners or the Australian College of Rural and Remote Medicine were offered to participating GPs. GPs were required to report the total number of consultations and the age, sex and vaccination status of all patients presenting with an ILI (defined below) for each week in the surveillance period and to take nose and throat swabs from patients with an ILI. An ILI was defined as fever (or history of feverishness), cough and fatigue/malaise. Completion of an evaluation questionnaire was also requested after weekly reporting had concluded.

The GP sentinel surveillance was conducted over a 22 week period in 2006 from 1 May to 1 October (weeks 18 to 39 inclusive). ILI activity was described using a set of threshold values: normal baseline activity (<2.5 ILI cases per 1,000 patients per week); normal seasonal activity (between 2.5 and 15); higher than expected activity (between 15 and 35); and epidemic activity (>35).[2]

GPs were asked to collect swabs from patients within 3 days of onset of ILI symptoms and forward them in viral transport medium to VIDRL with data on: the patient's age; vaccination status; date of illness onset; and the GPs clinical impression of the likelihood of influenza. Specimens were transported to VIDRL by a dedicated courier from metropolitan practices and through a network of commercial pathology laboratories from regional and rural practices. Specimens were tested at VIDRL using an in-house respiratory multiplex polymerase chain reaction (PCR), identifying influenza; adenovirus; picornavirus (enterovirus and rhinovirus); respiratory syncytial virus; and parainfluenza viruses.[3] In 2004, oligonucleotide primers to detect all known influenza viruses replaced primers aimed specifically at currently circulating H1 and H3 sub-types.

Melbourne Medical Locum Service

The MMLS is a 24-hour, seven days a week medical locum service for patients within an approximate 35 kilometre radius from central Melbourne. The MMLS provided data to VIDRL on the number cases with a final diagnosis reference to 'flu' or 'influenza'; the total number of call-outs each week; and ILI rates per 1,000 call-outs.

Notified laboratory-confirmed influenza

Medical practitioners and persons in charge of pathology services are required to notify cases of laboratory-confirmed influenza to the DHS within five days of the diagnosis under the *Health (Infectious Diseases) Regulations 2001*. In 2006, notifications were received from 10 laboratories. VIDRL accounted for the majority of the notifications (59%) followed by the Royal Children's Hospital (19%) and Monash Medical Centre (11%) laboratories. Data on cases with a notification date in 2006 were extracted for analysis from the DHS Notifiable Infectious Diseases Surveillance database.

Figure 1a. Distribution of sentinel surveillance sites in metropolitan Victoria

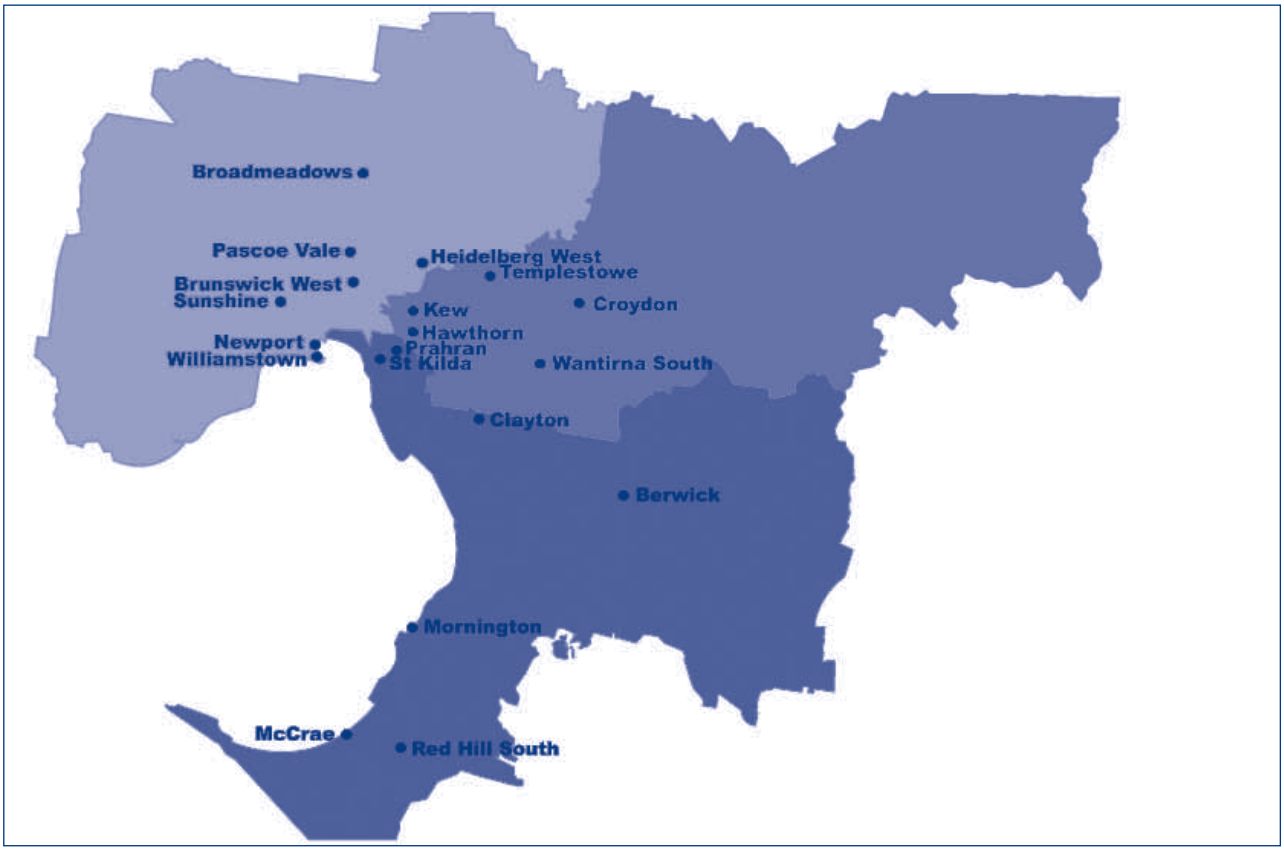
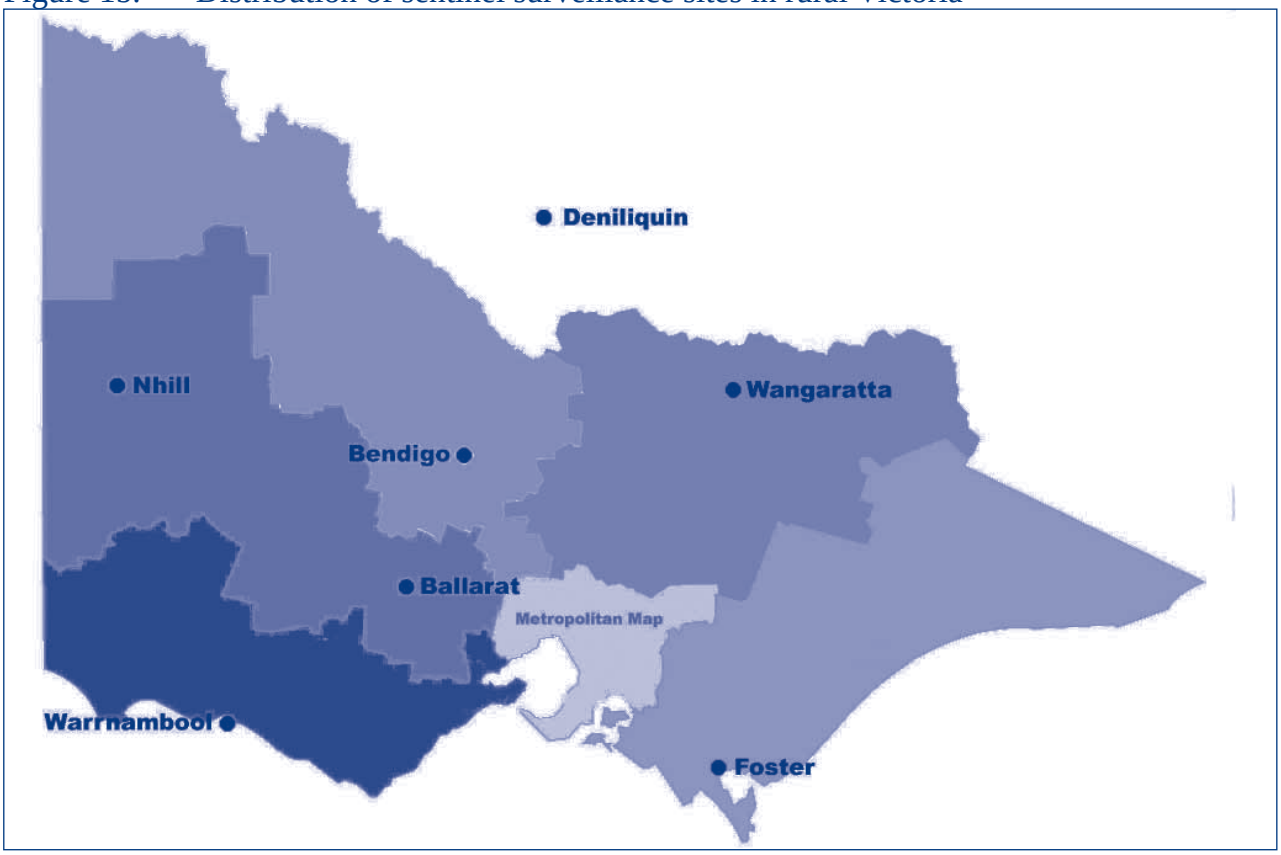


Figure 1b. Distribution of sentinel surveillance sites in rural Victoria



Data collation and reporting

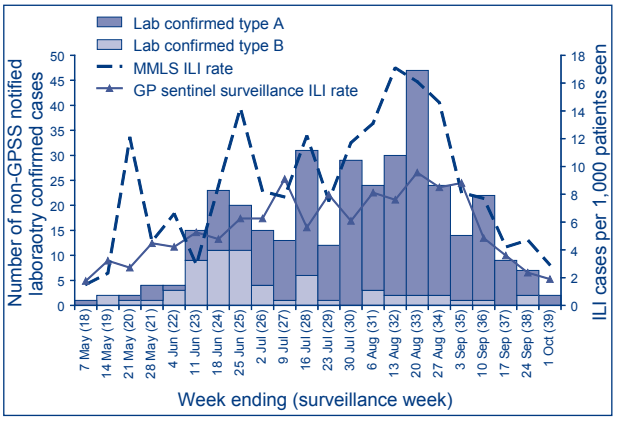
GP reports and MMLS data were collected and collated weekly. ILI surveillance data were reported to the DHS Communicable Disease Control Unit and the Australian Government Department of Health and Ageing weekly; structured summary reports were prepared and distributed fortnightly by email to GPs participating in the sentinel surveillance program, other interested health professionals and state and Australian Government departments of health. The summary reports were also posted on the VIDRL website (http://www.vidrl.org.au). Laboratory-confirmed influenza notifications data were updated daily and published on the DHS Communicable Disease Control Unit website (http://www.health.vic.gov.au/ideas/surveillance/daily.htm) in automated summary reports.

Results

ILI surveillance

An average of 50 of the 74 participating GPs (68%) returned the tally sheets each week (range 45 to 58). GPs reported seeing a total of 136,732 patients in the surveillance period with 765 reported to have an ILI (0.6% or an average rate of 5.6 cases per 1,000 consultations). The ILI rate generally increased over the surveillance period, peaking at 9.6 ILI cases per 1,000 consultations in week 33 (week ending 20 August)—corresponding with the peak in notified laboratory-confirmed cases—and decreasing to baseline levels by week 38 (week ending 24 September) (Figure 2). The average age of reported ILI cases was 33 years (range 0.5 to 83 years) and the male to female ratio was 1:1. There were 55 cases aged 65 years or older; 43 (78%) were reported as vaccinated.

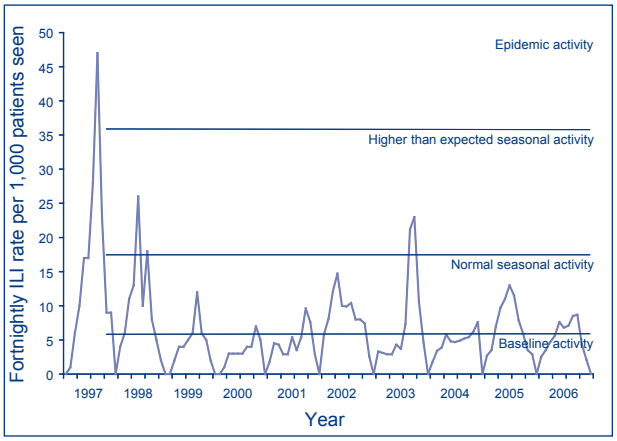
Figure 2. Notified cases of laboratory-confirmed influenza and GP sentinel surveillance and MMLS ILI rates by week, Victoria, 1 May to 1 October 2006



The MMLS ILI rate also showed a generalised increase over time, peaking at 17.1 ILI cases per 1,000 patients seen in week 32 (week ending 13 August) (Figure 2). The MMLS weekly ILI rates were generally higher—but tended to fluctuate more widely from week to week—compared to GP sentinel surveillance. The MMLS made 31,589 call-outs during the surveillance period. Of these, 268 were for an ILI; an average rate of 8.5 cases per 1,000 call-outs.

The 2006 influenza season (as measured by the GP sentinel surveillance ILI rate) was moderate relative to previous years (Figure 3). Rates were in the low to mid range of normal seasonal activity for most of the surveillance period and the peak rate was lower for only two other years (2000 and 2004) since 1997.

Figure 3. Fortnightly GP sentinel surveillance ILI rates, Victoria, 1997 to 2006



Laboratory surveillance

A total of 384 swabs were collected from GP sentinel surveillance patients with an ILI, of which 189 (49%) were positive for one of the respiratory viruses tested for in the multiplex PCR (Table). Almost 30% of total swabs (59% of those that were PCR positive for any virus) were positive for influenza A; 12% of swabs (25% of PCR positive swabs) were positive for picornavirus. Less than 10% were positive for other respiratory viruses, including influenza B. Most (85%) picornavirus diagnoses were made in the first half of the surveillance period whereas approximately three quarters of influenza A diagnoses were made between weeks 26 and 34 (26 June to 27 August). The other respiratory virus diagnoses were distributed throughout the surveillance period.

Table. Respiratory viruses detected from GP sentinel surveillance ILI patient swabs

Respiratory virus	n	Total swabs (%)	PCR positive swabs (%)
Influenza A	111	29	59
Influenza B	15	4	8
Picornavirus	47	12	25
Adenovirus	1	0.3	1
Parainfluenza virus	5	1	3
Respiratory syncytial virus	10	3	5
Total	189	49	100

A total of 408 cases of laboratory-confirmed influenza were notified to the Department of Human Services in 2006, of which 362 (89%) were made during the surveillance period from 1 May to 1 October. During this period, cases identified from GP sentinel surveillance comprised one-third of notifications and 11 cases (3%) were notified as a result of outbreak investigations. Laboratory-confirmed cases of influenza A showed a steady increase during the surveillance period before a sharp peak in week 33 (week ending 20 August), while influenza B notifications—which comprised only 18% of total influenza cases—peaked in weeks 24 and 25 (12 to 25 June) (Figure 2).

Stratification of laboratory-confirmed notifications by whether or not they were from sentinel surveillance sites showed little difference in trend for influenza type B notifications (Figure 4). However, for influenza type A notifications there was a bimodal distribution of notifications from GP sentinel surveillance with peaks in weeks 28 and 33 (weeks ending 16 July and 20 August respectively); notifications from other sources peaked in week 30 (week ending 30 July). The notification peaks from GP sentinel surveillance were mainly due to large numbers of notifications on single days: 16 cases on 10 July and 25 cases on 15 August. There was also a stark contrast in the age distributions for the different notification sources; 34% of cases notified from non-GP sentinel surveillance sites were aged less than 5 years and 55% were aged less than 20 years (Figure 5). The modal age group for GP sentinel surveillance laboratory-confirmed influenza cases was 35–39 years, although the distribution was much less skewed.

A total of 219 specimens were referred to the WHO Collaborating Centre for Reference and Research on Influenza in 2006; half were from VIDRL and the remainder from two metropolitan hospitals. Isolates

Figure 4. Laboratory-confirmed influenza by type and notification source, Victoria, 1 May to 1 October 2006

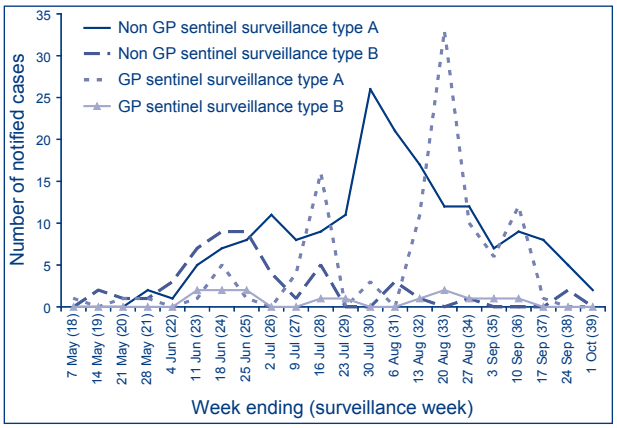
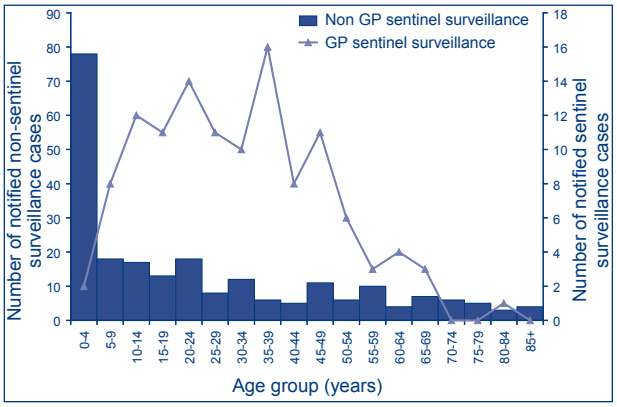


Figure 5. Laboratory-confirmed influenza by age group and notification source, Victoria, 1 May to 1 October 2006



from 135 specimens (62%) were recovered, of which 71% were influenza A and 29% were influenza B. Ninety-seven per cent of influenza A isolates were A/Wisconsin/67/2005-like (H3) and the remainder were A/New Caledonia/20/99-like (H1); all the influenza B samples were B/Malaysia/2506/2004-like. The influenza vaccine for 2006 contained A/New Caledonia/20/99(H1N1)-like virus, A/California/7/2004(H3N2)-like virus and B/Malaysia/2506/2004-like virus.[4]

Outbreak investigations

In 2006, the Communicable Disease Control Unit investigated two outbreaks of type A influenza infection in aged care facilities in July and September respectively. In the first outbreak there were 24 cases (6 laboratory confirmed) in 34 residents and staff, while in the second there was a total of 8 cases (5 laboratory confirmed) in 54 residents; all cases recovered.

Discussion

The 2006 influenza season in Victoria was relatively mild compared to previous years; ILI rates from GP sentinel surveillance remained within normal seasonal activity parameters. The seasonal peak appeared to occur in mid-August, with laboratory-confirmed notifications and ILI rates from GP sentinel surveillance peaking in week 33 and the ILI rate from the MMLS peaking in week 32 (weeks ending 20 and 13 August respectively). A rise in ILI rates as detected from community surveillance did not precede the rise in laboratory-confirmed notified cases as was observed in 2005.[5] There was greater variability in the MMLS ILI rate from week to week compared to the GP sentinel surveillance, although the reason for this is unclear. There was also some variability in the laboratory-confirmed influenza notifications—particularly in week 29 (week ending 23 July)—but this may be explained by the apparent batching of notifications from sentinel surveillance. When stratified, a single defined peak of notifications from non-sentinel surveillance sources occurred in week 30 (week ending 30 July)—which preceded the peaks in ILI rates for the MMLS and GP sentinel surveillance by two and three weeks respectively—whereas a bimodal peak in sentinel surveillance notifications was observed in weeks 28 and 33 (weeks ending 16 July and 20 August respectively).

The laboratory-confirmed surveillance data highlighted again in 2006 the different populations that are captured by the GP sentinel surveillance system and from other sources. Hospitals comprise a large proportion of non-sentinel surveillance notifiers and the proportion of notifications in the 0–4 year age group was also correspondingly high. This is most likely reflective of higher hospitalisation rates for influenza among this age group compared to older children[6] and better diagnostic follow-up for patients hospitalised with respiratory illness.

The notifications and strain typing data suggested two relatively distinct circulating influenza strains during 2006. The first half of the season was marked by circulation of influenza B, for which all typed isolates were B/Malaysia/2506/2004-like (and which was covered by the 2006 vaccine). From about week 26, influenza A/H3N2 became the dominant circulating subtype; nearly all typed influenza A viruses were A/Wisconsin/67/2005-like, which has been included in the 2007 vaccine. The recommended composition of influenza virus vaccines for use in the 2007 Southern Hemisphere influenza season is an A/New Caledonia/20/99(H1N1)-like virus, an A/Wisconsin/67/2005(H3N2)-like virus and a B/Malaysia/2506/2004-like virus.[7]

The GP Sentinel Surveillance Program is a cornerstone of Victoria's influenza surveillance and—as discussed above—provides much additional useful information about influenza in Victoria to that gained from passive notifiable laboratory-confirmed influenza surveillance. The system has several strengths. It has been shown to be useful for surveillance of established diseases using a small sample.[8] It is also a flexible system, defined as being able to adapt to changing information needs or operating conditions with little additional time, personnel or allocated funds.[9] The latter attribute will be demonstrated in 2007 with the addition of varicella-zoster virus (chickenpox and herpes zoster) to be added to the conditions under GP sentinel surveillance in Victoria.

Acknowledgements

We gratefully acknowledge the participation of general practitioners and their practice staff in the sentinel surveillance program; their support is critical to its success. We also thank others involved in the operation of the sentinel surveillance program, including the private pathology providers who facilitate transport of respiratory specimens from rural and regional general practices. Surveillance of laboratory-confirmed influenza is made possible through notifying laboratories (particularly the Viral Identification Laboratory at VIDRL) and medical practitioners throughout Victoria and the WHO Collaborating Centre for Reference and Research on Influenza which provides valuable influenza strain identification data. We also thank staff in the Communicable Disease Control Unit at Department of Human Services for data entry and follow-up of notifications data.

Author details

James E Fielding, Senior Epidemiologist[1]
Emma R Miller, Surveillance Coordinator/Epidemiologist[2]
Josie Adams, Director[3]
Bronwyn Hawking, Director[3]
Kristina Grant, Data Manager[4]
Heath A Kelly, Head[2]

1. Communicable Disease Control Unit, Department of Human Services, Melbourne, Victoria
2. Epidemiology Unit, Victorian Infectious Diseases Reference Laboratory, Melbourne, Victoria
3. Melbourne Medical Locum Service, Melbourne, Victoria
4. Victorian Infectious Diseases Reference Laboratory, Melbourne, Victoria

Corresponding author: Mr James E Fielding, Senior Epidemiologist, Communicable Disease Control Unit, Department of Human Services, 50 Lonsdale Street, Melbourne Victoria 3000. Telephone: +61 3 9096 5872. Facsimile: +613 9096 9174. Email: James.Fielding@dhs.vic.gov.au

References

1. Influenza Pandemic Planning Committee, Communicable Diseases Network Australia New Zealand. *A framework for an Australian Influenza Pandemic Plan.* Communicable Diseases Intelligence Technical Report Series No. 4. Canberra: Commonwealth Department of Health and Aged Care, June 1999.
2. Watts CG, Andrews RM, Druce JD, Kelly HA. Establishing thresholds for influenza surveillance in Victoria. *Aust N Z J Public Health* 2003;27:409–412.
3. Druce J, Tran T, Kelly H, Kaye M, Chibo D, Kostecki R, et al. Laboratory diagnosis and surveillance of human respiratory viruses by PCR in Victoria, Australia, 2002–2003. *J Med Virol* 2005;75:122–129.
4. World Health Organization. World Health Organization influenza vaccine recommendations for the Southern Hemisphere for 2006. Available from: http://www.influenzacentre.org/ Accessed on 9 January 2007.
5. Turner JL, Fielding JE, Clothier HJ, Kelly HA. Influenza surveillance in Victoria, 2005. *Commun Dis Intell* 2006;30:137–143.
6. Beard F, McIntyre P, Gidding H, Watson M. Influenza related hospitalisations in Sydney, New South Wales, Australia. *Arch Dis Child* 2006;91:20–25.
7. World Health Organization. Recommended composition of influenza virus vaccines for use in the 2007 Southern Hemisphere influenza season. Available from: http://www.who.int/csr/disease/influenza/recommendations-2007south/en/index.html Accessed on 9 January 2007.
8. Clothier H, Turner J, Hampson A, Kelly H. Geographic representativeness for sentinel influenza surveillance: implications for routine surveillance and pandemic preparedness. *Aust N Z J Public Health* 2006;30:337–341.
9. Centers for Disease Control and Prevention. Updated Guidelines for Evaluating Public Health Surveillance Systems. *MMWR Recomm Rep* 2001;50(RR–13);1–35.

Articles

Prevalence of antimicrobial resistances in common pathogenic Enterobacteriaceae in Australia, 2004: report from the Australian Group on Antimicrobial Resistance

Julie Pearson, John Turnidge, Clare Franklin, Jan Bell and the Australian Group on Antimicrobial Resistance

Abstract

Antibiotic resistance in 3 common pathogenic types of Enterobacteriaceae was examined in a point-prevalence study in 2004. Strains of *Escherichia coli*, *Klebsiella* and *Enterobacter* species were collected prospectively in 25 institutions in Australian capital cities and tested by broth microdilution to 12 β-lactams and 3 other antibiotics. Almost 22% of isolates tested were from blood cultures. In *E. coli*, acquired resistance to ampicillin and piperacillin was common (>40%), and clinically significant percentages of intermediate susceptibility and resistance (>8%) were observed to amoxycillin-clavulanate, cefazolin and trimethoprim. In *Klebsiella* species, clinically important acquired resistance (>8%) was seen to piperacillin, cephalothin and trimethoprim, while in *Enterobacter* species, this was found with piperacillin, ceftriaxone, ceftazidime and trimethoprim. Blood culture isolates had similar rates of resistance to isolates from other specimen sources. New South Wales/Australian Capital Territory (combined) tended to have higher percentages of resistance than the other states, which were otherwise comparable across the agents and species tested. Multi-resistance, defined as more than 3 acquired resistances to antibiotic classes, was found in 6.5% of *E. coli*, 8.3% in *Klebsiella* species and 16.9% of *Enterobacter* species. Co-resistance to ciprofloxacin, gentamicin and/or trimethoprim was common in isolates presumptively harbouring extended-spectrum ß-lactamases. Strains with extended-spectrum ß-lactamases, although common in other countries, appear to be at fairly low levels in Australia; less than 4% in *E. coli* and less than 9% in *Klebsiella* species. Rates in *Enterobacter* species were not able to be determined. Presumptive plasmid-borne AmpC ß-lactamases were seen at low levels across the country and carbapenemases have now been found for the first time in Australia in Enterobacteriaceae. Both of these types of resistance represent a significant threat to major last-line antibiotics. *Commun Dis Intell* 2007;31:106–112.

Keywords: antimicrobial resistance, *Escherichia coli,* Enterobacteriaceae*, Klebsiella*

Introduction

Emerging resistance in common pathogenic members of the Enterobacteriaceae is a world-wide phenomenon, and presents therapeutic problems for practitioners in the community and in hospital practice. The Australian Group on Antimicrobial Resistance (AGAR) commenced surveillance of the key Gram-negative pathogens, *Escherichia coli* and *Klebsiella* species in 1992. Surveys have been con-

ducted biennially since then. In 2004, another genus of Gram-negative pathogens in which resistance can be of clinical importance, *Enterobacter* species, was added. The objectives of the 2004 surveillance program were to:

- determine proportions of resistance to the main therapeutic agents in *Escherichia coli*, *Klebsiella* species and *Enterobacter* species in Australian institutions;
- examine the extent of co-resistance and multi-resistance in these species; and
- detect emerging resistance to newer last-line agents such as carbapenems.

All species surveyed are members of the family Enterobacteriaceae. This family contains the most important Gram-negative pathogens in a wide range of common conditions in both the community and in hospitals. The 3 groups surveyed are considered to be valuable sentinels for multi-resistance and emerging resistance.[1,2]

Resistances of particular interest include resistance to ß-lactams due to ß-lactamases, especially extended-spectrum ß-lactamases, which inactivate the reserve agents, the third-generation cephalosporins. Other resistances of interest include resistance to antibiotics commonly used in the community such as trimethoprim; resistance to agents important for serious infections, such as gentamicin; and resistance to reserve agents such as ciprofloxacin and meropenem.

Methods

Institutions

Twenty-five institutions from each state and territory of Australia participated in the Gram-negative 2004 AGAR survey. There were 9 institutions from New South Wales/Australian Capital Territory, 5 from Victoria/Tasmania, 4 from Queensland, 4 from Western Australia and 3 from South Australia. Each institution collected up to 25 *E. coli*, 25 *Klebsiella* species and 50 *Enterobacter* species from different patients. Limits were placed on specimen types for *E. coli* and *Klebsiella* species in order to maximise the number of isolates from more serious infections, such as bacteraemia.

Laboratory methods

Participating laboratories were required to meet standards for species identification. Susceptibility testing was performed using custom made panels prepared commercially by Dade-Behring Microscan® (West Sacramento, CA). The following agents were tested: ampicillin, amoxycillin-clavulanate, piperacillin, piperacillin-tazobactam, cephalothin, cefazolin, cefoxitin, ceftriaxone, ceftazidime, cefepime, aztreonam, meropenem, ciprofloxacin, gentamicin and trimethoprim. Interpretive criteria for susceptibility were those recommended by the Clinical and Laboratory Standards Institute (CLSI).[1] *E. coli* ATCC® 25922 and *E. coli* ATCC® 35218 were used for quality control strains. The presence of extended-spectrum ß-lactamases was tested for using published CLSI and other methods.[3,4]

Results

Source of isolates

The majority of isolates were from urine. There were 596 isolates of *E. coli*, 590 *Klebsiella* species and 1,204 *Enterobacter* species. Twenty-two per cent of isolates overall were from blood cultures. More than one third (35.2%) of the *E. coli* isolates were from blood cultures, with lower percentages for *Klebsiella* (24.7%) and *Enterobacter* species (13.2%). Urinary isolates accounted for almost half of the isolates (42.7%). Other sites of isolation reflected the high incidence of these species in nosocomial and pre- and post-operative surgical infections.

Susceptibility testing results

A summary of resistance rates for all antibiotics against the 3 groups of bacteria are shown in Tables 1 to 3. There were no significant differences between the states for any of the agents tested, with the exception of New South Wales/Australian Capital Territory which has higher percentages of acquired resistance to many drug classes in *Enterobacter* species compared to the other states. Furthermore, isolates causing bacteraemia had similar percentages of resistance to isolates from other specimen sources (data not shown).

Escherichia coli

Ampicillin resistance proportions have been high for many years at around 45%; we found 46.5% were resistant in 2004. Amoxycillin-clavulanate intermediate and resistant strains were a relatively low proportion at 8.4% (range 4.8–12.0%). Percentages of resistance to piperacillin and piperacillin-tazobactam reflected those of ampicillin and amoxycillin-clavulanate respectively. Cephalothin is known to have marginal activity against *E. coli*, while intermediate + resistant percentages to cefazolin resembled those of amoxycillin-clavulanate. The ciprofloxacin resistance proportion was less than 4%. Gentamicin resistance was fairly low but there was a moderate percentage of resistance to trimethoprim (Table 1).

Klebsiella species

Ampicillin resistance is normal in these species due to the presence of specific chromosomal ß-lactamases.

The percentage of resistance to amoxycillin-clavulanate and piperacillin-tazobactam was low (<5%). Percentages were substantially higher for first-generation cephalosporins cephalothin and cefazolin (~25%). Resistance to gentamicin was low, although higher than for *E. coli*. Resistance to ciprofloxacin and trimethoprim was less common than in *E. coli* (<2% and <10% respectively) (Table 2).

Enterobacter species

Ampicillin, amoxicillin-clavulanate and first-generation cephalosporins are generally considered inactive against *Enterobacter* species due to intrinsic chromosomal β-lactamases. Resistance to gentamicin was more common than that seen in *E. coli* and *Klebsiella* species. About 25% of strains were resistant to third-

Table 1. *Escherichia coli* (n = 596)

Antibiotic	Cat* %	NSW/ACT %	Qld/NT %	SA %	Vic/Tas %	WA %	Australia %
Ampicillin	R	50.2	52.7	49.3	33.6	47.0	46.5
Amoxycillin-clavulanate	I	6.8	8.8	5.3	3.2	10.0	6.7
	R	2.4	1.1	0.0	1.6	2.0	1.7
Piperacillin	R	42.0	44.0	45.3	28.8	44.0	40.3
Piperacillin-tazobactam	R	2.0	1.1	0.0	0.0	2.0	1.2
Cephalothin	I	27.8	20.9	36.0	36.8	21.0	28.5
	R	23.9	25.3	38.7	16.0	33.0	25.8
Cefazolin	I	2.9	1.1	6.7	0.0	10.0	3.7
	R	6.3	11.0	14.7	4.0	9.0	8.1
Ceftriaxone	NS	1.5	1.1	1.3	1.6	2.0	1.5
Ceftazidime	NS	1.5	0.0	1.3	1.6	1.0	1.2
Cefepime	NS	1.5	0.0	0.0	0.8	1.0	0.8
Meropenem	NS	0.0	0.0	0.0	0.0	0.0	0.0
Ciprofloxacin	NS	2.9	4.4	2.7	1.6	6.0	3.4
Gentamicin	R	2.4	3.3	2.7	0.0	4.0	2.3
Trimethoprim	R	18.0	23.1	10.7	9.6	21.0	16.6

* Category: R = resistant, I = intermediate, NS = not susceptible (intermediate + resistant).

Table 2. *Klebsiella* species (n = 590)

Antibiotic	Cat* %	NSW/ACT %	Qld/NT %	SA %	Vic/Tas %	WA %	Australia %
Ampicillin	R	82.2	97.9	86.5	74.8	81.4	83.6
Amoxycillin-clavulanate	I	4.0	2.1	6.8	2.4	4.1	3.7
	R	0.5	0.0	2.7	2.4	0.0	1.0
Piperacillin	R	23.8	35.1	43.2	13.8	14.4	24.4
Piperacillin-tazobactam	R	5.0	1.1	8.1	4.1	3.1	4.2
Cephalothin	I	7.9	3.2	10.8	9.8	8.2	8.0
	R	15.8	18.1	27.0	19.5	15.5	18.3
Cefazolin	I	4.5	2.1	5.4	13.8	4.1	6.1
	R	15.3	10.6	32.4	19.5	18.6	18.1
Ceftriaxone	NS	7.4	5.3	12.2	4.9	6.2	6.9
Ceftazidime	NS	3.5	3.2	5.4	4.1	2.1	3.6
Cefepime	NS	3.0	0.0	5.4	1.6	1.0	2.2
Meropenem	NS	0.0	0.0	0.0	0.8	0.0	0.2
Ciprofloxacin	NS	4.0	0.0	0.0	1.6	1.0	1.9
Gentamicin	R	1.5	6.4	8.1	5.7	2.1	4.1
Trimethoprim	R	9.9	10.6	10.8	8.1	9.3	9.7

* Category: R = resistant, I = intermediate, NS = not susceptible (intermediate + resistant).

generation cephalosporins, similar to proportions seen in the past. Levels of resistance to ciprofloxacin and trimethoprim were similar to those observed with the other two species (Table 3).

Extended-spectrum and plasmid-borne AmpC β-lactamases

The prevalence of presumptive extended-spectrum and plasmid-borne β-lactamases is shown in Table 4. Strains with minimum inhibitory concentrations (MIC) of ceftriaxone and/or ceftazidime above 1 mg/L were considered presumptive evidence of the presence of an extended-spectrum or related β–lactamase in *E. coli* and *Klebsiella* species. A cefepime MIC above 0.5 mg/L was considered presumptive of an extended-spectrum β-lactamase in *Enterobacter* species. About half of these were confirmed to be extended-spectrum ß-lactamases by the clavulanate enhancement test.

Plasmid-borne AmpC ß-lactamases are suspected when *E. coli* or *Klebsiella* species are not susceptible to cefoxitin. Three per cent of *E. coli* and 6.6% of *Klebsiella* species were not susceptible to cefoxitin (MIC >8 mg/L).

Carbapenemases

One strain of *Klebsiella pneumoniae* and 5 strains of *Enterbacter cloacae* were not susceptible to meropenem. The strains came from 4 institutions in 2 states. In 3 of these 6 strains, the presence of a carbapenemase (metallo-β-lactamase) was confirmed by molecular methods. These are among the first known recordings of metallo-β-lactamases in Australia.[5]

Multi-resistance

The rates of multiple resistances are shown in Table 5. Multi-resistance was defined as resistance to three or more classes of antibiotics to which the genus/species has no natural resistance. Multi-resistance was reasonably common, above 5% in all 3 species groups. It was above 15% for *Enterobacter* species.

Discussion

Amongst the most troublesome international trends in resistance in Enterobacteriaceae has been the emergence of extended-spectrum ß-lactamases (ESBLs), all of which are plasmid-borne. They have been predominantly a problem in hospital practice, and initially were more common in *Klebsiella* species than in *E. coli*. Recently, two new trends have emerged: the presence of ESBLs in *Enterobacter* species, and the emergence of specific types of ESBLs (so-called CTX-M enzymes). ESBLs are important as they compromise the efficacy of third-generation cephalosporins, which have been such a useful therapeutic alternative in hospital practice. Outbreaks of ESBL-producing *Klebsiella* species and *E. coli* have led some hospitals in Australia to severely restrict or abandon third-generation cephalosporin use. Overall ESBL rates in Australia remain low when compared to many other countries.[6] At least

Table 3. *Enterobacter* species (N = 1204)

Antibiotic	Cat* %	NSW/ACT %	Qld/NT %	SA %	Vic/Tas %	WA %	Australia %
Ampicillin	R	87.2	86.1	92.7	83.6	83.8	86.5
Amoxycillin-clavulanate	I	23.1	24.1	20.0	23.4	25.8	23.3
	R	70.9	67.5	76.0	70.5	68.2	70.5
Piperacillin	R	27.6	17.5	25.3	14.3	16.2	21.3
Piperacillin-tazobactam	R	7.0	4.2	8.0	4.5	6.1	6.1
Cephalothin	I	0.4	0.6	0.0	0.8	1.5	0.7
	R	98.2	98.8	100.0	98.0	96.0	98.1
Cefazolin	I	0.9	24.7	4.7	1.2	3.0	5.1
	R	94.4	72.9	93.3	95.1	93.9	91.4
Ceftriaxone	NS	28.9	20.5	28.0	20.5	24.2	25.2
Ceftazidime	NS	30.0	19.3	26.0	21.7	23.7	25.3
Cefepime	NS	6.1	3.0	10.0	4.9	2.5	5.3
Meropenem	NS	0.9	0.0	0.0	0.4	0.0	0.4
Ciprofloxacin	NS	4.5	0.6	0.7	0.8	1.0	2.2
Gentamicin	R	13.7	7.8	6.7	2.0	0.5	7.5
Trimethoprim	R	20.0	12.0	9.3	6.1	8.6	12.9

* Category: R = resistant, I = intermediate, NS = not susceptible (intermediate + resistant).

Table 4. Presumptive/confirmed extended-spectrum ß-lactamase production

Species and antibiotic	NSW/ACT %	Qld/NT %	SA %	Vic/Tas %	WA %	Australia %
E. coli						
Ceftriaxone >1	2.4	3.3	1.3	1.6	2.0	2.2
Ceftazidime >1	2.9	4.4	2.7	1.6	2.0	2.7
Aztreonam >8	2.9	2.2	1.3	1.6	1.0	2.0
Any of above	4.9	6.6	2.7	1.6	2.0	3.7
Confirmed	1.5	1.1	1.3	0.8	1.0	1.2
***Klebsiella* spp.**						
Ceftriaxone >1	8.4	6.4	13.5	6.5	6.2	8.0
Ceftazidime >1	5.0	5.3	6.8	5.7	4.1	5.3
Aztreonam >8	8.4	6.4	10.8	4.1	5.2	6.9
Any of above	9.4	6.4	13.5	7.3	8.2	8.8
Confirmed	6.9	4.3	8.1	4.9	2.1	5.4
***Enterobacter* spp.**						
Cefepime >0.5	23.5	15.7	21.3	13.1	11.6	18.1

Table 5. Multi-resistance: counts of numbers of strains versus the number of antibiotic classes to which genus/species was resistant

Species	Non-multi-resistant (Number of antibiotic classes)					Multi-resistant (Number of antibiotic classes)											
	0	1	2	3	%	4	5	6	7	8	9	10	11	12	13	14	%
*E. coli**	290	42	157	68	93.5	21	7	4		4	2				1		6.5
Klebsiella†	415	65	46	15	91.7	10	9	17	6	3	1	3					8.3
Enterobacter‡	787	72	80	61	83.1	65	43	53	28	12	2	1					16.9

* *Antibiotics included*: ampicillin, piperacillin, amoxycillin-clavulanate, piperacillin-tazobactam, cefazolin, cefoxitin, ceftriaxone, ceftazidime, cefepime, aztreonam, meropenem, ciprofloxacin, gentamicin, trimethoprim
Antibiotics excluded: cephalothin.

† *Antibiotics included*: piperacillin, amoxycillin-clavulanate, piperacillin-tazobactam, cephalothin, cefazolin, cefoxitin, ceftriaxone, ceftazidime, cefepime, aztreonam, meropenem, ciprofloxacin, gentamicin, trimethoprim
Antibiotics excluded: ampicillin.

‡ *Antibiotics included*: piperacillin, piperacillin-tazobactam, ceftriaxone, ceftazidime, cefepime, ciprofloxacin, gentamicin, trimethoprim *Antibiotics excluded*: ampicillin, amoxycillin-clavulanate, cephalothin, cefazolin, cefoxitin

some of this may be attributed to fairly tight controls on third-generation cephalosporin use, both in Australian hospitals and in the community. Where hospital prolonged outbreaks have occurred, control has usually been achieved by switching to a ß-lactamase inhibitor combination for the most common indications. The methods used in our study for detecting ESBLs are considered indicative only. The diversity of enzymes means that molecular methods are required to supplement our knowledge of which enzymes are circulating.

Based on the tests performed in this study, ESBLs appear most common in *Enterobacter* species, but the study design did not lend itself to determining the true prevalence of ESBLs in these species. Molecular studies would be required to confirm their presence in *Enterobacter* species because a considerable amount of resistance to third-generation cephalosporins in this species results from stable de-repression of the natural chromosomal cephalosporinase (of the AmpC type). ESBLs are more common in *Klebsiella* species than in *E. coli;* a situation that has prevailed in Australia since their original emergence here. The lower rate of confirmation of ESBLs suggests that current routine screening methods are inadequate because the extended range of substrates used in this study and the screening concentrations used are more likely to detect true ESBLs.

Plasmid-borne AmpC ß-lactamases have recently emerged internationally as a growing Gram-negative resistance problem. They are the result of mobilisation of natural chromosomally located genes from uncommon species onto transmissible plasmids and into the common pathogens. Already there are 6 separate classes. Like ESBLs these enzymes confer resistance to the important third-generation cephalosporins. Routine detection methods have not yet been effectively developed. Nevertheless, it is possible to exploit a special feature of these enzymes; their ability to inactivate the cephamycins, represented by cefoxitin. *Enterobacter* species already naturally possess AmpC enzymes: their chromosomal cephalosporinases. These enzymes are present in Australia, but their exact prevalence in unknown.[7]

Acquired carbapenemases, in particular metallo-ß-lactamases, were first described in *Pseudomonas aeruginosa* and *Acinetobacter baumannii*. They are now being seen for the first time among members of the Enterobacteriaceae, and some were detected in this study. These are among the first known incidences of metallo-ß-lactamases in Australia. These are particularly troublesome resistance mechanisms as they inactivate a broad range of ß-lactams including the last-line carbapenems. Recent experience in one institution has shown that this resistance mechanism can spread amongst a range of Gram-negatives.[5]

The most problematic Gram-negative pathogens are those with multiple acquired resistances. Although there is no agreed benchmark for the definition of multi-resistance in Enterobacteriaceae, we have chosen acquired resistance to more than 3 classes to define multi-resistance in our survey. For each species, antibiotics were excluded from the count if they were affected by natural resistance mechanisms, so that only true acquired resistances were included. For the purposes of this analysis, resistance included intermediate susceptibility when the tested range did not go beyond the susceptible category.

It is clear that multi-resistance is more common in *Enterobacter* species. Some clustering of resistance is also noticeable in *Klebsiella* and *Enterobacter* species, as acquired resistance to 6 agents was more common than acquired resistance to 5 agents.

Although this study is comprehensive in its coverage of Australia, and the methodology follows international standards, there are a small number of limitations to the data and its interpretation.

1. The data are not denominator controlled. There is currently no consensus on an appropriate denominator for such surveys. Institution size, throughput, patient complexity and local antibiotic use patterns very much determine the types of resistance likely to be observed. As such, simple denominators such as occupied bed days over the period of collection would not provide meaningful comparisons between institutions.

2. Apart from blood cultures and sterile site isolates, the clinical significance of the isolates cannot be ascertained with certainty. Every attempt has been made by the participating laboratories to ascertain the clinical significance of isolates; however, the laboratories are dependent on (sometimes very limited) clinical information supplied on request forms. Gathering detailed clinical information sufficient to make a judgment on significance would require much greater resources than were available for this survey.

3. Molecular analyses for resistances of importance were not undertaken. They would be of greatest interest in the emerging resistances: ESBLs, plasmid-borne AmpC β-lactamases and carbapenemases. The major role of interspecies transfer of these types of resistance genes means that molecular characterization of these 3 enzyme types should be planned for in future surveys.

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

Acknowledgements

This study was fully supported by a grant from the Australian Government Department of Health and Ageing.

The participating laboratories were from: Concord Hospital, Douglass Hanly Moir, John Hunter Hospital, Royal North Shore Hospital, Royal Prince Alfred Hospital, South West Area Pathology Service, Nepean Hospital, The Canberra Hospital, Westmead Hospital, The Alfred Hospital, Gribbles Pathology (Victoria), Royal Children's Hospital (Melbourne), Royal Hobart Hospital, St Vincent's Hospital (Melbourne), Princess Alexandra Hospital QHPS, Royal Brisbane Hospital QHPS, Royal Darwin Hospital, Sullivan Nicolaides Pathology (Brisbane), Fremantle Hospital PathWest, Queen Elizabeth II Medical Centre PathWest, Royal Perth Hospital PathWest, St John of God Hospital (Perth), Flinders Medical Centre, Institute for Medical and Veterinary Science, Women's and Children's Hospital.

Author details

Julie Pearson, Scientific Officer[1]
John Turnidge, Director[2]
Clare Franklin, Senior Scientist[3]
Jan Bell, Senior Scientist[4]

1. Australian Group on Antimicrobial Resistance, Department of Microbiology and Infectious Diseases, PathWest Laboratory Medicine, Royal Perth Hospital, Western Australia
2. Division of Laboratory Medicine, Women's and Children's Hospital, North Adelaide, South Australia
3 Microbiology Department, Alfred Hospital, Prahran, Victoria
4. Department of Microbiology and Infectious Diseases, Women's and Children's Hospital, North Adelaide, South Australia

Corresponding author: Prof. John Turnidge, Division of Laboratory Medicine, Women's and Children's Hospital, 72 King William Road, North Adelaide, SA 5062. Telephone: +61 8 8161 6873. Facsimile: +61 8 8161 6189. Email: john.turnidge@cywhs.sa.gov.au

References

1. Paterson DL. Resistance in Gram-negative bacteria: Enterobacteriaceae. *Am J Infect Control* 2006;35 Suppl 1:S20–S28.
2. Clinical and Laboratory Standards Institute. Performance standards for antimicrobial susceptibility testing. 16th informational supplement. M100–S16. CLSI, Wayne, Pa, 2006.
3. Clinical and Laboratory Standards Institute. Methods for Dilution Antimicrobial Susceptibility Tests for Bacteria That Grow Aerobically; Approved Standard – Seventh Edition. M7–A7. CLSI, Wayne, Pa, 2006
4. Bell JM, Turnidge JD, Jones RN; SENTRY Asia-Pacific Participants. Prevalence of extended-spectrum beta-lactamase-producing *Enterobacter cloacae* in the Asia-Pacific region: results from the SENTRY Antimicrobial Surveillance Program, 1998 to 2001. *Antimicrob Agents Chemother* 2003;47:3989–3993.
5. Peleg AY, Franklin C, Bell JM, Spelman DW. Dissemination of the metallo-beta-lactamase gene blaIMP-4 among gram-negative pathogens in a clinical setting in Australia. *Clin Infect Dis* 2005;41:1549–1556.
6. Hirakata Y, Matsuda J, Miyazaki Y, Kamihira S, Kawakami S, Miyazawa Y, et al. SENTRY Asia-Pacific Participants. Regional variation in the prevalence of extended-spectrum beta-lactamase-producing clinical isolates in the Asia-Pacific region (SENTRY 1998–2002). *Diagn Microbiol Infect Dis* 2005;52:323–329.
7. Bell JM, Chitsaz M, Turnidge JD, Barton M, Walters LJ, Jones RN. Prevalence and significance of a negative extended-spectrum ß-lactamase (ESBL) confirmation test after a positive ESBL screening test among *Escherichia coli* and *Klebsiella pneumoniae*: Results from the SENTRY Asia-Pacific surveillance program. *J Clin Microbiol* In press.

Public health management of increased incidence of meningococcal disease in the Australian Capital Territory: 2003 to 2004

Geetha Isaac-Toua, Charles Guest, Rona Hiam, Irene Passaris

Abstract

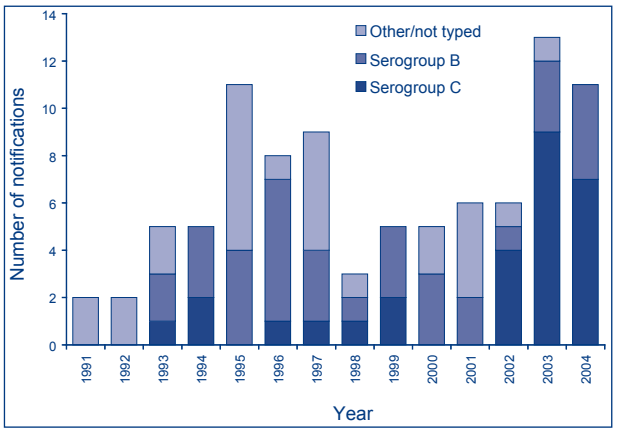
This paper describes a sudden increase of meningococcal notifications in the Australian Capital Territory within a 3 month period, and the public health strategies used to manage it. There were 15 cases of meningococcal disease notified to the Communicable Disease Control (CDC) section, Australian Capital Territory Health (ACT Health), between 6 November 2003 and 5 February 2004. This was much higher than the annual average of 6 cases. The cases were notified in 2 clusters. The first cluster of 8 cases, all serogroup C, was notified between 6 November to 8 December 2003. Seven of these cases had an identical phenotype C:2a:P1.4 suggesting a common source. The second cluster of 7 cases was notified between 30 December 2003 and 5 February 2004. Of these, 5 were serogroup B, 1 was serogroup W-135 and 1 was serogroup C, whose phenotype (C:2a:P1.4) was identical to the phenotype of the first cluster of serogroup C cases. Phenotypes were not available for the serogroup B cases. There were 4 main interventions developed to manage the increased incidence based on the epidemiology of the cases; these were implemented concurrently. Factors that supported investigation and management were good surveillance systems, quick turnover of laboratory tests, regular communication with relevant health agencies and maintaining public awareness. As the number of cases notified was much higher than the annual average, the possibility of a community outbreak was considered. The *Guidelines for the Early Clinical and Public Health Management of Meningococcal Disease in Australia* (national guidelines) were consulted to determine whether there was an outbreak and the influence this had on management is also discussed. *Commun Dis Intell* 2007;31:112–118.

Keywords: disease surveillance, meningococcal disease, outbreak, public health management, *Neisseria meningiditis,* prevention and control

Introduction

The average notification rate of meningococcal disease (including all serogroups) in the Australian Capital Territory between 1991 and 2002 was 1.9 cases per 100,000 population per year, with an annual average of 6 cases (range 2–11). The Australian average notification rate per year for the same period was 3.1 cases per 100,000 population.[1] The majority of notifications in the Australian Capital Territory were of serogroup B until 2002 when an increase in serogroup C cases was noted. This trend occurred Australia wide. All notifications in the Australian Capital Territory between 1991 and 2004 are presented in the Figure, with serogroup B and C cases highlighted. The obvious increase in notifications in 2003 and 2004 includes the cases discussed in this paper.

Figure. Notifications of meningococcal disease, Australian Capital Territory, 1991 to 2004



All cases of meningococcal disease in the Australian Capital Territory are notified to the Communicable Disease Centre, ACT Health. Close contacts as defined by the national guidelines[1] are traced and offered clearance antibiotics to eliminate carriage of the bacteria to prevent further transmission.

Free meningococcal serogroup C vaccine for certain age and risk groups became available in the Australian Capital Territory in early 2003 as part of the four-year National Meningococcal C Vaccination Program.[2] All school attendees aged 15–19 years were offered free vaccine as part of this program through special immunisation clinics run between August and December 2003. The program for non-school attendees aged 15–19 years was planned to commence from mid-December 2003. As of 2004, meningococcal serogroup C vaccine has been made available through general practitioners (GPs) for all children and young adults who turned 6 to 19 years in 2003.

Methods

Interviews

All cases of meningococcal disease notified during the period of increased incidence were interviewed within 24 hours of notification for identification of close contacts, epidemiological links and common risk factors as per the national guidelines.[1] Close contacts were offered clearance antibiotics to eliminate carriage of the bacteria to prevent further transmission, and if eligible, were offered meningococcal serogroup C vaccination.

Laboratory diagnosis

Initial diagnosis of meningococcal disease was conducted in the Australian Capital Territory by meningococcal specific polymerase chain reaction (PCR) testing[3] and serogrouping was done in Sydney by the New South Wales National Neisseria Network Reference Laboratory.[4] Serotyping and serosubtyping to determine phenotype were done by the South Western Sydney Area Pathology Services.[5,6] Samples that were PCR positive but culture negative were sent to the Melbourne Diagnostic Unit for genotyping to establish serotype and serosubtype.[7] Cases need to have the same microbiological classification established through phenotype to be included in the determination of outbreak notification rate as per the national guidelines.[1]

Results

There were 15 cases of meningococcal disease, notified in 2 clusters to CDC, ACT Health between 6 November 2003 and 5 February 2004. The first cluster was notified between 6 November and 8 December and the second cluster was notified between 30 December 2003 and 5 February 2004.

The first cluster of 8 cases were all of serogroup C. Seven of these cases were Australian Capital Territory residents while the eighth case was a New South Wales resident who worked as a taxi driver in the Australian Capital Territory. The first 4 cases were notified within a period of 6 days between 6 and 12 November. Two of these cases were notified on the same day. The remaining 4 cases were notified between 24 November and 5 December 2003. The 8 cases had an age range of 3–58 years (median age 18.5 years) with a male to female ratio of 1:1. Four cases were aged between 15–19 years. Three cases were not attending school and none were immunised against meningococcal serogroup C disease.

Phenotyping was done for the 7 Australian Capital Territory cases with all 7 returning an identical phenotype of C:2a:P1.4 suggesting the possibility of a common source. Interviews with the 8 cases did not reveal a common contact, but 5 of the cases

had visited the Australian Capital Territory Central Business District (CBD) during their incubation period, and 2 persons aged 15–19 years had visited the same group of nightclubs within the CBD.

The second cluster of 7 cases notified within 25 days of the first cluster, comprised 5 serogroup B cases, 1 serogroup C case and 1 serogroup W-135 case. The serogroup C case had the same phenotype as the 7 Australian Capital Territory cases in the first cluster and also had a definite epidemiological link to one of the cases in the first cluster. Phenotyping was not available for the serogroup B cases. Investigation and interviews with the 5 serogroup B cases did not reveal any common contact or epidemiological link. The W-135 serogroup case had been in contact with a family member who had returned from an area endemic for this serogroup[8] and was assumed to be part of the background Australian Capital Territory rate.

Public health management

Public health investigation and management commenced with the notification of the first 4 serogroup C cases of the first cluster. After extensive discussions with relevant agencies within and outside of ACT Health, 4 main interventions were developed and instituted concurrently with the aim of minimising transmission and preventing further cases. The response was coordinated by CDC, ACT Health, with continuing advice from the Infectious Diseases Unit of The Canberra Hospital and the Communicable Diseases Network Australia (CDNA).

Intervention 1: Free immunisation for all 15–19 year olds

At the time of the notifications, the establishment of immunisation clinics in the Australian Capital Territory as part of National Meningococcal C Vaccination Program[2] for school students aged 15–19 years, was close to completion. The program for non-school attendees aged 15–19 years was to commence within 2 weeks in mid-December. As the majority of notifications were amongst persons aged 15–19 years who were not in school, a decision was made to bring forward the commencement of the program for this group, and continue the program for persons aged 15–19 years in school. Free vaccine was made available for both groups through the special school immunisation clinics already in progress; through existing child immunisation clinics; and extended to general practices. The main emphasis was to promote immunisation through general practice due to impending school holidays and the closure of school immunisation clinics.

Intervention 2: Poster campaign

A poster targeting persons aged 15–19 years was developed to heighten awareness of the disease and to encourage uptake of the free vaccine. The poster was distributed to all nightclubs and other venues commonly accessed by this age group within the CBD, as there appeared to be a geographical link with this area.

Intervention 3: Media releases

There were several media releases from the Chief Health Officer during this period to increase public awareness of symptoms and availability of immunisation.

Intervention 4: Information/communication to relevant groups

The Medical Director of CDC, ACT Health updated relevant agencies, including the CDNA; CDC; and other public health units, on a regular basis through a series of email alerts. To increase sensitivity for diagnosis of meningococcal disease, general practices and hospitals within the Australian Capital Territory and surrounding regions were sent information regarding signs and symptoms and clinical management.

These interventions continued through the month after the notification of the first cluster of cases and were also used for the second cluster of cases.

Discussion

The only common risk factor identified through interviews with the first cluster of cases was accessing the Australian Capital Territory CBD and some common nightclubs in the CBD during the incubation period. The association with nightclubs was considered important as in 3 previous Australian meningococcal disease outbreaks, nightclubs were identified as a potential common source.[9–11] Whether there was a need to identify an ongoing common source to target interventions more specifically was discussed. The possibility of conducting a second level of interviews with close contacts of cases for this purpose was considered, particularly in regard to the older cases and the case aged less than 5 years. However, this option would have been resource intensive and would most likely have not changed public health management. It was therefore decided not to pursue this course.

It has been noted that maintaining high community awareness is important in the management of clus-

ters and outbreaks in order to allay public concern and to assist with the implementation of planned control measures such as a mass immunisation campaign.[1,12–14] Regular email alerts to relevant health professionals were aimed at keeping them informed, increasing sensitivity for early clinical diagnosis and management, and promoting immunisation in persons aged 15–19 years. The poster campaign and regular media alerts were aimed at raising public awareness, promoting health care seeking behaviour and increasing vaccine uptake in persons aged 15–19 years. As most of the cases were out of school and the school year was close to completion, specific targeting was not done through the school system. Locations considered to be associated with the outbreak and to be frequented by this age group were targeted instead.

There was extensive media coverage of 2 serogroup C cases, 1 of which was fatal while the other had partial amputation of a lower limb. This would most likely have heightened public awareness to the increased incidence and consequences of meningococcal disease. CDC, ACT Health received many phone calls from worried parents of the age group most affected, which suggests awareness of the situation. The impact that the poster campaign had on decreasing the incidence in the 15–19 age group is also difficult to estimate, as this could only be evaluated by conducting a survey amongst that age group.

A quick turnover of initial laboratory results with early confirmation of diagnosis, assisted with timely and appropriate public health management. The availability of phenotyping was a key factor in the decision making processes in relation to investigation and management, particularly with the first cluster of serogroup C cases with an identical phenotype, which suggested a common source. The importance of phenotyping was highlighted in the investigation and management of 2 of 3 outbreaks associated with nightclubs.[9,11] Six of 10 cases in a New South Wales outbreak with the same phenotype C:2a:P1.5, were linked to the same nightclub in Western Sydney,[11] while 4 of 5 notified cases of phenotype C:2a:P1.4 in a Victorian outbreak were associated with a nightclub in Portland.[9] Phenotyping for the second cluster of serogroup B cases may have been useful in establishing if they were linked as interviews did not reveal any common source or risk factors.

The 8 cases of meningococcal serogroup C disease (7 Australian Capital Territory cases and 1 New South Wales case) notified in the 1 month between November and December 2003 suggested the possibility of a community outbreak of meningococcal disease. There was extensive discussion on whether the increased notification rate constituted a community outbreak as per national guidelines. The national guidelines[1] define a community outbreak as '3 or more confirmed cases within a three-month interval, where the available microbiological characterisation of the organisms is the same, and incidence at least 10 cases per 100,000 total community population in the three-month interval'. Age group specific or social group specific notification rates can also be calculated, and are recommended[15,16] to establish if there is a problem in a specific population group, so that interventions can be tailored to that group.

There are 2 types of meningococcal disease outbreaks that can be declared as per the national guidelines,[1] one being a community outbreak and the other an organisational outbreak. An organisational outbreak is within a grouping of people that makes epidemiological sense, such as work colleagues within an organisation, school students or class mates and soldiers in military barracks. The threshold to declare an organisational outbreak is much lower than a community outbreak, and is defined as 'two or more probable cases with onset in a four-week interval in a grouping which makes epidemiological sense; or two or more confirmed cases with onset in a four week interval where the available microbiological characterisation of the organisms is the same in a grouping which makes epidemiological sense'.[1] The at-risk group is usually more clearly demarcated and identifiable in comparison to a community outbreak. These definitions in the guidelines are based on an arbitrarily set notification rate and provide a useful guide to develop appropriate management strategies.

Public health management of a community outbreak as per the national guidelines[1] includes mass immunisation of the at-risk population, provision of clearance antibiotics for close contacts and mass media to increase public awareness. It has been noted that mass immunisation of the population in which an outbreak is occurring may not be easy due to difficulties identifying the actual population at risk.[11,12,15] In addition, mass immunisation campaigns are resource intensive and can cause unwarranted public panic.[13]

Rates were calculated to establish whether the number of notifications in the first cluster between 6 November and 8 December 2003 fulfilled the criteria for an outbreak as per national guidelines.[1] As one of the criteria for a community outbreak was for cases to have identical microbiological characterisation, notification rates within a 3 month period were calculated for the first cluster of serogroup C cases for whom a common phenotype was identified (n=7, the New South Wales case was not included as the phenotype was not available). This notification rate was 8.64 cases per 100,000 (Australian Capital Territory population June 2003: 322,830) and was

slightly lower than the community outbreak rate as specified by the national guidelines (10 cases per 100,000 population within a 3-month period), but supported the possibility of an occurring outbreak. Assuming that the eighth case (the New South Wales resident for whom phenotyping was unavailable) was linked to this cluster as the case worked in the Australian Capital Territory, and adding the case into the calculation, made the notification rate 9.88 cases per 100,000 population. However, this represents an overestimation as the Australian Capital Territory non-resident working population is not included in the calculation.

With the notification of the eighth Australian Capital Territory serogroup C case with identical phenotype C:2a:P1.4 in January 2004 as part of the second cluster, the community notification rate over a 3 month period (6 November 2003 and 5 February 2004) was 9.88 cases per 100,000 population. Although this was on the borderline of a community outbreak within the Australian Capital Territory, due to the resource intensiveness of implementing community outbreak measures and possible public anxiety, a decision was made against declaring an outbreak.

As 50% of the serogroup C cases with identical phenotype were aged 15–19 years, an age-specific rate was also calculated; this was 65.64 cases per 100,000 (Australian Capital Territory population June 2003: 24,372). This notification rate fulfilled the criteria for a community outbreak in this age-group, and although the rate was very high, it represented 4 cases only. Management strategies already instituted were reviewed to make a decision on whether an outbreak in this age group needed to be declared. Two of the public health interventions implemented were specific to persons aged 15–19 years; these were access to free meningococcal serogroup C vaccine and developing targeted education campaigns. In addition, all close contacts had been provided with clearance antibiotics. By default, these interventions were outbreak measures within this age group. A decision was made not to declare an outbreak due to the smallness of numbers and to minimise public panic and anxiety.

The possibility of declaring a community outbreak was further complicated by having 2 different serogroup clusters notified and the one month interval in between the 2 clusters. The notification rate for serogroup B cases in the second cluster was 6.16 cases per 100,000 population within a 3 month period, which was lower than the required outbreak notification rate as per national guidelines. In addition to this, as phenotyping was unavailable, it would not have been accepted for the purposes of declaration of an outbreak.

In comparison, the declaration of an outbreak in an organisational setting and the implementation of outbreak management strategies such as mass immunisation could be assumed to be relatively straight forward as the at-risk group should be clearly identifiable. Anxiety and panic could be better managed as the risk group would be better demarcated. However, during the management of an organisational outbreak in a Brisbane boarding school, some discretionary judgements had to be made as some of the issues arising during the outbreak were not covered by the national guidelines.[1] Mass immunisation of all boarders and possible other at-risk groups associated with cases was instituted as a management strategy. Implementing this intervention on a large scale was feasible, but decisions about further defining an at-risk group with the notification of 2 subsequent cases who were not part of the boarding school but were associated indirectly, made definition of the at-risk population complicated.[17]

There were no more notifications after the second cluster of cases until June 2004. Six further cases of meningococcal serogroup C disease were notified between 5 June and 31 December 2004. The annual notification rates for 2003 and 2004 were 4.06 cases per 100,000 population and 3.43 cases per 100,000 population respectively, which was much higher than the average Australian Capital Territory rate. The higher than average incidence rate after clusters or outbreaks of meningococcal disease is not an uncommon finding. A sustained increase in incidence rate for a few years was noted after outbreaks in Canada[18] and after the Western Sydney outbreak of 1996.[11] For this reason, relevant health professionals in the Australian Capital Territory were encouraged to continue to have high sensitivity for possible cases of meningococcal disease.

Conclusions

It was obvious that there was a problem in the Australian Capital Territory with increased incidence of meningococcal disease and there was a need to intervene to decrease the incidence. Whether this was managed through the declaration of an outbreak and instituting outbreak measures as per the national guidelines,[1] was weighted against the community notification rate being on the borderline of an outbreak, the difficulty in identifying the actual at-risk group, and the logistics that would have been involved in implementing them. As these issues were not easy to resolve, an outbreak was not declared. If there were one, two or three additional cases (serogroup C, phenotype C:2a:P1.4) within the 3 month period, the notification rate would have crossed the community outbreak notification rate threshold and would have been 11.12, 12.46, and 13.60 cases per 100,000 population respectively. In

this situation, and taking into account that the last serogroup C case was fatal, serious consideration would have been given to declaring a community outbreak and instituting outbreak measures as per the national guidelines.[1]

In conclusion, careful consideration of the epidemiology of cases and targeted management appears to have been effective in decreasing and preventing further cases. Community outbreak notification rates as defined in the national guidelines were used as an arbitrary guide to develop suitable investigation and management processes. Good surveillance systems, the availability of relevant laboratory technology, regular public communication, and quick assessment, diagnosis and management of cases and contacts played an important role in the management of the increased incidence. We do not know whether declaration of an outbreak with the implementation of outbreak management strategies such as mass immunisation would have delivered any further benefits.

Acknowledgements

The authors would like to acknowledge the following agencies and persons for their assistance in the investigation and the public health management of the two clusters.

Members of the Communicable Diseases Control Section, in particular Riemke Kampen (Manager for Surveillance and Infection Control)

Paul Dugdale (Chief Health Officer)

The Communications and Marketing Unit, ACT Health

The Health Promotion Unit, ACT Health

ACT Pathology

The Infectious Diseases Unit, The Canberra Hospital

The Emergency Departments of The Canberra and Calvary hospitals

Infection Control Officers of The Canberra and Calvary hospitals.

Joanne Mercer and Robert Porritt, South Western Area Pathology Services (SWAPS), Sydney

Angelo Zaia, Melbourne Diagnostic Unit.

Author details

Geetha Isaac-Toua, PhD Scholar[1]
Charles Guest, Medical Director[2]
Rona Hiam, Public Health Registrar[3]
Irene Passaris, Manager,[4]

1. National Centre for Epidemiology and Population Health, Australian National University, Canberra, Australian Capital Territory
2. Health Protection Service, ACT Health, Canberra, Australian Capital Territory
3. The Canberra Hospital, ACT Health, Garran, Australian Capital Territory
4. Immunisation Services, Health Protection Service, ACT Health, Canberra, Australian Capital Territory

Corresponding author: Ms Geetha Isaac-Toua, PhD Scholar, National Centre for Epidemiology and Population Health, Building 62, Corner Miles and Eggleston Streets, Australian National University, Canberra 0200. Telephone +61 2 6125 0538. Facsimile: +61 2 6125 0740. Email: Geetha.Isaac-Toua@health.gov.au

References

1. Communicable Diseases Network Australia New Zealand. *Guidelines for the Early Clinical and Public Health Management of Meningococcal Disease in Australia.* Canberra: Commonwealth Department of Health and Ageing, June 2001.
2. Commonwealth Department of Health and Ageing. National Meningococcal C Vaccination Programme. 2003
3. Whiley DM, Crisante ME, Syrmis MW, Mackay IM, Sloots TP. Detection of *Neisseria meningitidis* in clinical samples by a duplex real-time PCR targeting the *por*A and *ctr*A genes. *Mol Diagn* 2003;7:141–145.
4. The National Neisseria Network. Meningococcal isolates surveillance Australia 1995. *Commun Dis Intell* 1995;19:286–289.
5. Wedege E, Hoiby E, Rosenqvist E, Froholm L. Serotyping and subtyping of *Neisseria meningitidis* isolates by co-agglutination, dot-blotting and ELISA. *J Med Microbiol*1990;31:195–201.
6. Hawkes R, Niday E, Gordon J. A dot-immunobinding assay for monoclonal and other antibodies. *Analyt Biochem* 1982;119:142–147.
7. Tribe DE, Zaia AM, Griffith JM, Robinson PM, Li HY, Taylor KN, et al. Increase in meningococcal disease associated with the emergence of a novel ST-11 variant of serogroup C *Neisseria meningitidis* in Victoria, Australia, 1999–2000. *Epidemiol Infect* 2002;128:7–14.
8. Decosas J, Koama JB. Chronicle of an outbreak foretold: meningococcal meningitis W-135 in Burkino Faso. *Lancet Infect Dis* 2002;2:763–765.
9. Seccul A. Meningococcal Disease Outbreak in Portland, December 2002: Report.
10. ABC online. Outbreak of meningococcal disease in Hobart nightclubs. *AM Archive* –29 September 2001.

11. Jelfs J, Jalaludin B, Munro R, Patel M, Kerr M, Daley D, et al. A cluster of meningococcal disease in Western Sydney, Australia initially associated with a nightclub. *Epidemiol Infect* 1998;120:263–270.

12. Patel MS, Collignon PJ, Watson CR, Condon RJ, Doherty RR. New guidelines for management and prevention of meningococcal disease in Australia. *Med J Aust* 1997;166:598–601.

13. Centers for Disease Control and Prevention. Control and prevention of serogroup C meningococcal disease: evaluation and management of suspected outbreaks: Recommendations of the Advisory Committee on Immunisation Practices. *MMWR Recomm Rep* 1997;46:13–21.

14. Robinson P, Taylor K, Tallis G, Carnie J, Rouch G, Griffith JM, et al. An outbreak of serogroup C meningococcal disease associated with a secondary school. *Commun Dis Intell* 2001;25:121–125.

15. National Health and Medical Research Council. *Guidelines for the control of meningococcal disease in Australia.* Canberra: Australian Government Publishing Service, 1997.

16. Chin J, editor. *Control of Communicable Diseases Manual* 17th edn. Washington: American Public Health Association, 2000:345.

17. Davison RP, Lovegrove DR, Selvey LA, Smith HV. Using the national guidelines to manage a meningococcal group C outbreak in a Brisbane boarding school – some discretionary judgements are needed. *Commun Dis Intell* 2003;27:520–523.

18. Whalen AM, Hockin JC, Ryan A, Ashton F. The changing epidemiology of invasive meningococcal disease in Canada, 1985 through 1992. *JAMA* 1995;273:2390–2394.

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

Nicola Stephens, Cameron Sault, Simon M Firestone, Diane Lightfoot, Cameron Bell

Abstract

This report describes one of the largest egg-associated outbreaks of foodborne illness in Australia for many years. Between June and December 2005, five outbreaks of *Salmonella* Typhimurium phage type 135 were identified in Tasmania, leading to 125 laboratory-confirmed cases. Public health investigations included case and food handler interviews, cohort studies, environmental health investigations of food businesses, microbiological testing, traceback, and inspections and drag swabbing of an egg farm. These investigations enabled identification of foods containing raw egg or foods contaminated through inadequate food handling and/or storage procedures as possible vehicles for infection. A particular poultry farm was reported as the common source of eggs. Interventions targeting the general public and food handlers to promote better handling of egg products, and advice to egg producers regarding harm minimisation strategies led to the series of outbreaks being brought under control. *Commun Dis Intell* 2007;31:118–124.

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

Introduction

Foodborne illness is a public health concern in all parts of the world. In Australia, an estimated 32% of gastroenteritis is foodborne, causing around 5 million illnesses, 4,000 hospitalisations and approximately 76 deaths annually.[1] Among known pathogens, enteropathogenic *Escherichia coli*, noroviruses, *Campylobacter* spp. and *Salmonella* spp. accounted for 88% of all foodborne disease in Australia in 2000.[1]

From 2001 to 2004, the average yearly rate of laboratory-confirmed *Salmonella* infections in Tasmania was less than that for Australia as a whole (31.2 versus 37.6 cases per 100,000 population respectively). During these 4 years, *S.* Mississippi was the most commonly reported *Salmonella* serotype in Tasmania, comprising 52% of the *Salmonella* notifications from this State. *S.* Mississippi (a group G *Salmonella*) is considered an environmental serovar occupying an ecological niche in native Tasmanian animals, and is commonly acquired from exposure to those animals and/or drinking untreated water.[2] *S.* Typhimurium (a group B *Salmonella*) was the next most commonly reported *Salmonella* serotype in Tasmania, comprising a further 22% of the

Salmonella notifications. Amongst these *S.* Typhimurium cases, phage types 9 and 135 were most commonly reported, with a combined average of 17 cases per year (3.5 cases per 100,000 population), of which an average of 8 were *S.* Typhimurium 135 (STm135) cases (1.7 cases per 100,000 population). Previous outbreaks of STm135 in Australia have been associated with the consumption of desserts containing raw egg products,[3,4,5] a bakery's cream piping-bag,[6] egg sandwiches,[7] chicken,[8,9] and a commercial orange juice.[10]

In June 2005, an increase in the number of laboratory notifications of salmonellosis was observed by the Communicable Diseases Prevention Unit of the Department of Health and Human Services Tasmania (DHHS). These were subsequently identified as STm135 but investigations at the time were unable to pinpoint a source. Several months later, there were a series of point-source outbreaks which caused a dramatic increase in *Salmonella* notifications.

An outbreak investigation consisting of a series of community case interviews and cohort studies was conducted in order to identify the source of infection and to enable public health intervention to occur.

Methods

There were a total of 5 outbreaks between June and December 2005, some occurring in group functions and others in restaurant or similar settings (Table 1). Methods used to investigate each outbreak varied and included case interviews, microbiological assessments, environmental investigations, and cohort studies where appropriate.

Case reports of salmonellosis in Tasmania are routinely investigated by local government Environmental Health Officers. However, given the magnitude of notifications in this outbreak, the DHHS outbreak investigation team conducted the majority of the case interviews. All case interviews were carried out by telephone. Community case interviews were conducted following the first peak in notifications, using a hypothesis-generating questionnaire. The questionnaire included a detailed 3-day food history and general questions about activities and foods consumed in the seven days prior to illness. The questionnaire was later expanded to include a 7-day food history. When the evidence from interviews and other lines of investigation were able to confirm a food business as the source of the outbreak, questionnaires were then modified to focus on the consumption of implicated products.

Faecal specimens were collected from individuals suffering from gastroenteric symptoms and tested for the presence of a range of pathogens including *Salmonella*. Limited *Salmonella* antigenic testing was conducted by laboratories in Tasmania. The isolates from cases of salmonellosis were then sent to the Microbiological Diagnostic Unit (MDU) Public Health Laboratory in Victoria for serotyping and phage typing. The tests conducted in Tasmania on the somatic (O) antigen of isolates collected during the initial increase in notifications showed that they were of group B *Salmonellae*. From this information

Table 1. Five outbreaks of *Salmonella* Typhimurium phage type 135 cases, Tasmania, June to December 2005

Outbreak number	Notification date	Number of cases	Method of investigation	Case definition*
1	16–27 June 2005	11	Case interviews, traceback	STm135 isolated from a faecal specimen
2	3–19 October 2005	63	Case interviews, cohort studies, food handling review, microbiological testing, traceback	STm135 isolated from a faecal specimen 'Probable cases' included in cohort if reported being ill following function attendance, with diarrhoea and two other clinically compatible symptoms*
3	28 October–2 November 2005	10	Case interviews, food handling review, microbiological testing, traceback	STm135 isolated from a faecal specimen
4	18–21 November 2005	5	Case interviews, food handling review, microbiological testing, traceback	STm135 isolated from a faecal specimen
5	1–20 December 2005	36	Case interviews, cohort studies, food handling review, microbiological testing, traceback	STm135 isolated from a faecal specimen 'Probable cases' were included if they reported being ill with symptoms of gastroenteritis following function attendance.

* Vomiting, nausea, abdominal cramps, lethargy, headache, fever or rigors.

the investigation focussed on group B *Salmonellae* in a timelier manner until more detailed typing information was obtained.

Case definitions were developed from information collected at interview and from laboratory specimens as shown in Table 1.

Environmental investigations

All food businesses identified in food histories of more than 1 case were investigated by environmental health and food safety officers. Food handling practices were reviewed and samples were collected from food products, raw ingredients, food preparation surfaces and equipment, for microbiological investigation.

Microbiological assessments

All human and non-human *Salmonella* isolates were sent to the MDU for confirmation, serotyping, subtyping and antibiotic resistance profiles.

Cohort studies

Of the 5 outbreaks, cohort studies were conducted in two: outbreaks 2 and 5 (Table 1). Two groups of individuals in outbreak 2 (cohorts 1 and 2) and 5 groups of individuals in outbreak 5 (cohorts 5, 8, 9, 10 and 11, Table 2) were investigated. The cohorts were assembled from guest lists and verbal consent was obtained from subjects prior to interview. Questionnaires were developed to collect data on food consumption, basic demographic data and symptoms.

Statistical methods

Data were entered into a Microsoft® Excel spreadsheet and analysed using Stata® version 8.0 (Stata Corporation, College Station, TX, USA) (Stata). Food exposures were expressed as dichotomous variables, and crude relative risks (RR) with 95% confidence intervals (CI) were calculated.

For cohort 1 of outbreak 2, a logistic regression model was fitted to the cohort study data and adjusted odds ratios (OR_{adj}) were calculated with 95% confidence intervals to adjust for possible confounding. Food exposure variables were only included in the model if they showed a strong univariate association with the dependant variable (illness due to STm135). Model fit was optimised by categorising age into 20-year age groups.

Following univariate statistical analysis of cohort study data from outbreak 5, a stratified analysis was conducted to clarify the influence of confounding and effect modification on results.

Results

Descriptive epidemiology

A total of 125 laboratory-confirmed cases of STm135 were notified across the 5 outbreaks (Figure 1). The age of cases ranged from <1 year to 86 years, with a median of 37 years (Figure 2). Females accounted for 55% of all laboratory-confirmed cases.

Outbreak 1

The first outbreak consisted of 11 sporadic community cases in the north of Tasmania (Table 1). The cause of illness in this outbreak could not be identified. Geographical clustering of cases suggested that a local food business or ingredient may have been the source however investigators were unable to link cases to a common source.

Outbreak 2

Outbreak 2 consisted of 53 sporadic cases in the community in the north of Tasmania and of 2 cohorts

Table 2. Food exposures associated with illness due to *Salmonella* Typhimurium phage type 135, from cohorts attending catered functions, outbreak 5, Tasmania, December 2005*

Food exposure	Cases/ people exposed	Cases / people not exposed	Relative Risk (95 % CI)	
Cohort 5				
Smoked salmon with potato salad and mayonnaise	18 / 25	6 / 15	1.8	(0.9, 3.5)
Cohort 8				
Chicken sandwich	12 / 15	0 / 2	undefined	
Beef sandwich	10 / 12	2 / 5	2.08	(0.7, 6.3)
Cohorts 9, 10 and 11 combined				
Cajun chicken / avocado sandwich	22 / 23	1 / 13	12.4	(1.9, 81.9)
Leg ham sandwich	18 / 25	5 / 15	2.2	(1.0, 4.6)
Scones	13 / 14	13 / 31	2.1	(1.4, 3.3)

* Analysis includes laboratory-confirmed and probable cases.

Figure 1. Epidemic curve of *Salmonella* Typhimurium phage type 135 cases, Tasmania, Australia, 1 January 2005 to 31 January 2006, by outbreak

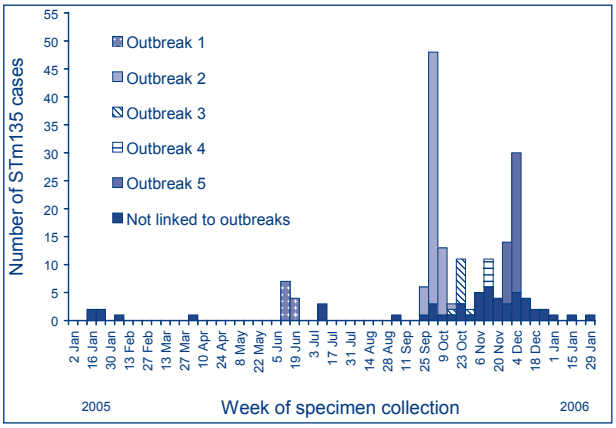
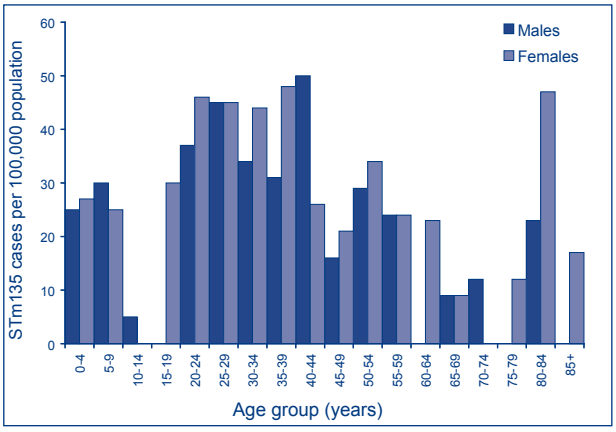


Figure 2. *Salmonella* Typhimurium phage type 135, all outbreak cases, Tasmania, 1 June to 31 December 2005, by age group and sex



(1 and 2) made up of 5 cases each, linked to two separate birthday party functions in the south of Tasmania (Table 1). Laboratory-confirmed cases (n=10) and probable cases (n=15) were included in both cohort studies. The community cases reported consuming products originating from 2 co-owned bakeries (Bakeries A and B) in the same city. Various sweet and savoury products, particularly cream-based products such as chocolate éclairs, were consumed. Cases associated with the functions all ate at least one product that had been prepared by the implicated bakeries. Table 3 shows the results of the univariate analysis of data collected from cohort 1. Three food items were associated with illness: a sponge-cream cake from Bakery A, and egg/bacon pies and pinwheels (small savoury scrolls) supplied by other unassociated bakeries. However, logistic regression showed that sponge-cream cake from Bakery A was the only food item significantly associated with illness (OR_{adj}=7.7, 95% CI: 1.7, 35.0).

Outbreak 3

All 10 cases in this outbreak had eaten at least one product at the same café in the south of Tasmania (Table 1). Products consumed included meals containing raw egg sauces and home-made hamburgers that some cases reported were obviously undercooked.

Outbreak 4

All 5 cases in this outbreak had eaten at least one product prepared at a third bakery in the north of Tasmania (Bakery C) (Table 1). A variety of products were reported as being consumed by cases.

Table 3. Association between food item consumption and illness due to *Salmonella* Typhimurium phage type 135, cohort 1, outbreak 2, Tasmania, 1 October 2005*

Food exposure	Cases/ people exposed	Cases/ people not exposed	Relative risk (95 % CI)		Adjusted odds ratio† (95 % CI)	
Foods from Bakery A						
Sponge cake	11 / 37	3 / 52	5.2	(1.5, 17.2)	7.7	(1.7, 35.0)
Mud cake	6 / 42	8 / 48	0.8	(0.3, 2.3)		
Savoury Toasts	6 / 30	8 / 57	1.4	(0.5, 3.7)		
Party-pies	5 / 21	9 /67	1.8	(0.7, 4.7)		
Quiches	3 / 19	10 / 67	1.1	(0.3, 3.5)		
Foods from other bakeries						
Egg/bacon pies	8 / 25	5 / 58	3.7	(1.3, 10.2)	3.2	(0.7, 13.6)
Pinwheels	6 / 17	8 / 68	3.0	(1.2, 7.5)	1.1	(0.2, 5.2)

* Analysis includes laboratory-confirmed and probable cases.

† Adjusted for age, sex, mud cake and savoury foods from Bakery A. The potential confounders (sex and all age group categories) were not strongly associated with the dependant variable.

Outbreak 5

Eleven separate groups of individuals including 36 cases ate at functions catered for by the same restaurant in the south of Tasmania over a period of 8 days (Tables 1 and 4). A summary of exposures found to be associated with illness for catered functions, by cohort, is shown in Table 2. Cohorts of cases who did not eat catered food were excluded due to unreliable recall of foods consumed, therefore results are reported only for 5 cohorts (5, 8, 9, 10 and 11). Consumption of an entrée of smoked salmon with potato salad and mayonnaise was associated with gastroenteritis in cohort 5. Consumption of chicken sandwiches in cohort 8 was associated with gastroenteritis. Three food exposures were associated with illness in cohorts 9, 10 and 11, chicken and avocado sandwiches, scones and leg ham sandwiches. Stratified analysis demonstrated that chicken and avocado sandwiches was the only food item consistently associated with gastroenteritis. Investigations by Food Safety Officers identified that raw egg had been used in the preparation of mayonnaise and mixed with avocado in the implicated food items in each cohort. Laboratory confirmed cases (n=36) and probable cases (n=40) were included in the analyses.

Environmental investigations

Extensive environmental investigations were undertaken at each implicated food business, and a number of raw ingredients and food products were collected for microbiological testing. STm135 was isolated from a disposable cream piping bag and a bench surface swab in Bakery A associated with outbreak 2. Staff interviews revealed that the disposable cream piping-bag had been re-used a number of times, potentially contaminating the cream layer of sponge cakes distributed to both functions in early October. STm135 was also isolated from the cream of a chocolate éclair distributed from Bakery A to another retail outlet. Bakeries A and B were found to share both the ingredients and preparation of their ready-to-eat foods. Samples collected during outbreaks 3 and 4 were negative for *Salmonella*. At the restaurant implicated in outbreak 5, STm135 was isolated from mayonnaise, tartare sauce and lettuce mix. These products were used in the making of the food items associated with illness.

Cross contamination issues were identified in all food businesses implicated in the series of outbreaks. Production demands which exceeded the physical capacity of the food businesses, combined with inadequate sanitisation and hygiene practices, were also contributing factors. The handling of products containing raw egg was identified to be a major issue. Both the café in outbreak 3 and the restaurant/caterer in outbreak 5 produced a variety of sauces made with raw eggs. Investigations revealed that these sauces were being kept and reused for up to 7 days. Raw egg was a component of the mayonnaise and tartare sauce which tested positive for STm135 in outbreak 5. Raw eggs were also used in the meat of hamburgers implicated in outbreak 3, the centres of which were reported to be undercooked, possibly due to their thickness.

Traceback of ingredients used by the food businesses implicated in these outbreaks confirmed that they all purchased their eggs from the same farm in Tasmania. This first became apparent during outbreak 3 when investigations demonstrated that eggs used by the café originated from the same farm that supplied Bakeries A and B identified during

Table 4. Eleven cohorts linked to either a function catered by, or with dining at, the same food business between 25 November and 2 December 2005 (outbreak 5), Tasmania

Cohort	Date of function	Number attended	Reported ill at interview		Tested positive for STm135	
			n	% attendees	n	% attendees
1	25 Nov	2	2	100	2	100
2	25 Nov	90	3	NA	2	2
3	25 Nov	3	3	100	2	67
4	25 Nov	2	2	NA	1	50
5	26 Nov	41	24	59	5	12
6	26 Nov	13	2	15	2	15
7	29/30 Nov	1	1	100	1	100
8	30 Nov	17	12	71	4	29
9*	2 Dec	6	5	83	5	83
10*	2 Dec	10	9	90	7	70
11*	2 Dec	28	12	43	5	21

* Analysed as one group due to identical menu.

NA Not applicable, not all attendees were interviewed.

outbreak 2. Traceback during outbreaks 4 and 5 also found that the eggs originated from the same farm. Visible external contamination of a number of eggs and packaging (dirt and other debris) was observed during the investigations of food businesses in outbreaks 2, 3 and 5, however microbiological samples from these raw eggs and exterior shells tested negative for *Salmonella*.

Management

In late October 2005, drag swabbing and a Hazard Analysis and Critical Control Point assessment (HACCP) were undertaken at the egg farm associated with the point source outbreaks. The drag swabbing methodology followed the New South Wales guidelines for voluntary *Salmonella* Enteritidis-free accreditation of egg farms.[11] Thirteen samples were submitted for microbial investigation. None were positive for *Salmonella*. Farm management issues that might have increased the risk of egg contamination included egg washing, storage, vermin control, and procedures for packaging and transportation. These issues were addressed in a 'whole of farm' quality assurance program that was being developed for the egg farm with the assistance of the Department of Primary Industry and Water (DPIW) at the time of the investigation. This quality assurance program has subsequently been implemented. Farm assessments (HACCP) and drag swabbing were repeated in December 2005 and January 2006 to check for the possibility of intermittent shedding of *Salmonella* by poultry. STm135 was subsequently isolated from samples taken from poultry faeces, spilled feed, and an egg conveyor belt in December, as well as from the surface of pulp grade eggs tested in January.

DHHS and DPIW sent a letter to all Tasmanian egg producers with recommendations on ways to reduce *Salmonella* contamination of eggs. As a precaution, DHHS issued media releases advising the community not to consume raw or under-cooked eggs. In a separate letter to all Tasmanian food businesses, DHHS made recommendations regarding the use of clean eggs ('free of visible external contamination'), refrigerated storage of eggs, and the safe preparation and handling of foods containing raw eggs. Following these media releases, a national supermarket chain changed to using single-use piping bags in their in-store bakeries (personal communication, M Kirk, OzFoodNet, December 2005). DHHS also sent information to general practitioners and emergency departments across the state to keep them informed about the outbreak and to request increased microbiological testing of patients presenting with gastroenteritis.

Following these interventions, the rate of notifications decreased with only 3 cases of STm135 notified in Tasmania during January 2006.

Microbiology

All *Salmonella* isolates from human and non-human sources were found to be antigenically identical and exhibited a phage reaction pattern that is designated as *S.* Typhimurium 135a by the Institute of Medical and Veterinary Science in South Australia.

Discussion

This report describes one of the largest egg-associated outbreaks in Australia for many years. Investigations identified several food businesses associated with point source outbreaks. Subsequently, traceback and farm level investigations supported the hypothesis that eggs were likely to be the food vehicle, although *Salmonella* was not isolated from market eggs. Interventions targeting food handlers and members of the public to raise awareness about safe handling of raw egg products and harm minimisation strategies on the farm from which eggs were reported to be sourced, led to the outbreak being controlled.

Ninety-one per cent of the cases in the 5 outbreaks (Table 1) were linked to food businesses supplied by a single egg farm. Each of these food businesses was found to have inadequate food handling and/or storage procedures that led to a potential *Salmonella* hazard from unclean eggs becoming an actual risk. Higher food handling standards with an emphasis on avoidance of cross-contamination could have prevented most if not all of these cases.

The food items from which positive microbial isolates were obtained were mostly dessert ingredients or sauces used in salad-based dishes. Even when contaminated by very small doses of *Salmonella*, these ingredients provide a fertile growth medium, especially if stored outside the refrigerator.

The available evidence from this outbreak suggests that, over a period of 6 months, a series of farm management issues led to eggs and egg containers leaving the identified farm carrying enough poultry faecal material to contaminate raw ingredients at the food businesses. However, no positive isolate from the outside of eggs actually in the human food chain was obtained, and the first series of drag swabs was negative. In spite of this, a low level of *Salmonella* intermittently shed by the farm's poultry, in conjunction with faecal contamination of eggs, is a likely explanation for our findings.

These findings illustrate the shared responsibility of primary food producers and food handlers to ensure the hygiene of the finished food product. Farm products such as eggs should never be regarded as 'sterile'.

The cooperation of the egg producer in these investigations was vital to understanding and ensuring a good public health outcome.

Acknowledgements

The authors would like to thank all those who participated in the outbreak investigation, in particular Lynne Andrewartha, Leanne Cleaver, David Coleman, Eric Johnson, Avner Misrachi, Roscoe Taylor, Kate Turner, the Environmental Health Officers and other staff at DHHS Tasmania; Christian Garland of FWE Health Services; Jane Hudspeth and other microbiologists of Tasmanian public and private laboratories; MDU staff; Mary Deeble; Martyn Kirk; Susan Bibby; our colleagues at DPIW; the egg producer; and food businesses who cooperated with our investigations; and all the cases and function attendees who responded willingly to our interviews. Thanks also to Kelly Shaw and Rod Andrewartha for helpful comments on the manuscript. Nicola Stephens and Cameron Sault are funded by OzFoodNet, which is an initiative of the Australian Government Department of Health and Ageing. Simon Firestone is funded by the Masters of Applied Epidemiology program, which is also an initiative of the Australian Government Department of Health and Ageing.

Author details

Nicola Stephens, Epidemiologist[1]
Cameron Sault, Research Officer[1]
Simon M Firestone, MAE student[2]
Diane Lightfoot, Senior Scientist[3]
Cameron Bell, Veterinary Officer[4]

1. Communicable Diseases Prevention Unit, Department of Health and Human Services, Hobart, Tasmania
2. National Centre for Epidemiology and Population Health, Australian National University, Canberra, Australian Capital Territory
3. Microbiological Diagnostic Unit, Public Health Laboratory, The University of Melbourne, Melbourne, Victoria
4. Animal Health and Welfare Branch, Department of Primary Industries and Water, Hobart, Tasmania

Corresponding author: Ms Nicola Stephens, Epidemiologist, OzFoodNet, Communicable Diseases Prevention Unit, Department of Health and Human Services, GPO Box 125, Hobart Tas 7001. Telephone: +61 3 6222 7627. Facsimile: +61 3 6222 7692. Email: nicola.stephens@dhhs.tas.gov.au

References

1. Hall G, Kirk MD, Becker N, Gregory JE, Unicomb L, Millard G, et al. Estimating foodborne gastroenteritis, Australia. *Emerg Infect Dis* 2005;11:1257–1264.
2. Ashbolt R, Kirk MD. *Salmonella* Mississippi infections in Tasmania: the role of native Australian animals and untreated drinking water. *Epidemiol Infect* 2006;134:1257-1265.
3. Hall R. Outbreak of gastroenteritis due to *Salmonella* Typhimurium phage type 135a following consumption of raw egg. *Commun Dis Intell* 2002;26:285–287.
4. Sarna M, Dowse G, Evans G, Guest C. An outbreak of *Salmonella* Typhimurium PT135 gastroenteritis associated with a minimally cooked dessert containing raw eggs. *Commun Dis Intell* 2002;26:32–37.
5. Tribe IG, Cowell D, Cameron P, Cameron S. An outbreak of *Salmonella* Typhimurium phage type 135 infection linked to the consumption of raw shell eggs in an aged care facility. *Commun Dis Intell* 2002;26:38–39.
6. Oakman T, Kolbe T, Hamilton I. An outbreak of *Salmonella* enterica serovar Typhimurium (*S.* Typhimurium) phage type 135a in the Greater Murray. *N S W Public Health Bull* 2003;14:125–127.
7. McCall BJ, Bell RJ, Neill AS, Micalizzi GR, Vakaci GR, Towner CD. An outbreak of *Salmonella* Typhimurium phage type 135a in a child care centre. *Commun Dis Intell* 2003;27:257–259.
8. Communicable Diseases Section. Surveillance of Notifiable Infectious Diseases in Victoria, 1998. Melbourne: Victorian Department of Human Services; 1999.
9. Communicable Diseases Section. Surveillance of Notifiable Infectious Diseases in Victoria, 2002. Melbourne: Victorian Department of Human Services; 2003.
10. Salmonellosis outbreak, South Australia. *Commun Dis Intell* 1999;23:73.
11. Guidelines NSW *Salmonella* accreditation. *Salmonella enteritidis* (SE). Accredited free and monitored layer farms. 1999. New South Wales Department of Primary Industries. 5 pp.

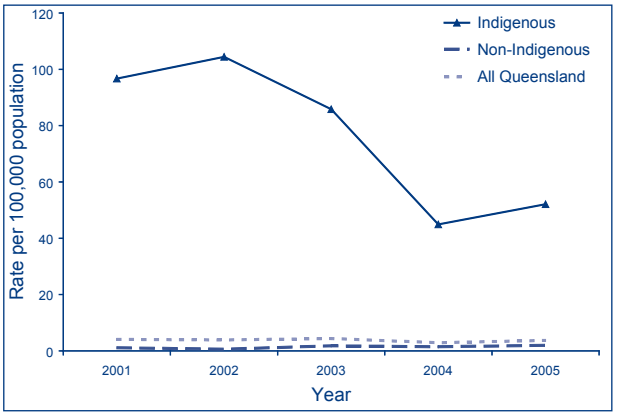
Short reports

SYPHILIS IN REMOTE NORTH QUEENSLAND

Patricia S Fagan, Fiona M Cannon

The notification rate for infectious syphilis (i.e. infection of less than 2 years duration) in Queensland has remained fairly static over the last 5 years but the pattern of infection appears to be changing (Figure 1). Between 2001 and 2005, the non-Indigenous rate climbed from 1.1 to 2.0 cases per 100,000 population, while the rate in the Aboriginal and Torres Strait Islander population across the state fell from 97 to 52 cases per 100,000 population – both statistically significant but divergent trends ($p<0.001$).[1]

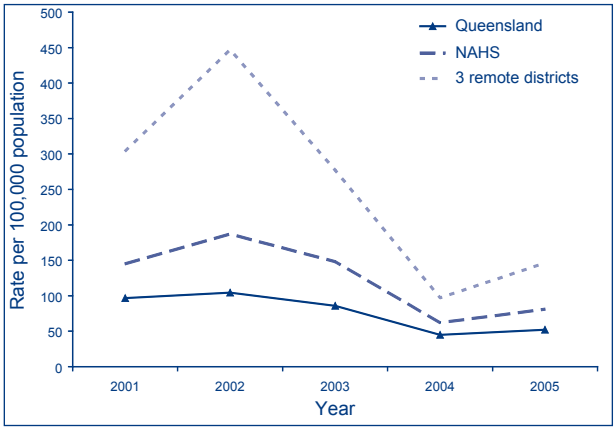
Figure 1. Notification rate of syphilis, Queensland, 2001 to 2005



A significant majority of Indigenous infectious syphilis notifications in Queensland arise in remote parts of the Northern Area Health Service (NAHS) in populations that historically have sustained a disproportionate burden of disease and where cases of congenital syphilis are notified almost every year.[2] Hence, we were keen to explore the implications for these remote populations of the changing epidemiology of syphilis in the state.

Figure 2 illustrates that the downward trend in Indigenous syphilis rates state-wide is amplified in 3 remote health service districts where 29% of the NAHS Aboriginal and Torres Strait Islander population reside (Map). In contrast, the notification rate for the Aboriginal and Torres Strait Islander population living in the NAHS but outside these remote districts, showed no significant trend over the same period ($p=0.092$), suggesting that changes within these districts were largely responsible for the observed fall in the Queensland Indigenous notification rate.

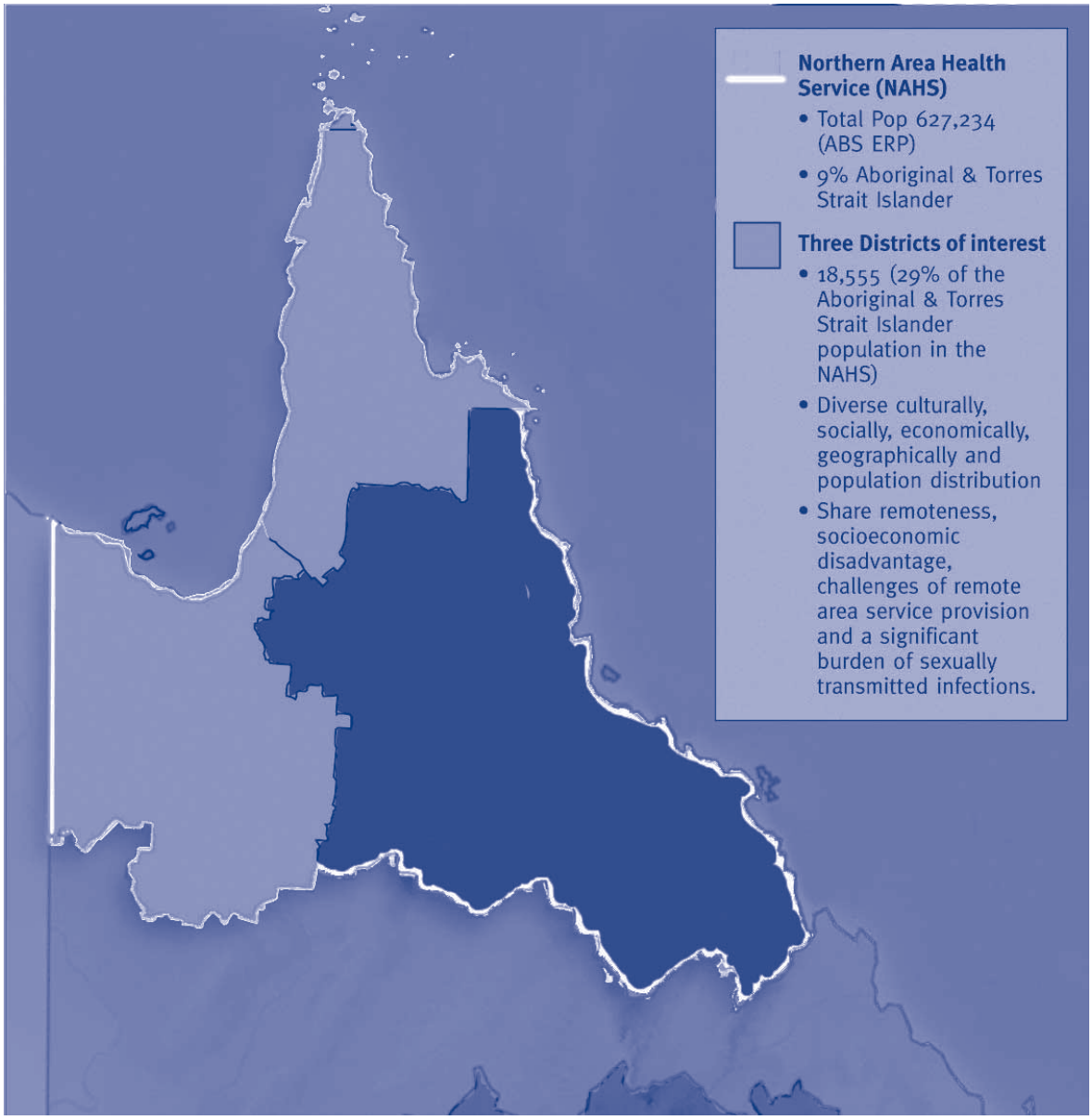
Figure 2. Notification rate of syphilis in Aboriginal and Torres Strait Islander populations, selected regions, 2001 to 2005



Relevant health system developments in the three remote districts

In recent years, the threat of an HIV epidemic has led to a sharper focus on bacterial sexually transmissible infection (STI) control as an HIV prevention strategy. As the diagnosis of syphilis in this setting usually relies on testing asymptomatic but 'at risk' individuals, a central plank of this strategy is to increase STI testing among the young 'at risk' population. Remote area primary health care providers are requested to offer all men and women aged 15–39 at least one test for syphilis, chlamydia and gonorrhoea each year. Queensland Health Pathology and Scientific Services (QHPSS) has been the sole pathology provider to all the remote (largely Indigenous) communities in the three districts during the period 2000 to 2005 with a small amount of private pathology service activity occurring in the more urban (non-Indigenous) areas of one district. Data from QHPSS describe increased syphilis testing from 4,054 to 6,119 tests for 2000 to 2005 with the increase in testing per capita showing a statistically significant trend in two of the three districts ($p<0.001$). The strategy may also have helped focus STI testing more in the 'at risk' age groups. Population level data for two of the districts indicates that 30% of the women and 21% of the men aged 15–39 years had had at least one syphilis serology test during 2005 (Fagan P, STI District reports, 2005). An assessment of testing activity over time in NAHS populations outside these three districts has not been undertaken and would be complicated by multiple providers and uncertain denominators. However, the finding of decreasing notifications over a period

Map. Remote districts where syphilis notifications are declining in Aboriginal and Torres Strait Islander populations



of increased testing in remote districts strengthens the argument for a reduced population level of disease in these areas.

Syphilis Register data allows the analysis of 'time to treatment' for all cases in the NAHS since 2001 (Table) – demonstrating an average annual improvement of 17% for each year since ($p<0.001$). This is good news for individuals with syphilis, and at the population level it decreases the duration of infectiousness and potentially reduces the number of new infections arising from a single source. Electronic data interchange (improving the timeliness of notifications), the enhanced surveillance and support provided by the Syphilis Register public health nurse in Cairns, and the increased focus on STI as a health issue in remote areas, may all be contributing to this sustained improvement in 'time to treatment'.

A final point ot note is that azithromycin is now widely used in north Queensland (usually a single oral dose of 1 gm) for the treatment of symptomatic and screen detected cases and for contacts of the other common bacterial STI.[3] When given at a higher dose (2 gm orally), it is also effective against *Treponema pallidum*.[4] It is possible that azithromycin, when used at the lower dose, may sometimes be adequate against very early treponemal infection and could contribute to a reduction in community prevalence.

Is syphilis declining in remote Indigenous populations in north Queensland?

Only time will tell if syphilis in remote Indigenous populations in north Queensland is declining. The

Table. Time to treatment for infectious syphilis in the Northern Area Health Service, by year of notification

Year	Number of cases	No record of treatment	Time to treatment (days) – all cases		
			Range	Median*	75% treated
2001	98	14	–1 to 1,469	19	147
2002	122	7	–5 to 928	12	21
2003	103	5	0 to 768	10	25
2004	48	3	–9 to 400	7	23
2005	63	5	0 to 547	7	17

p<0.001; Cox regression Hazard ratio = 1.17 (C.I. 1.08, 1.27).

factors outlined above suggest an improved health system response to syphilis but the downward trend in notifications needs to be interpreted with caution.

Firstly, despite increased testing being accompanied by a steady reduction in new cases over this period, one of the three districts went against this trend and recorded a 17% drop in syphilis testing since 2000 (QHPSS data).

The level of periodic testing coverage of an 'at risk' population that would be required (assuming comprehensive investigation and timely treatment of all identified cases) to achieve a specified reduction in community prevalence of syphilis, is uncertain. Certainly, the magnitude of the contribution to the downward trend in notifications that the level reported here (30% for women and 21% for men aged 15 to 39 years in 2005) is debatable.

Furthermore, the decrease in infectious syphilis has not yet translated into a noticeable change in congenital syphilis. Since 2001, there have been 12 cases of congenital syphilis notified in Queensland (spread evenly across the years), and two-thirds came from these three remote districts. Syphilis in pregnancy, long recognised as a significant cause of pregnancy loss and perinatal death in north Queensland,[5] is associated with a high population prevalence of infection.

Finally, the time period for which we have reliable data is relatively brief. The natural history of syphilis may be such that there are peaks (and troughs) in notifications over an 8 to 11 year cycle corresponding with new infections arising as herd immunity from earlier epidemics wanes.[6] If this is so, then 5 years is not a sufficient period over which to assess long term trends.

Despite the recent fall in notifications, syphilis persists in Aboriginal and Torres Strait Islander populations at unacceptably high levels – 52 cases per 100.000 population is many times higher than other Indigenous and non-Indigenous populations across the developed world.[7] Experience suggests that a sustained reduction in syphilis and the prevention of congenital syphilis rely on the maintenance of efforts in syphilis control, vigilant antenatal care and on-going collaboration between obstetric, paediatric and sexual health services.

Acknowledgement

We would like to thank TPHU staff, Ms Alexandra Raulli, epidemiologist, and Ms Fiona Tulip, data manager, for their assistance with data preparation and statistical analysis

Author details

Patricia S Fagan, Sexual Health Public Health Physician[1]
Fiona M Cannon, Public Health Nurse Syphilis Register[1]

1. Tropical Population Health Unit, Northern Area Health Service, Queensland Health, Cairns, Queensland

Corresponding author: Dr Patricia Fagan, PO Box 1103, Cairns, Qld, 4870. Telephone: +61 7 4050 3679. Email: patricia_fagan@health.qld.gov.au

References

1. Fagan PS, Cannon FM. Syphilis and genital ulcer disease in 2006: a Far North Queensland perspective. *Australasian Sexual Health Conference,* Melbourne 2006; poster presentation.
2. Sweeny AL. Diverging trends in infectious syphilis, Queensland Australia 2002–2005. *Australasian Epidemiological Association Conference*, Melbourne 2006; presentation.
3. Royal Flying Doctor Service; Queensland Health, North Queensland Workforce Unit. *Primary Clinical Care Manual.* 4th edn 2005. p. 375–394.
4. Hook EW, Martin DH, Stephens J, Smith BS, Smith K. A randomised, comparative pilot study of azithromycin versus benzathine penicillin G for treatment of early syphilis. *Sex Transm Dis* 2002;29:486–490.
5. Humphrey MD, Bradford DL. Congenital syphilis: still a reality in 1996. *Med J Aust* 1996;165:382–385.
6. Grassly NC, Fraser C, Garnett GP. Host immunity and synchronized epidemics across the United States. *Nature* 2005;433:417–421.
7. Department of Health and Human Services, National Center for HIV, STD and TB Prevention, Division of STD Prevention. *National Plan to eliminate syphilis from the USA.* May 2006.

Quarterly reports

OzFoodNet quarterly report, 1 October to 31 December 2006

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 October to 31 December 2006.

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 fourth quarter of 2006, OzFoodNet sites reported 370 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 7,955 people, of which 267 were hospitalised and 5 died. The majority (82%, n=306) of outbreaks resulted from infections suspected to be spread by person-to-person transmission (Figure 1). Fifty-nine per cent of these outbreaks occurred in aged care facilities, 22% in hospitals, 10% in child-care centres and 8% in various other settings. Norovirus was identified as a cause of illness in 102 of the outbreaks in aged care facilities and was suspected in many more.

Foodborne disease outbreaks

There were 34 outbreaks during the fourth quarter of 2006 where consumption of contaminated food was suspected or confirmed as the primary mode of transmission (Table). These outbreaks affected 756 people and resulted in 75 people being admitted to hospital. There were no deaths. This compares with 36 outbreaks for the fourth quarter of 2005 and 23 outbreaks in the previous quarter of 2006.

Salmonella was responsible for 11 outbreaks during the quarter, with *Salmonella* Typhimurium being the most common serotype. *S.* Typhimurium 44 was responsible for 3 outbreaks, *S.* Typhimurium 170/108 and *S.* Typhimurium 197 were each responsible for 2 outbreaks, and 1 outbreak was caused by *S.* Typhimurium 9. Each of these *S.* Typhimurium outbreaks were either confirmed or suspected to be associated with eating eggs or dishes containing eggs. The other *Salmonella* serotypes causing outbreaks were *S.* Saintpaul, *S.* Bareilly, and *S.* Litchfield. Norovirus was responsible for 5 outbreaks, *Campylobacter* for 3 outbreaks and 1 outbreak was caused by *Vibrio cholerae* O1 Ogawa El Tor.

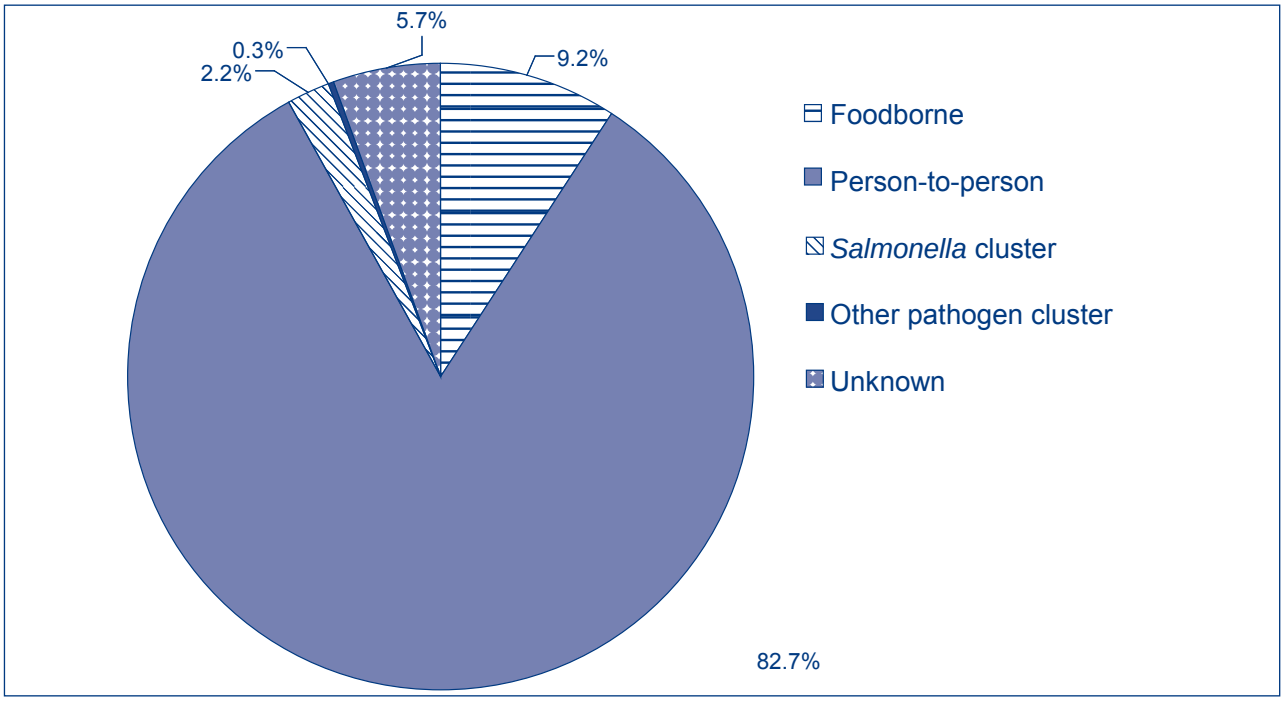
There were 5 toxin-related outbreaks during the quarter including *Clostridium perfringens* intoxication (2 outbreaks), ciguatera fish poisoning (2 outbreaks) and histamine poisoning (1 outbreak). One outbreak was suspected to be either *C. perfringens* or *Bacillus cereus* intoxication. The remaining 8 outbreaks were caused by unknown aetiological agents.

Sixteen outbreaks reported in the quarter were associated with food prepared by restaurants, 4 from contaminated primary produce, 3 with food prepared in private residences, 3 by commercial caterers, and 2 in aged care facilities. Single outbreaks were associated with food prepared by a bakery, institution, takeaway outlet, at a camp and a community-wide event. There was 1 outbreak where the food preparation setting was unknown, as multiple foods could have caused the outbreak.

To investigate these outbreaks, sites conducted 13 cohort studies and 6 case control studies, and collected descriptive data for 14 outbreaks. There was 1 outbreak where no individual patient data were available. Investigators obtained analytical epidemiological evidence in 6 outbreaks, microbiological with analytical epidemiological evidence in 7 outbreaks and microbiological evidence in 1 outbreak. For the remaining 20 outbreaks, investigators obtained descriptive epidemiological evidence implicating the food vehicle or suggesting foodborne transmission.

New South Wales reported 12 outbreaks of foodborne illness during the quarter. The aetiological agent was identified in 5 of the outbreaks, suspected in

Figure 1. Mode of transmission for outbreaks of gastrointestinal illness reported by OzFoodNet sites, 1 October to 31 December 2006



another and not identified in the remaining 6 outbreaks. *Clostridium perfringens* caused gastroenteritis in 80 people after a meal that included roast pork prepared by a takeaway outlet. *C. perfringens* (enterotoxin A) was isolated from stool samples and various leftover food but elevated results from samples of the roast pork suggested that it was the cause of the outbreak.

Vibrio cholerae O1 Ogawa El Tor was responsible for illness among 3 Italian women. The women ate raw whitebait fish imported from Indonesia while preparing a meal.

Campylobacter was responsible for illness among 3 residents of an aged care facility and thought to be due to undercooked chicken.

Salmonella Typhimurium 170/108 affected 47 people attending a hotel management training school. Laboratory and epidemiological evidence indicate that the use of raw egg in a white chocolate mousse was the cause of this outbreak.

The Australian Capital Territory reported 2 outbreaks of foodborne illness during the quarter. Eggs were identified as the cause of an outbreak of *Salmonella* Typhimurium 44 that affected 4 people. All cases ate free-range eggs purchased from a supermarket. One case was able to provide a brand name and these eggs came from a farm in New South Wales. The Australian Capital Territory also reported an outbreak of *S.* Typhimurium 170/108 that affected 13 people who had eaten meals dressed with a home-made mayonnaise containing raw egg. The farm that supplied the eggs was in New South Wales and an on-farm environmental sampling was positive for *S.* Typhimurium 170/108.

Victoria reported 7 outbreaks of foodborne illness during the quarter. Norovirus affected 57 patrons and staff during outbreaks associated with 3 restaurants where ill food handlers were the possible source.

Ten people were infected with *S.* Typhimurium 44 after eating a hazelnut gateau cake made with raw egg mousse filling. No other foods were consumed at the event and a sample of leftover cake was positive for *S.* Typhimurium 44.

During the quarter, Victoria experienced a community-wide outbreak of *S.* Typhimurium 44 suspected to be caused by contaminated eggs (Figure 2).

Clostridium perfringens was identified as the agent responsible for illness among 20 residents of an aged care facility but the source was unable to be identified.

Queensland reported 7 outbreaks of foodborne disease during the quarter. A cluster of 7 cases of *S.* Typhimurium 197 was identified in Queensland around December. All cases had eaten at a local restaurant but no common food vehicle could be identified from the epidemiological investigation. *Salmonella* was not detected in any of the food samples or environmental swabs taken at the restaurant. In a similar incident, another restaurant was linked to an outbreak of *S.* Typhimurium 197 among per-

Table. Outbreaks of foodborne disease reported by OzFoodNet sites,* October to December 2006

State	Month of outbreak	Setting prepared	Infection	Number affected	Evidence	Responsible vehicles
Multi-state	October	Contaminated primary produce	*Salmonella* Saintpaul	79	AM	Rockmelon
	November	Contaminated primary produce	*Salmonella* Litchfield	17	AM	Paw paw
ACT	November	Home	*Salmonella* Typhimurium 44	4	D	Eggs – free-range
	December	Restaurant	*Salmonella* Typhimurium 170/108	13	M	Eggs – free-range
NSW	October	Restaurant	Histamine poisoning	6	D	Yellowtail king fish fillets
		Restaurant	Unknown	5	D	Unknown
	November	Aged care facility	*Campylobacter*	3	AM	Undercooked chicken
		Institution	*Salmonella* Typhimurium 170/108	47	AM	Eggs – white chocolate mousse
		Commercial caterer	Suspected *Bacillus cereus* or *Clostridium perfringens*	14	A	Cooked chicken
		Restaurant	Unknown	15	D	Sandwiches
		Restaurant	Unknown	7	D	Banquet
		Home	*Vibrio cholerae* O1 Ogawa El Tor	3	D	Whitebait
	December	Takeaway	*Clostridium perfringens*	80	AM	Roast pork
		Restaurant	Unknown	24	D	Various Indian dishes – rice, beef madras, butter chicken, lamb rogan josh, veg curry
		Restaurant	Unknown	5	A	Unknown
		Commercial caterer	Unknown	25	D	Unknown
Qld	October	Contaminated primary produce	Ciguatoxin	4	D	Black kingfish
	November	Camp	*Campylobacter*	46	A	On-site water tank
	December	Restaurant	Norovirus	122	A	Chicken pesto & black forest stack
		Restaurant	*Salmonella* Bareilly	4	D	Unknown
		Restaurant	*Salmonella* Typhimurium 197	17	D	Eggs – suspected
		Restaurant	*Salmonella* Typhimurium 197	7	D	Eggs – suspected
		Restaurant	Unknown	9	D	Unknown
SA	December	Commercial caterer	*Campylobacter*	5	A	Chicken dish
		Bakery	*Salmonella* Typhimurium 9	15	AM	Eggs via a bakery product
Vic	October	Restaurant	Norovirus	15	D	Unknown
	November	Contaminated primary produce	Ciguatoxin	2	D	Coral perch or coral trout
		Restaurant	Norovirus	29	D	Unknown
		Restaurant	Norovirus	13	D	Unknown
		Home	*Salmonella* Typhimurium 44	10	AM	Eggs – hazelnut gateau cake made with raw egg mousse filling
	December	Aged care facility	*Clostridium perfringens*	20	D	Unknown
		Community	*Salmonella* Typhimurium 44	43	D	Eggs – suspected
WA	October	Unknown	Unknown	19	D	Unknown
	November	Restaurant	Norovirus	29	A	Salad

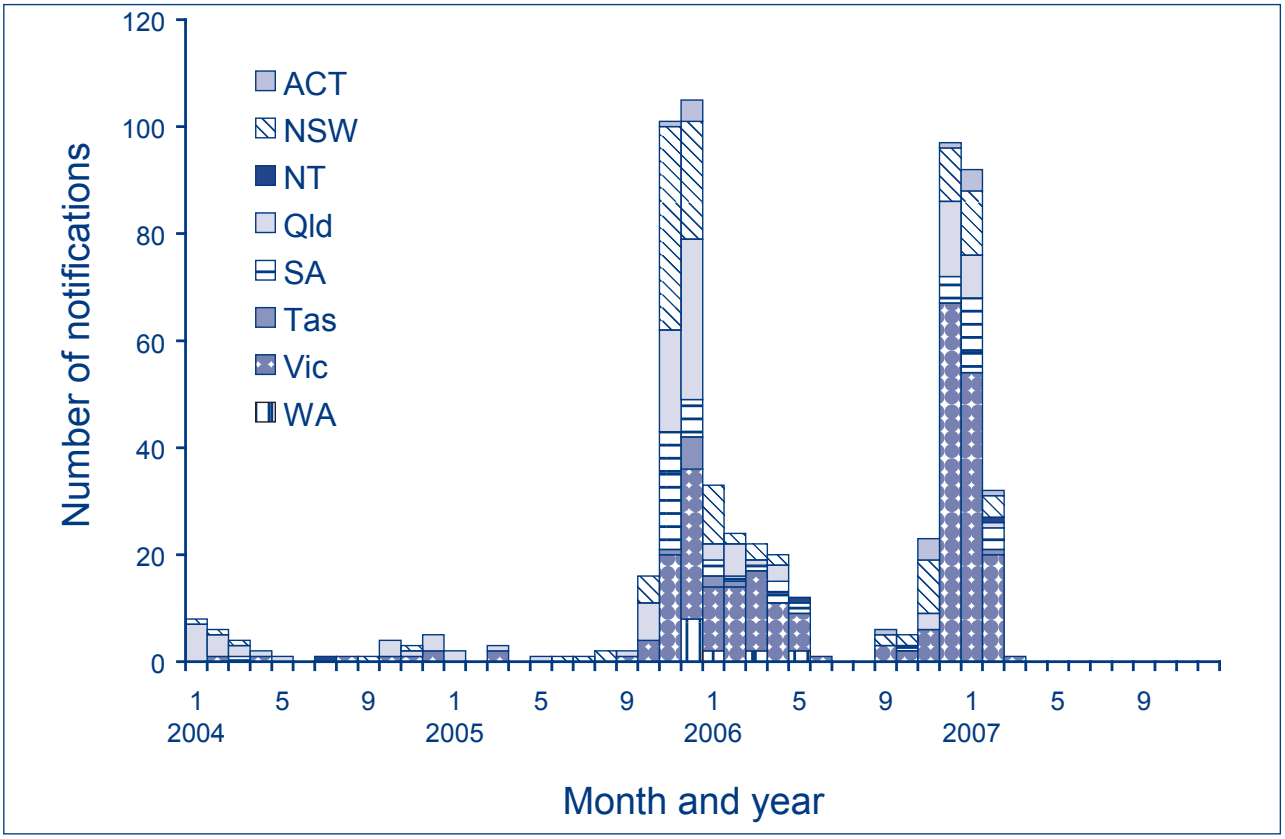
* No foodborne outbreaks were reported in the Northern Territory or Tasmania during the quarter.

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

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

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

Figure 2. *Salmonella* Typhimurium 44 notifications to the National Notifiable Diseases Surveillance System, Australia, 2005 to date, by month of diagnosis and state or territory



Analysed as at 16 March 2007.

sons attending 3 separate work functions on different days during mid-December. In total, 17 cases of salmonellosis were related to this restaurant. Cohort studies were conducted on two of the three functions but no food items were significantly associated with illness. An environmental inspection identified very poor food hygiene standards within the restaurant and it was closed until correction of these deficiencies. Based on the ecology of this phage type in Queensland, a traceback investigation of chicken and egg sources was conducted. Consequently, a strain of *S.* Typhimurium 197, which was not indistinguishable from those infecting cases, was detected from environmental samples taken at the egg farm.

Approximately 31% (122/400) of guests at a function at a Queensland coastal resort were ill in early December. A retrospective cohort study indicated that consumption of the entrée chicken pesto fillets (OR=14.9, 95%CI: 1.7 – 131.2, P=0.002) and the black forest stack (OR=2.9, 95%CI: 1.2 – 6.7, P=0.01) were significantly associated with the illness. Stool samples from 71% (17/24) of those submitted were positive for norovirus.

Western Australia reported 2 outbreaks of foodborne illness during the quarter. An outbreak of gastrointestinal illness was investigated among a group of people (19 cases) who were searching for a missing person in the bush near Perth. It is likely that a component of the meal provided for lunch caused the outbreak, however samples of this food were negative for common disease causing bacteria and toxins.

In November, an outbreak of norovirus affected 29 people attending events at a farm on 2 consecutive days. The food vehicle was a lettuce salad prepared by the family who owned the venue, 5 of whom had gastroenteritis in the preceding week and 3 were confirmed with norovirus infection.

South Australia reported 2 outbreaks during the quarter. Fifteen people infected with *Salmonella* Typhimurium 9 were notified during December 2006. Initial investigation identified 5 people that had consumed products from a bakery. A case control study showed that illness was strongly associated with eating at a bakery (OR=42 95%CI:3.9 – 1065.4, p=0.00009). Microbiological investigations found *S.* Typhimurium 9 on the outside of the eggs stored in the bakery and on several on-farm environmental

samples (the water and feeder, other environmental swabs, and a number of eggs). Cross contamination of multiple bakery products with *S.* Typhimurium 9 from the surface of eggs was suspected as the cause of this outbreak.

Campylobacter affected 5 people who had attended a private party and eaten food provided by a commercial caterer. A cohort study showed that a chicken dish was associated with the illness. Four of the 5 cases had eaten this dish but none had been consumed by the other remaining well party goers.

The Northern Territory and Tasmania did not report any foodborne outbreaks occurring in the fourth quarter of 2006.

Multi-state investigations

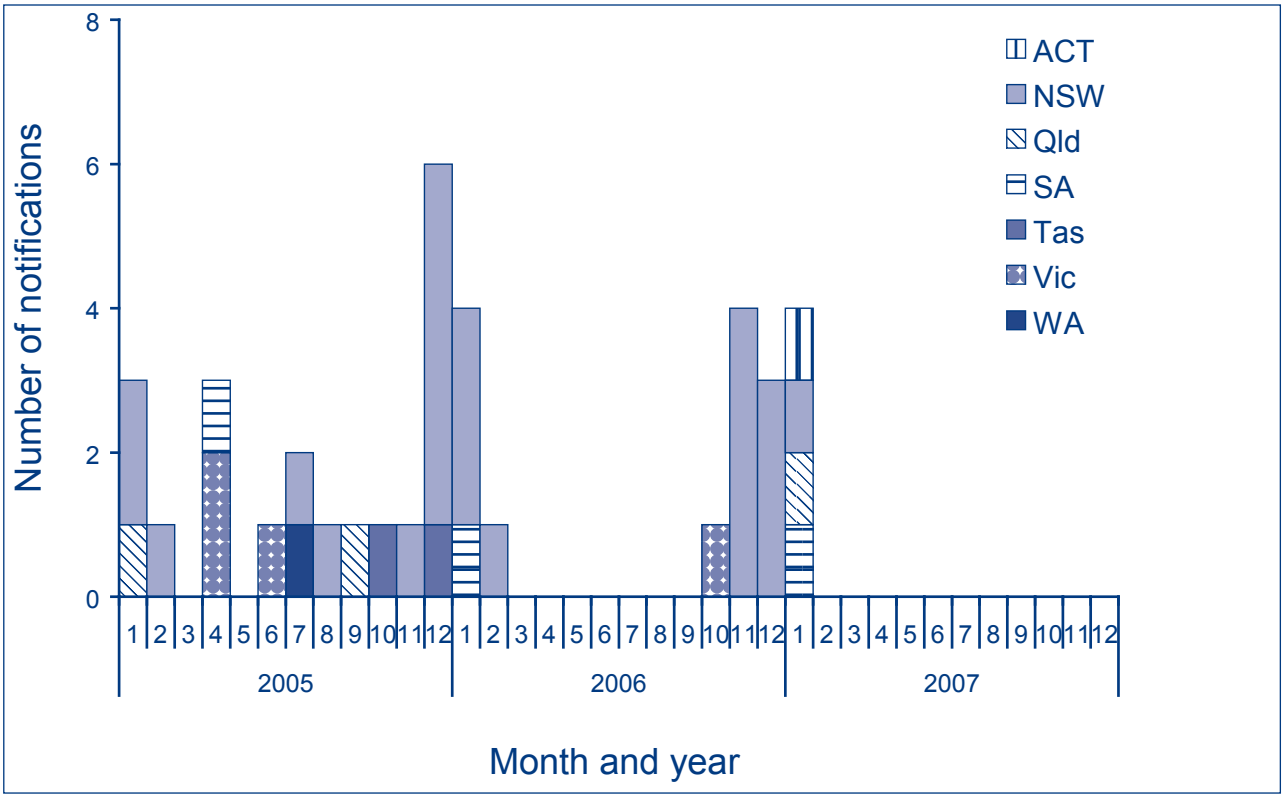
During the quarter there were 2 multi-state investigations of foodborne illness. OzFoodNet investigated a multi-state outbreak of *Salmonella* Saintpaul associated with contaminated rockmelon. Initial hypothesis generating interviews were conducted in New South Wales after routine surveillance detected an increase in notifications of *S.* Saintpaul infections. This process identified that 79% (11/14) of people had consumed rockmelon in the 7 days prior to onset of illness. Comparison data indicated that 28% of controls consumed rockmelon and watermelon in the 7 days prior to interview. The Australian Capital Territory and Victoria reported similar results from interviews with their cases. These findings prompted a case control study across the 3 jurisdictions which found that ill people were around 20 times more likely to have eaten rockmelon (OR 19.5, 95%CI:4.2 – 89.3, p value <0.001). *S.* Saintpaul was detected on rockmelon sold at retail and environmental sampling on farms detected a number of other *Salmonella* serotypes, though not *S.* Saintpaul.

Salmonella Litchfield was responsible for an outbreak in Western Australia and Queensland. A case-control study found an association between the consumption of paw paw and illness (OR=32.8, 95%CI: 2.7–884, p value <0.01). Food sampling showed that paw paw sold in retail outlets in Perth was contaminated with *S.* Litchfield, however the source of the paw paw contamination on the farms was not found.

Cluster investigations

In late 2006, New South Wales reported an increase in haemolytic uraemic syndrome (HUS) and Shiga toxin-producing *E. coli* (STEC) notifications, with 7 HUS and 4 STEC notifications (Figure 3). Three HUS cases had STEC toxin detected in stool specimens collected after the initial clinical diagnosis.

Figure 3. Haemolytic uraemic syndrome notifications reported to the National Notifiable Diseases Surveillance System, Australia, 2003 to date, by month of diagnosis and state or territory



Two were *E. coli* O157 and one was *E. coli* O55. Causative pathogens were not identified for the other 5 cases, although each of the cases reported a history of bloody diarrhoea prior to onset of HUS symptoms. One case died, however the cause of death was attributed to their underlying medical condition. Cases were geographically dispersed throughout New South Wales. The median age of cases was 6 years (range 1–27 years), with males representing 56% of this cluster of cases. OzFoodNet staff in the Hunter/New England area conducted interviews of 8 HUS/STEC cases but the investigation was unable to identify specific food items, supermarkets or suppliers as the source of this outbreak.

New South Wales reported an increase in the number of notifications of patients with *Salmonella* Montevideo during November. The patients resided throughout the Sydney metropolitan area and the Hunter/New England Area. No clustering was identified among cases to date. The investigation is ongoing and new cases will be interviewed using a standard questionnaire in an attempt to identify common exposures.

Comments

Between October and December 2006, all outbreaks of *Salmonella* Typhimurium were suspected to be associated with dishes containing raw or undercooked eggs. This represented 24% (8/34) of all foodborne disease outbreaks during the quarter. Australia does not have S. Enteritidis in egg-laying flocks. S. Enteritidis can cause transovarian infections of chickens to eggs. However, *S.* Typhimurium has caused large outbreaks associated with eggs and the frequency of these outbreaks is a concern to public health agencies.[1,2] Food Standards Australia New Zealand has established a committee to develop a national primary production and processing standard for eggs (http://www.foodstandards.gov.au/thecode/primaryproductionprocessingstandards/).

The 2 multi-state outbreak investigations associated with contaminated primary produce highlight the role of fresh fruits and vegetables in causing foodborne disease outbreaks. Since fresh produce is often eaten without cooking, its outbreak potential is significant once it becomes contaminated. There are many potential points of contamination of fresh produce between the farm and the consumer's table. In the 2 outbreaks reported this quarter, investigations of farms producing rockmelons and paw paws revealed multiple *Salmonella* serotypes from a wide range of environmental samples. Of particular note, the water used to wash the produce during processing at each farm tested positive for various *Salmonella* serotypes. This highlights that any water used to wash or rinse fresh produce should be of potable quality. In each outbreak, the implicated fresh produce tested positive for the infectious agent causing the outbreak, although concentrations were thought to be low and contamination infrequent. This highlights the need for appropriate prevention messages for the public in order to render these products safe to consume. Recent outbreaks in the United States of America[3] further highlight the importance of fresh produce as a cause of human illness.

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.

The OzFoodNet Working Group is *(in alphabetical order)*: Robert Bell (Qld), Caron Bowen (NSW), Barry Combs (SA), Craig Dalton (Hunter New England), Emily Davis (DoHA) Gerard Fitzsimmons (DoHA), Kathleen Fullerton (DoHA) Robyn Gibbs (WA), Joy Gregory (Vic), Gillian Hall (NCEPH), Michelle Harlock (NT), Geoff Hogg (MDU), Martyn Kirk (DoHA), Karin Lalor (Vic), Beth Lord (WA), Tony Merritt (Hunter New England), Sally Munnoch (Hunter New England), Jennie Musto (NSW), Lillian Mwanri (SA), Rhonda Owen (DoHA), Chris Oxenford (ACT), Nancy Pedersen (DAFF), Nevada Pingault (WA), Jane Raupach (SA), Mark Salter (FSANZ), Minda Sarna (WA), Cameron Sault (TAS), Nicola Stephens (Tas), Russell Stafford (Qld), Chris Sturrock (FSANZ, NCEPH), Hassan Vally (NCEPH), Kate Ward (NSW), Tory Worgan (Hunter New England).

Author details

Correspondence: Mr Gerard Fitzsimmons, Epidemiologist, OzFoodNet, Office of Health Protection, Australian Government Department of Health and Ageing, GPO Box 9848, MDP 14, Canberra, ACT 2601. Telephone: +61 2 6289 8124. Facsimile: +61 2 6289 7100. Email: gerard.fitzsimmons@health.gov.au

References

1. OzFoodNet Working Group. Burden and causes of foodborne disease in Australia: Annual report of the OzFoodNet network, 2005. *Commun Dis Intell* 2006;30:278–300.
2. Dalton CB, Gregory J, Kirk MD, Stafford RJ, Givney R, Kraa E, et al. Foodborne disease outbreaks in Australia, 1995 to 2000. *Commun Dis Intell* 2004;28:211–224.
3. Centers for Disease Control and Prevention. Ongoing multistate outbreak of *Escherichia coli* serotype O157:H7 infections associated with consumption of fresh spinach—United States, September 2006. *MMWR Morb Mortal Wkly Rep* 2006;55:1045–1046.

Communicable diseases surveillance

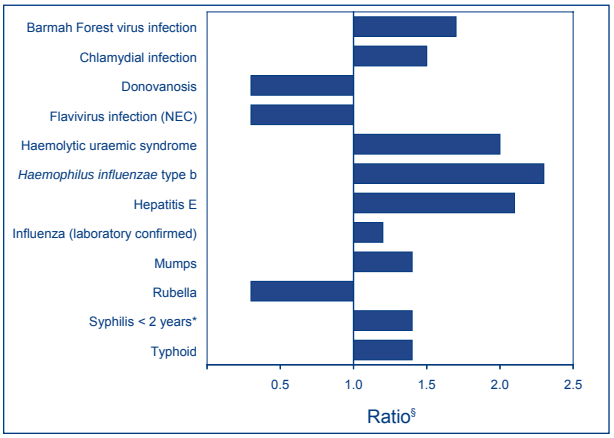
Highlights for 4th quarter, 2006

Communicable diseases surveillance highlights reports 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. The 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 fourth quarter of 2006, compared with the five-year mean for the same period. The following diseases were above the five-year mean: cholera,* *Haemophilus influenzae* type b infection, hepatitis E, haemolytic uraemic syndrome, Barmah Forest virus infection and chlamydial infection. Diseases for which the number of notifications was below the five-year mean for the same period include flavivirus infection (NEC), rubella and donovanosis.

Figure 1. Selected diseases*,† from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 October to 31 December 2006 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.

† Cholera is not shown due to scale issues.

‡ Some Victorian data for this period may be incomplete.

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

Gastrointestinal diseases

Haemolytic uraemic syndrome

There were 9 notifications of haemolytic uraemic syndrome between 1 October and 31 December 2006, which is twice the five-year mean for the fourth quarter. Eight of the reported cases were from New South Wales. The cases were investigated but no links were found.

Four cases were reported as infected with Shiga-like toxin-producing *Escherichia coli*/verotoxin-producing *E. coli*, one of which was identified as serotype O157:H7.

Hepatitis E

There were 5 cases of hepatitis E notified in the fourth quarter of 2006, which was 2.1 times the five-year mean for the period. Six cases were also notified in the previous period, compared to only 2 cases in the fourth quarter of 2005.

Four of the cases were notified in New South Wales. The cases were reported for 3 men and 2 women, ranging in age from 20 to 34 years. Two cases were imported from overseas: one from Bangladesh and the other was a student from India.

* There were 3 cases of cholera for the quarter, which is 15 times the five-year mean. Cholera is not shown in Figure 1 due to scale issues.

Typhoid

There were 21 cases of typhoid in this reporting period, which was 1.4 times the five-year mean. Half of the cases (11) were reported in New South Wales. Twenty cases had information on place of acquisition; 16 cases were acquired overseas and 4 were acquired locally (3 in New South Wales and 1 in Victoria).

Quarantinable diseases

Cholera

Three cases of cholera were notified in the fourth quarter of 2006. The cases were 3 elderly women (aged 71, 71 and 84) in Sydney, New South Wales, who suffered from diarrhoea in November 2006. The infecting organism was identified as toxin-producing *Vibrio cholerae* 01 Ogawa El Tor. The only common exposure among the 3 women was the consumption of raw whitebait. Investigations by the NSW Food Authority found that the whitebait implicated as the vehicle for infection was imported from Indonesia, and a media release advising people to avoid eating raw whitebait was issued. No additional cases of cholera were discovered, and the 3 women all recovered.[1]

These 3 cases represent all cholera notifications reported in Australia in 2006. The average number of cases over the last 5 years is 0.2 cases for the fourth quarter and 3.6 cases per calendar year (ranging from 1 case in 2003 to 5 cases in both 2001 and 2004).

Cholera is one of 7 human diseases subject to quarantine controls in Australia and it is one of the diseases reportable to the World Health Organization. Apart from 1 case of laboratory acquired cholera in 1996, all cases reported since the commencement of the NNDSS in 1991 have been imported.[2]

Sexually transmissible infections

Donovanosis

There was only one notification of donovanosis infection between 1 October and 31 December 2006, which was 0.3 times the five-year mean. There were only 3 cases notified in 2006, compared to an average of 18 cases per year over the previous 5 years. This decline follows the implementation of *The National Donovanosis (Elimination) Eradication Project 2001–2004*. This project led to enhanced surveillance and improved diagnosis and treatment, resulting in declining notifications of donovanosis.[3]

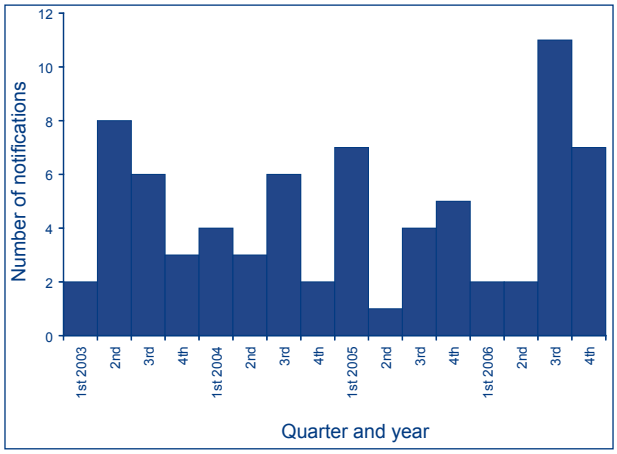
All 3 cases notified in 2006 were Indigenous persons aged between 30 and 60 years; 2 males from the Northern Territory and 1 female from Queensland.

Vaccine preventable diseases

Haemophilus influenzae type b infection

There were 7 notifications of *Haemophilus influenzae* type b (Hib) infection between 1 October and 31 December 2006, which was 2.3 times the five-year mean. This was less than the number notified in the previous period, 1 June to 30 September 2006 (11 cases) (Figure 2).

Figure 2. Notifications of *Haemophilus influenzae* type b infection, 2003 to 2006



Cases came from Queensland (3), New South Wales (2) and the Northern Territory (1). Four of the cases were in females. One of the cases was in an infant aged less than 1 year, with an additional 3 cases in children aged 1 to 5 years.

Indigenous status was recorded for 6 of the 7 cases; 3 notifications were in Indigenous people, including 2 children aged less than 2 years.

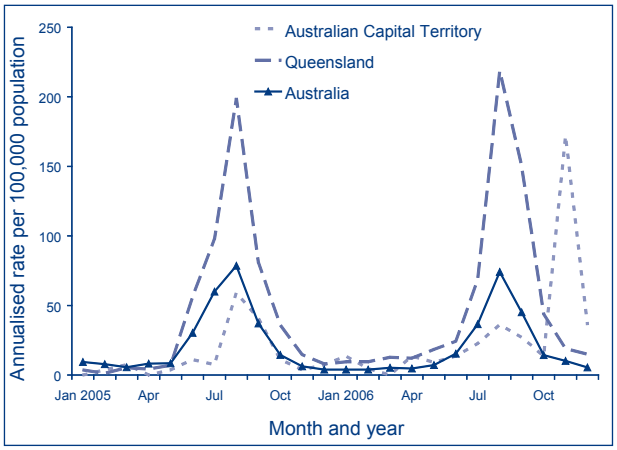
Routine vaccination against Hib became available in Australia in 1993. Vaccination status was available for all 4 of the cases who were eligible for Hib immunisation; 3 cases were fully vaccinated for age, and the other partially vaccinated for age.

Influenza

There were 420 notifications of laboratory-confirmed influenza in the fourth quarter of 2006, which was 1.2 times the five-year mean for the period. Half of the cases (212) were from Queensland. Figure 3 shows the notification rate of influenza in Queensland for 2005 and 2006. The mean age of onset was 43 years and the median age of onset was 46 years (40 years for males and 48 years for females). The highest proportion of cases was reported in children aged less than 5 years (12.9% or 54 cases).

Of particular note was an outbreak of influenza in an aged care facillity in the Australian Capital Territory (Figure 3). Between 11 October and 6 December, 77 people (55 of 132 residents and 22 of 173 staff) in an aged care facility reported symptoms of influenza-like illness. Of these, 19 people (18 residents and 1 staff) were found to have laboratory-confirmed influenza A infection.[4]

Figure 3. Notification rates of laboratory-confirmed influenza, Australian Capital Territory, Queensland and Australia, 2005 to 2006



Of those with laboratory-confirmed influenza A, 6 residents and 2 staff were immunised with the 2006–07 influenza vaccine prior to the outbreak.[4]

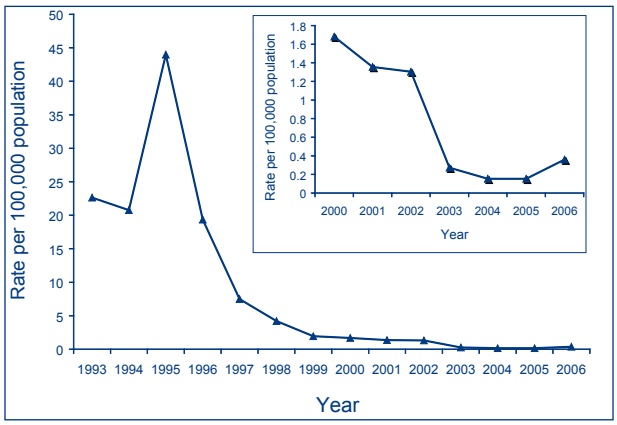
Ten resident deaths were associated with the outbreak. The mean age at death was 88 years and the median 91 years (range of 75 to 100 years). Two of the residents who died were fully vaccinated with the 2006–07 influenza vaccine.[4]

The outbreak control strategy included vaccination clinics, enhanced infection control and isolation of cases. Prophylactic treatment through administration of Oseltamivir was recommended to residents through their medical practitioners and provided to asymptomatic staff. The public health response also included laboratory investigation of suspect cases, social distancing and other measures to assist containment.[4]

Rubella

There were 9 cases of rubella reported for the period 1 October to 31 December 2006, which was 0.3 times the five-year mean. Six cases were reported for males and 3 for females. Rubella is more common in males than females; in 1971, vaccination against rubella was introduced for adolescent girls, but not boys. In 1989 however, with the introduction of the measles-mumps-rubella (MMR) vaccine (infant dose in 1989 and adolescent dose in 1994), both boys and girls are vaccinated against the disease.[5] Following epidemics of rubella in the early 1990s, notification rates have continued to decline (Figure 4). This is partly attributable to the Measles Control Campaign in late 1998.[6] The Measles Control Campaign had three main components: moving the second dose of the MMR vaccine from 10–16 years to 4–5 years; providing catch-up doses to children aged 5–12 years; and sending reminder letters to parents of preschool-aged children.[7]

Figure 4. Rubella notification rates, Australia, 1993 to 2006



Rubella notifications increased from 31 in 2005 to 60 in 2006. This was due to notifications from New South Wales increasing from 10 cases in 2005 to 37 in 2006. The New South Wales cases were mainly from South Eastern and Central Sydney and concentrated in those aged 15 to 44 years, however there was no single identifiable source for the increase in notifications (Mark Bartlett, personal communication).

The 3 female cases were aged 18, 22 and 23 years. Vaccination status was only available for the 18-year-old, who was not vaccinated.

Vectorborne diseases

Barmah Forest virus and Ross River virus infections

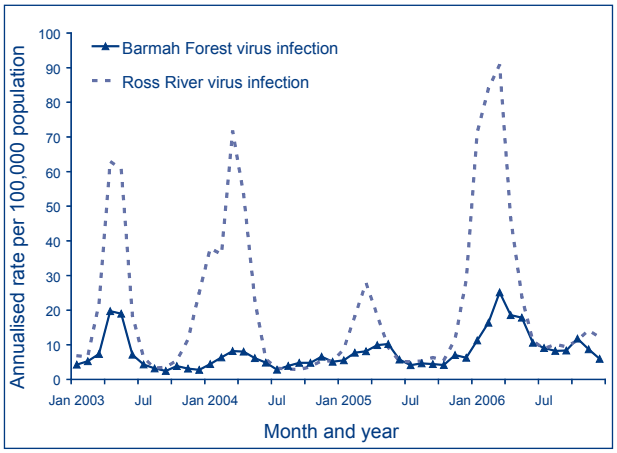
There were 364 notifications of Barmah Forest virus (BFV) infection and 520 notifications of Ross River virus (RRV) infection in the fourth quarter of 2006, which were 1.7 and 1.3 times the five-year mean, respectively. The majority of notifications came from Queensland (60% BFV and 34% RRV), New South Wales (26% BFV and 25% RRV) and Western Australia (22% RRV). While only 16 notifications of BFV and 43 notifications of RRV came

from the Northern Territory, the annualised rates were substantially higher than in other jurisdictions at 31.1 cases per 100,000 population for BFV (compared to 21.6 in Queensland and 5.5 in New South Wales) and 83.5 cases per 100,000 population (compared to 21.9 in Western Australia, 17.4 in Queensland and 7.6 in New South Wales).

Barmah Forest virus infection was reported more often for females than males (224 notifications versus 140). BFV notification rates were highest for males aged 50–59 years and females aged 30–39 years (9.5 and 13.0 cases per 100,000 population, respectively). Similarly, more notifications of Ross River virus infection were reported for females than males (285 and 235 notifications respectively). RRV notification rates were highest in women aged 30–39 years and in men aged 70–79 years (20.2 and 15.8 cases per 100,000 population, respectively).

Figure 5 shows national notification rates for Barmah Forest virus and Ross River virus from 2003 to 2006. Both diseases are seasonal, with notification rates peaking nationally in early autumn. Ross River virus infection rates are consistently higher than those for Barmah Forest virus in the peak season. The rates for both diseases were increased above historical levels in 2006.

Figure 5. Barmah Forest virus and Ross River virus infection notification rates, Australia, January 2003 to December 2006



References

1. NSW Public Health Bulletin, January 2007, in press.
2. A case of cholera [case report]. *Commun Dis Intell* 1998;22:154.
3. Department of Health and Ageing. National Sexually Transmissible Infections Strategy 2005–2008. Available from: http://www.health.gov.au/internet/wcms/publishing.nsf/content/health-pubhlth-strateg-hiv_hepc-hiv-index.htm/$file/sti_strategy.pdf Accessed on 5 February 2006.
4. Deeble M, Guest C. Influenza, nursing home – Australia (Canberra)(03). Available from: http://www.promedmail.org/pls/promed/f?p=2400:1001:::NO::F2400_P1001_BACK_PAGE,F2400_P1001_PUB_MAIL_ID:1000%2C35444 Accessed on 26 February 2006.
5. National Health and Medical Research Council. The Australian Immunisation Handbook. 8th edn. Canberra: Commonwealth of Australia; 2003. p288. Available from: http://www9.health.gov.au/immhandbook/pdf/handbook.pdf Accessed on 28 February 2006.
6. National Health and Medical Research Council. The Australian Immunisation Handbook. 8th edn. Canberra: Commonwealth of Australia; 2003. p214. Available from: http://www9.health.gov.au/immhandbook/pdf/handbook.pdf Accessed on 28 February 2006.
7. Turnbull F, Burgess M, Achat H, McIntyre P, Gidding H, Hull B, Wang H. Australian Measles Control Campaign 1998 Evaluation Report. Available from: http://www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/530B21812D3C4705CA25719D00183412/$File/archive-measles-control-evaluation.pdf Accessed on 28 February 2006.

Acknowledgments

Thanks to Mark Bartlett, Jennie Musto and Reimke Kampen for their assistance.

Tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 32,497 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 October and 31 December 2006 (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 October to 31 December 2006, by date of onset*

Disease	State or territory								Total 4th quarter 2006[†]	Total 3rd quarter 2006	Total 4th quarter 2005	Last 5 years mean 4th quarter	Year to date 2006	Last 5 years YTD mean	Ratio[‡]
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis B (incident)	5	14	4	12	2	3	28	12	80	66	53	338.6	297	71.0	1.1
Hepatitis B (unspecified)	20	757	56	234	73	12	402	199	1,753	1,905	1,468	6,551.0	6,663	1,575.4	1.1
Hepatitis C (incident)	13	2	1	0	10	2	43	24	95	92	90	499.0	428	117.8	0.8
Hepatitis C (unspecified)	38	1,577	58	756	130	68	609	265	3,501	3,681	2,916	14,746.0	13,189	3,494.2	1.0
Hepatitis D	0	2	0	2	0	0	1	0	5	10	6	25.6	31	4.2	1.2
Gastrointestinal diseases															
Botulism	0	0	0	1	0	0	0	0	1	0	0	1.3	1	0.0	NA
Campylobacteriosis[§]	117	NN	59	1,021	882	204	1,549	540	4,372	3,898	4,987	15,733.2	15,365	4,569.2	1.0
Cryptosporidiosis[‖]	0	123	25	81	36	6	76	76	423	305	792	2,211.2	3,181	447.6	0.9
Haemolytic uraemic syndrome	0	8	0	0	0	0	1	0	9	0	8	13.4	14	4.6	2.0
Hepatitis A	0	10	1	3	0	3	11	23	51	56	71	402.2	277	97.8	0.5
Hepatitis E	0	4	0	0	0	0	1	0	5	6	2	19.4	22	2.4	2.1
Listeriosis	1	4	0	2	1	0	2	3	13	14	18	63.2	59	16.2	0.8
Salmonellosis (NEC)	48	569	122	580	137	40	381	206	2,083	1,242	2,446	7,660.8	8,250	1,954.0	1.1
Shigellosis	0	20	30	15	10	0	23	16	114	103	168	553.4	542	125.4	0.9
SLTEC, VTEC[¶]	0	2	0	4	10	0	4	0	20	16	24	58.4	69	15.4	1.3
Typhoid	0	10	0	1	1	1	5	2	20	15	9	65.4	75	14.0	1.4
Quarantinable diseases															
Cholera	0	3	0	0	0	0	0	0	3	0	0	3.6	3	0.2	15.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	NA
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	NA
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	NA
Tularemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	NA
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	NA
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	NA

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 October to 31 December 2006, by date of onset,* *continued*

Disease	State or territory								Total 4th quarter 2006†	Total 3rd quarter 2006	Total 4th quarter 2005	Last 5 years mean 4th quarter	Year to date 2006	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection**	212	2,881	496	2,955	750	264	2,462	1,436	11,456	11,413	9,964	30,656.8	46,424	7,619.2	1.5
Donovanosis	0	0	1	0	0	0	0	0	1	0	3	17.6	3	3.8	0.3
Gonococcal infection	9	379	370	320	73	0	299	395	1,845	1,899	2,070	6,960.2	8,517	1,707.2	1.1
Syphilis (all)	7	288	50	78	6	2	190	48	669	653	532	2,087.8	2,592	522.4	1.3
Syphilis < two years duration	0	42	35	41	0	0	76	21	215	175	146	622.5	747	154.0	1.4
Syphilis >two years or unspecified duration	7	246	15	37	6	2	114	27	454	478	386	1,838.8	1,845	460.8	1.0
Syphilis - congenital	0	0	1	0	0	0	0	0	1	1	3	16.0	12	4.0	0.3
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0.0	NA
Haemophilus influenzae type b	0	2	1	3	0	0	1	0	7	11	5	20.4	22	3.0	2.3
Influenza (laboratory confirmed)‖	49	90	12	212	12	3	18	24	420	2,166	416	3,032.8	3,145	361.4	1.2
Measles	0	3	0	1	0	0	3	3	10	2	2	64.2	125	15.0	0.7
Mumps	0	18	0	7	5	0	2	5	37	90	38	121.0	260	25.8	1.4
Pertussis	13	372	6	492	142	9	150	30	1,214	4,563	2,805	8,035.8	10,921	2,568.8	0.5
Pneumococcal disease (invasive)‖	6	101	4	41	19	9	70	34	284	536	342	2,117.4	1,432	472.4	0.6
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	NA
Rubella	1	5	0	1	1	0	0	1	9	26	3	127.2	58	33.0	0.3
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	0	1.4	0	0.2	0.0
Tetanus	0	0	0	0	0	0	0	0	0	0	1	3.6	1	1.0	0.0
Varicella infections (chickenpox)	0	NN	73	178	297	4	0	178	730	416	13	NA	1,482	NA	NA
Varicella infections (unspecified)	0	NN	2	771	120	4	0	151	1,048	939	141	NA	3,705	NA	NA
Varicella zoster infections	0	NN	29	141	157	15	0	156	498	314	7	NA	1,147	NA	NA
Vectorborne diseases															
Barmah Forest virus infection	0	93	16	218	15	0	0	22	364	357	296	1,169.4	2,108	209.0	1.7
Dengue	0	9	4	13	4	0	2	1	33	38	48	347.4	184	66.6	0.5
Flavivirus infection (NEC)	0	0	0	3	0	0	0	0	3	4	4	62.0	32	10.2	0.3
Japanese encephalitis virus‖	0	0	0	0	0	0	0	0	0	0	0	0.4	0	0.0	NA
Kunjin virus‖	0	0	0	0	0	0	0	0	0	0	0	7.2	3	0.6	0.0
Malaria	1	30	14	44	4	5	20	29	147	209	143	634.0	780	138.2	1.1
Murray Valley encephalitis virus‖	0	0	0	0	0	0	0	0	0	0	0	2.2	1	0.0	NA
Ross River virus infection	0	129	43	176	47	1	12	112	520	395	777	3,057.4	5,472	405.2	1.3

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 October to 31 December 2006, by date of onset,* *continued*

Disease	State or territory								Total 4th quarter 2006†	Total 3rd quarter 2006	Total 4th quarter 2005	Last 5 years mean 4th quarter	Year to date 2006	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax‖	0	0	0	0	0	0	0	0	0	0	0	0.0	1	0.0	NA
Australian bat lyssavirus‖	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	NA
Brucellosis	0	4	0	7	0	0	0	0	11	14	18	31.8	44	10.8	1.0
Leptospirosis	0	1	0	13	0	1	2	1	18	19	28	169.0	145	29.8	0.6
Lyssavirus unspecified‖	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	NA
Ornithosis	0	19	0	0	0	0	13	2	34	43	31	190.6	160	42.6	0.8
Q fever	0	45	1	42	1	0	4	2	95	114	83	574.4	393	141.4	0.7
Other bacterial infections															
Legionellosis	0	10	0	10	19	1	16	34	90	81	87	321.8	344	87.4	1.0
Leprosy	0	0	0	0	0	0	0	0	0	2	1	7.4	5	1.2	0.0
Meningococcal infection††	1	21	1	12	6	1	19	5	66	111	105	547.6	319	129.4	0.5
Tuberculosis	6	125	7	45	24	1	96	33	337	331	318	1,143.0	1,232	325.6	1.0
Total	547	7,730	1,487	8,495	2,995	659	6,515	4,068	32,497	36,156	31,342	110,507.9	139,539	27,477.6	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 >2years 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'.

‖ Notifiable from January 2001 only. Ratio and mean calculations are based on the last five years.

¶ 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.

Table 3. Notification rates of diseases, 1 October to 31 December 2006, 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)	6.1	0.8	7.8	1.2	0.5	2.5	2.2	2.3	1.6
Hepatitis B (unspecified)	24.4	44.4	108.8	23.2	18.8	9.8	31.7	39.0	34.1
Hepatitis C (incident)	15.8	0.1	1.9	0.0	2.6	1.6	3.4	4.7	1.8
Hepatitis C (unspecified)	46.3	92.5	112.7	74.9	33.5	55.7	48.0	51.9	68.1
Hepatitis D	0.0	0.1	0.0	0.2	0.0	0.0	0.1	0.0	0.1
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis†	142.6	NN	114.6	101.2	227.3	167.0	122.0	105.7	127.3
Cryptosporidiosis	0.0	7.2	48.6	8.0	9.3	4.9	6.0	14.9	8.2
Haemolytic uraemic syndrome	0.0	0.5	0.0	0.0	0.0	0.0	0.1	0.0	0.2
Hepatitis A	0.0	0.6	1.9	0.3	0.0	2.5	0.9	4.5	1.0
Hepatitis E	0.0	0.2	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Listeriosis	1.2	0.2	0.0	0.2	0.3	0.0	0.2	0.6	0.3
Salmonellosis (NEC)	58.5	33.4	237.0	57.5	35.3	32.7	30.0	40.3	40.5
Shigellosis	0.0	1.2	58.3	1.5	2.6	0.0	1.8	3.1	2.2
SLTEC, VTEC‡	0.0	0.1	0.0	0.4	2.6	0.0	0.3	0.0	0.4
Typhoid	0.0	0.6	0.0	0.1	0.3	0.8	0.4	0.4	0.4
Quarantinable diseases									
Cholera	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1
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§	258.4	169.0	963.5	292.9	193.3	216.1	193.9	281.2	223.0
Donovanosis	0.0	0.0	1.9	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	11.0	22.2	718.7	31.7	18.8	0.0	23.6	77.3	35.9
Syphilis (all)	8.5	16.9	97.1	7.7	1.5	1.6	15.0	9.4	13.0
Syphilis <2 years duration	0.0	2.5	68.0	4.1	0.0	0.0	6.0	4.1	4.2
Syphilis >2 years or unspecified duration	8.5	14.4	29.1	3.7	1.5	1.6	9.0	5.3	8.8
Syphilis - congenital	0.0	0.0	1.9	0.0	0.0	0.0	0.0	0.0	0.0
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	1.9	0.3	0.0	0.0	0.1	0.0	0.1
Influenza (laboratory confirmed)	59.7	5.3	23.3	21.0	3.1	2.5	1.4	4.7	8.2
Measles	0.0	0.2	0.0	0.1	0.0	0.0	0.2	0.6	0.2
Mumps	0.0	1.1	0.0	0.7	1.3	0.0	0.2	1.0	0.7
Pertussis	15.8	21.8	11.7	48.8	36.6	7.4	11.8	5.9	23.6
Pneumococcal disease (invasive)	7.3	5.9	7.8	4.1	4.9	7.4	5.5	6.7	5.5
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	1.2	0.3	0.0	0.1	0.3	0.0	0.0	0.2	0.2
Rubella - congenital	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 October to 31 December 2006, by state or territory, (annualised rate per 100,000 population), *continued*

Disease*	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vaccine preventable diseases, continued									
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Varicella infections (chickenpox)	0.0	NN	141.8	17.6	76.5	3.3	0.0	34.9	21.3
Varicella infections (unspecified)	0.0	NN	3.9	76.4	30.9	3.3	0.0	29.6	30.5
Varicella zoster infections	0.0	NN	56.3	14.0	40.5	12.3	0.0	30.5	14.5
Vectorborne diseases									
Barmah Forest virus infection	0.0	5.5	31.1	21.6	3.9	0.0	0.0	4.3	7.1
Dengue	0.0	0.5	7.8	1.3	1.0	0.0	0.2	0.2	0.6
Flavivirus infection (NEC)	0.0	0.0	0.0	0.3	0.0	0.0	0.0	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	1.8	27.2	4.4	1.0	4.1	1.6	5.7	2.9
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	0.0	7.6	83.5	17.4	12.1	0.8	0.9	21.9	10.1
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	0.2	0.0	0.7	0.0	0.0	0.0	0.0	0.2
Leptospirosis	0.0	0.1	0.0	1.3	0.0	0.8	0.2	0.2	0.4
Lyssavirus unspecified	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	1.1	0.0	0.0	0.0	0.0	1.0	0.4	0.7
Q fever	0.0	2.6	1.9	4.2	0.3	0.0	0.3	0.4	1.8
Other bacterial infections									
Legionellosis	0.0	0.6	0.0	1.0	4.9	0.8	1.3	6.7	1.8
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection‖	1.2	1.2	1.9	1.2	1.5	0.8	1.5	1.0	1.3
Tuberculosis	7.3	7.3	13.6	4.5	6.2	0.8	7.6	6.5	6.6

* 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.

There were 2,699 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 October to 31 December 2006 (Tables 4 and 5).

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 October to 31 December 2006, and total reports for the year†

	State or territory								This period 2006	This period 2005	Year to date 2006	Year to date 2005
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Mealses virus	0	0	0	0	0	0	3	0	3	4	57	8
Mumps virus	0	0	0	0	0	0	1	0	1	13	26	38
Hepatitis viruses												
Hepatitis A virus	0	2	0	0	0	0	0	5	7	15	31	53
Hepatitis D virus	0	0	0	0	2	0	0	0	2	2	7	14
Hepatitis E virus	0	0	0	0	0	0	1	0	1	1	6	12
Arboviruses												
Ross River virus	0	1	10	8	11	0	3	7	40	119	1,061	452
Barmah Forest virus	0	1	0	12	8	0	0	0	21	27	287	185
Flavivirus (unspecified)	0	0	0	4	0	0	0	0	4	8	47	37
Adenoviruses												
Adenovirus not typed/ pending	2	69	0	0	26	0	28	0	125	182	615	680
Herpes viruses												
Herpes virus type 6	0	0	0	0	0	0	2	0	2	1	4	2
Cytomegalovirus	4	40	0	6	50	1	20	0	121	313	859	1,042
Varicella-zoster virus	0	24	0	48	54	1	10	0	137	388	1,034	1,499
Epstein-Barr virus	0	2	22	36	76	0	6	115	257	605	1,440	2,148
Other DNA viruses												
Poxvirus group not typed	0	0	0	0	0	0	1	0	1	0	3	2
Parvovirus	0	0	0	16	14	1	2	0	33	79	182	202
Picornavirus family												
Coxsackievirus A9	0	5	0	0	0	0	0	0	5	1	16	3
Echovirus type 34	0	1	0	0	0	0	0	0	1	0	1	0
Echovirus type 3	0	1	0	0	0	0	0	0	1	0	3	0
Rhinovirus (all types)	0	48	0	0	2	0	0	0	50	83	192	329
Enterovirus not typed/ pending	0	6	0	0	0	1	0	0	7	46	101	187
Ortho/paramyxoviruses												
Influenza A virus	0	17	0	3	9	0	7	0	36	61	336	708
Influenza B virus	0	4	0	0	2	0	0	0	6	27	172	257
Parainfluenza virus type 2	0	1	0	0	1	0	0	0	2	3	14	49
Parainfluenza virus type 3	0	50	0	4	25	0	24	0	103	116	217	390
Respiratory syncytial virus	0	32	0	6	5	2	11	0	56	129	1,803	1,679
Other RNA viruses												
HTLV-1	0	0	0	0	2	0	0	0	2	3	6	9
Rotavirus	0	157	0	0	95	13	132	1	398	271	1,267	1,270
Norwalk agent	0	5	0	0	0	0	423	0	428	104	1,538	267

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 October to 31 December 2006, and total reports for the year,† *continued*

	State or territory								This period 2006	This period 2005	Year to date 2006	Year to date 2005
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other pathogens												
Chlamydia trachomatis not typed	3	112	0	119	197	13	11	0	455	1,271	3,867	5,049
Chlamydia psittaci	0	0	0	0	0	0	17	0	17	15	60	53
Chlamydia spp typing pending	0	1	0	0	0	0	0	0	1	0	1	0
Mycoplasma pneumoniae	0	4	3	20	24	5	40	29	125	382	1,026	1,309
Mycoplasma hominis	0	3	0	0	0	0	0	0	3	3	23	7
Coxiella burnetii (Q fever)	0	0	1	0	5	0	4	0	10	41	103	162
Rickettsia tsutsugamushi	0	0	0	0	1	0	1	0	2	25	25	71
Rickettsia - spotted fever group	0	0	0	0	1	0	0	0	1	58	86	236
Streptococcus group A	0	0	0	24	0	0	16	0	40	178	369	609
Bordetella pertussis	0	5	0	14	56	0	10	0	85	406	1,309	1,573
Legionella pneumophila	0	0	0	0	0	0	3	0	3	6	28	23
Legionella longbeachae	0	0	0	0	0	0	6	0	6	14	21	51
Legionella species	0	0	0	0	0	0	1	0	1	1	1	1
Cryptococcus species	0	0	0	1	1	0	0	0	2	12	21	41
Leptospira species	0	0	0	1	1	0	0	0	2	10	18	33
Treponema pallidum	0	40	0	28	25	0	0	0	93	251	781	1,086
Toxoplasma gondii	0	0	0	0	2	0	1	0	3	14	39	45
Total	9	631	36	350	695	37	784	157	2,699	5,288	19,103	21,871

* 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 October to 31 December 2006*

State or territory	Laboratory	October 2006	November 2006	December 2006	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	116	55		171
	New Children's Hospital, Westmead	116	79	36	231
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	27	18	5	50
	South West Area Pathology Service, Liverpool	103	46	24	173
Queensland	Queensland Medical Laboratory, West End	1	368	1	370
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	38	655	2	695
Tasmania	Northern Tasmanian Pathology Service, Launceston	14	15	6	35
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Monash Medical Centre, Melbourne	5	10	11	26
	Royal Children's Hospital, Melbourne	153	52	15	220
	Victorian Infectious Diseases Reference Laboratory, Fairfield	151	314	73	538
Western Australia	PathCentre Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	67	80	43	190
Total		791	1,692	216	2,699

* The complete list of laboratories reporting for the 12 months, January to December 2006, 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 Research and Health Promotion Unit of the Royal Australian College of General Practitioners operates the Australian Sentinel Practice Research Network (ASPREN). ASPREN is a network of general practitioners who report presentations of defined medical conditions each week. The aim of ASPREN is to provide an indicator of the burden of disease in the primary health setting and to detect trends in consultation rates.

There are currently about 40 general practitioners participating in the network from all states and territories. Seventy-five per cent of these are in metropolitan areas and the remainder are rural based. Between 3,000 and 4,000 consultations are recorded each week.

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published.

In 2006, six conditions are being monitored, four of which are related to communicable diseases. These include influenza, gastroenteritis, varicella and shingles. Definitions of these conditions were published in Commun Dis Intell *2007;31:162.*

Data on influenza-like illness and gastroenteritis from 1 October to 31 December 2006 compared with 2005 are shown as the rate per 1,000 consultations in Figures 1 and 2, respectively.

Figure 1. Consultation rates for influenza-like illness, ASPREN, 1 January to 31 December 2006, by week of report

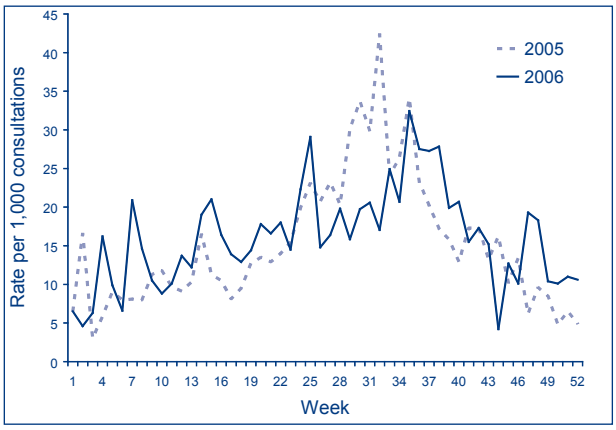
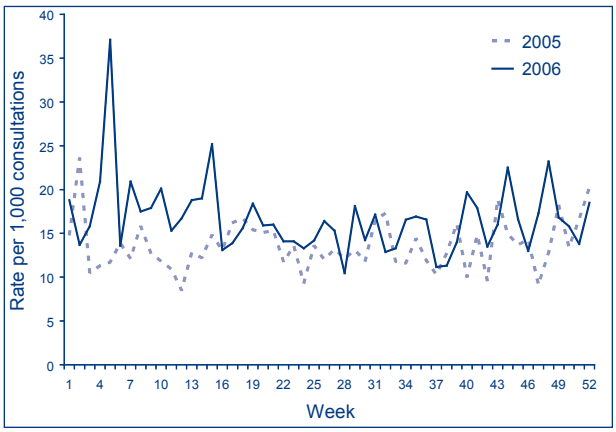


Figure 2. Consultation rates for gastroenteritis, ASPREN, 1 January to 31 December 2006, by week of report



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 July to 30 September 2005, at 24 months of age for the cohort born between 1 July to 30 September 2004, and at 6 years of age for the cohort born between 1 July to 30 September 2000 according to the National Immunisation Program Schedule.

For information about the Australian Childhood Immunisation Register see Surveillance systems reported in CDI, *published in* Commun Dis Intell *2007;31:165 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.

Reporting period 1 July to 30 September 2006

Immunisation coverage for children 'fully immunised' at 12 months of age for Australia increased marginally by 0.4 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 no significant movements in coverage for individual vaccines by state or territory.

Immunisation coverage for children 'fully immunised' at 24 months of age for Australia increased marginally from the last quarter by 0.2 percentage points to 92.4% (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 'fully immunised' at 24 months of age has been higher than the 12 months coverage estimate since the 18 month DTPa booster was removed from the immunisation schedule in September 2003.

It is also notable that, for the 2 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% and 95.1% respectively at 24 months versus 92.2% and 92.0% 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 is likely to be a minimum estimate.

Table 3 shows immunisation coverage estimates for children at 6 years of age for Australia and by state or territory. For the second consecutive quarter, 'fully immunised' coverage for Australia increased significantly by 1.8 percentage points (a total increase of 5.3 percentage points in 2 quarters) and is now at the highest level ever recorded since coverage at 6 years of age was first reported in early 2003. Coverage increased in all jurisdictions and for all individual vaccines with the greatest increase in the Northern Territory and Western Australia, by 5.9 and 4 percentage points, respectively. A possible factor in this increase in coverage at 6 years of age is the introduction of the multi-valent combination vaccine DTP-IPV onto the schedule in November 2005, reducing the number of vaccines to be recorded from three to two. Other factors which may have had an impact at the local level include promotional campaigns centred around child care or school entry, or data cleaning activities.

Table 1. Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 July to 30 September 2005; assessment date 31 December 2006

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,071	23,311	909	14,333	4,550	1,564	16,508	6,745	68,991
Diphtheria, tetanus, pertussis (%)	92.2	92.0	92.8	91.4	92.1	94.3	92.9	90.8	92.0
Poliomyelitis (%)	92.2	91.8	92.8	91.4	92.0	94.0	92.8	90.8	92.0
Haemophilus influenzae type b (%)	96.1	95.3	96.4	93.7	94.8	96.4	95.1	94.5	94.8
Hepatitis B (%)	96.1	95.3	96.6	93.5	94.5	96.4	95.0	94.3	94.7
Fully immunised (%)	91.9	91.5	92.3	90.1	91.1	94.0	91.7	90.2	91.2
Change in fully immunised since last quarter (%)	+1.2	+0.6	+1.7	-0.3	+0.6	+0.2	+0.3	+0.9	+0.4

Table 2. Percentage of children immunised at 2 years of age, preliminary results by disease and state or territory for the birth cohort 1 July to 30 September 2004; assessment date 31 December 2006*

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,077	22,296	844	13,513	4,330	1,509	16,194	6,505	66,268
Diphtheria, tetanus, pertussis (%)	95.5	95.0	96.7	94.7	94.9	97.0	96.1	94.0	95.2
Poliomyelitis (%)	95.5	94.9	96.5	94.6	94.9	97.0	96.0	94.0	95.1
Haemophilus influenzae type b (%)	94.9	93.7	95.1	93.5	93.7	96.0	94.8	92.7	93.9
Measles, mumps, rubella (%)	94.5	93.7	96.2	93.5	93.9	95.0	95.0	92.7	94.0
Hepatitis B(%)	95.9	95.8	97.5	95.5	95.9	97.2	96.4	94.7	95.8
Fully immunised (%)	93.5	92.1	94.4	91.8	92.4	94.5	93.6	90.8	92.4
Change in fully immunised since last quarter (%)	-0.3	+0.7	-0.1	+0.2	+1.2	+0.7	-0.0	-0.5	+0.2

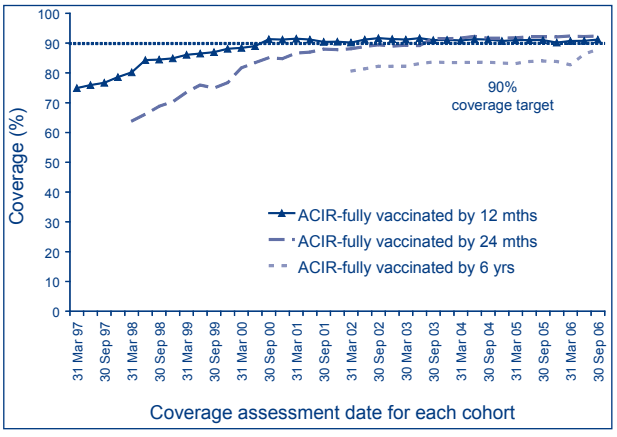
* The 12 months age data for this cohort was published in *Commun Dis Intell* 2006;30:157.

Table 3. Percentage of children immunised at 6 years of age, preliminary results by disease and state or territory for the birth cohort 1 July to 30 September 2000; assessment date 31 December 2006

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,064	22,649	815	13,963	4,599	1,569	16,277	6,861	67,797
Diphtheria, tetanus, pertussis (%)	89.7	88.8	88.8	88.4	87.6	89.9	90.8	85.6	88.8
Poliomyelitis (%)	90.1	88.7	88.8	88.6	87.5	89.9	90.8	85.6	88.8
Measles, mumps, rubella (%)	90.2	88.8	88.8	88.6	87.2	89.7	90.8	85.6	88.8
Fully immunised (%)[1]	89.4	87.9	88.1	87.6	86.6	89.3	90.1	84.7	88.0
Change in fully immunised since last quarter (%)	+1.8	+1.7	+5.9	+1.4	+2.0	+0.7	+1.1	+4.0	+1.8

Figure 3 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 2 years for all age groups. The recent increase in coverage at 6 years of age, described in the previous paragraph, is apparent in the Figure. It should be noted that, currently, coverage for the vaccines added to the National Immunisation Program since 2003 (pneumococcal conjugate at 2, 4 and 6 months; meningococcal C conjugate at 12 months; and varicella at 18 months) are not included in the coverage estimates at 12 or 24 months of age.

Figure 3. Trends in vaccination coverage, Australia, 1997 to 2006, by age cohorts



Gonococcal surveillance

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

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various States and Territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5% 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;31:163.*

Reporting period 1 July to 30 September 2006

The AGSP laboratories received a total of 869 gonococcal isolates of which 854 remained viable for susceptibility testing. This was about 10% less than the 968 gonococci reported for the same period in 2005. About one third of this total was from New South Wales, 21% from Victoria, 16% each from the Northern Territory and Queensland, 11% from Western Australia and 5% from South Australia. There were 2 isolates each from Tasmania and the Australian Capital Territory.

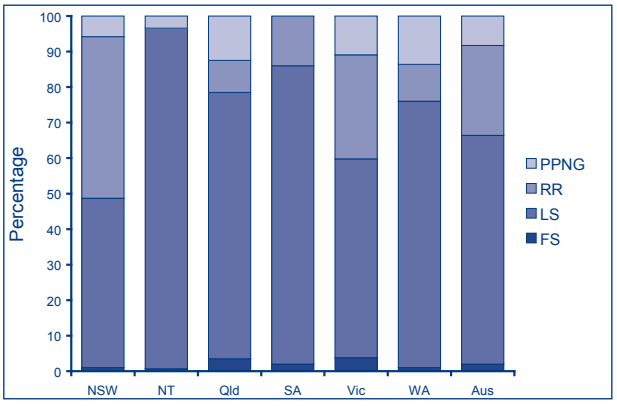
Penicillins

In this quarter, 303 (35.5%) of the 854 isolates examined were penicillin resistant by one or more mechanisms. Seventy-six (8.9%) were penicillinase producing *N. gonorrhoeae* (PPNG) and 227 (26.6%) resistant by chromosomal mechanisms, (CMRNG). The proportion of all strains resistant to the penicillins by any mechanism ranged from 4.3% in the Northern Territory to 50% in New South Wales and Victoria. High rates of penicillin resistance were also found in South Australia (37.8%) and Queensland (31%), with a lower rate (13.4%) in Western Australia.

Figure 4 shows the proportions of gonococci fully sensitive (MIC ≤ 0.03 mg/L), less sensitive (MIC 0.06–0.5 mg/L), relatively resistant (MIC ≥1 mg/L) or else penicillinase producing (PPNG) aggregated for Australia and by state or territory. A high proportion those strains classified as PPNG or else resistant by chromosomal mechanisms fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

In New South Wales, most of the penicillin resistance was due to CMRNG (117, 42.4%) with 21 PPNG (7.6%). A similar distribution was present in Victoria with 20 PPNG (11%) and 70 CMRNG (38.7%). This disparity was not quite as pronounced in other centres. The proportion of CMRNG in Queensland increased to 17% while 14% were PPNG. In South Australia, 16% were PPNG and 21% were CMRNG. In Western Australia, PPNG and CMRNG each accounted for 6.7% of all 89 isolates. PPNG were also present in Tasmania and the Northern Territory (1 and 4 isolates respectively). No PPNG were detected in the Australian Capital Territory. CMRNG were present in Tasmania (1 isolate), the Northern Territory (2) and there was a single CMRNG from the Australian Capital Territory.

Figure 4. Categorisation of gonococci isolated in Australia, 1 July to 30 September 2006, 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

Four isolates with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected. Three were found in Queensland and 1 in New South Wales. All 4 isolates were penicillin resistant by chromosomal mechanisms and were also quinolone resistant (ciprofloxacin MICs 4–16 mg/L). It is emphasised that no treatment failures have been documented locally when a 250 mg IM dose of ceftriaxone has been used.

Spectinomycin

All isolates susceptible to this injectable agent.

Quinolone antibiotics

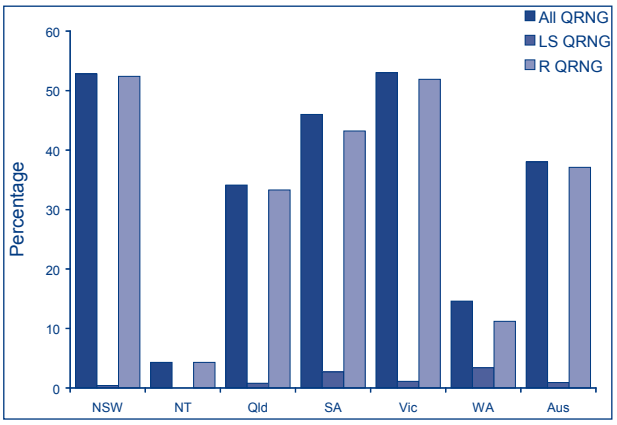
The number (325) and percentage (38%) of quinolone resistant *N. gonorrhoeae* (QRNG) detected in this quarter was the highest proportion of QRNG found in this program to date. In the third quarter of 2004 there were 200 QRNG; 24% of all gonococci tested, and in this quarter in 2005, the number (335) was higher but the proportion (35.5%) slightly lower. The majority of QRNG (317 of 325, 97.5%) exhibited higher-level resistance to ciprofloxacin of 1 mg/L or more. QRNG are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06–0.5 mg/L) or resistant (MIC ≥ 1 mg/L) groups.

QRNG were detected in all states and territories (Figure 5). The highest proportion of QRNG was found in Victoria where 96 QRNG accounted for 53% of all gonococci tested. In South Australia, there were 17 QRNG (46% of isolates), in New South Wales 146 QRNG (52.8%), Queensland 44 (34%), Western Australia 13 (14.6%), with 6 QRNG detected in the Northern Territory, 2 in Tasmania and 1 in the Australian Capital Territory.

High level tetracycline resistance

The number (102) and proportion (11.9%) of high level tetracycline resistant *N. gonorrhoeae* (TRNG) detected was lower than that recorded in this quarter in 2005 (156, 16.6%). TRNG were found in all states and territories except for Tasmania and the Australian Capital Territory and represented between 5% (Northern Territory) and 26% of isolates (Western Australia).

Figure 5. The distribution of quinolone resistant isolates of *Neiserria gonorrhoeae* in Australia by jurisdiction, 1 July to 30 September 2006



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

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 October to 31 December 2006 are included in Tables 6 and 7. Data include cases reported and entered by 19 January 2006. 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;31:164–165.*

Reporting period 1 October to 30 September 2006

There were 1,873 reports to NEPSS of human *Salmonella* infection in the fourth quarter of 2006; 64% more than in third quarter of 2006, and around 10% more than the 10-year historical mean for this period. An increase in reports of both sporadic and outbreak-associated human salmonellosis from late spring through summer is typical of seasonal trends in the incidence of salmonellosis in Australia.

During the fourth quarter of 2006, the 25 most common *Salmonella* types in Australia accounted for 1,243 cases; 66% of all reported human *Salmonella* infections. Nineteen of the 25 most common *Salmonella* infections in the fourth quarter of 2006 were also among those most commonly reported in preceding quarter.

S. Saintpaul was by far the most common *Salmonella* in Australia, with the recent excess of cases largely due to widespread outbreaks associated with fresh produce. *S.* Typhimurium phage types 170, 135 and 44 were next most common, particularly in New South Wales and Victoria. *S.* Typhimurium phage type 170 emerged in late 2001, and despite declining markedly each winter, reappears regularly as a prominent cause of human disease during the warmer months. The increase in *S.* Typhimurium phage type 44 cases is more recent.

Other salmonellae manifesting increases over the recent historical average include *S.* Typhimurium phage type 197 (in Queensland), *S.* Montevideo and *S.* Wangata (New South Wales), *S.* Litchfield (Western Australia) and *S.* Havana (New South Wales).

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

Table 6. Top 25 *Salmonella* types identified in Australia, 1 October to 31 December 2006, by state or territory

National rank	*Salmonella* type	State or territory								Total 4th quarter 2006	Last 10 years mean 4th quarter	Year to date 2006	Year to date 2005
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
1	*S.* Saintpaul	10	81	9	79	1	7	44	7	238	78	561	437
2	*S.* Typhimurium PT 170	13	57	0	15	0	0	47	2	134	61	409	472
3	*S.* Typhimurium PT 135	5	43	5	21	2	4	30	9	119	168	667	805
4	*S.* Typhimurium PT 44	5	23	0	14	3	0	67	0	112	37	237	230
5	*S.* Typhimurium PT 9	1	25	0	17	21	0	12	5	81	121	353	434
6	*S.* Typhimurium PT 197	0	10	0	46	0	2	2	1	61	24	146	546
7	*S.* Virchow PT 8	1	8	5	29	0	0	4	1	48	49	266	249
8	*S.* Birkenhead	0	23	0	20	0	0	1	0	44	63	263	219
9	*S.* Chester	0	10	2	12	1	0	6	6	37	37	158	185
10	*S.* Montevideo	0	22	0	1	0	0	9	2	34	10	64	21
11	*S.* Muenchen	0	4	1	15	0	0	0	11	31	27	153	147
12	*S.* Oranienburg	1	5	3	8	6	1	2	3	29	16	161	101
13	*S.* Stanley	0	9	0	7	0	0	9	4	29	15	102	69
14	*S.* Infantis	0	10	0	1	6	0	6	5	28	29	173	170
15	*S.* Litchfield	0	1	5	6	0	0	0	16	28	9	51	35
16	*S.* Typhimurium PT 12	1	8	0	0	0	0	9	7	25	16	118	117
17	*S.* Aberdeen	0	1	0	20	0	0	2	0	23	22	146	151
18	*S.* Typhimurium PT 135a	0	0	4	0	18	1	0	0	23	6	66	27
19	*S.* Hvittingfoss	0	3	0	12	0	0	3	1	19	20	133	185
20	*S.* Anatum	0	0	5	7	1	0	0	5	18	20	107	79
21	*S.* Potsdam	0	1	3	8	1	0	1	3	17	19	83	49
22	*S.* Waycross	0	7	0	9	0	0	1	0	17	18	140	115
23	*S.* Havana	0	8	2	2	0	0	3	2	17	11	42	39
24	*S.* Singapore	0	11	1	1	1	0	0	2	16	13	54	37
25	*S.* Enteritidis PT 6a	0	3	0	0	0	2	5	5	15	8	52	90

Table 6. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 October to 31 December 2006, as reported to 19 January 2007

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total all *Salmonella* for quarter	49	523	93	495	116	38	386	173	1,873
Total contributing *Salmonella* types	20	110	46	96	41	15	101	70	235

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 *2007;31:162–163.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 July to 30 September 2006, as reported to 30 December 2006, 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 July to 30 September 2006, 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 2006	This period 2005	YTD 2006	YTD 2005
HIV diagnoses	Female	0	1	0	7	0	0	5	11	24	26	85	73
	Male	0	41	0	31	1	0	59	13	145	199	548	649
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	0	42	0	38	1	0	66	24	171	226	636	723
AIDS diagnoses	Female	0	0	0	1	0	0	3	1	5	11	15	26
	Male	0	17	0	3	2	0	19	2	43	43	119	137
	Total*	0	17	0	4	2	0	23	3	49	54	136	163
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	1	3	3
	Male	0	6	0	2	2	0	10	0	20	17	49	45
	Total*	0	6	0	2	2	0	10	0	20	18	54	48

* 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 30 September 2006, and reported by 31 December 2006, by sex and state or territory

	Sex	State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	32	844	18	261	94	8	356	203	1,816
	Male	259	13,301	128	2,685	899	95	5,187	1,202	23,756
	Not reported	0	231	0	0	0	0	22	0	253
	Total*	291	14,405	146	2,955	994	103	5,587	1,412	25,893
AIDS diagnoses	Female	10	250	3	70	32	4	110	38	517
	Male	93	5,375	43	1,022	399	50	1,984	423	9,389
	Total*	103	5,642	46	1,094	432	54	2,106	463	9,940
AIDS deaths	Female	7	136	1	42	20	2	60	24	292
	Male	74	3,572	26	661	276	32	1,410	292	6,343
	Total*	81	3,719	27	705	296	34	1,479	317	6,658

* Totals include people whose sex was reported as transgender.

OVERSEAS BRIEFS

Reporting period 1 October to 31 December 2006

Avian influenza

The World Health Organization (WHO) confirmed 11 cases of avian influenza (H5N1) in humans including 9 deaths between 1 October and 31 December 2006,[1] bringing the total number of WHO-confirmed cases for 2006 to 116 including 80 deaths.[2]

There was no evidence of human-to-human transmission of avian influenza during the reporting period. The Egyptian (4) and Indonesian (6) cases were all known to have had exposure to, or close contact with sick poultry.[1] The Chinese (1) case kept domestic poultry but the health status of the birds is unclear.[1]

Since the beginning of the current outbreak of avian influenza in November 2003, peak incidence has generally occurred between January and April, therefore a rise in the number of confirmed cases could be expected in the first quarter of 2007.

Chikungunya

Outbreaks of chikungunya were reported from Sri Lanka and the Maldives between October and December 2006, following major outbreaks in neighbouring India and a number of Indian Ocean islands earlier in the year. Sri Lanka reported 5,000 suspected cases in November[3] (confirmation of the presence of the virus was obtained for 5 blood samples[4]). Between early November and 19 December 2006, 135 suspected cases were also reported from the Maldives.[5] In 2006, there were no deaths directly attributable to chikungunya infection confirmed by the WHO anywhere in the world.[6]

Imported cases of chikungunya were reported in the United Kingdom (106 between January and October 2006),[7] Taiwan (1),[8] the United States of America (28)[8] and Spain (7)[9] in 2006. Most cases were linked to travel to known chikungunya endemic areas, although the case that was imported to Taiwan was reportedly from Singapore, where no recent chikungunya outbreaks have been recorded.

Cholera

WHO estimates that the officially reported cases of cholera represent around 5–10% of actual cases worldwide due to widespread under-reporting and poor surveillance systems. During the reporting period, new and continuing outbreaks of cholera or watery diarrhoeal syndrome were reported from China, India and a number of African countries: Angola, Burundi, Chad, Democratic Republic of the Congo, Ethiopia, Guinea, Kenya, Liberia, Malawi, Mozambique, Niger, Nigeria, Senegal, Sierra Leone, Somalia, Sudan, Tanzania, Uganda and Zambia. All of these countries reported outbreaks of cholera during 2005 with the exceptions of Angola, Ethiopia, Somalia and Sudan, which did not record significant outbreaks between 2000 and 2005,[10,11] but are all considered cholera-endemic.

The outbreak in Angola (beginning in February 2006) has become the largest outbreak in the country in more than a decade. The WHO confirmed 11,346 cases including 2,499 deaths between 4 September and 16 December 2006.[12] The outbreak is continuing.

Dengue fever and dengue haemorrhagic fever

Year-to-date figures released in the current reporting period show that epidemics of dengue fever and the more severe form of the disease, dengue haemorrhagic fever (DHF) continued to be a major public health problem in 2006. Globally, around 500,000 cases of DHF (mainly children) require hospitalisation each year, with CFRs ranging between <1% and 20% depending on the level of care provided.

Major outbreaks of dengue fever were reported from India, Pakistan and Taiwan. Dengue is considered endemic in all of these countries. In the year to 25 November 2006, the Taiwanese Department of Health reported 2,051 cases of dengue fever, a 234% increase over numbers reported for the same period in 2005. Laboratory evidence from 864 cases showed that 97 were imported (32 from Vietnam, 18 from Indonesia and the remainder from other South-east Asian countries, South Asia, El Salvador and Madagascar) and the remaining 767 cases were domestic in origin.[13]

Malaria

Between 27 September[14] and 22 December 2006,[15] the Ministry of Health confirmed 107 cases of *Plasmodium falciparum* malaria in Jamaica's first outbreak since it was certified malaria-free in 1966. All cases occurred within the city of Kingston or the neighbouring provinces. Public health measures have been put in place to control vectors, to improve public awareness and to strengthen surveillance in an effort to contain the outbreak and return to malaria-free status (assessed by the WHO as the interruption of transmission[16]). Authorities are still investigating the travel histories of early cases to determine whether the source of the outbreak was imported or domestic.

Meningococcal disease

Between 1 September and 8 November 2006, the WHO reported 231 suspect cases (including 16 deaths) of meningococcal meningitis in Greater Yei County, South Sudan.[17] Five specimens tested positive for the bacteria *Neisseria meningitidis* serogroup A.[17] The meningitis belt of sub-Saharan Africa stretches from Senegal in the west to Ethiopia in the east: South Sudan lies within this area[18] where outbreaks of meningitis are common during the dry season (between December and June). Enhanced surveillance, investigation and a large-scale vaccination campaign have been implemented by a task force incorporating local and international agencies.[17]

Methicillin-resistant *Staphylococcus aureus*

Data reported from the European Anti-Microbial Resistance Surveillance System (EARSS) shows an increasing prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA) in Europe. Some 3 million hospital-acquired infections occur each year in the region.[7]

In the United Kingdom, 8 cases (including 2 fatal) of Panton-Valentine leukocidin (PVL) positive community-associated MRSA were reported amongst patients and staff of a Midlands hospital and their household contacts since September 2006.[7] Two of the cases were fatal. *S. aureus* strains that carry the gene for PVL are associated with a higher incidence of cases with abscesses and tissue necrosis.[7]

Poliomyelitis

As of 19 December 2006, polio remains endemic in 4 countries: Afghanistan, India, Nigeria and Pakistan.[19] Between 3 October and 31 December 2006, cases of polio have also been reported in a number of countries that were previously polio-free but have been re-infected since 2003: Bangladesh (2), Cameroon (1), Democratic Republic of the Congo (4), Ethiopia (3), Kenya (1), Niger (2), and Somalia (2).[19]

Endemic countries

Cross-border polio transmission between Afghanistan and Pakistan remains a key difficulty in achieving polio eradication in the region, however most areas are now polio-free.[19] New cases have been reported from both countries between 3 October and 31 December 2006, with 3 cases reported from Afghanistan and 16 cases reported from Pakistan.

India reported a 10-fold increase in polio cases in 2006 compared with 2005, with 62 cases confirmed during the current reporting period.[20] The state of Utter Pradesh has been the epicentre of the outbreak. Supplementary immunisation activities were conducted on 12 November 2006, covering nearly 80% of the country by area.[19]

In 2006, 57% of the world's reported polio cases occurred in Nigeria, with 6 northern states accounting for half of the world's cases.[16] Between 3 October and 31 December 2006, 215 cases were reported.[19] Polio vaccination coverage in Nigeria is rising: in the third quarter of 2006, estimated vaccination coverage was 72%, compared with 60% in the last quarter of 2005. However, the effects of increased vaccination coverage on lowering new case numbers

may be slow because greater than 90% immunisation of children and young people is needed to interrupt transmission.[21]

Re-infected countries

Among re-infected countries, the focus for global polio eradication in 2007 is in Central Africa (Angola and Democratic Republic of the Congo), the horn of Africa (Somalia and Ethiopia) and Bangladesh. Transmission of wild-polio viruses is still occurring in these areas,[22] although at lower levels than in countries where polio is still considered endemic.

Rift Valley fever

An outbreak centred in the Garissa district began in mid-December, with 32 cases of Rift Valley fever including 19 deaths (CFR – 59.4%) confirmed by the WHO in the 2 weeks to 27 December 2006.[23] The last major outbreak of Rift Valley fever in Kenya was in 1997–1998 with more than 8,000 reported cases, including at least 350 deaths.[24] Data from cases early in the current outbreak suggest that they had acquired the disease from the consumption of raw milk or under-cooked meat from infected animals, rather than from mosquitos. Control measures being undertaken by local authorities include mass vaccinations of livestock, education campaigns including recommendations on boiling milk and cooking meat, spraying to control vectors, and the distribution of mosquito nets.[23]

Yellow fever

During the current reporting period, the WHO reported cases of yellow fever from Togo (3) and Cote d'Ivore (2).[25] Both of these countries are considered to be endemic for yellow fever; however Togo is not listed as a 'declared place' for yellow fever under Australia's *Quarantine Act (1908)*. Vaccination campaigns were carried out by the local Ministries of Health in conjunction with WHO in Cote d'Ivoire and are planned for Togo in January 2007.

References

1. World Health Organisation update; 3, 11, 16 and 31 October 2006, 13 and 29 November 2006, 27 December 2006, 10 January 2007.
2. World Health Organization, *Cumulative Number of Confirmed Human Cases of Avian Influenza A/(H5N1) reported to WHO*, 6 February 2007.
3. *ProMED* 29 November 2006.
4. *ProMed* 22 November 2006.
5. *ProMED* 23 December 2006.
6. *ProMED* 13 October 2006.
7. *CDR Weekly,* 14 December 2006.
8. *ProMED* 25 November 2006.
9. *ProMED* 20 December 2006.
10. World Health Organization, *Information for Travellers*, Chapter 5 Available from: http://whqlibdoc.who.int/publications/2005/9241580364_chap5.pdf
11. WHO *Wkly Epidemiol Rec* 4 August 2006.
12. WHO *Wkly Epidemiol Rec* 6 October, 3 and 17 November, 15 December 2006 and 12 January 2007.
13. *Pro-MED* 11 December 2006.
14. *Eurosurveillance* 2006 11(12) 061214.
15. CDC Outbreak Notice updated 22 December 2006.
16. World Health Organization http://www.who.int/malaria/docs/malariaeliminationagenda.pdf
17. World Health Organization update 21 November 2006.
18. World Health Organization. Meningitis Fact Sheet http://www.who.int/mediacentre/factsheets/fs141/en/
19. Wild Poliovirus Weekly updates 18 October 2006–1 January 2007.
20. Global Polio Eradication Initiative Monthly Situation Report 28 November 2006.
21. WHO Wkly Epidemiol Rec 8 December 2006.
22. Global Polio Eradication Initiative Monthly Situation Report 16 January 2007.
23. WHO update, 27 December 2006.
24. GIDEON http://www.gideononline.net/ Accessed on 22 December 2006.
25. WHO update, 19 October, 19 December 2006.

Other reports

Communicable Diseases Intelligence INSTRUCTIONS FOR AUTHORS

Communicable Diseases Intelligence (*CDI*) is published quarterly (March, June, September and December) by the Surveillance Branch, Office of Health Protection, Australian Government Department of Health and Ageing. The aim of *CDI* is to disseminate information on the epidemiology of communicable disease in Australia. *CDI* invites contributions dealing with any aspect of communicable disease epidemiology, surveillance or prevention and control in Australia. Submissions can be in the form of original articles, short reports, surveillance summaries, reviews or correspondence.

Manuscripts for submission

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

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

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

Authorship

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

Types of manuscript

Articles

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

Short reports

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

Surveillance summaries

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

Outbreak reports

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

Case reports

Brief unstructured reports of 500 to 1,000 words on unique cases of communicable disease will be considered based on their public health significance. Authors must note the instructions on the protection of patient's right to privacy (see Ethics commit-

tee approvals and patient's right to privacy below). Some discussion of the significance of the case for communicable disease control should be included.

Letters to the Editor

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

Document preparation

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

In addition:

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

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

Tables

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

Figures and illustrations

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

Figures

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

Illustrations

- Electronic copies of computer-generated illustrations should be saved in Adobe Photoshop, or similar graphic software in one of the following graphic formats: JPEG, EPS, GIF, or TIFF.
- Electronic versions of photos need to be at least 300 dpi. Black and white illustrations or photographs can be included if required.
- Use a sans serif font for figures. Symbols, lettering and numbering should be clear and large enough to be legible when reduced.

References

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

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

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

Ethics committee approvals and patients' rights to privacy

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

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

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

Review process

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

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

Short reports are not subject to peer review.

Copyright

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

Submission of manuscripts

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

Requests for further information can be obtained either by telephone to (02) 6289 8245, by facsimile: (02) 6289 7100 or by email to the address above.

Surveillance systems reported in *CDI*, 2007

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

In Australia, communicable diseases surveillance systems exist at national, state and local levels. State and local surveillance systems are crucial to the timely and effective detection and management of outbreaks and in assisting in the effective implementation of national policies. The national surveillance system combines some of the data collected from state and territory-based systems to provide an overview at a national level. Specific functions of the national surveillance system include: detection and management of outbreaks affecting more than one jurisdiction; monitoring of the need for and impact of national control programs; guidance of national policy development and resource allocation; and description of the epidemiology of rare diseases for which there are only a few notifications in each jurisdiction. National surveillance also assists in quarantine activities and facilitates international collaborations such as reporting to the World Health Organization.

Surveillance has been defined by the World Health Organization as the 'continuing scrutiny of all aspects of the occurrence and spread of disease that are pertinent to effective control.' It is characterised by 'methods distinguished by their practicability, uniformity, and frequently by their rapidity, rather than complete accuracy.'[1] Although some surveil-

lance schemes aim for complete case ascertainment, others include only a proportion of all cases of the conditions under surveillance, and these samples are subject to systematic and other biases. Results generated from surveillance schemes must be interpreted with caution, particularly when comparing results between schemes, between different geographical areas or jurisdictions and over time. Surveillance data may also differ from data on communicable diseases gathered in other settings.

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

Other surveillance schemes for which *CDI* publishes annual reports include tuberculosis notifications (*Commun Dis Intell* 2007; this issue), the Australian Mycobacterium Reference Laboratory Network (*Commun Dis Intell* 2007; this issue), invasive pneumococcal disease surveillance (*Commun Dis Intell* 2007; this issue), the National Arbovirus and Malaria Advisory Committee (*Commun Dis Intell* 2006;30:411–429), and the Australian Rotavirus Surveillance Program (*Commun Dis Intell* 2006;30:434–438).

National Notifiable Diseases Surveillance System

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

The system coordinates the national surveillance of more than 60 communicable diseases or disease groups endorsed by the CDNA. Under this scheme, notifications are made from doctors and laboratories to state or territory health authorities under the provisions of the public health legislation in their jurisdiction. Electronic, de-identified unit records of notifications are supplied to the Australian Government Department of Health and Ageing for collation, analysis and reporting in *CDI*.

Data provided for each notification include a unique record reference number, state or territory, disease code, date of onset, date of notification to the relevant health authority, sex, age, indigenous status and postcode of residence. Additional data include: infecting organism and subtype; the diagnosis method; full details of vaccination where appropriate; resident location; dates of onset, specimen collection, notification and date when notification was received by health authorities; outbreak reference number; how the case was found; whether the case was confirmed; and whether the case was imported from overseas.

Aggregated data are presented on the *Communicable Diseases Australia* Internet site and updated three times a week (www.health.gov.au/cda). Data are published in *CDI* every quarter and in an annual report. The reports include numbers of notifications for each disease by state or territory, and totals for Australia for the current period, the year to date, and for the corresponding period of the previous year. The national total for each disease is compared with the average number of notifications over the previous five years in the same period. A commentary on the notification data is included with the tables in each issue of *CDI* and graphs are used to illustrate important aspects of the data.

HIV infection and AIDS surveillance is conducted by the National Centre for HIV Epidemiology and Clinical Research and is reported in the HIV and AIDS surveillance reports (see below).

Australian Sentinel Practice Research Network

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

There are currently about 40 general practitioners participating in the network from most states. Seventy-five per cent of these are in metropolitan areas and the remainder are rural. Between 3,000 and 4,000 consultations are recorded each week.

The list of conditions is reviewed annually by the ASPREN Director and an annual report is published. In 2007, six conditions are being monitored; four are related to communicable disease issues. These include influenza, gastroenteritis, chickenpox and shingles. Data for communicable diseases are published in *CDI* every quarter. Data are presented in graphic format as the rate of reporting per 1,000 consultations per week. The conditions are defined as follows:

Influenza-like illness – record once only per patient

Must have the following: cough; fatigue; and fever.

Gastroenteritis – record once only per patient

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

Chickenpox – record once only per patient

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

Shingles – record once only per patient

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

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

HIV and AIDS surveillance

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

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

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

Each year from 1997, the NCHECR has published the *HIV/AIDS, Viral Hepatitis and Sexually Transmissible Infections in Australia Annual Surveillance Report*. The annual surveillance report, available through www.med.unsw.edu.au/nchecr/, provides a comprehensive analysis and interpretation of surveillance data on HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia.

National Influenza Surveillance Scheme

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

- Since 2001, laboratory-confirmed influenza has been a notifiable disease in all Australian states and territories (except South Australia) and reported in the National Notifiable Diseases Surveillance System (see above).
- In 2007, six sentinel general practitioner schemes contribute reports of influenza-like illness: the Australian Sentinel Practice Research Network, the Tropical Influenza Surveillance from the Northern Territory, the New South Wales Sentinel General Practice Scheme, the Victorian Sentinel General Practice Scheme, Queensland and Western Australian sentinel general practices.
- The Laboratory Virology and Serology Reporting Scheme laboratory reports of influenza diagnoses including virus type.

The results of each of the schemes are published together fortnightly throughout the influenza season (May to October) on the *Communicable Diseases Australia* Website as the Australian Influenza Report.

Annual reports on influenza in Australia are published in *CDI* each year (*Commun Dis Intell* 2006;30:189–200). These reports include the above data as well as absenteeism data from a major national employer, hospitalisation and mortality data and influenza typing data from the WHO Collaborating Centre for Influenza Reference and Research.

Sentinel Chicken Surveillance Programme

The Sentinel Chicken Surveillance Programme is used to provide an early warning of increased flavivirus activity in Australia. The main viruses of

concern are Murray Valley encephalitis (MVEV) and Kunjin viruses. MVEV causes the disease Murray Valley encephalitis (formerly known as Australian encephalitis), a potentially fatal disease in humans. Encephalitis is less frequent in cases of Kunjin virus infection and these encephalitis cases have a lower rate of severe sequelae.

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

Currently, flocks are maintained in the north of Western Australia, the Northern Territory, New South Wales and in Victoria. The flocks in Western Australia and the Northern Territory are tested all year round but those in New South Wales and Victoria are tested only in the summer months, during the main MVEV risk season. Results are posted on the National Arbovirus Surveillance Website by state representatives. A yearly summary is presented in *CDI* (*Commun Dis Intell* 2006;30:411–429*)*.

Australian Gonococcal Surveillance Programme

The Australian Gonococcal Surveillance Programme (AGSP) is a continuing program to monitor antimicrobial resistance in *Neisseria gonorrhoeae* and includes the reference laboratories in all states and territories. These laboratories report data on sensitivity to an agreed core group of antimicrobial agents on a quarterly basis and provide an expanded analysis as an annual report in *CDI* (*Commun Dis Intell* 2006;30:205–210). The antibiotics that are currently routinely surveyed are the penicillins, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens. One main purpose of the AGSP is to help define standard protocols for antibiotic treatment of gonococcal infection. When *in vitro* resistance to a recommended agent is demonstrated in 5% or more of isolates, it is usual to reconsider the inclusion of that agent in current treatment schedules. Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level resistance to the tetracyclines and intermittent surveys of azithromycin resistance are conducted. Comparability of data is achieved by means of a standardised system of MIC testing and a program-specific quality assurance process.

Australian Meningococcal Surveillance Programme

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

Data are reported annually and quarterly in *CDI*. Data in the quarterly reports are restricted to a description of the number of cases per jurisdiction, and serogroup where known. A full analysis of laboratory-confirmed cases of IMD, including phenotyping and antibiotic susceptibility data are published annually (*Commun Dis Intell* 2006;30:211–221).

Laboratory Virology and Serology Reporting Scheme

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

LabVISE data should be interpreted with caution. The number and type of reports received is subject to a number of biases. These include the number of participating laboratories, which has varied over time. The locations of participating laboratories also create bias, as some jurisdictions are better represented than others. Also changes in diagnostic practices, particularly the introduction of new testing methodologies, may affect laboratory reports. The ability of laboratory tests to distinguish acute

from chronic or past infection must also be considered in interpretation of the data. Although changes in incidence cannot be determined with precision from this data, general trends can be observed, for example with respect to seasonality and the age-sex distribution of patients. See review in *Commun Dis Intell* 2002;26:323–374).

Australian Paediatric Surveillance Unit

The Australian Paediatric Surveillance Unit (APSU) conducts national, active surveillance of uncommon conditions of childhood, including infectious, genetic, mental health, and vaccine preventable diseases and childhood injuries. Communicable diseases currently under surveillance include: acute flaccid paralysis (to identify potential cases of poliovirus infection); congenital cytomegalovirus infection; congenital rubella; perinatal exposure to HIV, HIV infection and AIDS; neonatal herpes simplex virus infection; hepatitis C virus infection; B group *Streptococcus* sepsis; and neonatal, congenital and severe complications of varicella virus infection. A study of intussusception due to rotavirus infection or rotavirus vaccination is planned for 2007.

The primary objectives of the APSU are to document the number of Australian children aged under 15 years, newly diagnosed with specified conditions; their geographic distribution; clinical features; current management; and outcome. Contributors to the APSU are clinicians known to be working in paediatrics and child health in Australia. In 2005, over 1,100 clinicians participated in the surveillance of 16 conditions through the APSU, with an overall monthly response rate of 93%. APSU is a unit of the Royal Australasian College of Physicians, and its activities are supported by the Department of Health and Ageing; the Faculty of Medicine, University of Sydney; and the National Health and Medical Research Council Enabling Grant 402784. For further information please contact the APSU Director, Professor Elizabeth Elliott on telephone: +61 2 9845 3005, facsimile +61 2 9845 3082 or email: apsu@chw.edu.au

National Enteric Pathogens Surveillance System

Since 1980, the National Enteric Pathogens Surveillance Scheme (NEPSS) has collected, analysed and disseminated data on human enteric bacterial infections diagnosed in Australia. These pathogens include *Salmonella*, *Escherichia coli*, *Vibrio*, *Yersinia*, *Plesiomonas*, *Aeromonas* and *Campylobacter*.

Communicable Diseases Intelligence NEPSS quarterly reports include only *Salmonella*. NEPSS receives reports of *Salmonella* isolates submitted from primary diagnostic laboratories throughout Australia to any of the five serotyping laboratories, two of which (MDU and IMVS) also perform phage typing.

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 a *Salmonella* from the clinical sample.

NEPSS is operated by the Microbiological Diagnostic Unit — Public Health Laboratory, Department of Microbiology and Immunology, The University of Melbourne; and is overseen by a Steering Committee of state, territory and Commonwealth stakeholders. NEPSS may be contacted at the Microbiological Diagnostic Unit, by telephone +61 3 8344 5701, facsimile +61 3 8344 7833 or email joanp@unimelb.edu.au

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

Australian Childhood Immunisation Register

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

From the data finally entered onto the ACIR, Medicare Australia provides regular quarterly coverage reports at the national and state level. Coverage for these reports is calculated using the cohort method described in *Commun Dis Intell* 1998;22:36–37. With this method, a cohort of children is defined by date of birth in three-month groups. This birth cohort has the immunisation status of its members assessed at the three key milestones of 12 months, 24 months and 6 years of age. Analysis of coverage is undertaken three months after the due date for completion of each milestone, so that time is available for processing notifications and the impact on coverage estimates of delayed notification to the ACIR is minimised. Only children enrolled with Medicare are included in order to minimise inaccuracies in coverage estimates due to duplicate records.

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

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

OzFoodNet: enhanced foodborne disease surveillance

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

OzFoodNet reports quarterly on investigations of gastroenteritis outbreaks and clusters of disease potentially related to food. Annual reports have been produced and published in *CDI* (*Commun Dis Intell* 2006;30:278–300) since 2002. Data are reported from all Australian jurisdictions.

References

1. Last JM. A dictionary of epidemiology. New York: Oxford University Press, 1988.
2. Hall R. Notifiable diseases surveillance, 1917 to 1991. *Commun Dis Intell* 1993;226–236.

ERRATUM

The Communicable Diseases Network Australia National Arbovirus and Malaria Advisory Committee annual report published in the last issue of *Communicable Diseases Intelligence* contained errors in Tables 1 and 2 on page 420 (*Commun Dis Intell* 2006;30:420). The following tables contain the correct data.

Table 1. Number and proportion of Barmah Forest virus infections with corresponding Ross River virus infections, Australia, 1 July 2000 to 30 June 2006, by year of diagnosis and state or territory

State or territory	2000–01			2001–02			2002–03			2003–04			2004–05			2005–06		
	dual n	BFV n	BFV % dual	dual n	BFV n	BFV % dual	dual n	BFV n	BFV % dual	dual n	BFV n	BFV % dual	dual n	BFV n	BFV % dual	dual n	BFV n	BFV % dual
ACT	0	2	0%	0	0	0%	0	1	0%	0	0	0%	0	2	0%	0	7	0%
NSW	9	375	2%	3	378	1%	15	423	4%	13	350	4%	13	438	3%	64	626	10%
NT	2	33	6%	1	25	4%	0	18	0%	1	15	7%	5	44	11%	8	98	8%
Qld	19	602	3%	17	418	4%	86	805	11%	53	543	10%	53	676	8%	127	804	16%
SA	2	17	12%	0	4	0%	0	1	0%	0	4	0%	2	20	10%	14	163	9%
Tas	0	1	0%	0	0	0%	0	0	0%	0	0	0%	0	0	0%	0	1	0%
Vic	3	21	14%	0	58	0%	0	16	0%	0	5	0%	0	22	0%	1	25	4%
WA	5	81	6%	2	45	4%	2	25	8%	7	49	14%	2	69	3%	21	171	12%
Total	40	1,132	4%	23	928	2%	103	1,289	8%	74	966	8%	75	1,271	6%	235	1,895	12%

Table 2. Number and proportion of Ross River virus infections with corresponding Barmah Forest virus infections, Australia, 1 July 2000 to 30 June 2006, by year of diagnosis and state or territory

State or territory	2000–01			2001–02			2002–03			2003–04			2004–05			2005–06		
	dual n	RRV n	RRV % dual	dual n	RRV n	RRV % dual	dual n	RRV n	RRV % dual	dual n	RRV n	RRV % dual	dual n	RRV n	RRV % dual	dual n	RRV n	RRV % dual
ACT	0	14	0%	0	0	0%	0	0	0%	0	6	0%	0	4	0%	0	10	0%
NSW	9	773	1%	3	217	1%	15	453	3%	13	654	2%	13	441	3%	64	1,275	5%
NT	2	233	1%	1	71	1%	0	134	0%	1	203	0%	5	180	3%	8	267	3%
Qld	19	1,717	1%	17	944	2%	86	2,391	4%	53	2,123	2%	53	1,013	5%	127	2,470	5%
SA	2	271	1%	0	57	0%	0	20	0%	0	51	0%	2	50	4%	14	354	4%
Tas	0	11	0%	0	120	0%	0	2	0%	0	21	0%	0	5	0%	0	14	0%
Vic	3	375	1%	0	43	0%	0	14	0%	0	91	0%	0	38	0%	1	252	0%
WA	5	236	2%	2	130	2%	2	146	1%	7	1,583	0%	2	144	1%	21	873	2%
Total	40	3,630	1%	23	1,582	1%	103	3,160	3%	74	4,732	2%	75	1,875	4%	235	5,515	4%