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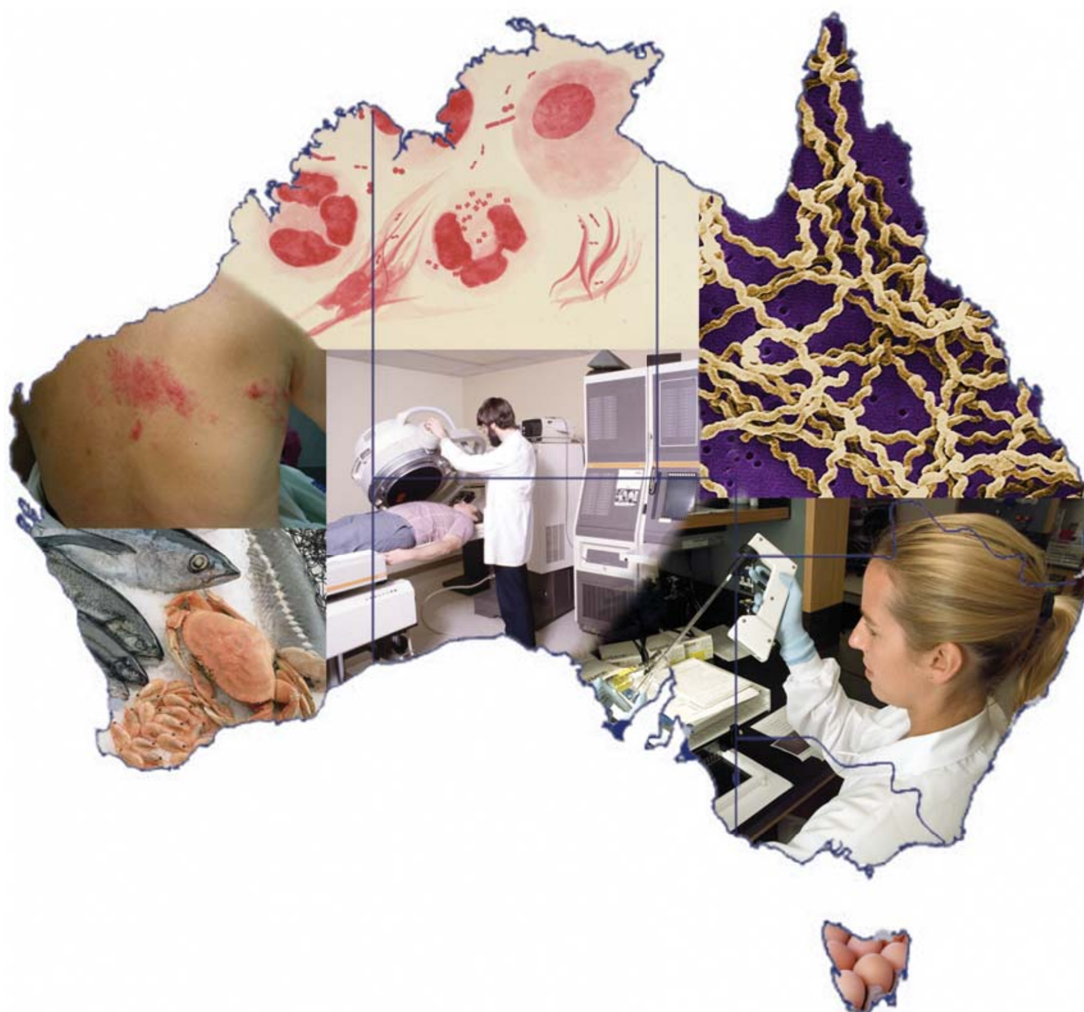
Communicable Diseases Intelligence



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2006

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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* 2005;29:95–97.

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Front cover: Surveillance Section, Australian Government Department of Health and Ageing.
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Clockwise from top left: Urethral exudate containing *Neisseria gonorrhoeae,* CDC; Electron micrograph of *Leptospira sp.*, Janice Carr, CDC; Pulsed-field Gel Electrophoresis test for tuberculosis, James Gathany, CDC, raw eggs are a common cause of *Salmonella*; chest x-ray; seafood is a common cause of foodborne illness; shingles.

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Australia's notifiable diseases status, 2004, Annual report of the National Notifiable Diseases Surveillance System

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Australian Gonococcal Surveillance Programme

Australian Meningococcal Surveillance Programme

Australian Sentinel Practice Research Network

Australian Quarantine Inspection Service

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Abstract

In 2004, 60 diseases and conditions were nationally notifiable in Australia. States and Territories reported a total of 110,929 cases of communicable diseases to the National Notifiable Diseases Surveillance System (NNDSS): an increase of 4 per cent on the number of notifications in 2003. In 2004, the most frequently notified diseases were sexually transmissible infections (46,762 cases; 42% of total notifications), gastrointestinal diseases (25,247 cases; 23% of total notifications) and bloodborne diseases (19,191 cases; 17% of total notifications). There were 13,206 notifications of vaccine preventable diseases, 6,000 notifications of vectorborne diseases, 1,799 notifications of other bacterial infections (includes, legionellosis, leprosy, meningococcal infections and tuberculosis) and 877 notifications of zoonotic diseases. *Commun Dis Intell* 2006;30:1–79.

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Abbreviations used in this report

AFP	Acute flaccid paralysis
AIDS	Acquired immune deficiency syndrome
AGSP	Australian Gonococcal Surveillance Programme
ASPREN	Australian Sentinel Practice Research Network
ASVS	Australian Standard Vaccination Schedule
BFV	Barmah Forest virus
CDI	*Communicable Diseases Intelligence*
CDNA	Communicable Diseases Network Australia
DENV	Dengue
DSS	Dengue Shock Syndrome
DoHA	Australian Government Department of Health and Ageing
Hib	*Haemophilus influenzae* type b
HIV	Human immunodeficiency virus
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
MMR	Measles-mumps-rubella
MVEV	Murray Valley encephalitis virus
NAQS	Northern Australia Quarantine Strategy
NCHECR	National Centre in HIV Epidemiology and Clinical Research
NEC	Not elsewhere classified
NN	Not notifiable
NNDSS	National Notifiable Diseases Surveillance System
NPA	Northern peninsula area
PCR	Polymerase chain reaction
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, 2004, 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 two 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, which 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 2004 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.

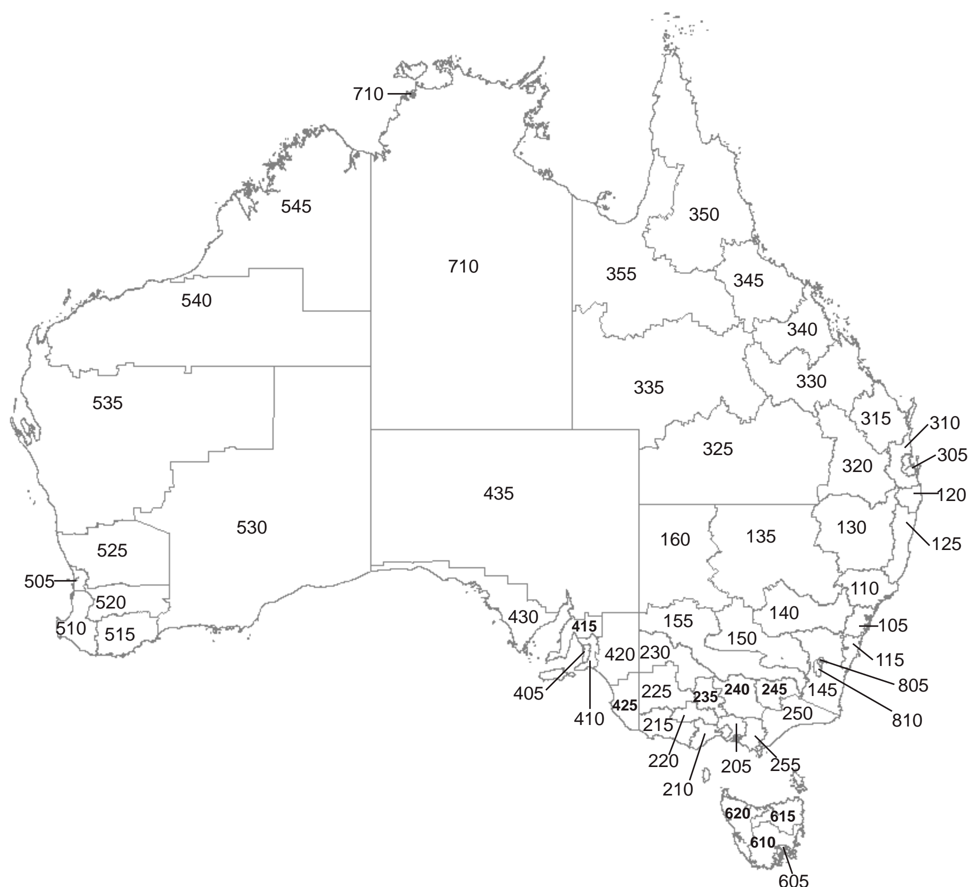
The geographical distribution of selected diseases was mapped using MapInfo software. Maps were based on the postcode of residence of each patient aggregated to the appropriate Statistical Division (Map 1). Rates for the different Statistical Divisions were ordered into six groups — the highest value, the lowest value above zero, those equal to zero, and the intermediate values sorted into three equal-sized groups. The Statistical Divisions in each of the two territories, the Australian Capital Territory and the Northern Territory were combined to calculate rates for each 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. The annual report of the NNDSS, *Australia's notifiable diseases status*, provides yearly summaries of notifications.

Notes on interpretation

The present report is based on 2004 '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 July 2005. 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.

Map 1. Australian Bureau of Statistics Statistical Divisions, and population by Statistical Division, 2004



Statistical Division		Population
Australian Capital Territory		
805	Canberra*	324,021
New South Wales		
105	Sydney	4,232,078
110	Hunter	604,420
115	Illawarra	410,148
120	Richmond-Tweed	223,875
125	Mid-North Coast	291,865
130	Northern	179,121
135	North Western	118,733
140	Central West	179,232
145	South Eastern	200,530
150	Murrumbidgee	153,143
155	Murray	114,644
160	Far West	23,686
Northern Territory		
705	Darwin	109,478
710	NT - balance	90,435
Queensland		
305	Brisbane	1,774,890
310	Moreton	797,696
315	Wide Bay-Burnett	250,253

Statistical Division		Population
Queensland, continued		
320	Darling Downs	218,484
325	South West	26,952
330	Fitzroy	187,916
335	Central West	12,239
340	Mackay	143,699
345	Northern	200,909
350	Far North	234,849
355	North West	33,900
South Australia		
405	Adelaide	1,124,315
410	Outer Adelaide	121,448
415	Yorke & Lower North	44,682
420	Murray Lands	68,571
425	South East	63,040
430	Eyre	34,560
435	Northern	77,634
Tasmania		
605	Greater Hobart	202,138
610	Southern	35,459
615	Northern	136,638
620	Mersey-Lyell	107,893
910	*Other territories*	2,670

Statistical Division		Population
Victoria		
205	Melbourne	3,600,080
210	Barwon	266,112
215	Western District	101,008
220	Central Highlands	146,185
225	Wimmera	50,812
230	Mallee	91,619
235	Loddon	173,231
240	Goulburn	201,042
245	Ovens-Murray	96.098
250	East Gippsland	82,276
255	Gippsland	164,316
Western Australia		
505	Perth	1,457,639
510	South West	211,918
515	Lower Great Southern	53,656
520	Upper Great Southern	18,068
525	Midlands	52,659
530	South Eastern	54,289
535	Central	59,663
540	Pilbara	39,311
545	Kimberley	35,001
Total Australia		20,111,227

* Includes Statistical Division 810 "ACT – balance."

Analyses in this report were based on the date of disease onset in an attempt to estimate disease activity 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 2004, 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[1] of data in this report is summarised in Appendix 3. The case's sex was complete in 99.7 per cent of notifications and date of birth in 99.8 per cent of notifications. In 2004, nationally, Indigenous status[2] was complete in 46 per cent of notifications, and varied by jurisdiction. Indigenous status was complete for 92 per cent of data reported in the Northern Territory, 89 per cent in South Australia, 66 per cent in Western Australia and 52 per cent in Victoria. In the remaining jurisdictions, less than 50 per cent of data were complete for Indigenous status.

Data completeness on Indigenous status also varied by disease; in notifications of tuberculosis (TB), *Haemophilus influenzae* type b, meningococcal disease, infectious syphilis and hepatitis A were more than 90 per cent complete for Indigenous status, while in notifications of other diseases such as chlamydial infection and salmonellosis, data completeness was 41 per cent.

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1 Definition of completeness = (Number with valid data/total notifications x 100

2 Data completeness = (Total notifications – Indigenous status 'Not stated or missing')/total notifications 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

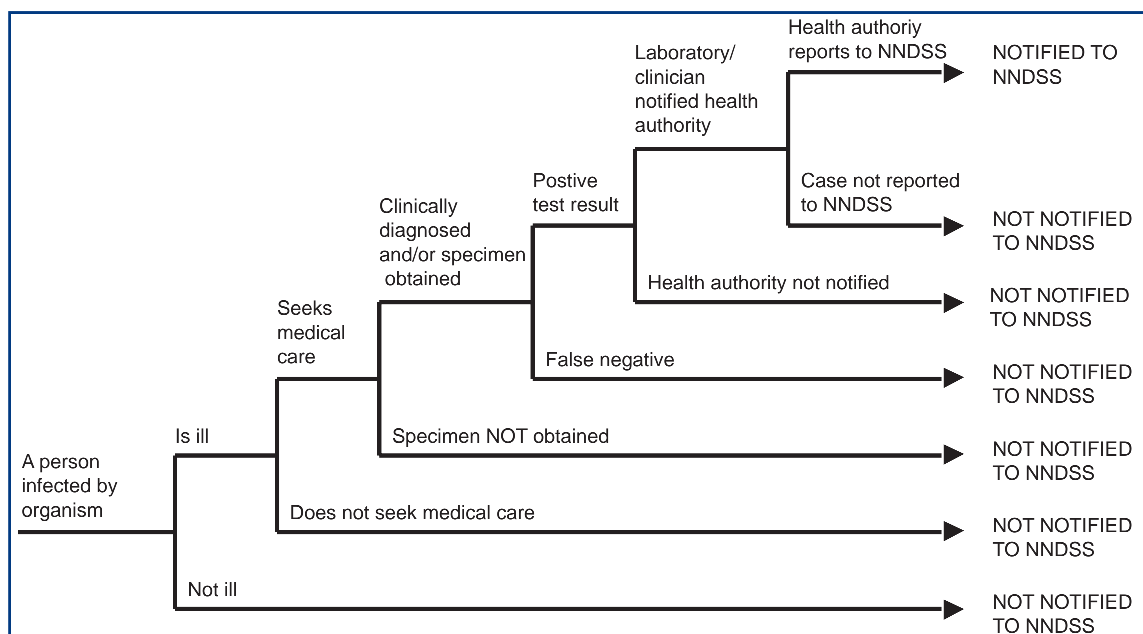


Table 1. Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2004

Disease	Data received from
Bloodborne diseases	
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)*	All jurisdictions except NT
Hepatitis C (incident)	All jurisdictions except Qld and NT
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 infections (NEC)[‖]	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis (all categories)	All jurisdictions
Syphilis < 2 years duration	All jurisdictions
Syphilis > 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, 2004, *continued*

Disease	Data received from
Vectorborne diseases	
Barmah Forest virus infection	All jurisdictions
Dengue	All jurisdictions
Flavivirus (NEC)**	All jurisdictions except ACT
Japanese encephalitis virus	All jurisdictions
Kunjin virus††	All jurisdictions except ACT
Malaria	All jurisdictions
Murray Valley encephalitis virus	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 (NEC)	All jurisdictions
Ornithosis‡‡	All jurisdictions
Q fever	All jurisdictions except ACT
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 the Northern Territory and 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.

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

** Flavivirus (NEC) replaces Arbovirus (NEC) from 1 January 2004.

†† In the Australian Capital Territory, Murray Valley encephalitis virus and Kunjin 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.

NEC Not elsewhere classified.

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. States and territories may have case definitions which reflect their local public health needs. These may be the same as or more comprehensive than the CDNA case definitions.

In 2004, not all jurisdictions implemented the CDNA case definitions (Queensland did not implement the CDNA case definitions in 2004 and New South Wales introduced it in August 2004). This has to be kept in mind when comparing data across time and between jurisdictions.

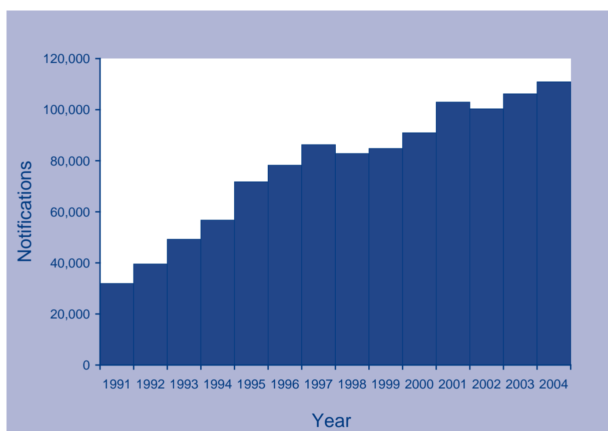
Results

Summary of 2004 data

There were 110,929 communicable disease notifications received by NNDSS in 2004 (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 2000 to 2004 are shown in Table 4.

In 2004, the total number of notifications was the highest recorded in NNDSS since the system began in 1991. There was an increase of 4 per cent compared to the total number of notifications in 2003 (Figure 2).

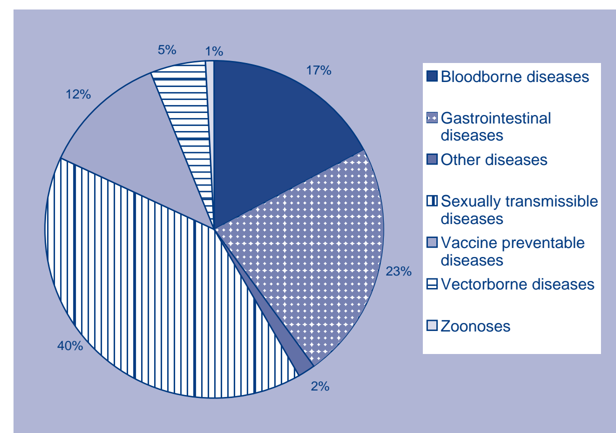
Figure 2. Trends in notifications received by the National Notifiable Diseases Surveillance System, Australia, 1991 to 2004



In 2004, the most frequently notified diseases were sexually transmissible infections (44,604 notifications, 40 per cent of total notifications), gastrointestinal diseases (25,247 notifications, 23%) and bloodborne diseases (19,191 notifications, 17%). There were 13,206 notifications of vaccine preventable diseases; 6,000 notifications of vectorborne diseases; 1,799 notification of other bacterial infections and 877 notifications of zoonotic diseases (Figure 3).

The major changes in communicable disease notifications in 2004 are shown in Figure 4 as the ratio of notifications in 2004 to the mean number of notifications for the previous five years. The number of notifications of chlamydial infections and hepatitis E

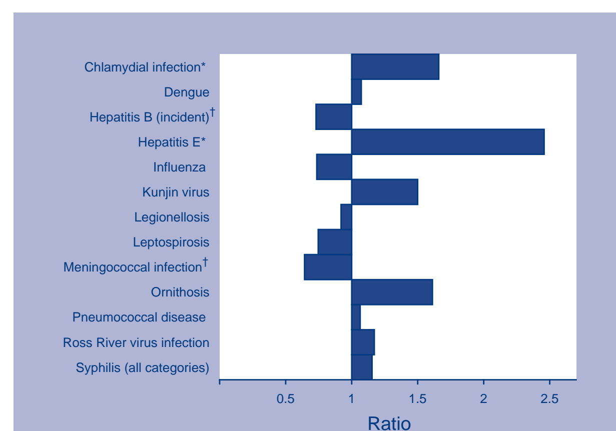
Figure 3. Notifications to the National Notifiable Diseases Surveillance System, Australia, 2004, by disease category



infections surpassed the expected range (5-year mean plus two standard deviations). Notifications of hepatitis B (incident) and meningococcal infections were below the expected range (5-year mean minus two standard deviations). Notifications for the remaining diseases were within the historical range.

In the financial year 2003–04, there were 92,892 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 1.4 per cent of all hospital separations in that period. A further 56,675 separations were recorded with a principal diagnosis of influenza or pneumonia (ICD10–AM J10–J18).[1]

Figure 4. Comparison of total notifications of selected diseases reported to the National Notifiable Diseases Surveillance System in 2004, with the previous five-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)..

Table 2. Notifications of communicable diseases, Australia, 2004, by state or territory

Disease	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis B (incident)	9	53	8	44	8	18	106	29	275
Hepatitis B (unspecified)*	47	2,851	2	761	260	59	1,482	399	5,861
Hepatitis C (incident)	7	60	NN	NN	60	24	89	121	361
Hepatitis C (unspecified)*,†	209	4,906	271	2,480	555	287	2,898	1,061	12,667
Hepatitis D	0	14	0	10	0	0	3	0	27
Gastrointestinal diseases									
Botulism	0	1	0	0	0	0	0	0	1
Campylobacteriosis‡	371	NN	219	3,715	1,844	609	6,317	1,933	15,008
Cryptosporidiosis	6	327	113	602	74	18	309	124	1,573
Haemolytic uraemic syndrome	0	9	1	1	2	0	1	1	15
Hepatitis A	1	139	13	22	11	1	71	57	315
Hepatitis E	0	8	0	4	0	1	12	3	28
Listeriosis	1	30	1	7	2	1	14	9	65
Salmonellosis (NEC)	97	2,153	393	2,580	496	119	1,134	635	7,607
Shigellosis	2	96	119	61	54	3	70	113	518
SLTEC, VTEC‡,§	0	3	0	9	28	0	4	0	44
Typhoid	1	39	0	9	1	0	18	5	73
Quarantinable diseases									
Cholera	0	1	0	1	0	0	2	1	5
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	0	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 transmissible infections									
Chlamydial infections (NEC)‖	619	10,020	1,640	8,121	2,241	620	7,609	4,319	35,189
Donovanosis	0	0	6	3	0	0	0	1	10
Gonococcal infection	35	1,446	1,588	1,096	357	28	1,129	1,419	7,098
Syphilis (all categories)	12	1,039	284	290	23	14	427	207	2,296
Syphilis < 2 years duration	4	294	57	92	8	2	89	50	596
Syphilis > 2 years or unknown duration	7	744	104	198	1	12	338	157	1,561
Syphilis – congenital	0	0	6	4	0	0	1	0	11
Vaccine preventable diseases									
Diphtheria	0	0	0	0	0	0	0	0	0
Haemophilus influenzae type b	0	5	3	3	2	1	1	0	15
Influenza (laboratory confirmed)¶	1	1,012	41	561	69	3	203	183	2,073
Measles	0	12	3	0	6	0	15	9	45
Mumps	3	67	0	16	4	0	2	10	102
Pertussis	122	3,549	29	942	928	37	853	2,097	8,557
Pneumococcal disease (invasive)	55	908	93	477	198	56	389	199	2,375
Rubella	0	17	0	10	2	0	1	3	33
Rubella – congenital	0	1	0	0	0	0	0	0	1
Tetanus	0	0	0	3	2	0	0	0	5

Table 2. Notifications of communicable diseases, Australia, 2004, by state or territory, *continued*

Disease	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vectorborne diseases									
Barmah Forest virus infection	2	402	22	535	6	0	16	69	1,052
Dengue	6	31	19	249	4	1	9	7	326
Flavivirus (NEC)**	0	1	0	45	0	0	3	0	49
Japanese encephalitis virus	0	0	0	1	0	0	0	0	1
Kunjin virus††	NN	0	0	11	0	0	1	0	12
Malaria	16	101	41	263	20	15	67	36	559
Murray Valley encephalitis virus	0	0	1	0	0	0	0	0	1
Ross River virus infection	6	700	235	1,795	53	20	92	1,099	4,000
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	7	0	26	0	0	3	0	36
Leptospirosis	0	40	2	110	1	0	8	5	166
Ornithosis‡‡	0	81	0	3	5	0	146	0	235
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0
Q fever	2	223	3	137	38	0	28	9	440
Other bacterial infections									
Legionellosis	1	82	2	31	45	1	98	50	310
Leprosy	0	3	1	1	0	0	0	0	5
Meningococcal infection§§	11	153	12	81	13	18	79	41	408
Tuberculosis	14	431	28	129	60	11	322	81	1,076
Total	1,656	31,021	5,199	25,249	7,472	1,965	24,032	14,335	110,929

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

† In the Northern Territory and 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.

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

** Flavivirus (NEC) replaces 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 rates of communicable diseases, Australia, 2004, by state and territory (per 100,000 population)

Disease	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis B (incident)	2.8	0.8	4.0	1.1	0.5	3.7	2.1	1.5	1.4
Hepatitis B (unspecified)*	14.5	42.4	1.0	19.6	16.9	12.2	29.8	20.1	29.1
Hepatitis C (incident)	2.2	0.9	NN	NN	3.9	5.0	1.8	6.1	2.3
Hepatitis C (unspecified)*,†	64.5	72.9	135.6	63.9	36.2	59.5	58.3	53.5	63.0
Hepatitis D	0.0	0.2	0.0	0.3	0.0	0.0	0.1	0.0	0.1
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis‡	114.5	NN	109.5	95.7	120.2	126.3	127.0	97.5	112.2
Cryptosporidiosis	1.9	4.9	56.5	15.5	4.8	3.7	6.2	6.3	7.8
Haemolytic uraemic syndrome	0.0	0.1	0.5	0.0	0.1	0.0	0.0	0.1	0.1
Hepatitis A	0.3	2.1	6.5	0.6	0.7	0.2	1.4	2.9	1.6
Hepatitis E	0.0	0.1	0.0	0.1	0.0	0.2	0.3	0.2	0.1
Listeriosis	0.3	0.4	0.5	0.2	0.1	0.2	0.3	0.5	0.3
Salmonellosis (NEC)	29.9	32.0	196.6	66.5	32.3	24.7	22.8	32.0	37.8
Shigellosis	0.6	1.4	59.5	1.6	3.5	0.6	1.4	5.7	2.6
SLTEC, VTEC§	0.0	0.0	0.0	0.2	1.8	0.0	0.1	0.0	0.2
Typhoid	0.3	0.6	0.0	0.2	0.1	0.0	0.4	0.3	0.4
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	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	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 infections (NEC)‖	191.0	148.9	820.4	209.2	146.1	128.6	153.0	217.9	175.0
Donovanosis	0.0	0.0	3.0	0.1	0.0	0.0	0.0	0.1	0.1
Gonococcal infection	10.8	21.5	794.3	28.2	23.3	5.8	22.7	71.6	35.3
Syphilis (all categories)	3.7	15.4	142.1	7.5	1.5	2.9	8.6	10.4	11.4
Syphilis < 2 years duration	1.2	4.4	28.5	2.4	0.5	0.4	1.8	2.5	3.0
Syphilis > 2 years or unknown duration	2.2	11.1	52.0	5.1	0.1	2.5	6.8	7.9	7.8
Syphilis – congenital	0.0	0.0	3.0	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	1.5	0.1	0.1	0.2	0.0	0.0	0.1
Influenza (laboratory confirmed)¶	0.3	15.0	20.5	14.5	4.5	0.6	4.1	9.2	10.3
Measles	0.0	0.2	1.5	0.0	0.4	0.0	0.3	0.5	0.2
Mumps	0.9	1.0	0.0	0.4	0.3	0.0	0.0	0.5	0.5
Pertussis	37.7	52.7	14.5	24.3	60.5	7.7	17.2	105.8	42.5
Pneumococcal disease (invasive)	17.0	13.4	46.5	12.3	12.9	11.6	7.8	10.0	11.5
Rubella	0.0	0.3	0.0	0.3	0.1	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
Tetanus	0.0	0.0	0.0	0.1	0.1	0.0	0.0	0.0	0.0

Table 3. Notification rates of communicable diseases, Australia, 2004, by state and territory (per 100,000 population), *continued*

Disease	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vectorborne diseases									
Barmah Forest virus infection	0.6	6.0	11.0	13.8	0.4	0.0	0.3	3.5	5.2
Dengue	1.9	0.5	9.5	6.4	0.3	0.2	0.2	0.4	1.6
Flavivirus (NEC)**	0.0	0.3	0.0	1.2	0.0	0.0	0.1	0.0	0.3
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	0.3	0.0	0.0	0.0	0.0	0.1
Malaria	4.9	1.5	20.5	6.8	1.3	3.1	1.3	1.8	2.8
Murray Valley encephalitis virus	0.0	0.0	0.5	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	1.9	10.4	117.6	46.2	3.5	4.1	1.9	55.4	19.9
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	0.7	0.0	0.0	0.1	0.0	0.2
Leptospirosis	0.0	0.6	1.0	2.8	0.1	0.0	0.2	0.3	0.8
Ornithosis‡‡	0.0	1.2	0.0	0.1	0.3	0.0	2.9	0.0	1.2
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Q fever	0.6	3.3	1.5	3.5	2.5	0.0	0.6	0.5	2.2
Other bacterial infections									
Legionellosis	0.3	1.2	1.0	0.8	2.9	0.2	2.0	2.5	1.5
Leprosy	0.0	0.0	0.5	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection§§	3.4	2.3	6.0	2.1	0.8	3.7	1.6	2.1	2.0
Tuberculosis	4.3	6.4	14.0	3.3	3.9	2.3	6.5	4.1	5.4

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

† In the Northern Territory and 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.

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

** Flavivirus (NEC) replaces 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 rates (per 100,000 population), of communicable diseases, Australia, 2000 to 2004

Disease	Notifications					Rate per 100,000 population				
	2000	2001	2002	2003	2004	2000	2001	2002	2003	2004
Bloodborne diseases										
Hepatitis B (incident)	410	418	392	349	275	2.1	2.2	2.0	1.8	1.4
Hepatitis B (unspecified)*	7,321	8,747	6,677	6,637	5,861	38.2	45.1	34.0	33.4	29.1
Hepatitis C (incident)	504	703	448	477	361	3.3	4.5	2.8	3.0	2.3
Hepatitis C (unspecified)[*,†]	19,110	19,792	15,906	13,911	12,667	99.8	102.0	81.0	70.0	63.0
Hepatitis D	26	20	23	28	27	0.1	0.1	0.1	0.1	0.1
Gastrointestinal diseases										
Botulism	2	2	0	1	1	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis[‡]	13,661	16,134	14,736	15,323	15,008	107.8	125.7	113.3	116.2	112.2
Cryptosporidiosis	1,144	1,621	3,272	1,225	1,573	6.0	8.3	16.7	6.2	7.8
Haemolytic uraemic syndrome	14	4	11	15	15	0.1	0.0	0.1	0.1	0.1
Hepatitis A	806	538	392	439	315	4.2	2.8	2.0	2.2	1.6
Hepatitis E	9	14	12	14	28	0.0	0.1	0.1	0.1	0.1
Listeriosis	66	64	62	70	65	0.3	0.3	0.3	0.4	0.3
Salmonellosis (NEC)	6,099	7,036	7,848	7,042	7,607	31.8	36.2	40.0	35.4	37.8
Shigellosis	490	567	507	444	518	2.6	2.9	2.6	2.2	2.6
SLTEC, VTEC[§]	43	45	59	52	44	0.2	0.2	0.3	0.3	0.2
Typhoid	56	81	70	51	73	0.3	0.4	0.4	0.3	0.4
Quarantinable diseases										
Cholera	2	4	5	2	5	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
Smallpox	–	–	–	–	0	–	–	–	–	0.0
Tularaemia	–	–	–	–	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 transmissible infections										
Chlamydial infections (NEC)[‖]	16,809	20,265	24,426	30,437	35,189	87.8	104.4	124.4	153.2	175.0
Donovanosis	22	32	16	16	10	0.1	0.2	0.1	0.1	0.1
Gonococcal infection	5,862	6,254	6,433	6,828	7,098	30.6	32.2	32.8	34.4	35.3
Syphilis (all categories)	2,028	1,846	2,015	2,012	2,296	10.6	9.5	10.3	10.1	11.4
Syphilis < 2 years duration	235	203	374	480	596	1.2	1.0	1.9	2.4	3.0
Syphilis > 2 years or unknown duration	1497	711	1115	1180	1561	7.8	3.7	5.7	5.9	7.8
Syphilis – congenital	4	21	18	15	11	0.0	0.1	0.1	0.1	0.1
Vaccine preventable diseases										
Diphtheria	0	1	0	0	0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	26	20	31	23	15	0.1	0.1	0.2	0.1	0.1
Influenza (laboratory confirmed)[¶]	–	1,291	3,674	3,491	2,073	–	6.7	18.7	17.6	10.3
Measles	108	140	32	98	45	0.6	0.7	0.2	0.5	0.2
Mumps	212	117	69	82	102	1.1	0.6	0.4	0.4	0.5
Pertussis	5,711	9,325	5,570	5,159	8,557	29.8	48.0	28.4	26.0	42.5
Pneumococcal disease (invasive)	–	1,795	2,430	2,303	2,375	–	9.2	12.4	11.6	11.8
Rubella	313	266	254	55	33	1.6	1.4	1.3	0.3	0.2
Rubella – congenital	0	0	1	3	1	0.0	0.0	0.0	0.0	0.0
Tetanus	8	3	4	4	5	0.0	0.0	0.0	0.0	0.0

Table 4. Notifications and notification rates (per 100,000 population), of communicable diseases, Australia, 2000 to 2004, *continued*

Disease	Notifications					Rate per 100,000 population				
	2000	2001	2002	2003	2004	2000	2001	2002	2003	2004
Vectorborne diseases										
Barmah Forest virus infection	616	1,148	896	1,369	1,052	3.2	5.9	4.6	6.9	5.2
Dengue	197	180	169	854	326	1.0	0.9	0.9	4.3	1.6
Flavivirus (NEC)**	65	38	73	61	49	0.3	0.2	0.4	0.3	0.3
Japanese encephalitis virus	–	0	0	1	1	–	0.0	0.0	0.0	0.0
Kunjin virus††	–	5	0	19	12	–	0.0	0.0	0.1	0.1
Malaria	967	717	469	598	559	5.0	3.7	2.4	3.0	2.8
Murray Valley encephalitis virus	16	6	2	0	1	0.1	0.0	0.0	0.0	0.0
Ross River virus infection	4,160	3,256	1,458	3,832	4,000	21.7	16.8	7.4	19.3	19.9
Zoonoses										
Anthrax	–	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
Brucellosis	28	21	39	19	36	0.1	0.1	0.2	0.1	0.2
Leptospirosis	249	249	163	132	166	1.3	1.3	0.8	0.7	0.8
Ornithosis‡‡	99	136	212	201	235	0.5	0.7	1.1	1.0	1.2
Lyssavirus (NEC)	–	0	0	0	0	–	0.0	0.0	0.0	0.0
Q fever	548	685	784	583	440	2.9	3.5	4.0	2.9	2.2
Other bacterial infections										
Legionellosis	470	309	317	340	310	2.5	1.6	1.6	1.7	1.5
Leprosy	4	9	6	5	5	0.0	0.0	0.0	0.0	0.0
Meningococcal infection§§	622	700	686	578	408	3.2	3.6	3.5	2.9	2.0
Tuberculosis	581	963	1,051	993	1,076	3.0	5.0	5.4	5.0	5.4
Total	90,143	105,588	101,718	106,193	110,929					

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

† In the Northern Territory and 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.

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

** Flavivirus (NEC) replaces 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.

– The condition was not nationally notifiable in that year.

Bloodborne diseases

In 2004, bloodborne viruses reported to the NNDSS included 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: http://www.med.unsw.edu.au/nchecr

When reported to NNDSS, newly acquired (incident) hepatitis B and hepatitis C infections were differentiated from those where the timing of disease acquisition was unknown (unspecified). As considerable time may have elapsed between the date of disease acquisition and the date an unspecified hepatitis infection is first diagnosed, the analysis of hepatitis B (unspecified) and hepatitis C (unspecified) infections is by date of diagnosis, which is the earliest of specimen, notification or notification received dates supplied.

Hepatitis B

Incident hepatitis B notifications

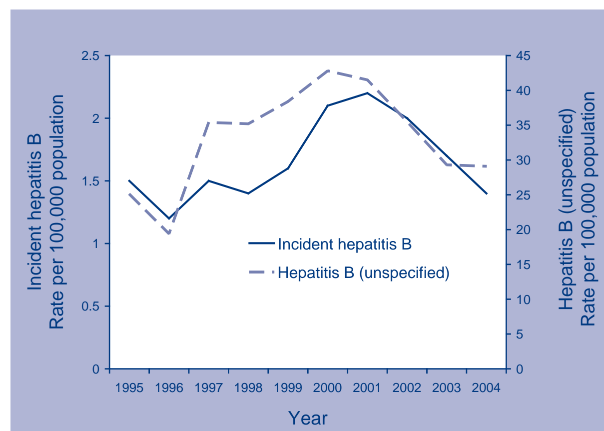
Case definition – Hepatitis B (incident)

*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 2004, 275 incident hepatitis B infections were reported to the NNDSS, giving a national notification rate of 1.4 cases per 100,000 population. The highest rates were reported from the Northern Territory (4 cases per 100,000 population) and Tasmania (3.7 cases per 100,000 population). The rate of notification of incident hepatitis B infection increased from 1.5 in 1995 to 2.2 in 2002 and declined to 1.4 per 100,000 population in 2004 (Figure 5).

Figure 5. Trends in notification rates incident hepatitis B and hepatitis B (unspecified), Australia, 1995 to 2004*

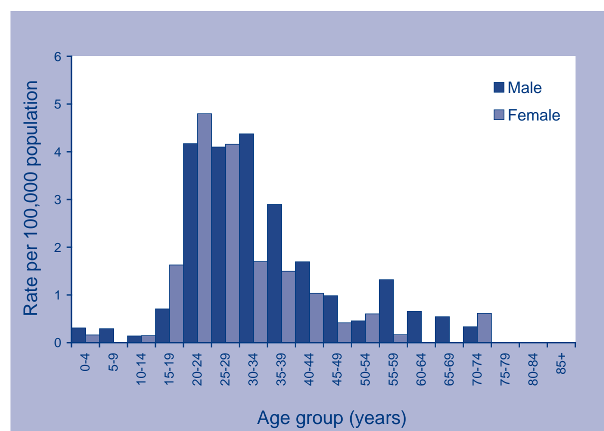


* Year of onset for incident hepatitis B and year of report for hepatitis B (unspecified) notifications.

The increased rates of newly acquired hepatitis B infection in 2000–2002 were attributed to increased transmission among injecting drug users in Victoria, followed by a decline in transmission between 2002 and 2004 during a heroin 'drought' (Greg Dore, personal communication).

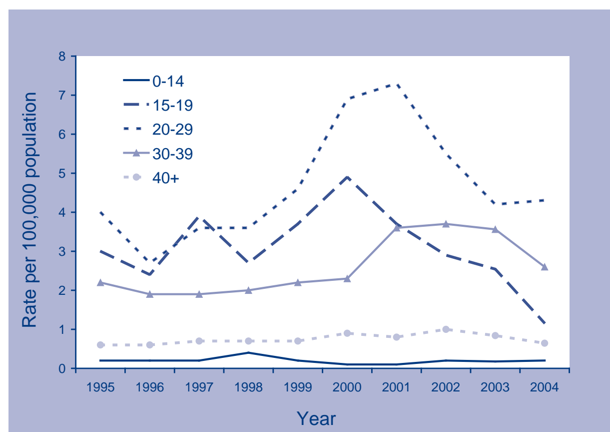
In 2004, the highest rate of incident hepatitis B infection was in the 20–24 year age group for females (4.8 cases per 100,000 population) and in the 30–34 year age group for males (4.2 cases per 100,000 population, Figure 6). Overall, infections in males exceeded those in females, with a male to female ratio of 1.4:1.

Figure 6. Notification rate for incident hepatitis B infections, Australia, 2004, by age group and sex



Trends in incident hepatitis B infection by year and age group are shown in Figure 7.

Figure 7. Trends in notification rates of incident hepatitis B infections, Australia, 1995 to 2004, by age group



In the past five years, rates of incident hepatitis B notifications fell by 75 per cent among cases in the 15–19 year age group and by 38 per cent among cases in the 20–24 year age group. The reported source of exposure for cases of incident hepatitis B infection in 2004 was reported from South Australia, Tasmania and Victoria (Table 5).

The proportion of newly acquired hepatitis B infections associated with injecting drug use increased from 44 per cent in 2002 to 53 per cent in 2004. By contrast, the proportion of newly acquired hepatitis B infections associated with sexual contact declined from 26 per cent in 2002 to 22 per cent in 2004.[2]

Table 5. Incident hepatitis B infection, Australia,* 2004, by exposure category

Exposure category	Number	Percentage
Injecting drug use	74	52.8
Sexual contact	31	22.2
Male homosexual contact	*1*	
Heterosexual contact	*30*	
Blood/tissue recipient	0	
Skin penetration procedure	0	
Healthcare exposure	0	
Household contact	1	0.7
Other	1	0.7
Undetermined	33	23.6
Total	140	100.0

* Data from South Australia, Tasmania and Victoria only, (National Centre in HIV Epidemiology and Clinical Research, 2005[2]).

Hepatitis B (unspecified) notifications

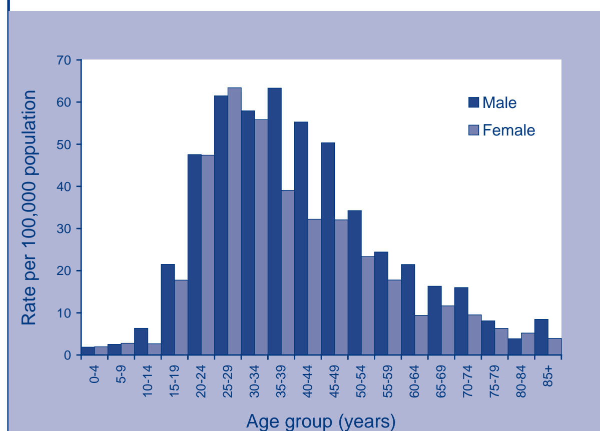
Case definition – Hepatitis B – unspecified

*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 2004, 5,861 cases of hepatitis B (unspecified) infection were notified to NNDSS, giving a rate of 29.1 cases per 100,000 population. New South Wales (42.4 cases per 100,000 population) and Victoria (29.8 cases per 100,000 population) recorded the highest notification rates. The male to female ratio was 1.3:1. Among males, the highest notification rate was in the 35–39 year age group (63.3 cases per 100,000 population), whereas among females, the highest notification rate was in the 25–29 year age group (63.4 cases per 100,000 population, (Figure 8). The rate of notification of hepatitis B (unspecified) infection increased from 19.4 in 1996 to 42.8 in 2000 and declined to 29.1 cases per 100,000 population in 2004 (Figure 8).

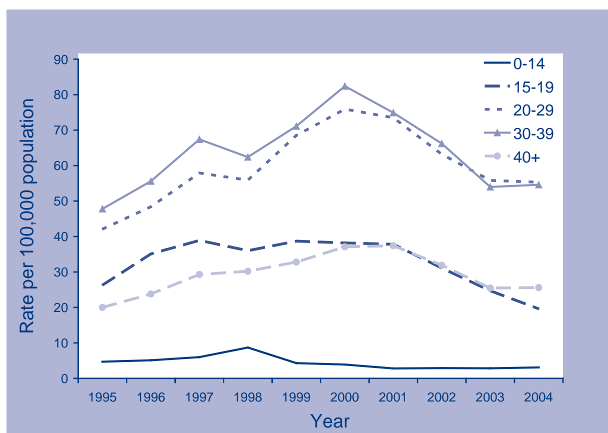
Figure 8. Notification rate for hepatitis B (unspecified) infections, Australia, 2004, by age group and sex*



Trends in hepatitis B (unspecified) infection by age group and year are shown in Figure 9.

Rates of hepatitis B (unspecified) notifications in 2000–2004 fell by 49 per cent among cases in the 15–19 year age group, 27 per cent in the 20–29 year age range and 22 per cent in the 30–39 year age range. Rates in other age groups remained relatively stable.

Figure 9. Trends in notification rates of hepatitis B (unspecified) infections, Australia, 1995 to 2004, by age group*



In 2004, 28 cases of HBV infection (3 incident and 25 unspecified) in children in the 0–4 year age group were reported. Approximately 95 per cent of infants born in 2004 received hepatitis B vaccination in Australia.[3]

Hepatitis C

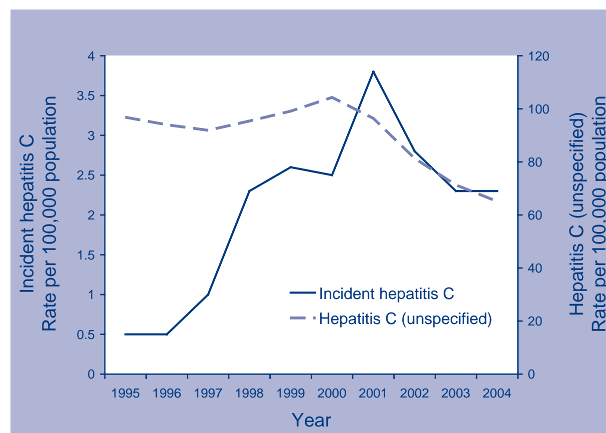
Incident hepatitis C notifications

> *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.*

The number of incident hepatitis C notifications as a reflection of the incidence of hepatitis C in Australia should be interpreted with caution. It is known that the notification rate vastly underestimates the true incidence of hepatitis C.

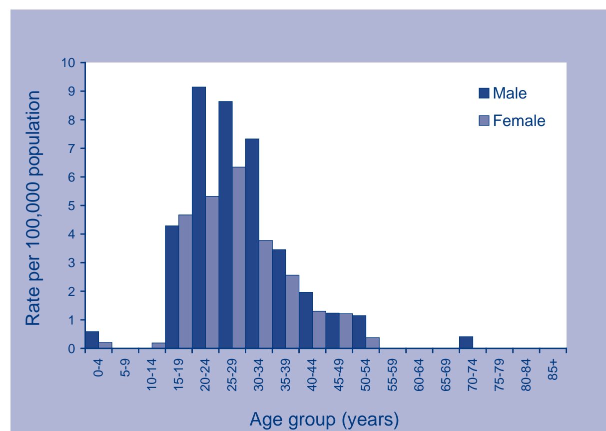
A total of 361 incident cases of hepatitis C with an onset date in 2004 were notified, giving a rate of 2.3 cases per 100,000 population (Figure 10). The proportion of all hepatitis C notifications in 2004 that were documented as incident cases was 2.7 per cent. The highest rate of incident hepatitis C infection was reported from Western Australia (6.1 cases per 100,000 population).

Figure 10. Trends in notification rates, incident and hepatitis C (unspecified) infection, Australia, 1995 to 2004



In 2004, the highest rate of incident hepatitis C notification was in the 20–24 year age group for males (9.1 cases per 100,000 population) and the 25–29 year age group for females (6.3 cases per 100,000 population, Figure 11). Overall, the male to female ratio was 1.5:1.

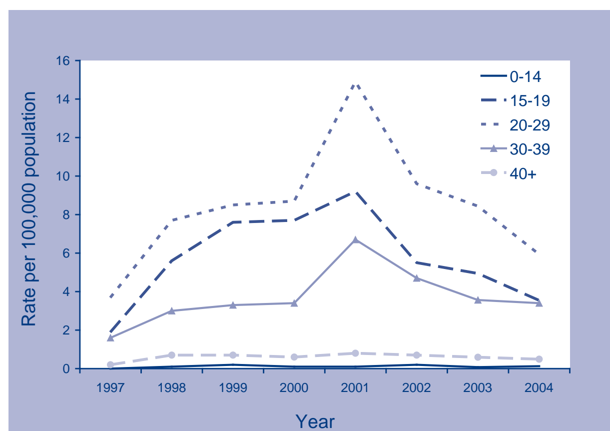
Figure 11. Notification rate for incident hepatitis C infections, Australia, 2004, by age group and sex



Trends in the age distribution of incident hepatitis C infection are shown in Figure 12.

The notification rates for incident hepatitis C declined from 2001 to 2004 by 62 per cent in the 15–19 year age group, 60 per cent in the 20–29 year age group and 50 per cent in the 30–39 year age group (Figure 12).

Figure 12. Trends in notification rates of incident hepatitis C infections, Australia, 1997 to 2004, by age group



The exposure history of cases of incident hepatitis C was collected in the Australian Capital Territory, South Australia, Tasmania, Victoria and Western Australia in 2004 (Table 6). At least 70 per cent of incident hepatitis C infections in 2004 were among injecting drug users.

In 2004, an estimated 259,570 people were living with hepatitis C in Australia. Of these 65,300 people cleared their infection, 153,300 had chronic hepatitis C and early liver disease (stage 0/1), 32,800 had chronic hepatitis C infection and moderate liver disease (stage 2/3) and 8,160 were living with hepatitis C related cirrhosis.[2]

Table 6. Incident hepatitis C infection, Australia,* 2004, by exposure category

Exposure category	Number	Percentage
Injecting drug use	210	70.0
Sexual contact	13	4.3
Blood/tissue recipient	4	1.3
Skin penetration procedure	7	2.3
Healthcare exposure	2	0.6
Household contact	1	0.3
Other	9	3.0
Undetermined	54	18.0
Total	300	100.0

* Data from the Australian Capital Territory, South Australia, Tasmania, Victoria and Western Australia only, (National Centre in HIV Epidemiology and Clinical Research, 2005[2])

Hepatitis C (unspecified) notifications

Case definition – Hepatitis C (unspecified)

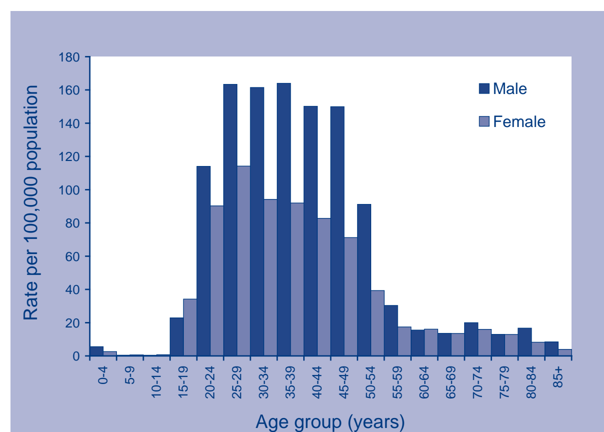
*Only **confirmed cases** are reported.*

***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.*

National notification rates of hepatitis C (unspecified) infection ranged between 96 and 104 cases per 100,000 population in 1995–2001. The national rate declined to 81.3 in 2002 and to 63.7 cases per 100,000 population in 2004 (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 2004, 12,667 hepatitis C (unspecified) infections were notified to NNDSS, giving a notification rate of 63 cases per 100,000 population. Of the total notifications of hepatitis C (unspecified), 39 per cent were from New South Wales, but the Northern Territory had the highest notification rate (135.6 cases per 100,000 population). The male to female ratio was 1.6:1. The highest reporting rates were in the 35–39 year age group for males (164 cases per 100,000 population), and in the 25–29 year age group for females (114.2 cases per 100,000 population, Figure 13).

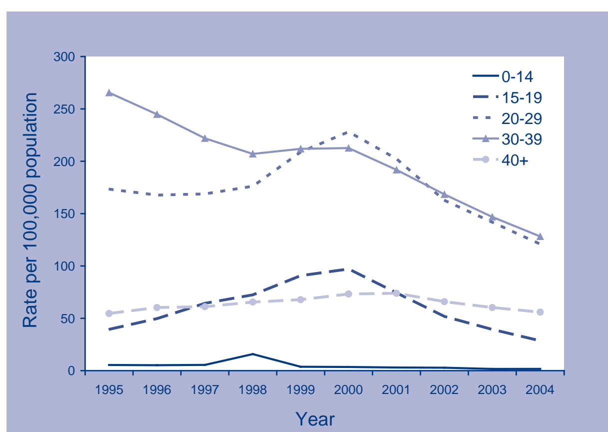
Figure 13. Notification rate for hepatitis C (unspecified) infections, Australia, 2004, by age group and sex



Trends in the age distribution of hepatitis C (unspecified) infections are shown in Figure 14.

Between 2000 and 2004, the notification rates of hepatitis C (unspecified) fell by 71 per cent among cases in the 15–19 year age group, suggesting declining hepatitis C incidence among young people with a history of injecting drug use. Notification rates of hepatitis C (unspecified) also fell in the same period by 47 per cent among cases in the 20–29 year age range and by 40 per cent in the 30–39 year age range. Rates in the other age groups have remained relatively stable during this period.

Figure 14. Trends in notification rates of hepatitis C (unspecified) infections, Australia, 1995 to 2004, by age group



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 superinfection 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 27 notifications of hepatitis D to the NNDSS in 2004 giving a notification rate of 0.1 cases per 100,000 population. Of the 27 notifications, 14 were reported from New South Wales, 10 from Queensland and 3 from Victoria. The majority (19/27, 70%) of cases were males, with the highest number of cases reported in 40–44 and 45–49 year age groups.

Gastrointestinal diseases

In 2004, gastrointestinal diseases that were notified to NNDSS were: botulism, campylobacteriosis, cryptosporidiosis, haemolytic uraemic syndrome (HUS), hepatitis A, hepatitis E, listeriosis, salmonellosis, shigellosis, Shiga toxin-producing *Escherichia coli*/verotoxigenic *E. coli* (STEC/VTEC) infections and typhoid.

Notifications of gastrointestinal diseases increased by 2 per cent; from 24,676 in 2003 to 25,248 in 2004 (Table 4). Compared with 2003, there was a decrease in the number of notifications of campylobacteriosis (2%), hepatitis A (28%), listeriosis (7%) and STEC (15%) in 2004. On the other hand, increases were reported for cryptosporidiosis (28%), hepatitis E (107%), salmonellosis (8%), shigellosis (17%) and typhoid (43%). The reported changes in the number of notifications were within the expected range (i.e. within the five year mean and two standard deviations) except for hepatitis E which had an excess of 13 cases above the upper 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).*

One case of infant botulism in a female, less than 12 months old was reported to NNDSS in 2004 (Table 2). Since the commencement of the surveillance of botulism in 1992 there have been six cases of infant botulism reported, but no classic foodborne botulism has been reported in Australia since NNDSS commenced collecting data on botulism in 1992.

Case definition – Campylobacteriosis

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

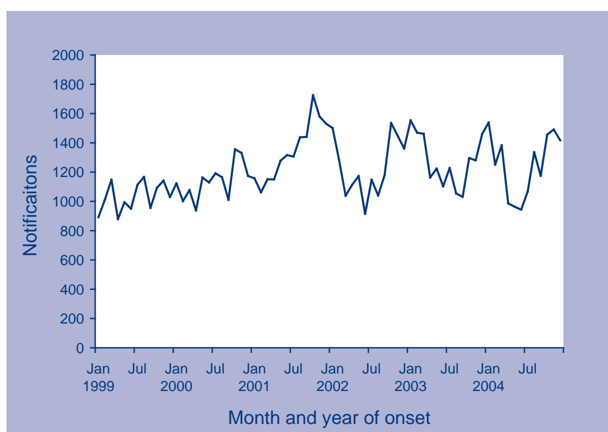
***Confirmed case:** Requires isolation or detection of* Campylobacter *species.*

Campylobacteriosis

There were 15,008 notifications of campylobacteriosis in Australia in 2004. Campylobacteriosis is notifiable in all jurisdictions, except New South Wales. The national rate of notifications in 2004 was 112 cases per 100,000 population; a marginal decrease compared with the rate reported in 2003 (116 cases per 100,000 population). All jurisdictions with the exception of Victoria reported decreases in notifications, with South Australia reporting the largest decrease (30%). Victoria reported a 12 per cent increase in notifications, and had the highest notification rate in 2004 (127 cases per 100,000 population).

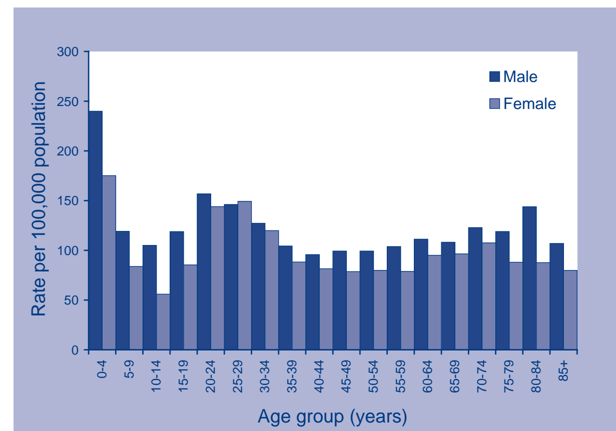
Monthly notifications of campylobacteriosis in 2004, consistent with previous years (1999 to 2003), peaked in the third quarter of the year in late winter/early spring (Figure 15). In 2004, seven *Campylobacter* related outbreaks were identified, of which four were suspected to be foodborne.[4] These suspected foodborne outbreaks occurred in an aged care facility, restaurant and food takeaway settings.

Figure 15. Trends in notifications of campylobacteriosis, Australia, 1999 to 2004, by month of onset



Children aged 0–4 years had the highest notification rate of campylobacteriosis (Figure 16). In this age group notification rates were higher in males (243 cases per 100,000 population) than in females (175 cases per 100,000 population). The overall male to female ratio, as in previous years, was 1.2:1.

Figure 16. Notification rates of campylobacteriosis, Australia, 2004, by age group and sex



Cryptosporidiosis

Case definitions – Cryptosporidiosis

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

Confirmed case: Requires detection of Cryptosporidium *oocytes.*

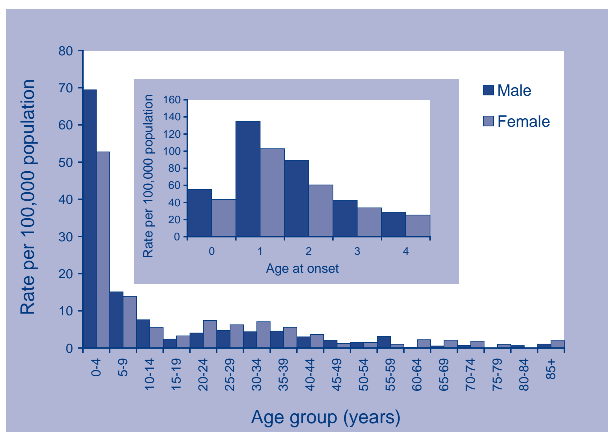
In 2004, a total of 1,573 cases of cryptosporidiosis were reported to NNDSS, a notification rate of 8 cases per 100,000 population, which represents an increase of 28 per cent on the 1,225 cases reported in 2003.

New South Wales, the Northern Territory, Queensland, and Victoria reported increases in cryptosporidiosis notifications, with the largest increase in Queensland (276%). The Northern Territory and Queensland had notification rates above the national average at 57 and 16 cases per 100,000 population, respectively.

Fifty per cent of cryptosporidiosis cases notified in 2004 were under the age of five years. Compared to 2003, the notification rate in this age group increased by 24 per cent in 2004. With a notification rate of 61 cases per 100,000 population, children under the age of four years continue to have the highest notification rate of cryptosporidiosis. Within

this age group one-year-old males had the highest notification rate at 130 cases per 100,000 population (Figure 17).

Figure 17. Notification rates of cryptosporidiosis, Australia, 2004, by age group and sex

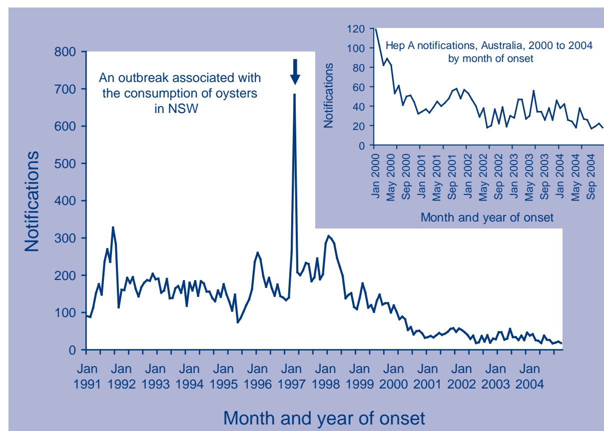


Hepatitis A

> *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 315 cases of hepatitis A reported to NNDSS in 2004, a notification rate of 2 cases per 100,000 population. The notifications of hepatitis A have steadily decreased over the last decade, but remained stable in the period 2002 through 2004 (Figure 18).

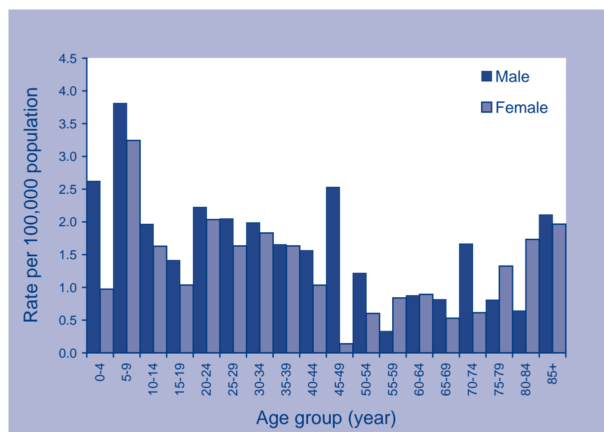
Figure 18. Trends in notifications of hepatitis A, Australia, 1991 to 2004, by month of notification



Compared to 2003, hepatitis A notification rates decreased in all jurisdictions (ranging from 15% in South Australia to 92% in Tasmania) except in New South Wales where an increase of 9 per cent was reported. The Northern Territory had the highest notification rate (7 cases per 100,000 population) followed by New South Wales (3 cases per 100,000 population).

Males, with a rate of 1.8 cases per 100,000 population had a higher notification rate of hepatitis A than females (1.3 cases per 100,000 population). The highest age specific rate of hepatitis A notifications among males and females was in the 5–9 year age group (3.8 cases and 2.8 cases per 100,000 population, respectively) (Figure 19).

Figure 19. Notification rates of hepatitis A, Australia, 2004, by age group and sex



In 2004, Indigenous Australians had the highest burden of hepatitis A. In 2004, Indigenous status of 90 per cent of cases was complete and 11 per cent of cases were Indigenous (Table 7).

Hepatitis A is commonly spread from person to person or from contaminated food or water. Where information on risk factors was known (in 22% of all notifications), overseas travel and household contact with a case were the main risk factors for hepatitis A infection (Table 8).

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.*

Table 7. Hepatitis A notifications, Australia, 2004, by Indigenous status

State or territory	Indigenous	Non-Indigenous	Unknown	Total	% Indigenous (of total)
ACT	0	0	1	1	–
NSW	1	113	25	139	0.7
NT	5	8	0	13	38.5
Qld	0	20	2	22	–
SA	1	10	0	11	9.1
Tas	0	0	1	1	–
Vic	0	67	4	71	–
WA	28	28	1	57	49.1
Total	35	246	34	315	11.1

Table 8. Risk exposures associated with hepatitis A virus infection, Australia, 2004, by state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total	1	139	13	22	11	1	71	57	315
Number of case with known risk factors*	0	57	6	11	6	0	41	21	142
Injecting drug use	–	0	0	0	1	0	4	–	7
Household/close contact of case	–	14	2	2	1	0	11	9	39
Overseas travel	–	41	1	12	3	0	28	9	94
Childcare	–	2	3	0	0	0	0	1	6
Homosexual contact	–	–	0	0	0	0	1	0	1
Sex worker	–	–	0	0	0	0	0	0	0
Other†	–	–	0	0	2	0	0	0	2

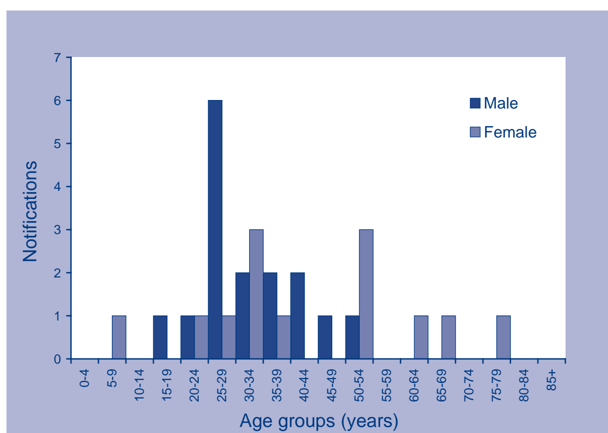
* Number of risk factors may not add up to the totals as exposures are not mutually exclusive hence more than one exposure per person is possible.

† Includes association with persons from country where hepatitis A is endemic and, living in areas where hepatitis A is endemic.

– Not assessed.

There were 28 cases of hepatitis E reported to NNDSS in 2004, an increase of 100 per cent on the number of cases reported in 2003. Twelve cases were reported in Victoria, eight in New South Wales, four in Queensland, three in Western Australia and one in Tasmania. The male to female ratio was 1.2:1. Cases were aged between 5 and 79 years (Figure 20). Data on countries visited were available for 26/28 cases with overseas travel and showed that 18 had travelled to India, two to Bangladesh and one each to China, Indonesia, Peru, Vietnam, Thailand and New Zealand.

Figure 20. Notification rates of hepatitis E, Australia, 2004, by age group and sex



Hepatitis E virus is transmitted enterically. In non-industrialised countries, where sanitation is poor water-borne transmission of hepatitis E occurs, while in industrialised countries zoonotic transmission (from pigs to humans) has been recorded. In Australia, locally acquired hepatitis E was reported in the early 1990s.[5]

Listeriosis

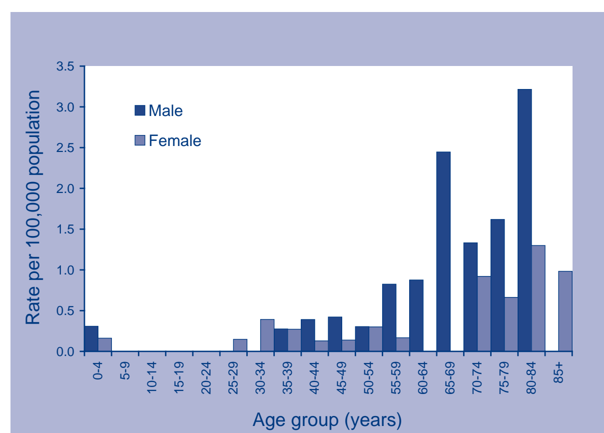
Case definitions – Listeriosis

*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 2004, 65 cases of listeriosis were reported to NNDSS, a notification rate of 0.3 cases per 100,000 population. Listeriosis notifications have been stable at this rate since 1998. In 2004, 71 per cent of listeriosis cases were aged over 50 years, with the highest notification rate in the 80–84 year age group in males and females (Figure 21).

Figure 21. Notification rates of listeriosis, Australia, 2004, by age group and sex



In 2004, there were seven listeriosis cases of materno-foetal origin and one foetal death was reported.[6] Health outcome for 29 cases was known, and of these, four cases all aged over 66 died. No common-source outbreaks of listeriosis were identified during 2004.[6]

Salmonellosis (non-typhoidal)

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).*

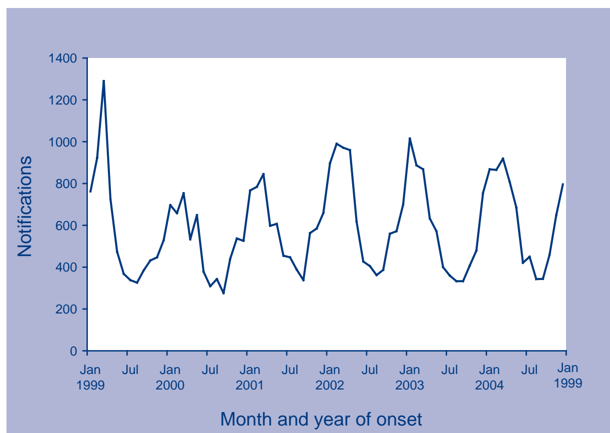
A total of 7,607 salmonellosis cases were reported to NNDSS in 2004, a rate of 37.8 cases per 100,000 population and a 7 per cent increase from the rate reported in 2003 (35.4 cases per 100,000 population). During the five year period, 1998–2003, the highest national notification rate was 40 cases per 100,000 population in 2002.

The Northern Territory and Queensland had notification rates 5 and 1.6 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 2004, the Kimberley Statistical Division of Western Australia had the highest notification rate at 309 cases per 100,000 population. The same Statistical Division had a notification rate of 323 cases per 100,000 population in 2003.

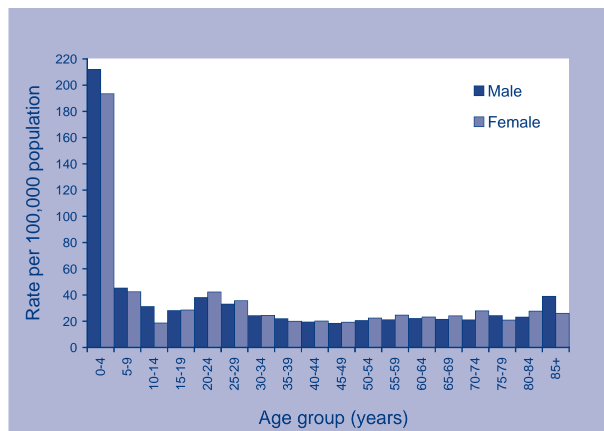
As in previous years, reports of salmonellosis peaked during summer (January to March) (Figure 22). Thirty-five per cent of salmonellosis cases in 2004 had dates of onset during the first quarter of the year.

Figure 22. Trends in notifications of salmonellosis, Australia, 1999 to 2004, by month of onset

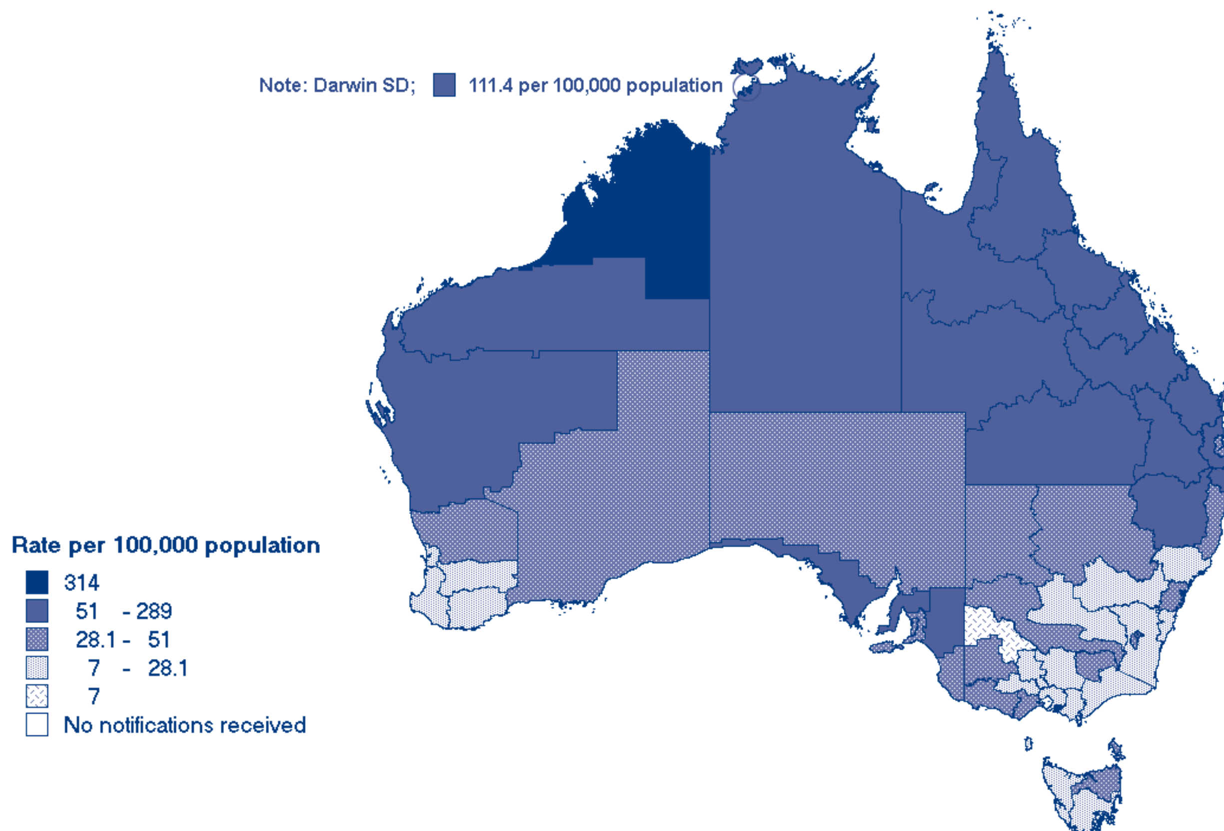


As in 2003, the highest rate of notification was in children aged between 0–4 years: 32 per cent of salmonellosis notifications were in this age group (Figure 23).

Figure 23. Notification rates of salmonellosis, Australia, 2004, by age group and sex



Map 2. Notification rates of salmonellosis, Australia, 2004, by Statistical Division of residence



The National Enteric Pathogens Surveillance Scheme reported serovars for 7,771 isolates in 2004.[7] The 10 most frequently isolated serovars and phage types of *Salmonella,* which accounted for 43 per cent of all isolates, are shown in Table 9. Nationally, *Salmonella* Typhimurium 135, *Salmonella* Typhimurium 170 and *S.* Saintpaul were the three most frequently isolated serovars/phage types. In 2003, *S.* Saintpaul was ranked fourth among the most notified serovars. *S.* Typhimurium 12 was for the first time, in the top 10 serovars in 2004, replacing *Salmonella* Typhimurium 290.

In 2004, there was little change to the distribution of *Salmonella* serovars reported in 2003. The most commonly reported serovars in Queensland, Tasmania, and the Northern Territory were *S.* Virchow 8 (9% of salmonellosis notifications), *S.* Mississippi (52% of salmonellosis notifications) and *S.* Ball (15% of salmonellosis notifications), respectively. Typhimurium was the most commonly reported serovar in the rest of the jurisdictions. Typhimurium 170 accounted for 55 per cent of cases in the Australian Capital Territory, 17 per cent in New South Wales, 11 per cent in Victoria, and 13 per cent in South Australia. In Western Australia, Typhimurium 135 was the most commonly notified phage type, making 12 per cent of salmonellosis notifications.

Outbreaks and clusters of salmonellosis

In 2004, OzFoodNet reported 118 foodborne disease outbreaks of which 29 were attributable to *S.* Typhimurium infection. These outbreaks affected 599 persons and resulted in 74 hospitalisations. Of the six significant foodborne outbreaks (affecting 50 or more persons each) in 2004, two were due to Typhimurium: phage types 12 in New South Wales and phage type 9 in Victoria. The outbreak that occurred in New South Wales was a community-wide outbreak. In this outbreak, investigators found that the consumption of home prepared chicken was the main risk factor for *S.* Typhimurium 12 infection. The outbreak in Victoria was associated with the consumption of food from a pizza restaurant. *S.* Typhimurium 9 was isolated from several foods, suggesting that there had been cross contamination of foods.[8]

Shigellosis

Case definitions – Shigellosis

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

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

In 2004, a total of 518 cases of shigellosis were reported to NNDSS, a notification rate of 2.6 cases per 100,000 population. This rate was 18 per cent higher than the rate reported in 2003 (2.2 cases per 100,000 population), but it was within the five year average (Table 4). The Northern Territory continued to have the highest notification rate at 59.5 cases per 100,000 population, but this was a decrease by 10 per cent in notification rates compared to 2003.

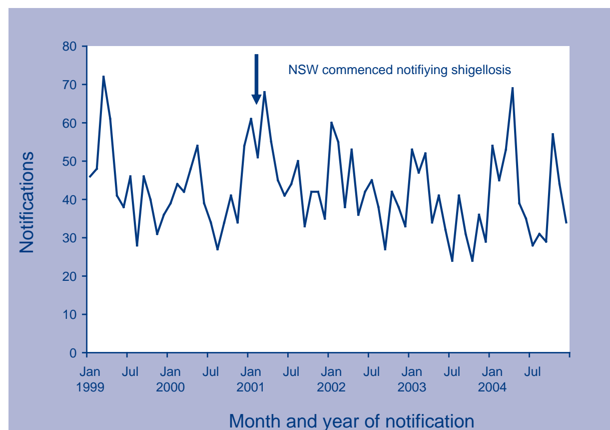
Table 9. Top 10 human isolates of *Salmonella,* Australia, 2004

Organism	State or territory									Total (%)
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust	
S. Typhimurium 170	33	357	2	50	0	4	129	2	577	7.4
S. Typhimurium 135	5	189	3	180	18	1	92	75	563	7.2
S. Saintpaul	1	41	49	226	13	2	20	42	394	5.1
S. Typhimurium 9	6	119	0	43	44	4	130	14	360	4.6
S. Virchow 8	4	43	1	248	9	2	26	0	333	4.3
S. Typhimurium 197	9	48	0	147	2	0	61	1	268	3.4
S. Birkenhead	1	80	1	167	1	1	11	1	263	3.4
S. Typhimurium 12	3	172	0	30	8	0	18	2	233	3.0
S. Chester	2	34	12	87	20	1	11	23	190	2.4
S. Infantis	6	59	7	11	21	1	43	10	158	2.0
Sub Total	70	1,142	75	1,189	136	16	541	170	3,339	43.0
Other isolates	37	1,005	296	1,559	392	104	598	441	4,432	57.0
Total	107	2,147	371	2,748	528	120	1139	611	7,771	100

Source: National Enteric Pathogens Surveillance System.

Nationally, notifications of the disease remained stable over the last five years (Figure 24). The male to female ratio remained at 0.8:1.

Figure 24. Trends in notifications of shigellosis, Australia, 1999 to 2004, by month of onset

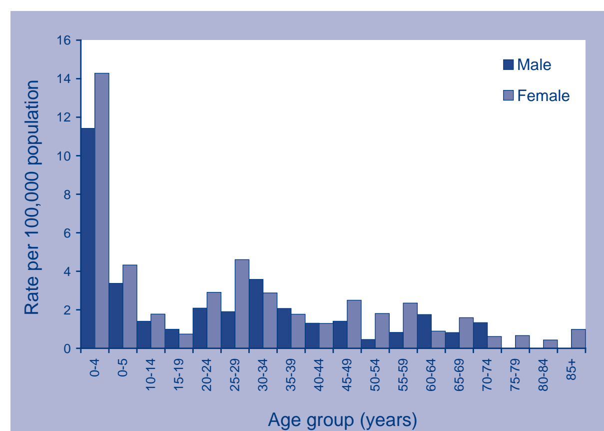


Children under the age of four years represented 31 per cent of shigellosis notifications (Figure 25). This age group had a notification rate of 13 cases per 100,000 population, which is five times the national rate and an increase of 18 per cent compared to the rate reported in 2003 (11 cases per 100,000 population).

Indigenous populations continue to have the highest burden of shigellosis. In 2004, of the notifications of shigellosis where Indigenous status of cases was complete (64% of all cases) 37 per cent were identified as Indigenous. In the Northern Territory (where 98% of notifications had the Indigenous status of the case recorded), 82 per cent of shigellosis cases were Indigenous people.

Shigella flexneri and *Shigella sonnei* infections accounted for about 50 per cent and 48 per cent of shigellosis, respectively in 2004 (Table 10).

Figure 25. Notification rates of shigellosis, Australia, 2004, by age group and sex



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

> *Case definitions – Shiga toxin-producing/verotoxin-producing* Escherichia coli *(STEC/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 STEC/VTEC is isolated in the context of haemolytic uraemic syndrome (HUS), it should be notified as STEC/VTEC and HUS.*

Table 10. *Shigella* infections, Australia, 2004, by serogroup and state or territory

Organism	State or territory								Total	Per cent
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
S. boydii		2		1	1		2	1	7	1.9
S. dysenteriae		1		1				1	3	0.8
S. flexneri		32		16	39	2	28	67	184	49.5
S. sonnei	2	59		30	13	1	37	36	178	47.8
Sub Total	2	94	0	48	53	3	67	105	372	100.0
Unknown	0	2	119	13	1	0	3	8	146	–
Total	2	96	119	61	54	3	70	113	518	–

There were 44 cases of SLTEC/VTEC reported to NNDSS in 2004. With a notification rate of 0.2 cases per 100,000 population, the rate of SLTEC/VTEC notifications remained stable compared to 2003. Seventy-three per cent of cases were notified in South Australia (1.8 cases per 100,000 population), where bloody stools are routinely tested by polymerase chain reaction (PCR) for genes coding for Shiga toxin. New South Wales, Queensland, and Victoria were the only other jurisdictions that notified SLTEC/VTEC. OzFoodNet reported that among typed *E. coli* (67% of all notifications) 15 per cent were subtype O157, 16 per cent were subtype O11 and 13 per cent were O26.[6]

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 STEC/VTEC is isolated in the context of HUS, it should be notified as both STEC/VTEC and HUS.*

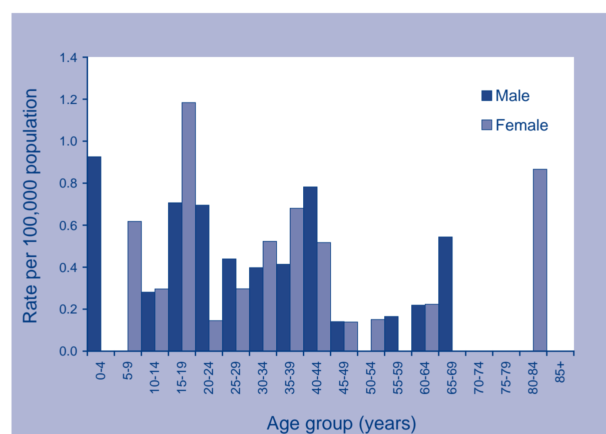
In 2004, 15 cases of HUS were reported to NNDSS, a rate of 0.1 cases per 100,000 population, the same rate as in 2003. No HUS cases were notified in the Australian Capital Territory, Tasmania, the Northern Territory or Western Australia. Among the 15 cases of HUS notified in 2004, six were males. The median age among males was 19 years (range 2–54 years) and among females was 34 years (range 0–82 years). STEC was isolated in three cases of HUS.

Typhoid

> *Case definitions – Typhoid fever*
>
> *Only **confirmed cases** are reported.*
>
> ***Confirmed case:** Requires isolation or detection of* Salmonella *Typhi.*

In 2004, there were 73 notifications of typhoid, a rate of 0.4 cases per 100,000 population, representing an increase of 43 per cent compared to 2003. The largest increase, compared to 2003, occurred in New South Wales (increase of 143%). Nationally, the male to female ratio was 1:1, with the highest notification rates in males aged 0–4 years (0.9 cases per 100,000 population) and in females aged 15–19 years (1.2 cases per 100,000 population) (Figure 26). The National Enteric Pathogen Surveillance Scheme identified 71 *Salmonella* Typhi isolates, 68 of which were from Australian residents. Of the 68 Australian residents, 17 had no travel history recorded, two had not travelled, and the remaining 49 cases had travelled outside Australia in South East Asia, Africa, Europe, Pacific Islands, and South America.[7]

Figure 26. Notification rates of typhoid, Australia, 2004, by age group and sex



Quarantinable diseases

Human diseases covered by the Quarantine Act 1908, and notifiable in 2004 were cholera, plague, rabies, yellow fever, smallpox, highly pathogenic avian influenza in humans (HPAIH), severe acute respiratory syndrome (SARS) and four 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 2004, there were five cases of cholera notified in Australia, two from Victoria, and one each from New South Wales, Queensland and Western Australia. Four of these cases acquired their disease overseas: one in Indonesia, one in the Philippines, and two in India. The place of acquisition of the fifth case was unknown.

All five notifications were *Vibrio cholerae* serogroup O1. There were two El Tor biotype notifications and two Ogawa serotypes reported. Table 11 summarises the serogroups, biotypes, serotypes and toxin producing status of these notifications.

In 2004, there were several suspected cases of SARS reported by jurisdictions. Enhanced surveillance by general practitioners and hospitals in Australia resulted in the testing of five people with fever, respiratory symptoms and history of travel to China. All tests for SARS were negative.

Cholera, plague, rabies, yellow fever, SARS, HPAIH 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 the Australian Government Department of Health and Ageing Website at: http://www.health.gov.au/internet/wcms/Publishing.nsf/Content/health-pubhlth-strateg-quaranti-index.htm

Sexually transmissible infections

In 2004, sexually transmissible infections (STIs) reported to NNDSS were chlamydial infection, donovanosis, gonococcal infections and for the first time two categories of syphilis: syphilis – infectious (primary, secondary and early latent) less than 2 years duration and syphilis – of greater than 2 years or unknown duration. The NNDSS also received reports on congenital syphilis. These conditions were notifiable in all states and territories.

Other national surveillance systems that monitor STI 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 the NNDSS between 2000 and 2004 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, 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 per cent of notifications. These data however, have to be interpreted cautiously as STI screening occurs predominantly in specific high-risk groups including Indigenous populations. Similarly, rates between males and females need to be interpreted cautiously as rates of testing for STI differ between the sexes.

Table 11. Cholera notifications 2004, Australia, by notifying jurisdiction and case details

Notifying jurisdiction	Sex	Age at onset	*Vibrio cholerae* serogroup/biotype/serotype	Toxin production	Country of acquisition
NSW	Male	45	*Vibrio cholerae* O1 El Tor	Unknown	Philippines
Qld	Female	50	*Vibrio cholerae* O1	Unknown	Unknown
Vic	Female	23	*Vibrio cholerae* O1 Ogawa	Not reported	India
Vic	Female	34	*Vibrio cholerae* O1 El Tor Ogawa	Not reported	India
WA	Male	33	*Vibrio cholerae* O1	Unknown	Indonesia

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 continues to be the most commonly notified disease in 2004. A total of 35,189 notifications of chlamydial infection were received by the NNDSS; a rate of 175 cases per 100,000 population. This was the highest rate since surveillance of the condition commenced in 1991, and represents an increase of 14 per cent on the rate reported in 2003 (153 cases per 100,000 population). Between 2000 and 2004, chlamydial infection notification rates increased from 88 to 175 cases per 100,000 population, an increase of 99 per cent (Table 4).

Chlamydial infection notification rates were higher than the national average in the Northern Territory (820 cases per 100,000 population), Western Australia (218 cases per 100,000 population), Queensland (209 cases per 100,000 population) and the Australian Capital Territory (191 cases per 100,000 population) (Table 3). New South Wales had the largest percentage increase in 2004 compared to 2003 (27% increase). At the regional level, the Northern Territory excluding Darwin had the highest chlamydial infection notification rate at 1,691 cases per 100,000 population (Map 3).

In 2004, notification rates of chlamydial infection in males and females were 142 and 206 cases per 100,000 population respectively. In 2004, notification rates increased by 14 per cent in males and by 15 per cent in females compared to 2003. The male to female ratio remained at 0.7:1 as in the previous year. Rates in females markedly exceeded those in males in the 15–19 and 20–24 age groups with ratios of 1:4 and 1:2 respectively (Figure 27).

Trends in age and sex specific notification rates between 2000 and 2004 show increases in all age groups between 15 and 34 years in both males and females (Figure 28). Since 2000, the highest average annual percentage increase occurred in the 20–24 age group (23% in males and 21% in females). However, in 2004 the annual rate of increase declined relative to 2003, for all age groups. In the 20–24 age

Map 3. Notification rates of chlamydial infection, Australia, 2004, by Statistical Division

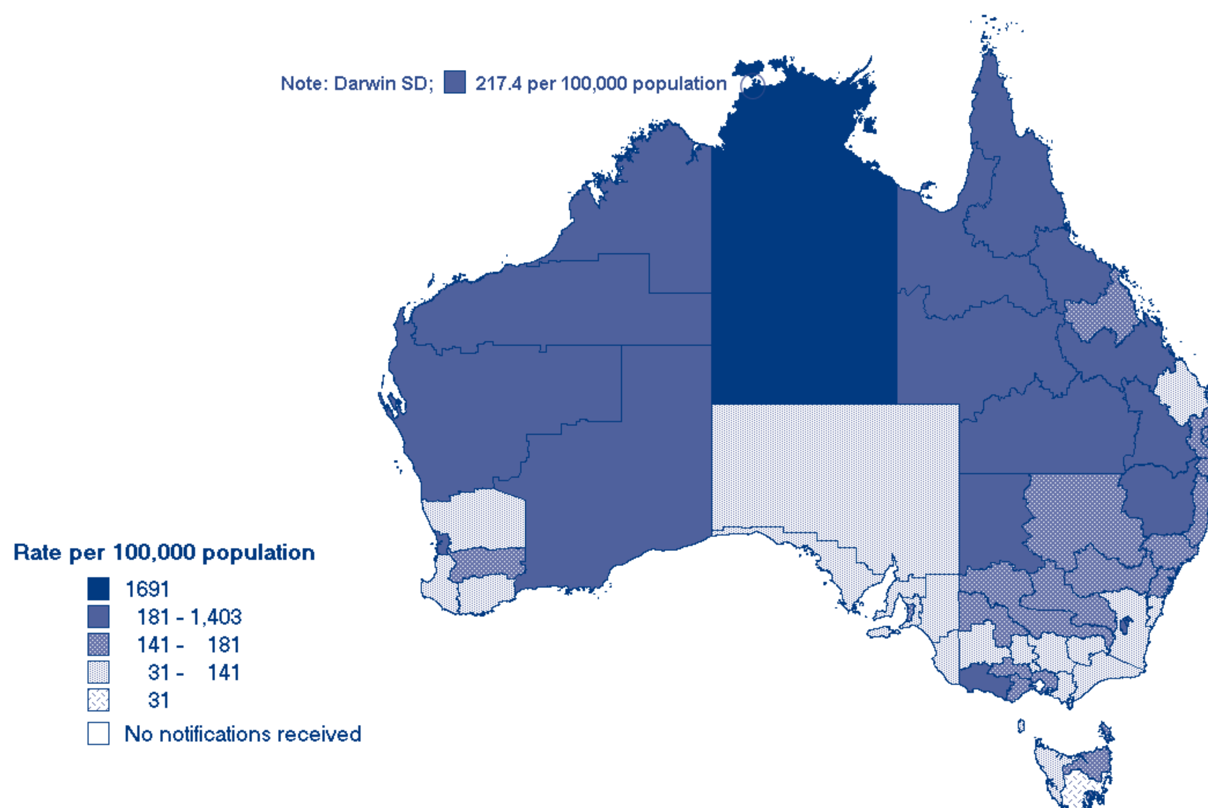


Figure 27. Notification rates of chlamydial infections, Australia, 2004, by age group and sex

Figure 28. Trends in notification rates of chlamydial infection in persons aged 10–39 years, Australia, 2000 to 2004, by age group and sex

group the annual rate increase dropped from 27 per cent to 14 per cent in males and from 26 per cent to 15 per cent in females.

In 2004, data on Indigenous status was complete in 59 per cent of cases of chlamydial infection, higher than the 43 per cent reported in 2003. The combined chlamydial infection notifications in four jurisdictions with greater than 50 per cent completeness of Indigenous status (the Northern Territory, South Australia, Western Australia, and Victoria) show that in 2004, the age adjusted notification rate was 1,159 cases per 100,000 Indigenous population, and 178 cases per 100,000 non-Indigenous population (Table 12). The age adjusted rate ratio of Indigenous to non-Indigenous was 7:1.

Although surveillance data continues to show substantial increases in chlamydial infection notifications nationally, 2004 data suggests that the rate of increase has declined. As a large proportion of cases with genital chlamydial infection are asymptomatic, notification rates for this disease are particularly

Table 12. Trends in age adjusted notification rates of chlamydial infections, the Northern Territory, South Australia, Western Australia, and Victoria, 2000 to 2004, by Indigenous status*

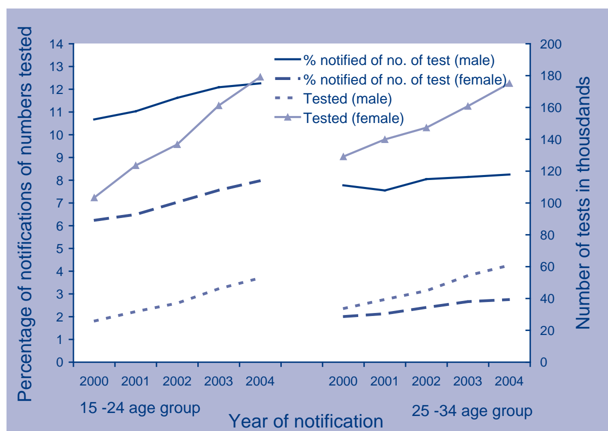
Year	NT			SA			Vic			WA			All		
	Indigenous Rate	Non-Indigenous Rate	Ratio†	Indigenous Rate	Non-Indigenous Rate	Ratio†	Indigenous Rate	Non-Indigenous Rate	Ratio†	Indigenous Rate	Non-Indigenous Rate	Ratio†	Indigenous Rate	Non-Indigenous Rate	Ratio†
2000	1,177.5	271.1	4.3	556.6	60.4	9.2	–	–	–	974.6	115.5	8.4	905.5	98.1	9.2
2001	1,412.2	348.9	4.0	452.3	95.6	4.7	–	–	–	1,028.8	119.1	8.6	994.9	118.3	8.4
2002	1,511.4	435.4	3.5	548.3	117.1	4.7	–	–	–	930.4	145.7	6.4	1,014.6	145.0	7.0
2003	1,796.1	449.8	4.0	568.3	130.6	4.4	135.1	145.3	0.9	1,211.7	171.6	7.1	1,102.1	153.4	7.2
2004	1,793.0	444.8	4.0	294.7	111.9	2.6	205.2	170.6	1.2	1,280.3	202.4	6.3	1,158.5	177.8	6.5

* The rates in non-Indigenous peoples include diagnoses in people whose Indigenous status was not reported.

† Ratio of Indigenous to non-Indigenous.

susceptible to the overall rate of testing as well as the targeted testing of certain population sub-groups. Thus this apparent abatement therefore may reflect changes in surveillance practices and public health interventions such as targeted health promotion. Data from Medicare Australia (http://www.medicare-australia.gov.au/statistics/) show that the number of diagnostic tests performed for *Chlamydia trachomatis* continued to increase in 2004, but relative to 2003, the rate of increase in testing declined: 24 to 17 per cent in the 15–24 age group, 24 to 15 per cent in males in the 25–34 age group, but remained unchanged in females in this age group (Figure 29). Using the number of tests as the denominator and the number of notifications as the numerator, from 2000 through 2004 the percentage notified of the number tested in the 15–24 and in the 25–34 year age groups remained stable in both males and females (Figure 29).

Figure 29. Number of diagnostic tests for *Chlamydia trachomatis* and the proportion notified among 15–24 and 25–34 year age groups, Australia, 2000 to 2004, by sex



Data source: National Notifiable Diseases Surveillance System and Medicare Australia data.

Subject to the limitations of this ecological analysis and the inherent limitations of Medicare Australia data sets (which do not include tests from public laboratories), this analysis suggests that an increase in the number of tests for *Chlamydia* may in part account for the increase in notifications. Similarly, the data also suggests that slight decline in the rate of increase in testing may in part account for the decline in the rate of increase observed in notifications.

Donovanosis

Case definition – Donovanosis

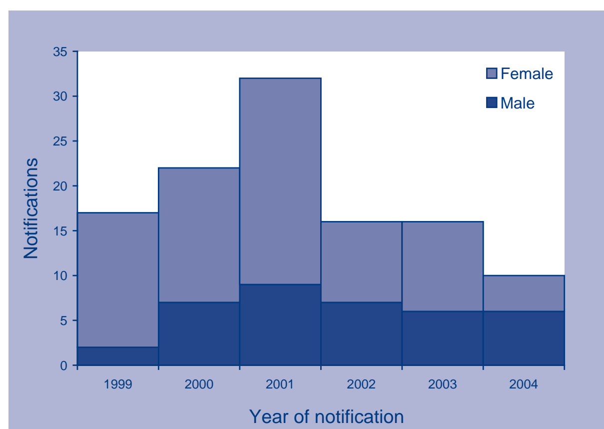
Both ***confirmed cases*** *and* ***probable cases*** *are reported.*

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 relatively uncommon, it is a disease of public health importance in Australia because it predominantly occurs in Indigenous communities. It has been identified as a potential co-factor in HIV transmission, and it is preventable.[9,10] Donovanosis is targeted for elimination from Australia through the donovanosis elimination project. In 2004, 10 cases of donovanosis, six male and four female, were reported to the NNDSS. Nine cases of the total were Indigenous: five in the Northern Territory, three in Queensland and one in Western Australia. One non-Indigenous case was reported in the Northern Territory. In 2003, a total of 16 cases, all Indigenous, six males and 10 females, were notified (Figure 30). Cases ranged in age from 18 years to 74 years and the majority were aged 15–39 years.

Figure 30. Number of notifications of donovanosis, Australia, 1999 to 2004, by sex



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 2004, 7,098 notifications of gonococcal infection were received by NNDSS. This represents a rate of 35 cases per 100,000 population, an increase of 3 per cent from the rate reported in 2003 (33 cases per 100,000 population). Nationally, there were increases in the notification rates in males (by 8%) and females (by 5%). The male to female ratio in 2004 was 2:1, unchanged in the previous three years (2001 to 2003).

The highest notification rate in 2004 was in the Northern Territory at 794 cases per 100,000 population (Table 3), while the largest increase in the notification rate in 2004 (compared to 2003) occurred in Tasmania. In Tasmania a 21 per cent overall increase in notification rates was reported: 44 per cent increase in males and 1 per cent increase in females. In 2004 nationally, gonococcal infection rates for males and females were 47 and 22 cases per 100,000 population, respectively. The exception to this pattern was the Northern Territory, where females had higher notification rates than males (621 versus 882 cases per 100,000 population). The regional distribution of gonococcal infection notifications shows that the highest notification rate occurred in the Northern Territory (excluding Darwin) at 1,821 cases per 100,000 population (Map 4).

Notification rates for gonococcal infection in males exceeded those in females in all age groups except in the 10–14 and 15–19 year age groups (Figure 31). Trends in sex specific notification rates show that the increase in rates in males in the 15–19 and 20–24 age groups has continued, although there was some abatement in the increase in the male 25–29 year age group. In females, there were no marked changes in rates, with only a slight increase in rates in the 35–39 year age group (Figure 32).

Map 4. Notification rates of gonococcal infection, Australia, 2004, by Statistical Division of residence

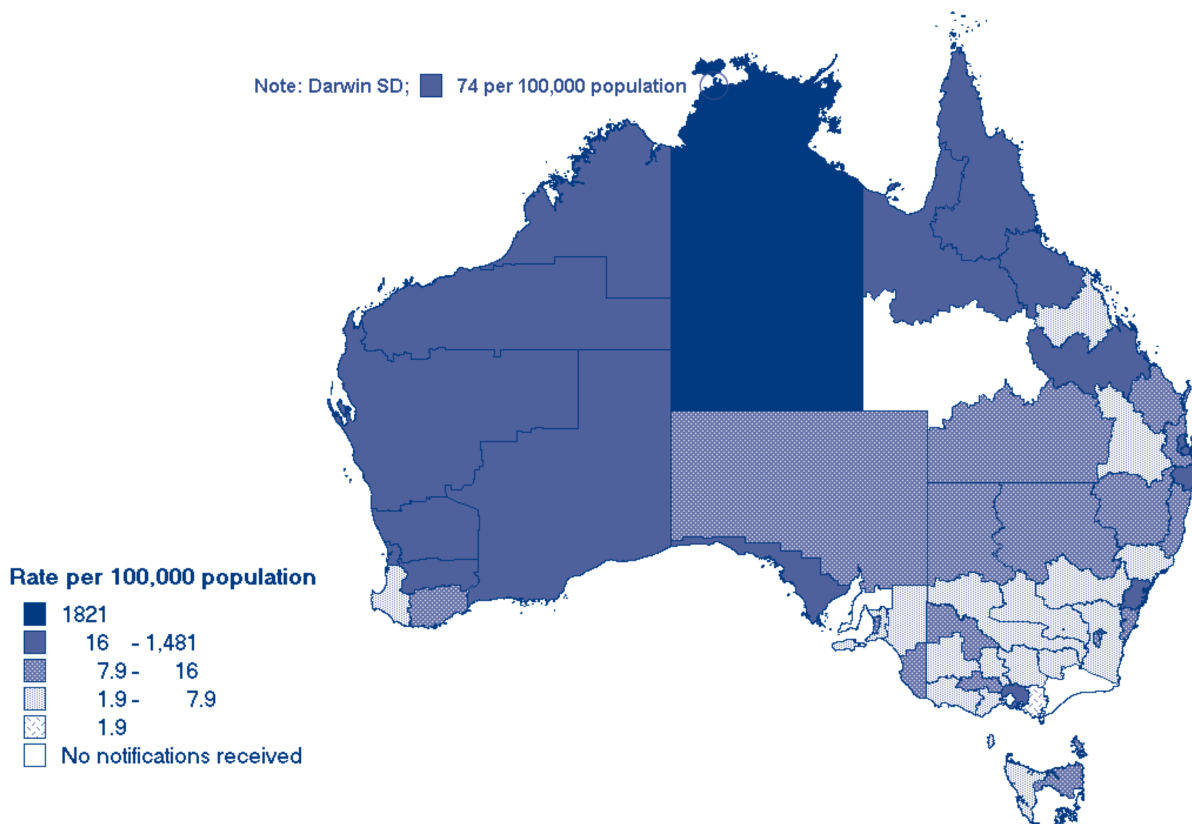


Figure 31. Notification rates of gonococcal infection, Australia, 2004, by age group and sex

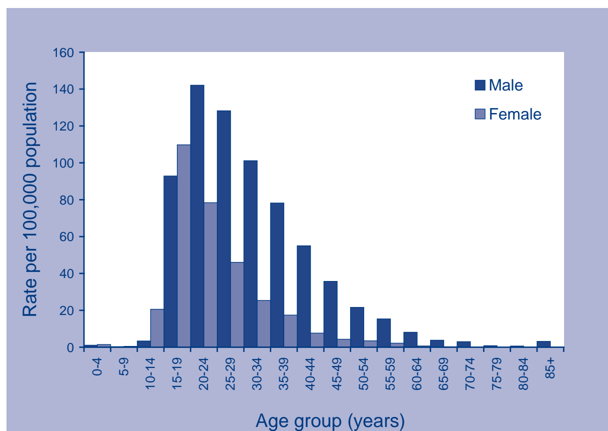
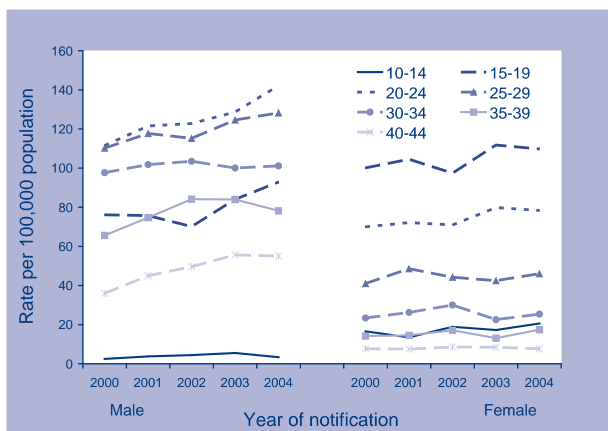


Figure 32. Trends in notification rates of gonococcal infection in persons aged 15–39 years, Australia, 2000 to 2004, by age group and sex



In 2004, the data completeness (66%) of Indigenous status of gonococcal infection notifications was similar to that in 2003. The combined gonococcal infection notifications of four jurisdictions with more than 50 per cent data completeness of Indigenous status (the Northern Territory, South Australia, Western Australia and Victoria) shows that in 2004, the age adjusted notification rate was 1,351 cases per 100,000 Indigenous population and 26 cases per 100,000 non-Indigenous population: a ratio of Indigenous to non-Indigenous of 52:1 (Table 13).

Other surveillance of gonococcal infections

The Australian Gonococcal Surveillance Program (AGSP) is the national surveillance system of antibiotic susceptibility of gonococcal isolates. In each state and territory, a network of reference laboratories determine susceptibility of isolates to a core group of antibiotics using a standard methodology. The following is the summary of their 2004 report.

Table 13. Trends in age adjusted notification rates of gonococcal infection, the Northern Territory, South Australia, Western Australia, and Victoria, 2000 to 2004, by Indigenous status*

Year	NT			SA			Vic			WA			All		
	Indigenous Rate	Non-Indigenous Rate	Ratio†	Indigenous Rate	Non-Indigenous Rate	Ratio†	Indigenous Rate	Non-Indigenous Rate	Ratio†	Indigenous Rate	Non-Indigenous Rate	Ratio†	Indigenous Rate	Non-Indigenous Rate	Ratio†
2000	1,777.1	147.2	12.1	616.1	6.5	94.6	8.5	19.0	0.5	1,249.5	30.8	40.6	1,244.3	24.9	50.0
2001	2,091.8	213.3	9.8	411.2	7.2	57.1	0.0	17.6	0.0	1,557.8	17.7	87.9	1,449.2	21.0	68.9
2002	2,057.2	258.5	8.0	356.1	8.1	44.0	42.3	18.1	2.3	1,262.5	31.8	39.7	1,313.2	30.6	42.9
2003	2,019.8	181.7	11.1	371.0	14.4	25.8	25.2	26.6	0.9	1,295.0	33.2	39.0	1,191.1	28.3	42.0
2004	2,339.0	170.3	13.7	291.7	8.5	34.3	33.8	25.2	1.3	1,320.9	29.2	45.2	1,351.1	25.9	52.1

* The rates in non-Indigenous peoples include diagnoses in people whose Indigenous status was not reported.

† Ratio of Indigenous to non-Indigenous.

In 2004, a total of 3,640 isolates of gonococci were tested for antibiotic susceptibility. Eighty-five per cent of isolates were from men, of which 76 per cent were obtained from the urethra, 13 per cent from the rectum and 8 per cent from the larynx. In females, 92 per cent of isolates were obtained from the cervix.

Trends in the proportion of isolates resistant to penicillin, quinolones and tetracycline are shown in Table 14. In 2004, the proportion of isolates resistant to penicillin by plasmid mediated resistance and chromosomally mediated resistance increased by 23 and 17 per cent, respectively. Quinolone resistance also increased by 61 per cent, 92 per cent of which were resistant at a higher 'minimal inhibitory concentration' (MIC) (1 mg/L or more). This is of concern as quinolones (e.g. 500 mg of ciprofloxacin), still used for treatment in Australia, will not be effective in high level quinolone resistant isolates.

In 27 per cent of infections by strains with plasmid mediated resistance to penicillin and in 64 per cent of infections by strains resistant to quinolone, information on country where resistant strains were acquired were available. This showed that 48 per cent (51/106) of plasmid mediated resistance were locally acquired with the rest acquired from South or South East Asia. Sixty per cent of quinolone resistant strains were acquired locally and the remaining 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 (all categories)

In 2004, all jurisdictions began reporting to NNDSS syphilis infections categorised as: infectious syphilis of less than two years duration; and syphilis of more than two years or unknown duration. Detailed analysis will be reported for the two categories, as well as for syphilis of *all categories* for the purpose of showing trends in keeping with reports in previous years.

In 2004, a total of 2,296 cases of syphilis infection of all categories were reported, representing a notification rate of 11.4 cases per 100,000 population, an increase of 13 per cent on the 10.1 cases per 100,000 reported in 2003 (Table 3). The Northern Territory continues to have the highest notification rate of syphilis (142 cases per 100,000 population), although in 2004 the rate was lower by 13 per cent from the previous year. In 2004, there were increases in notification rates only in New South Wales (by 24%), in Western Australia (by 43%) and in Victoria (by 18%). At the regional level, the highest notification rate was in the Kimberley Statistical Division of Western Australia at 344 cases per 100,000 population (Map 5).

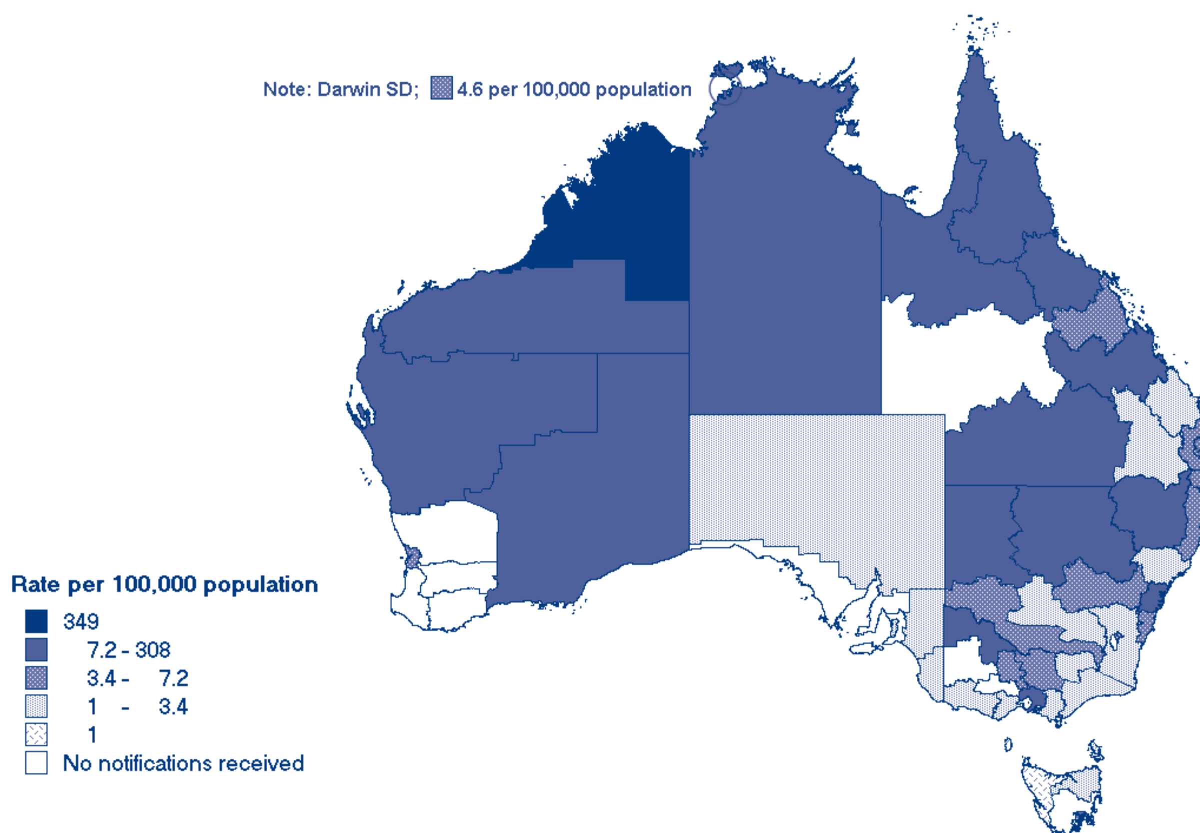
Table 14. Proportion of gonococcal isolates showing antibiotic resistance, Australia, 1998 to 2004

Year	Penicillin resistance (% resistant)		Quinolone resistance (% resistant)	High level tetracycline (% resistant)
	Plasmid mediated	Chromosomally mediated		
1998	5.3	21.8	5.2	NR
1999	7.4	14.3	17.2	7.9
2000	8.7	10.6	17.8	9.1
2001	7.5	15.3	17.5	9.4
2002	7.1	10.9	10.0	11.4
2003	9.0	9.0	14.4	11.2
2004	11.1	10.6	23.3	13.8

Source: Australian Gonococcal Surveillance Programme.

NR Not reported.

Map 5. Notification rates of syphilis infection, Australia, 2004, by Statistical Division of residence



Syphilis – infectious (primary, secondary and early latent), 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.*

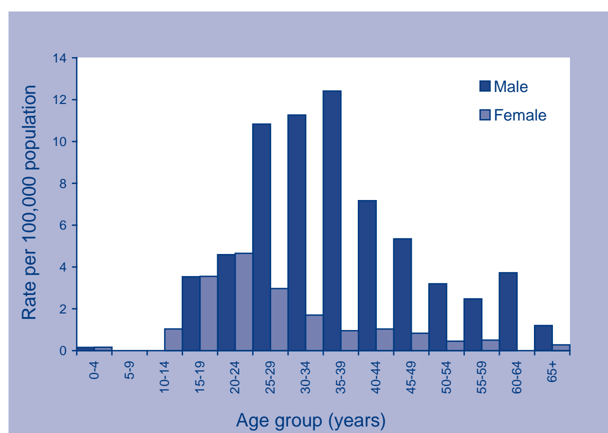
In 2004, a total of 596 cases of syphilis of less than two years duration were reported. This represents a notification rate of 3 cases per 100,000 population. The Northern Territory had the highest notification rate at 28.5 cases per 100,000 population in 2004 (Table 15).

Table 15. Number and rates of notifications of syphilis of less than two years duration Australia, 2004, by state or territory and sex

	Male		Female		Total	
	n	Rate	n	Rate	n	Rate
ACT	3	1.9	1	0.6	4	1.2
NSW	257	7.7	37	1.1	294	4.4
NT	27	25.7	30	31.7	57	28.5
Qld	69	3.6	23	1.2	92	2.4
SA	4	0.5	4	0.5	8	0.5
Tas	2	0.8	2	0.8	4	0.8
Vic	81	3.3	8	0.3	89	1.8
WA	25	2.5	25	2.5	50	2.5
Total	468	4.7	128	1.3	596	3.0

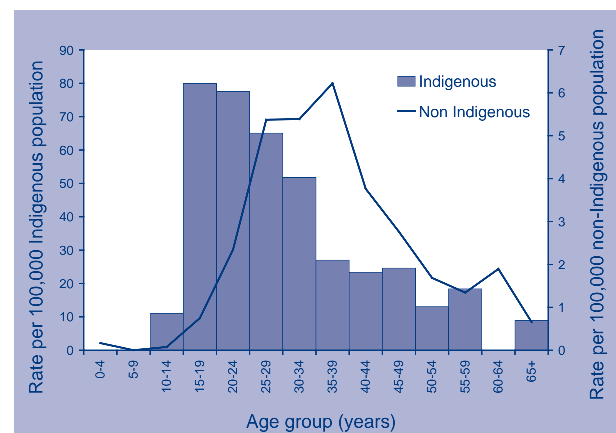
The notification rates of syphilis of less than two years duration for males and females were 4.7 and 1.3 cases per 100,000 population respectively. Notification rates were higher in males than in females in all jurisdictions except in the Northern Territory, where females had higher rates (26 versus 32 cases per 100,000 population). Nationally, the male to female ratio was 4:1. Notification rates in males peaked in the 35–39 year age group and in females in the 20–24 year age group (Figure 33).

Figure 33. Notification rates of syphilis of less than two years duration, Australia, 2004, by age group and sex



Data on Indigenous status was complete in 92 per cent of cases of syphilis of less than two years duration. The age adjusted notification rate was 37 cases per 100,000 Indigenous population, and 3 cases per 100,000 non-Indigenous population: a ratio of Indigenous to non-Indigenous of 14:1. Age specific notification rates show that compared to the non-Indigenous population, rates of syphilis of less than two years duration in the Indigenous population are in an order of magnitude higher and peak in a younger age group (Figure 34).

Figure 34. Notification rates of syphilis of less than two years duration, Australia, 2004, by Indigenous status



Syphilis of more than two years or unknown duration

Case definition – Syphilis of more than two 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 nonreactive AND the absence of a history of documented previous adequate treatment of syphilis, or endemic treponemal disease (e.g. Yaws).*

In 2004, a total of 1,561 cases of syphilis of more than two years or unknown duration were reported: a notification rate of 7.7 cases per 100,000 population. The Northern Territory had the highest notification rate at 52 cases per 100,000 population (Table 3).

In 2004, 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 (Table 16). Notification rates were higher in males in all jurisdictions except in the Northern Territory, where both sexes had equivalent notification rates (51.3 and 52.8 cases per 100,000 population for females and males, respectively. Nationally, the male to female ratio was 1.8:1. Notification rates in males and females were similar in the younger age groups up to 30–34 years (Figure 35). In females, the rate peaked in the 30–34 age group while in males it remained high from 35 years (Figure 35).

Data on Indigenous status was complete in 53 per cent of cases of syphilis of more than two years or unknown duration. The combined age adjusted rate for the jurisdictions with greater than 50 per cent data completeness of Indigenous status (all jurisdictions except New South Wales and the Australian Capital Territory) was 136 cases per 100,000 Indigenous population, and 5 cases per 100,000 non-Indigenous population: a ratio of Indigenous to non- Indigenous of 27:1. Age specific notification rates showed a similar pattern with age and no single distinct peak for either Indigenous or non-Indigenous groups. Overall, rates in the Indigenous population were higher than those in the non-Indigenous by an order of magnitude (Figure 36).

Figure 35. Notification rate of syphilis of more than two years or unknown duration, Australia, 2004, by age group and sex

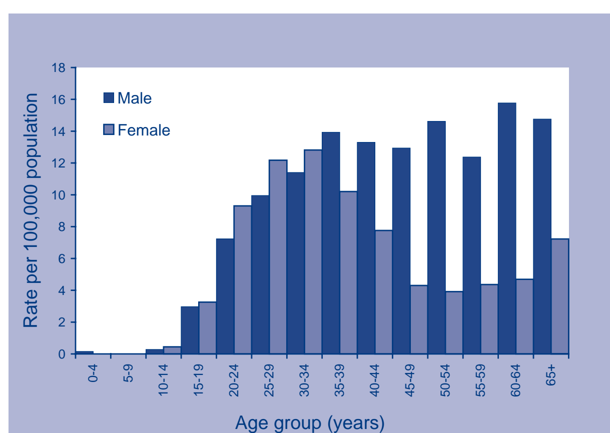


Figure 36. Notification rate of syphilis of more than two years or unknown duration, Australia, 2004, by Indigenous status

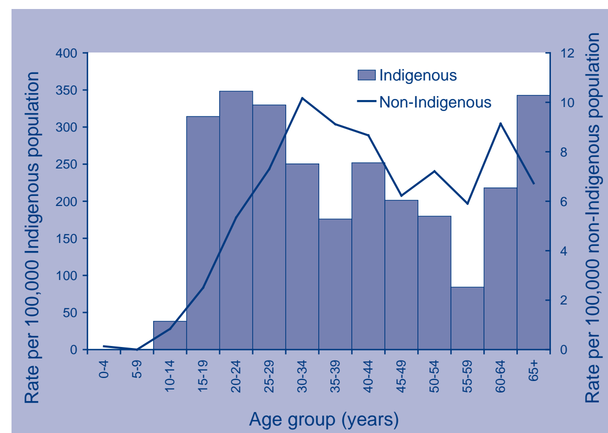


Table 16. Number and rate of notifications of syphilis of more than two years or unknown duration, Australia, 2004, by state or territory and sex

State or territory	Male		Female		Total	
	n	Rate	n	Rate	n	Rate
ACT	7	4.4	0	0	7	2.2
NSW	459	13.7	283	8.4	742	11.0
NT	54	51.3	50	52.8	104	52.0
Qld	109	5.6	89	4.6	198	5.1
SA	1	0.1	0	0.0	1	0.1
Tas	6	2.5	6	2.5	12	2.5
Vic	204	8.3	128	5.1	332	6.7
WA	101	10.2	56	5.7	157	7.9
Total	941	9.4	612	6.1	1,553	7.7

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*

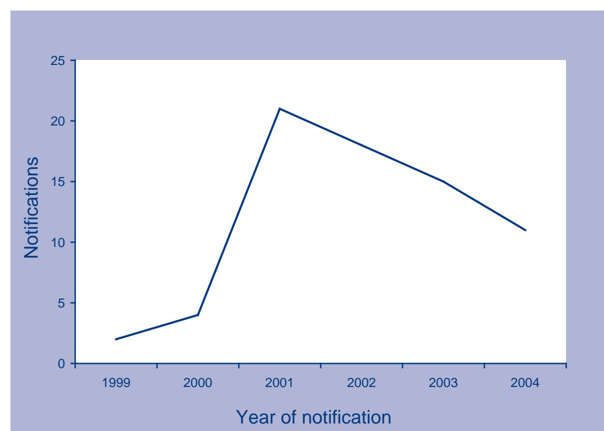
c. *Evidence of congenital syphilis on long bone X-ray*

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 11 cases of congenital syphilis notified in 2004, 10 males and one female. Six of the cases were reported in the Northern Territory, four in Queensland and one in Victoria. All but two cases were Indigenous. There has been a gradual decline in the number of congenital syphilis notified since the peak in 2001 (Figure 37). In the Northern Territory where the rates of infectious syphilis of less than 2 years duration are highest, the highest numbers of cases of congenital syphilis continue to be reported. The occurrence of congenital syphilis could be reduced by improving access to early pre-natal care.

Figure 37. Trends in notifications of congenital syphilis, Australia, 1999 to 2004



Vaccine preventable diseases

Introduction

This section summarises the national notification data for influenza and diseases targeted by the Australian Standard Vaccination Schedule (ASVS) except varicella in 2004. These include diphtheria, *Haemophilus influenzae* type b infection, measles, mumps, pertussis, invasive pneumococcal disease, poliomyelitis, rubella and tetanus. Notifications for hepatitis B and meningococcal disease, which are also targeted by the ASVS, 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.

The main change to the ASVS relevant to this reporting period was the removal of the fourth dose of the DTPa vaccine, due at 18 months of age, which occurred in September 2003. In 2004, Western Australia and New South Wales ran school-based programs to deliver dTpA vaccine to adolescents.

There were 13,206 notifications of vaccine preventable diseases (VPDs) with onset dates in 2004; 11.9 per cent of the total notifications to NNDSS. Pertussis was the most commonly notified VPD (8,557 or 65% 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 2004. The last case of diphtheria reported in Australia was a case of cutaneous diphtheria in 2001.

Haemophilus influenzae type b

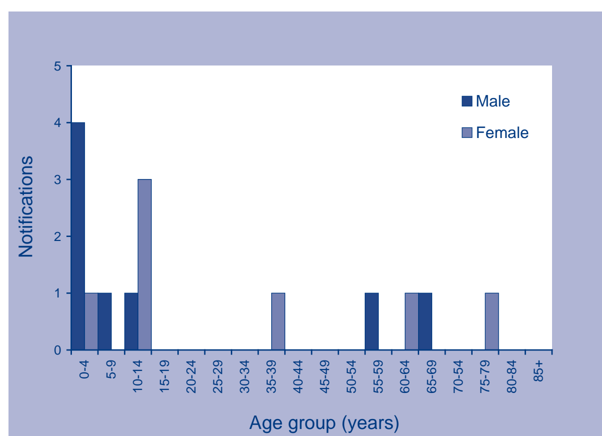
Case definition – Haemophilus influenzae *type b*

*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.*

Notifications of *Haemophilus influenzae* type b (Hib) have fallen more than 30-fold since 1991 due to the impact of Hib conjugate vaccines.[11] There were 15 notifications of Hib disease in 2004, a rate of 0.1 cases per 100,000 population. This is eight (35%) fewer cases than reported in 2003, and is the lowest number of notifications recorded since national surveillance began in 1991. Five cases (33% of total cases) were in children aged less than five years and two were infants aged less than one year (Figure 38). There were eight cases in males and seven in females, (male: female ratio 1.1:1).

Figure 38. Notifications of *Haemophilus influenzae* type b infection, Australia, 2004 by age group and sex



The Northern Territory had the highest notification rate (1.5 cases per 100,000 population, 3 cases) although most cases were from New South Wales (n=5).

Of the 14 cases with a known Indigenous status, two were Indigenous and 12 were non-Indigenous. Indigenous children now make up a greater proportion of cases than in the pre-immunisation era.[11] In a review of vaccine preventable disease in Indigenous people, 2000 to 2002, Menzies, *et al* observed a notification rate of Hib in Indigenous which was 9.7 times that in non-Indigenous people.[12] In 2004,

the Hib notification rate was 0.4 per 100,000 in Indigenous people and 0.06 per 100,000 in non-Indigenous people—a ratio of 6.7:1.

Cases under the age of 15 years were eligible for vaccination. The vaccination status of 9 of these 10 cases was known—two were unvaccinated, one partially vaccinated and six met the definition for vaccine failure, having received at least 2 doses under the age of 12 months. Of the vaccine failures, 3 (50%) were aged under 5 years.

A recent evaluation of the impact of Hib vaccination on Hib meningitis in Far North Queensland shows a dramatic decline in the incidence of this disease. In the four years prior to the addition of Hib vaccines to the ASVS, there were 28 cases of Hib meningitis in Far North Queensland and the rate of disease was 3.5 times greater in Indigenous children compared with non-Indigenous children. Since 1993, there has only been a single case of Hib meningitis, which was in a non-Indigenous child. The authors of this study estimated that in their region, Hib vaccination had prevented 70 cases of disease, five deaths and 12 cases with neurological sequelae.[13]

Influenza (laboratory confirmed)

> *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 2,073 reports of laboratory-confirmed influenza in 2004, a rate of 10.3 cases per 100,000 population. Notifications of influenza showed a peak in September 2004 (Figure 39).

Children aged less than 5 years made up 21 per cent of all notifications and had a notification rate of 34.8 cases per 100,000 population (Figure 40). Children aged less than 1 year had the highest rates (63.1 cases per 100,000 population). The overall male to female ratio was 1:1.

There were 72 notifications of influenza in Indigenous people in 2004. This gives a notification rate for influenza of 14.8 per 100,000 compared with 10 per 100,000 in non-Indigenous people—a rate ratio of 1.5:1. A higher rate of hospitalisation for influenza in Indigenous people was noted between 2000 and 2002.[12]

Figure 39. Notifications of laboratory-confirmed influenza, Australia, 2004, by month of onset

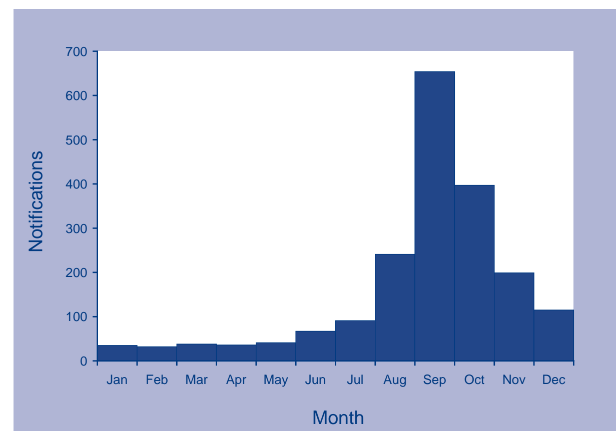
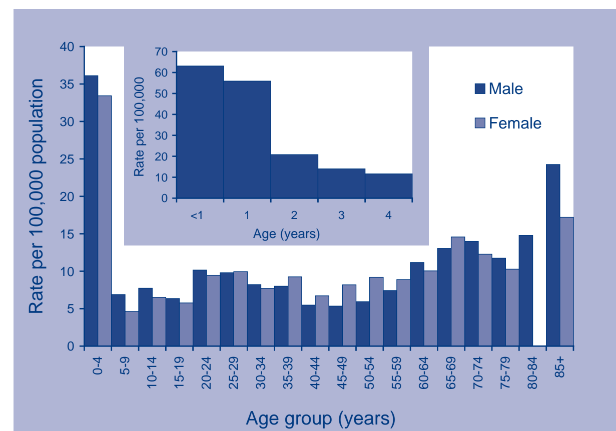


Figure 40. Notification rate of laboratory-confirmed influenza, Australia, 2004, by age group and sex



In 2004, 1,896 (91%) of notifications had serotype data. Of these 79 per cent (1,493) were influenza A and 21 per cent (403) were influenza B.

Of 454 isolates analysed at the WHO Collaborating Centre for Reference and Research on Influenza in 2004, 342 were A(H3N2), 3 were A(H1N1) strains and 108 were influenza B. The majority of A(H3N2) viruses were A/Fujian/411/2002(H3N2)-like with significant antigenic drift and were similar to the recent A/Wellington/1/2004 isolate.

In 2004, 79 per cent of those aged 65 years and over in Australia received influenza vaccination.[14]

There were a number of outbreaks of influenza in 2004. Two outbreaks occurred in army barracks, one in Victoria and another in Queensland. There were 13 outbreaks of influenza-like illness in 12 aged care facilities in New South Wales, marked by high attack rates (76% in residents and 42% in staff) and a case fatality rate of 14 per cent.

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 45 measles cases in 2004, including 43 confirmed and 2 probable cases; a national rate of 0.2 cases per 100,000 population. This was a 54 per cent decrease compared with 2003 when 98 cases were notified, and is the second lowest annual rate for Australia since national surveillance began in 1991 (Figure 41). The highest rate was in the Northern Territory with 1.5 cases per 100,000 population (3 cases), while the largest number of cases were reported from Victoria (15 cases, 0.3 cases per 100,000 population). In 2004 there were no cases reported from the Australian Capital Territory, Queensland or Tasmania (Tables 2 and 3).

Notification rates were highest in the 25–29 year age group (1.2 cases per 100,000 population), followed by the 0–4 and 30–34 year age groups (0.5 cases per 100,000 population, Figure 42). There were only six cases in the under 5 year age group and three were aged less than 1 year (0.8 cases per 100,000 population).

Figure 41. Notifications of measles, Australia, 1997 to 2004, by month of onset

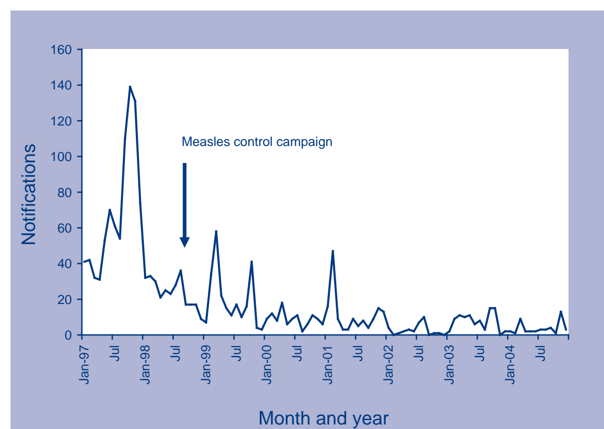
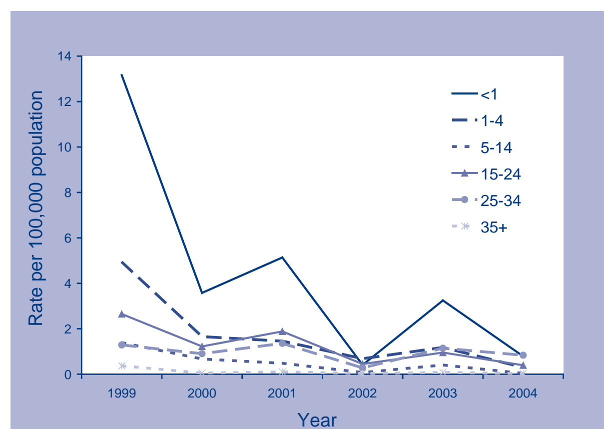


Figure 42 shows trends in measles notification rates by age group. In 2004 the largest proportion of measles cases occurred in adults, which reflects the success of measles vaccination programs in children and adolescents. A recent review suggests that indigenous transmission of measles has been interrupted and that Australia is making good progress toward measles elimination.[15]

Figure 42. Trends in notification rates of measles, Australia, 1999 to 2004, by age group



Of the 45 measles cases reported in 2004, 21 (46%) occurred in six outbreaks in three states (Table 17). The index case in four of the six outbreaks acquired their infection outside Australia.

The outbreak in Western Australia is significant because all six cases were in Indigenous people and there was no link to a confirmed imported index case. These were the only measles notification in Indigenous people in 2004, giving a rate of 1.2 per 100,000 population compared with 0.2 per 100,000 in non-Indigenous people (a rate ratio of 6:1).

Table 17. Outbreaks and clusters of measles, Australia,* 2004

State or territory	Month of onset	Number of linked cases (including index case)	Place of acquisition of infection in index case
New South Wales	Mar	2	Overseas
New South Wales	Mar	4	Overseas
Victoria	Apr	2	Overseas
Victoria	July	4	Victoria
Victoria	Nov	3	Overseas
Western Australia	Nov	6	Not identified

* There were no cases of measles reported in 2004 in the Australian Capital Territory, Queensland or Tasmania

The vaccination status was recorded for 25 of the 42 cases born after the introduction of measles vaccination in 1970: 19 were unvaccinated, four were partially vaccinated and two were fully vaccinated for age. Both 'fully vaccinated for age' cases had only received a single dose of measles-mumps-rubella (MMR) vaccine: one was a 1-year-old child who was fully vaccinated for age and the other was a 16-year-old who should be regarded as partially vaccinated.

Mumps

Case definition – Mumps

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

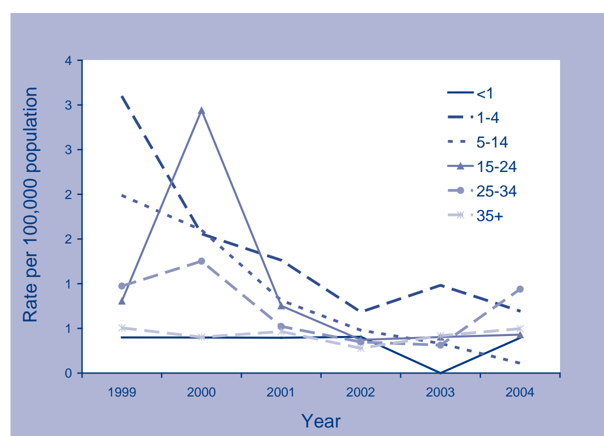
***Confirmed case:** Requires isolation of mumps virus or detection of mumps virus by nucleic acid testing or IgG seroconversion or significant increase in antibodies or a significant increase in antibody level, or a fourfold or greater rise in titre to mumps virus (except where there has been recent 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 2004, there were 102 notifications of mumps, a rate of 0.5 cases per 100,000 population. This was a 24 per cent increase on the 82 cases reported in 2003. Unlike 2003 when there was a preponderance of cases in males (male:female ratio 1.5:1), the male:female ratio in 2004 was 1:1.

The highest rates were in the 25–29 year age group (1.3 cases per 100,000 population). The rate for the 0–4 year age group (0.6 cases per 100,000 population) was similar to that seen in 2003.

Trends in age group notification rates for mumps (Figure 43) show an increase in the rates in the 25–34 year age group since 2003. Increases in mumps in England and Wales, predominately among older teenagers and young adults who had not received two doses of MMR vaccine, have also been observed.[16]

Figure 43. Trends in notification rates for mumps, Australia, 2004, by age group



Pertussis

Case definition – Pertussis

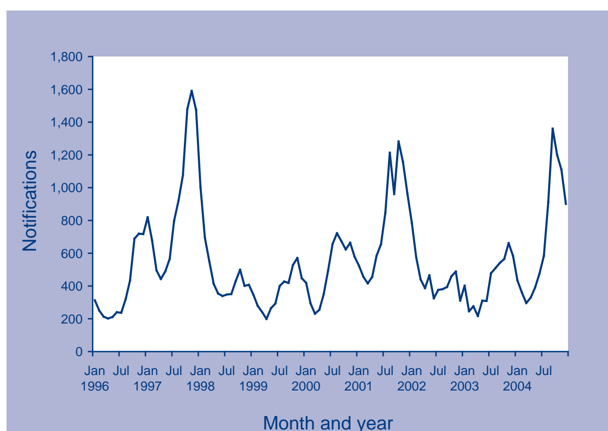
*Both **confirmed cases** and **probable cases** are notified.*

***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.*

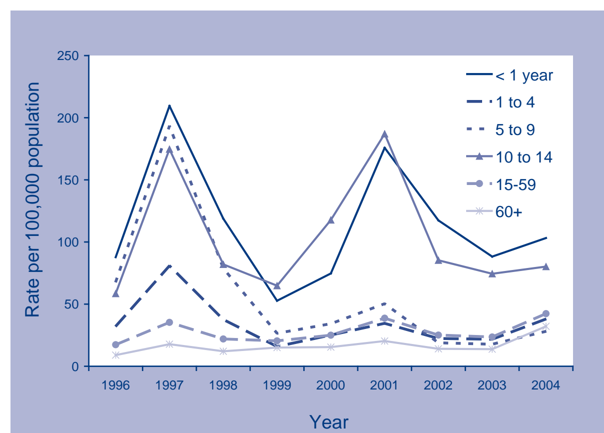
Pertussis continues to be the most common vaccine preventable illness in Australia, with periodic epidemics occurring at intervals of 3 to 5 years on a background of endemic circulation (Figure 44). In 2004, there were 8,557 cases (42.5 cases per 100,000 population) notified to NNDSS. Of these, 7,638 were confirmed and 649 were probable cases, while the status of the remaining 270 cases was unknown.

Figure 44. Notifications of pertussis, Australia, 1996 to 2004, by month of onset



The highest notification rates were among children aged <1 year (262 cases, 103.1 cases per 100,000 population) and those in the 10–14 year age group (1,112 cases, 80.2 cases per 100,000 population) (Figure 45). The notification rate in persons aged 60 years and over rose dramatically between 2003 and 2004 (13.8 versus 33.6 cases per 100,000 population). This is in contrast to the relatively steady annual rates previously seen in this age group. In 2004, 74 per cent of pertussis cases were aged 15 years or over. Although severe morbidity and mortality are less likely in these age groups, they are an important pertussis reservoir, facilitating transmission to children too young to be fully vaccinated. The overall male to female ratio was 0.8:1.

Figure 45. Trends in notification rates for pertussis, Australia, 1996 to 2004 by age group



Notification rates of pertussis varied considerably by geographic location (Map 6). At the State/Territory level, rates were highest in Western Australia (105.8 cases per 100,000 population) and lowest in Tasmania (7.7 cases per 100,000 population).

There was an outbreak of pertussis in the Western Australia in 2004, where the notification rate was the highest since 1997. A relatively large proportion of notifications were in secondary school students, so a mass vaccination campaign with dTpa was instituted in secondary schools. New South Wales also had a school-based dTpa campaign in 2004. New South Wales and South Australia recorded rates of pertussis for all ages above the national average in 2004 (Figure 46).

Map 6. Notification rates of pertussis, Australia, 2004, by Statistical Division of residence

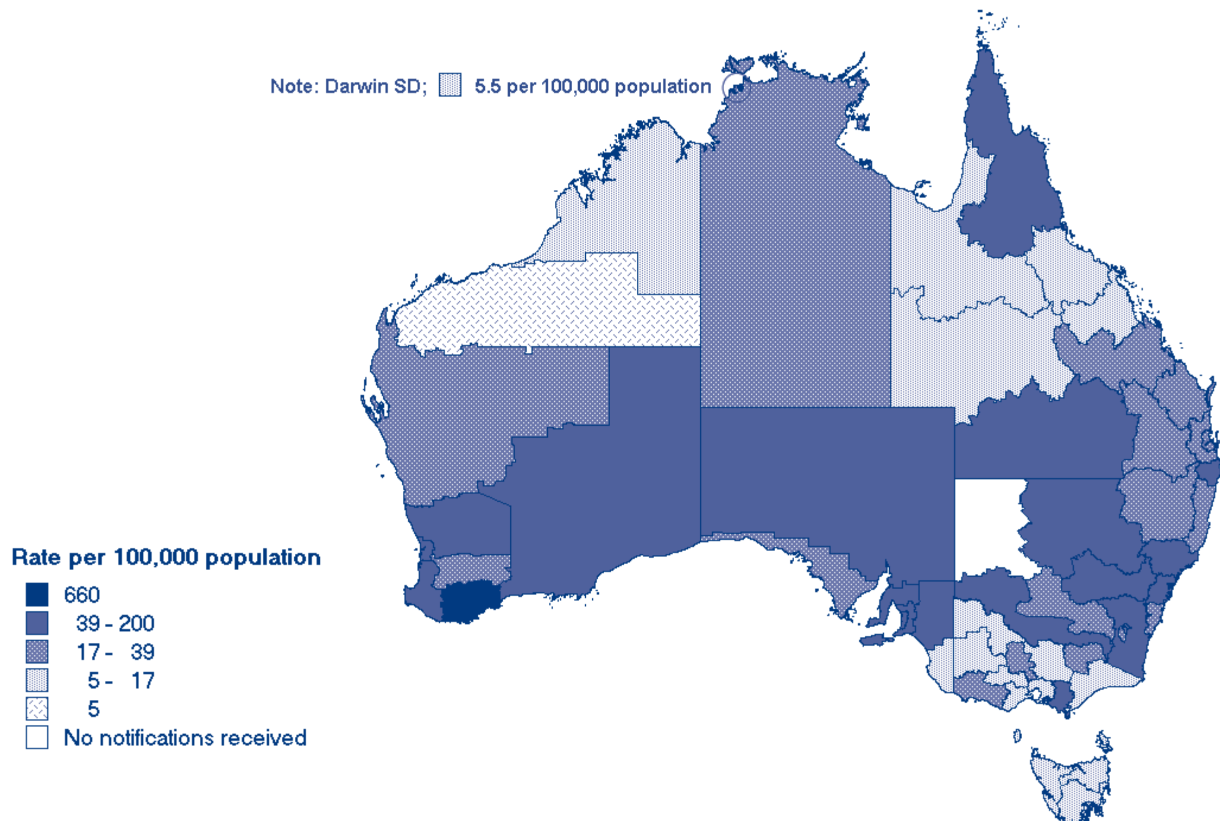
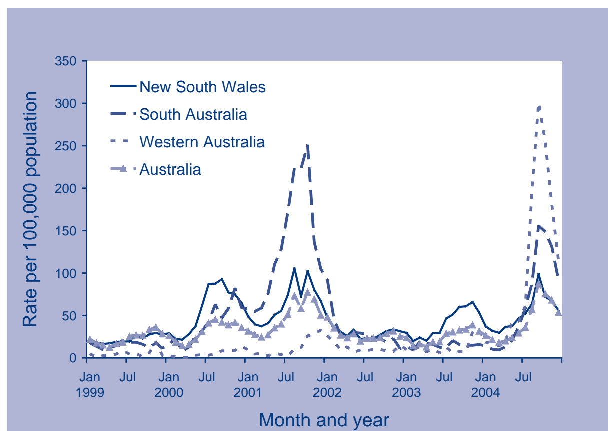


Figure 46. Notification rates of pertussis, New South Wales, South Australia, Western Australia and Australia, 1999 to 2004, by month of notification



There were 153 cases who were identified as Indigenous (31.6 cases per 100,000 population) and 8,227 who were identified as non-Indigenous (41.9 cases per 100,000 population). The Indigenous pertussis notification rate ratio for all ages was therefore 0.75, but it is important to note that previous analyses have shown that, in the age groups where the disease is most severe, there were higher rates in Indigenous compared to non-Indigenous populations. For example, in 2000–2002 the notification rate ratio for children aged 0–4 years was 1.7, and 2.6 for those aged less than one year.[12]

A review of cough symptoms in children in Sydney has provided evidence of cases of pertussis which are not notified. Clinically diagnosed pertussis was estimated to be between 5 and 20 times the notification rates.[17]

Invasive pneumococcal disease

Case definition – Invasive pneumococcal disease

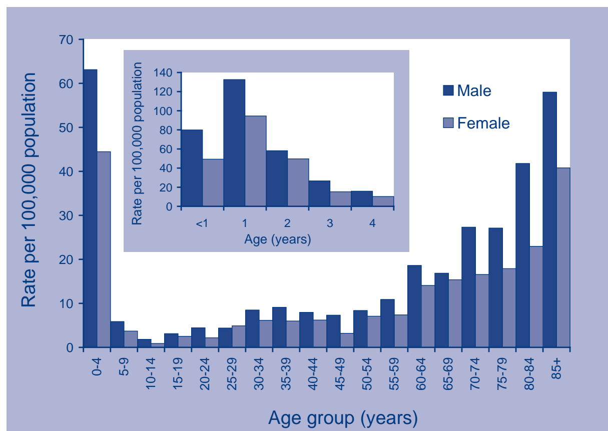
*Only **confirmed cases** are notified.*

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

There were 2,375 notifications of invasive pneumococcal disease (IPD) in Australia in 2004 giving a rate of 11.8 cases per 100,000 population. While the largest number of cases were reported from New South Wales, Queensland and Victoria (Table 1), the highest rate was in the Northern Territory (47 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 2004, IPD remained largely a disease of the very young and very old. The highest rates of disease were among children aged less than 5 years (54.3 cases per 100,000 population, with peak rates in 1-year-olds, 114 cases per 100,000 population) and adults aged more than 85 years (46.3 cases per 100,000 population) (Figure 47). There were more cases among males, with a male to female ratio of 1.4:1.

Figure 47. Notification rate for invasive pneumococcal disease, Australia, 2004, by age group and sex



There were 174 cases of IPD in Indigenous people (35.9 cases per 100,000 population) and 2,201 in non-Indigenous people (11.2 cases per 100,000 population), an Indigenous:non-Indigenous ratio of 3.2:1.

Additional data were collected on cases of invasive pneumococcal disease in all Australian jurisdictions during 2004. Analyses of these data can be found in the IPD annual report in this issue.[18]

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 2004.

There were 62 notifications of acute flaccid paralysis (AFP) reported in 2004. Of these 49 occurred in children aged less than 15 years. This represents an AFP notification rate of 1.2 cases per 100,000 children aged less than 15 years and meets the WHO indicator target for adequate AFP reporting. One infant AFP case had Sabin-like polioviruses 1 and 2 isolated from stool. The Polio Expert Committee classified this case as infant botulism based on the detection of *Clostridium botulinum* serotype B toxin and isolation of *C. botulinum* serotype B organism from a faecal sample.[19]

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*

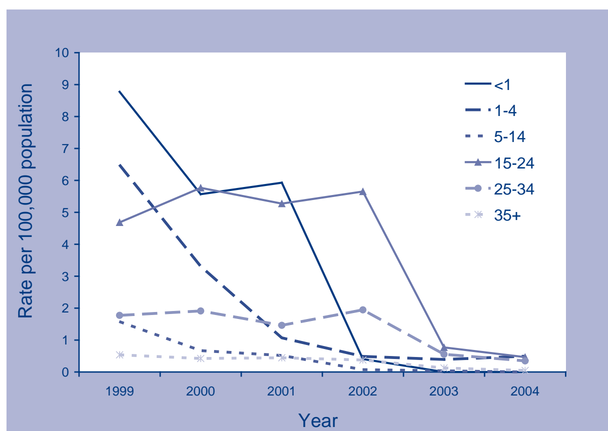
***Epidemiological evidence:** An epidemiological link is established when there is: 1. Contact between two people involving a plausible mode of transmission at a time when: a) one of them is likely to be infectious (about one week before to at least four days after appearance of rash) AND b) the other 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 2004, there were 33 notifications for rubella: 32 confirmed and one probable case; a notification rate of 0.2 cases per 100,000 population. This is the lowest rate on record and a 40 per cent reduction on 2003 (55 notifications, 0.3 cases per 100,000 population). In 2004, New South Wales accounted for 52 per cent of all notified cases of rubella (17 cases, notification rate 0.3 cases per 100,000 population) and Queensland 30 per cent (10 cases, 0.3 cases per 100,000 population).

The male to female ratio of notified cases in 2004 was 1:1 in contrast to the male predominance seen in 1999 (male:female ratio 1.4:1), 2002 (male:female ratio 3.0:1) and 2003 (male:female ratio 1.6:1).

Figure 48 shows trends in rubella notification rates in different age groups. The rates in older teenagers and young adults continued to decline in 2004.

Figure 48. Trends in notification rates for rubella, Australia, 2004, by age group and sex



There was a single case of congenital rubella reported from New South Wales in 2004. The child was born to an unvaccinated overseas-born mother. Altogether there were 14 cases of rubella notified from women of child-bearing age (15–49 years) in 2004.

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 2004, there were five notifications of tetanus. Four were female and one was male and all were aged over 60 years.

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 2004 are shown in Table 18, Table 19, and Table 20, respectively. Over the four quarters, there were no significant changes in coverage for all three age groups. Coverage of all vaccines used to assess 'fully immunised' status at 24 months of age was higher than for the other two age groups. Coverage for all vaccines at 6 years of age remains significantly lower (8–9 percentage points) than at 12 and 24 months and still is of concern.

Vectorborne diseases

During 2004, there were 6,000 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.

Alphavirues

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 vectors for Barmah Forest virus and Ross River virus, which facilitate the transmission of these viruses in diverse environments (freshwater habitats, coastal regions, salt marshes, floodwaters, established wetlands and urban areas).[20]

Table 18. Percentage of Australian children born in 2003 immunised according to data available on the Australian Childhood Immunisation Register, estimate at one year of age

Vaccine	Birth cohort			
	1 Jan–31 Mar 2003	1 Apr– 30 Jun 2003	1 Jul–30 Sep 2003	1 Oct–31 Dec 2003
DTP	92.3	92.7	92.6	92.2
OPV	92.2	92.6	92.5	92.0
Hib	94.5	94.8	94.8	94.4
Hepatitis B	94.7	94.9	95.0	94.7
Fully immunised	90.9	91.3	91.2	90.7

Table 19. Percentage of Australian children born in 2002 immunised according to data available on the Australian Childhood Immunisation Register, estimate at two years of age

Vaccine	Birth cohort			
	1 Jan–31 Mar 2002	1 Apr–30 Jun 2002	1 Jul–30 Sep 2002	1 Oct–31 Dec 2002
DTP	95.5	95.3	95.0	94.9
OPV	94.9	95.2	95.0	94.8
Hib	93.4	93.8	93.4	93.2
MMR	93.5	93.9	93.6	93.4
Hepatitis B	95.7	95.9	95.4	95.5
Fully immunised	91.7	92.3	91.7	91.7

Table 20. Percentage of Australian children born in 1998 immunised according to data available on the Australian Childhood Immunisation Register, estimate at six years of age

Vaccine	Birth cohort			
	1 Jan–31 Mar 1998	1 Apr–30 Jun 1998	1 Jul–30 Sep 1998	1 Oct–31 Dec 1998
DTP	85.2	85.4	85.2	84.7
OPV	85.2	85.3	85.2	84.8
MMR	84.8	84.8	84.8	84.6
Fully immunised	83.5	83.6	83.6	83.3

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 1,052 notifications of Barmah Forest virus (BFV) infection notified to NNDSS in 2004, which accounts for 18 per cent of the total mosquito-borne disease notifications for the reporting period. Eighty-nine per cent of BFV notifications were reported from Queensland (n=535) and New South Wales (n=402).

The highest rates of BFV notifications were reported by Queensland (13.8 cases per 100,000 population), the Northern Territory (11 cases per 100,000 population) and New South Wales (6 cases per 100,000 population). The national BFV notification rate was 5.2 cases per 100,000 population which was the third highest since 1999. Figure 49 shows that there was a peak in the BFV notification rate in Queensland in March 2004 (26.6 cases per 100,000 population). The Northern Territory reported a peak

BFV notification rate in May 2004 (24.4 cases per 100,000 population), whereas New South Wales reported a peak BFV notification rate in April 2004 (9.8 cases per 100,000 population). The peak BFV notification rates in 2004 for Queensland and New South Wales represent a 60–66 per cent reduction from the previous peak notification rates in 2003.

The highest rate of BFV infection in 2004, was in the mid-North Coast area of New South Wales (67.5 cases per 100,000 population, Map 7).

Figure 50 shows the age and sex distribution of BFV notifications. The national rate of notifications for BFV was highest amongst the 50–54 year age group (10.4 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 rates (12.4 cases per 100,000 population). The highest age-specific BFV notification rate in females was recorded in the 45–49 year age group (9 cases per 100,000 population).

Figure 49. Notification rates for Barmah Forest virus infection, select jurisdictions, January 1999 to December 2004, by month and year of onset

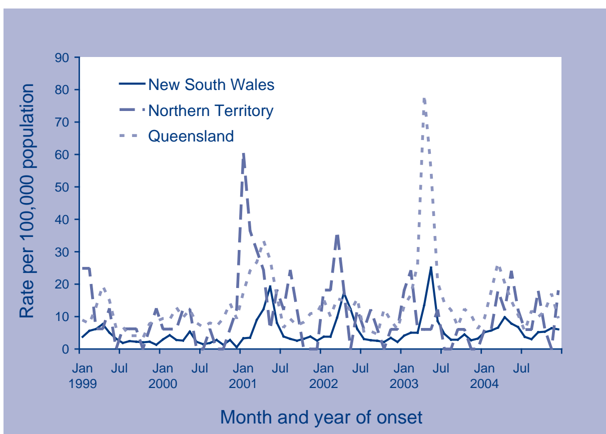
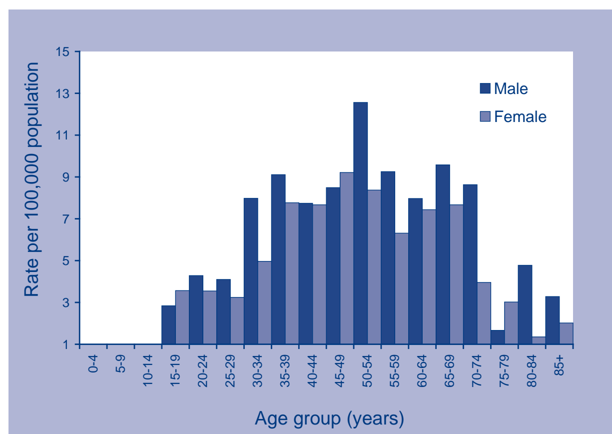
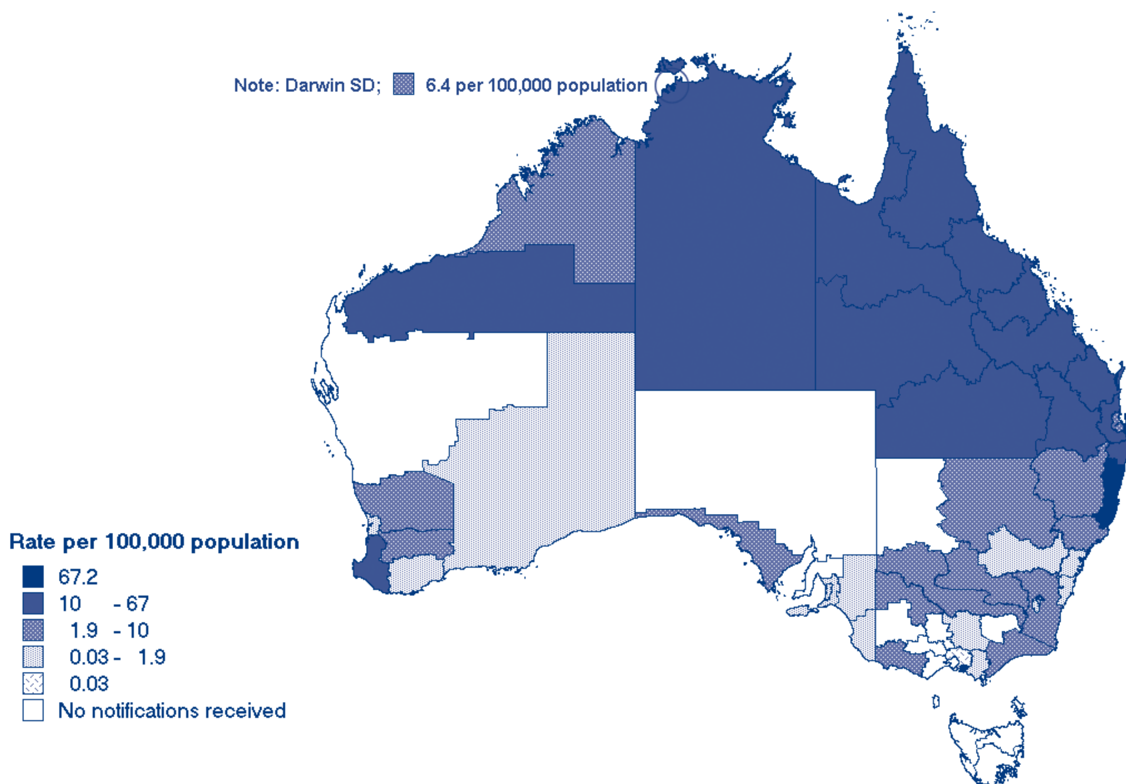


Figure 50. Notification rates for Barmah Forest virus infections, Australia, 2004, by age group and sex



Map 7. Notification rates for Barmah Forest virus infection, Australia, 2004, by Statistical Division of residence



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 four-fold or greater rise in titre to Ross River virus, OR detection of Ross River virus-specific IgM.*

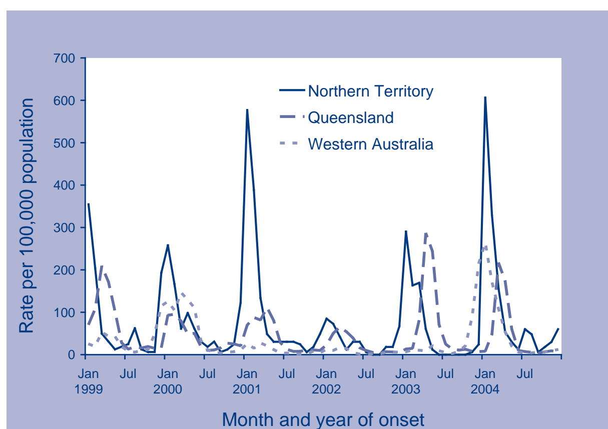
There were 4,000 notifications of Ross River virus (RRV) infection reported to NNDSS in 2004, which accounts for two-thirds (67%) of the total mosquito-borne disease notifications received in 2004.

The highest contributors to RRV notifications in 2004 were Queensland (45%, n=1,795), Western Australia (27%, n=1,099), and New South Wales (18%, n=700). The highest rates of infection were reported by the Northern Territory (117.6 cases per 100,000 population), Western Australia (55.4 cases per 100,000), and Queensland (46.2 cases per 100,000 population). The 2004 national RRV notification rate (19.9 cases per 100,000) was the third highest RRV notification rate reported to NNDSS since 1999.

Map 8 shows that the highest rate of RRV infection in 2004, was in the Kimberley region area of Western Australia (202.8 cases per 100,000 population).

RRV infection notifications in the Northern Territory peaked in January 2004 at 606.3 cases per 100,000 population (Figure 51). This was the highest rate since 1999, closely resembling the RRV peak notification rate and profile in the Northern Territory in January 2001. Queensland reported the peak notification rate for RRV in March 2004 at 216.7 cases per 100,000 population, and this was a 24 per cent reduction from the peak notification rate for April 2003 (286.3 cases per 100,000 population).

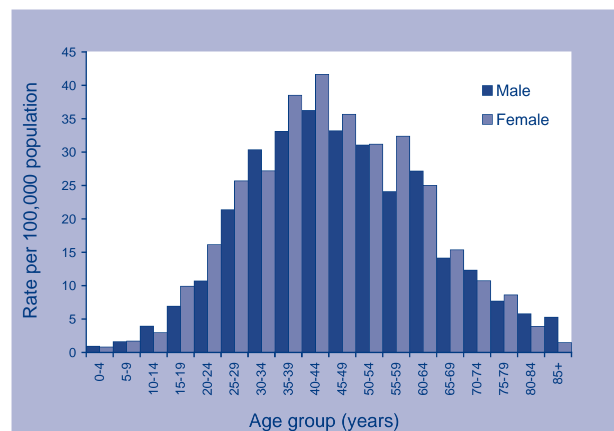
Figure 51. Notification rates for Ross River virus infection, select jurisdictions, 1999 to 2004, by month and season of onset



In Western Australia, a state-wide outbreak of RRV peaked in January 2004 at 263.9 cases per 100,000 population which was the largest recorded outbreak of RRV in Western Australia[21,22] despite early warning through media and publicity channels. The predisposing environmental, entomological and virological aspects of the outbreak have been described elsewhere.[22,23]

The age and sex distribution of RRV notifications are shown in Figure 52. The notification rates were highest in the 40–44 age group (38.9 cases per 100,000 population) and the female to male ratio was 1:0.9.

Figure 52. Notification rates for Ross River virus infection, Australia, 2004, by age group and sex

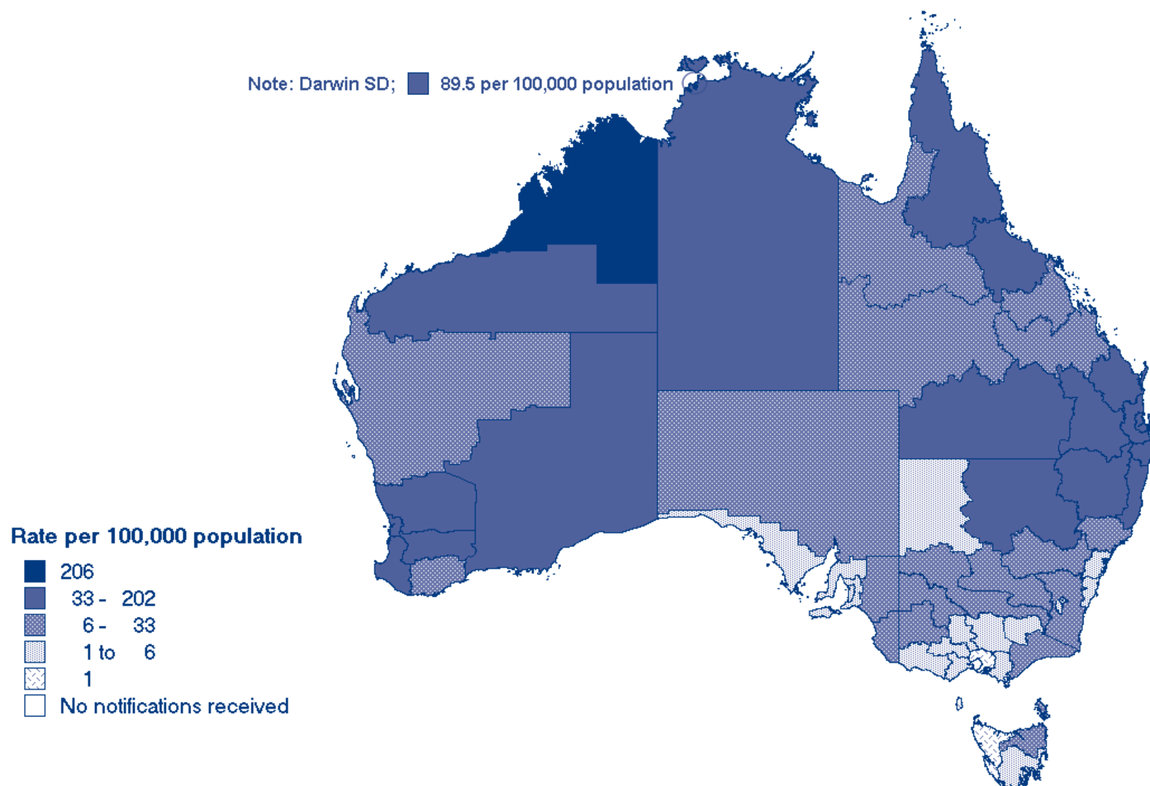


Flaviviruses

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 virus (MVEV), Kunjin virus (KUNV), Japanese encephalitis and dengue viruses.

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.[24] 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,[25–27] and the latest report was published in *CDI* in 2005 as part of the National Arbovirus and Malaria Advisory Committee annual report, 2004–05.[28]

Map 8. Notification rates for Ross River virus infections, Australia, 2004, by Statistical Division of residence



Murray Valley encephalitis virus

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 meningo-encephalitis 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.

In April 2004, there was one notification of MVEV from Central Australia, when an 11-month-old infant with an onset of symptoms in March 2004 was hospitalised in Alice Springs for one week, and then transferred to South Australia. The infant developed serious neurological sequelae and after a long and debilitating illness, died from complications from MVEV. The Health Department of the Northern Territory government issued a general seasonal warning for

MVEV and KUNV for the Alice Springs region and other regions in January 2004, and for the Top End in March 2004 after sentinel chicken seroconversions in the Leanyer swamp area near Darwin, and in April for the whole of the Northern Territory after notification of the MVEV case.

Kunjin virus

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 were 12 notifications of KUNV during 2004, with 11 of the cases reported from Queensland. These 11 cases were symptomatic with a mild febrile illness but without encephalitis. Of the 11 cases, nine were reported in January and February 2004 and it is likely that these cases were identified because of increased testing undertaken in north Queensland due to the major dengue outbreak (Jeffrey Hanna, personal communication). There is nothing to indicate any genuine increase in human health risk from Kunjin virus activity during that time.

The other jurisdiction to report a KUNV notification in 2004 was Victoria. In October 2004, a 35-year-old female was notified as having acquired KUNV infection. The person lived in metropolitan Melbourne, but a detailed investigation did not reveal any likely exposure within Victoria, nor was there any other evidence of KUNV activity. She had travelled extensively overseas and it is assumed that she acquired KUNV or a closely-related virus while overseas.

Dengue virus infection

Case definition – dengue virus

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

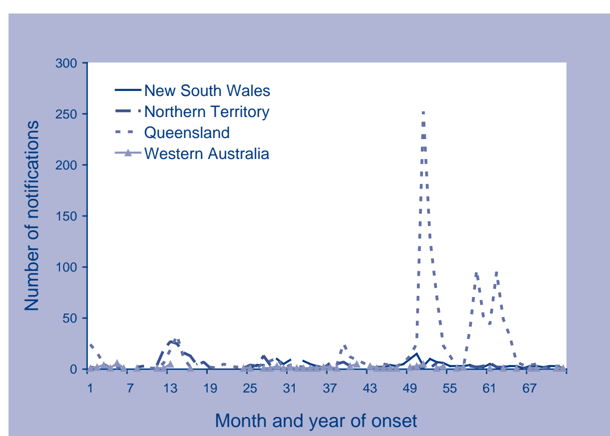
***Confirmed case:** Requires isolation of dengue virus, OR detection of dengue virus by nucleic acid testing, OR IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to dengue virus, proven by neutralisation or another specific test, OR detection of dengue 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 2004, there were 326 notifications of dengue (DENV) reported to NNDSS, of which Queensland reported 249 notifications (76%). The only locally acquired notifications were reported by Queensland (n=181), while other jurisdictions reported imported cases from overseas (n=70), or from unknown sources (n=74). Queensland reported a peak in DENV notifications in November 2003 and February 2004 (95–97 cases). These were much lower than the previous peak of 252 notifications in March 2003 (Figure 53).

Figure 53. Notifications of dengue (locally acquired and imported cases), select jurisdictions, January 1998 to June 2005, by month and year of onset



The Queensland notifications resulted from outbreaks that began in late 2003 in Cairns, Townsville and the Torres Strait islands. A summary of identified outbreaks of locally acquired cases is shown in Table 21.

Dengue serotype 2 was the major serogroup circulating in Queensland during these outbreaks. A 40-year-old Torres Strait Islander woman and 70-year-old man died from dengue shock syndrome (DSS) in February and March 2004, respectively, and it has been suggested that the primary infection for these two cases occurred in 1981,[29] when there was a dengue serotype 1 epidemic. The deaths from DSS were the first from locally acquired dengue in Australia for 100 years.

An incursion of the mosquito vector for DENV, *Aedes aegypti*, occurred in Tennant Creek in the Northern Territory in February 2004.[30,31] This species of mosquito has not been endemic in the Northern Territory since 1955.[32] Mosquito control activities including fogging in residential and public places, distribution of surface sprays, removal of water-filled receptacles and residual insecticide spraying were initiated along with public awareness campaigns.[33] No human cases of dengue were reported in Tennant Creek.

The age and sex distribution of DENV notifications is shown in Figure 54. Most cases in males occurred in the 30–34 year age group (25 cases), and in females in the 25–29 year age group (24 cases).

Figure 54. Notifications of dengue (locally acquired and imported cases), Australia, 2004, by age group and sex

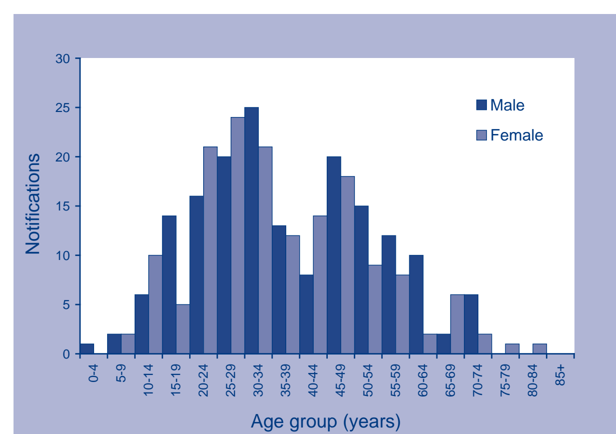


Table 21. Outbreaks of locally acquired cases of dengue, Queensland, 2003 to 2004

Year	Location	Reported cases	Duration (weeks)	Type
2003–04	Cairns, Townsville, Torres	536	69	Dengue 2
2003–04	Torres, Cairns	356	41	Dengue 2
2004	Torres	1	1	Dengue 2

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

Japanese encephalitis virus

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 was one case of Japanese encephalitis virus (JEV) notified in February 2004, when Queensland reported that a 66-year-old male acquired JEV from Papua New Guinea. There have been nine other cases of JEV reported to NNDSS since 1995, although JEV was not nationally notifiable until 2001. Four of these nine 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 four cases were reported as acquired from overseas countries.

The Australian Quarantine and Inspection Service, through the Northern Australia Quarantine Strategy (NAQS) program, conducted monitoring for JEV for the 2004 wet season using sentinel pigs at sites on Badu Island in Torres Strait and its northern peninsula area (NPA) site at Injinoo airport in Cape York Peninsula. The five sentinel pigs on Badu Island all seroconverted (based on results of testing at Queensland Health Scientific Services and the CSIRO Australian Animal Health Laboratory). JEV was also identified through the detection of RNA by TaqMan polymerase chain reaction in a pool of culicine mosquitoes collected in a Banks trap on Badu Island. This was collaborative mosquito trapping performed by NAQS for Queensland Health.

The five NPA sentinel pigs located at Injinoo Airport, all seroconverted to JEV (based on results of testing at Queensland Health Scientific Services and the CSIRO Australian Animal Health Laboratory). This is the second time that JEV has been detected on the mainland; the first detection was in 1998. As a follow up to this mainland detection, the Queensland Health Tropical Public Health Unit conducted mosquito trapping at various sites in the NPA. A total of 147 pools, comprising 23,144 mosquitoes, were processed using the JEV-specific TaqMan RT-PCR. Pools were comprised of up to 200 mosquitoes. There was one positive pool of 200 mosquitoes obtained from a trap set at Bamaga rubbish tip. There was inconclusive serological evidence of exposure to JEV in feral pigs sampled by NAQS on the west coast of Cape York Peninsula in July 2004. The time of exposure could not be determined, but it is unlikely to be linked to the 1998 incursion.

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 49 flavivirus (NEC) notifications during 2004. These include flavivirus infections (e.g. MVEV and KUNV) where serology was unable to differentiate between the different viruses.

Queensland reported 46 of the 49 flavivirus (NEC) notifications, of which there were six each of Kokobera and Stratford viruses, one KUNV notification and the remaining 33 notifications were of unknown flavivirus type.

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 559 notifications of malaria in Australia in 2004. The majority of cases were reported by Queensland (47%, n=263), New South Wales (18%, n=101), and Victoria (12%, n=67). There were no reports of locally acquired malaria during the reporting period.

The largest number of malaria notifications was reported amongst males in the 20–24 year age group, and in females in the 25–29 year age group (Figure 55). The male to female ratio was 2:1.

Figure 55. Notifications of malaria, Australia, 2004, by age group and sex

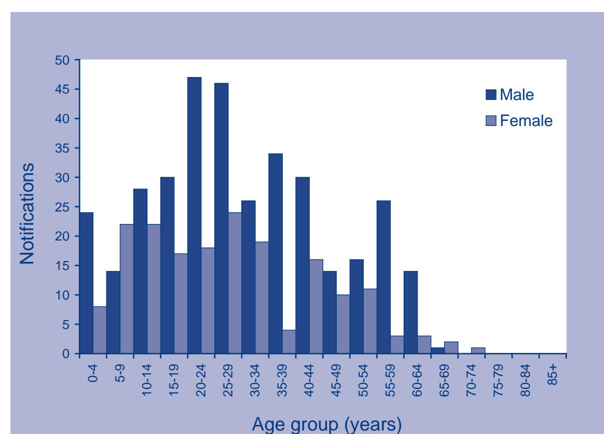


Table 22 shows that the infecting *Plasmodium* species were reported for 91 per cent of malaria notifications in 2004. Of these 559 notifications, *P. falciparum* (48%, n=270) and *P. vivax* (44%, n=248) were the predominant species while untyped *Plasmodium* species accounted for 2 per cent (n=9). The remaining cases were *P. ovale* (4%, n=20) and *P. malariae* (1%, n=7). It should be noted that mixed infections (<1%, n=5) are underestimated due to the variation in reporting practice in different states and territories.

Zoonoses

Zoonoses are diseases and infections naturally transmitted between non-human vertebrate animals and humans.[34] Animal hosts play an essential role in maintaining the infection in nature, and humans are only accidental hosts.[35] Strikingly, 75 per cent of emerging infectious diseases have been identified as zoonotic in origin.[36] In 2004, zoonotic diseases notifiable to the NNDSS were anthrax, Australian bat lyssaviral or lyssaviral (unspecified) infection, brucellosis, leptospirosis, ornithosis and Q fever. During 2004, a total of 877 notifications of zoonotic disease (0.8% 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.*

Table 22. Malaria notifications in Australia, 2004, by parasite type and jurisdiction

Parasite type	Type (%)	State or territory								Australia
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Plasmodium species	2.0	0	0	0	1	0	1	0	7	9
Plasmodium falciparum	48.0	2	44	31	137	14	9	13	20	270
Plasmodium malariae	1.0	1	0	2	3	0	0	0	1	7
Plasmodium ovale	4.0	1	5	0	4	1	0	4	5	20
Plasmodium vivax	44.0	12	50	8	118	3	5	50	2	248
Mixed infection (unspecified)*	0.2	–	0	–	–	0	0	0	1	1
Mixed *P. falciparum* and *P. vivax**	0.7	–	2	–	–	2	0	0	0	4
Mixed *P. falciparum* and *P. ovale**	0.0	–	0	–	–	0	0	0	0	0
Mixed *P. falciparum* and *P. malariae**	0.0	–	0	–	–	0	0	0	0	0
Total	100	16	101	41	263	20	15	67	36	559

* New South Wales, South Australia, Tasmania, Victoria, 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.

Following the deliberate release of anthrax spores in the United States of America in 2001, anthrax became a nationally notifiable disease in Australia. In 2004, no cases of anthrax were notified. 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. Cases only occur sporadically, mostly in partially vaccinated animals.

In 2004, 15 outbreaks of anthrax were reported in livestock (13 from New South Wales and 2 from Victoria). Only one of these outbreaks was from outside the known anthrax endemic areas, on a farm that was part of an old stock route leading to the endemic zone in New South Wales. In all instances the usual protocols of quarantine, disinfection of contaminated ground, carcass incineration, and vaccination of the herd and neighbouring herds were implemented. All animal movements from affected properties were traced and there was no risk of further spread of disease.

Australian bat lyssaviral and lyssaviral (unspecified) infections

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.

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.*

No new cases of either Australian bat lyssaviral or lyssaviral (unspecified) infections were notified during 2004. Two cases of human infection with Australian bat lyssavirus, in 1996 and 1998, occurred following close contact between bat-handlers and infected bats. Both resulted in the death of the infected person.

There are two 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.[37] Research into the genetic sequences of lyssaviruses isolated from different groups of bats using molecular methods suggests that the virus has been associated with bats in Australia for more than 1,500 years.[38] That is, the virus was well established before European colonisation, and its recent 'emergence' is in all likelihood due to changes in human behaviour and encroachment on bat habitats.

Brucellosis

Case definition – Brucellosis

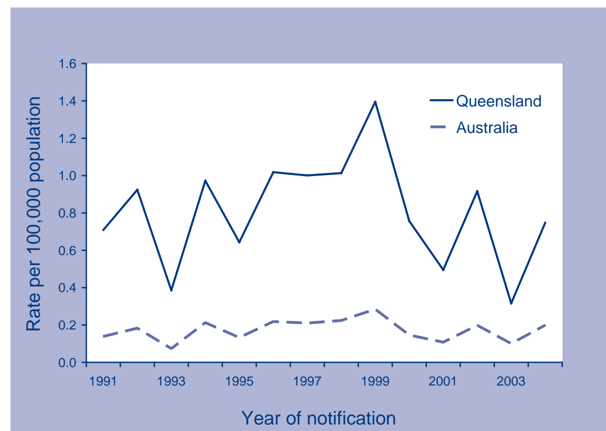
*Only **confirmed cases** are reported.*

***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.*

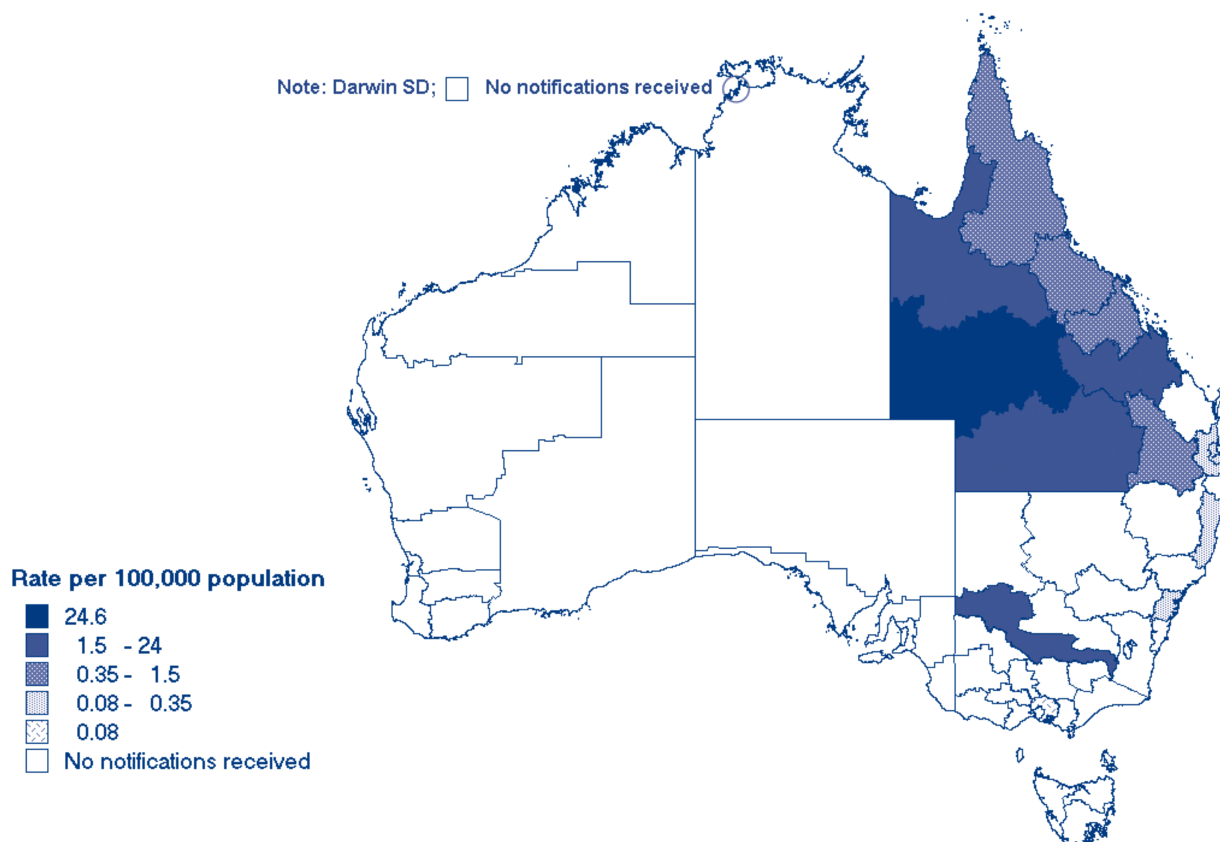
In 2004, 36 cases of brucellosis were reported to the NNDSS, giving a national notification rate of 0.2 cases per 100,000 population. This number of notifications lies in the middle of the range observed over the previous 13 years (13–54 notifications). Most cases were reported from Queensland (26 cases; 72 per cent; Map 9), with a further 19 per cent of cases reported from New South Wales (7 cases), and 8 per cent of cases reported from Victoria (3 cases). There is little evidence of a trend in the national or Queensland notification rates of brucellosis over the last 13 years (Figure 56). Most cases were male (n=32, male to female ratio 6.4:1), and of these, 22 were aged between 20 and 39 years.

Among the nine reported cases for whom species data were available, four cases (all from Queensland) were identified as *Br. suis* which is endemic in feral pigs in Australia. Four cases were identified as *Br. Melitensis* (all overseas acquired). Ovine and caprine brucellosis (*Brucella melitensis*) has never been reported in Australian sheep or goats.[39] One case was identified as *Br. abortus* ('undulant fever'); which was presumably acquired overseas. Bovine brucellosis (*Brucella abortus*) was eradicated from the Australian cattle herd in 1989[39] and is presently considered an exotic animal disease in Australia.

Figure 56. Trends in notification rates of brucellosis, Australia and Queensland, 1991 to 2004



Map 9. Notification rates of brucellosis, Australia 2004, by Statistical Division of residence



Leptospirosis

Case definition – Leptospirosis

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

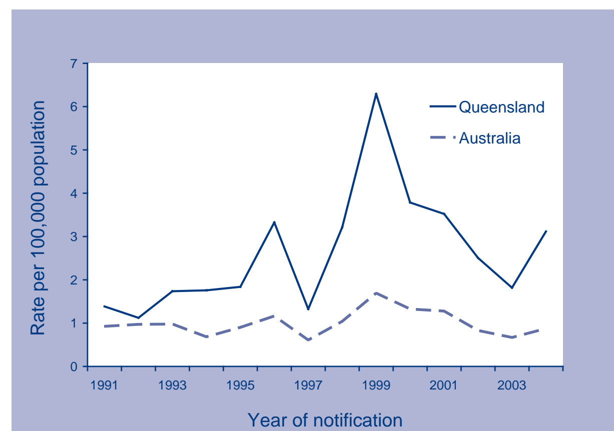
***Confirmed case:** Requires isolation of pathogenic* Leptospira *species, OR a fourfold or greater rise in* Leptospira *agglutination titre between acute and convalescent phase sera obtained at least two weeks apart and preferably conducted at the same laboratory, OR a single* Leptospira *micro agglutination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

Leptospirosis is caused by the spirochaete, *Leptospira*. Nationally, 166 notifications of leptospirosis were received during 2004 (0.9 cases per 100,000 population). This rate is relatively low compared to the previous years but is 31 per cent higher than the national rate in 2003 (Figure 57).

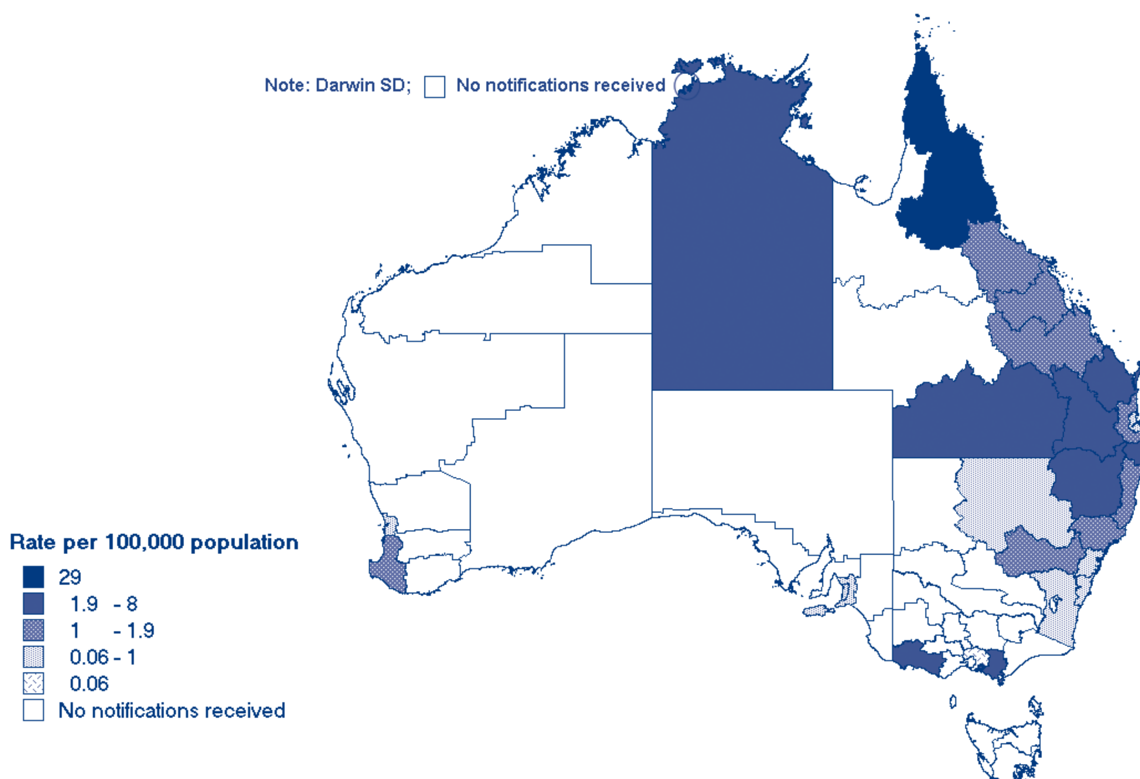
In 2004, the notification rate was highest in Queensland (110 notifications, 2.8 cases per 100,000 population), the Northern Territory (1 notification, 1.0 cases per 100,000 population) and New South Wales (40 notifications, 0.6 cases per 100,000 population). Forty per cent of all notifications were from Far North Queensland (Map 10); the notification rate in this Statistical Division of residence was 28.8 cases per 100,000 population.

Most cases were male (n=151, male to female ratio 10.1:1). There was little evidence that rates of notification varied between age groups.

Figure 57. Trends in notification rates of leptospirosis, Australia and Queensland, 1991 to 2004



Map 10. Notification rates of leptospirosis, Australia, 2004, by Statistical Division of residence



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.

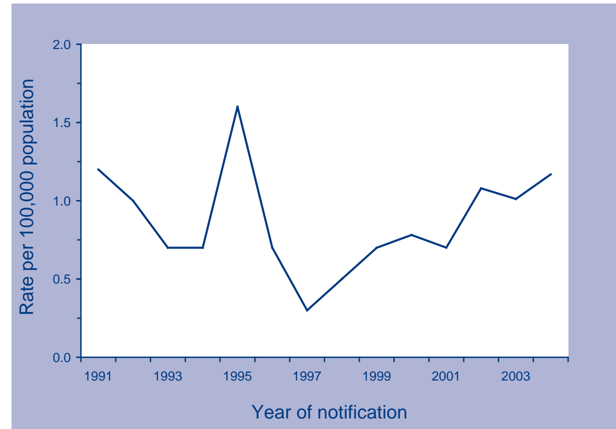
Probable case: *Requires a single high total antibody level or detection of IgM antibody to* C. psittaci *by MIF, OR a single high total antibody titre to* Chlamydia *species demonstrated by complement fixation (CF) in at least one sample obtained at least two weeks after onset of symptoms, OR a fourfold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

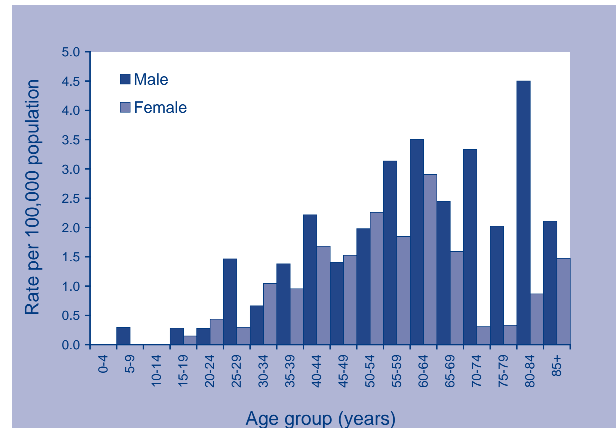
In 2004, there were 235 ornithosis infections notified to NNDSS, giving a national rate of 1.2 cases per 100,000 population. This represents the highest number of notifications in a 12 month period since NNDSS records began in 1991. The national rate of notifications has steadily increased since 1997 (Figure 58).

Figure 58. Trends in notification rates of ornithosis, Australia, 1991 to 2004



Victoria had the highest number of notifications (146 notifications, 2.9 cases per 100,000 population). Notifications also occurred in New South Wales (81 notifications), South Australia (5 notifications) and Queensland (3 notifications). The majority of cases were male (n=138, male to female ratio 1.4:1). The highest reporting rates were in the 80–84 year age group for males (7 notifications, 4.5 cases per 100,000 population) and in the 60–64 year age group for females (13 notifications, 2.9 cases per 100,000 population) (Figure 59).

Figure 59. Notification rates of ornithosis, Australia, 2004, by age group and sex



During 2004, three outbreaks of ornithosis and one death were reported. The first outbreak occurred at a Victorian poultry farm in February. There were 26 cases (14 confirmed and 12 probable) notified, nearly all of whom had worked in the on-site abattoir. In this outbreak, the ratio of males to females was 2:1, and the median age was 43 years (range 17 to 69 years). The second outbreak involving four poultry processing workers occurred at a game processing plant. The three males and one female were aged from 37 to 56 years (median 53 years), two were hospitalised. The other identified outbreak was in New South Wales where eight cases were linked to a pet shop. The one death was reported from South Australia in a female in the 45–49 year age group.

Infection of parrots with *Chlamydia psittaci* has been traditionally known as psittacosis, whereas infection in domestic poultry, waterfowl, pigeons and finches has been called ornithosis. In the past human cases of *C. psittaci* infection have been described as psittacosis, which has led to the common misconception that this disease is associated only with exposure to diseased psittacine birds (i.e. parrots). Subclinical infection with *C. psittaci* is common in numerous wild and domesticated bird species in Australia.[40] Epizootics of clinical disease in commercial flocks and domestic bird collections can be initiated through stresses such as poor animal husbandry.[40] Furthermore, poor biosecurity of commercial poultry flocks can lead to contact with infected native birds (or their excretions) leading to establishment of latent infection within the flock. The two reported outbreaks involving poultry production workers emphasise the need for increased awareness within animal production industries of appropriate animal husbandry and occupational health and safety. Spillovers of a commonly subclinical avian disease from poultry into human populations, possibly emanating from wild bird reservoirs, is concerning given the present highly pathogenic avian influenza (HPAI) epidemic in South East Asia.

Reported rates of ornithosis have repeatedly been 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, although an outbreak investigation in rural Victoria in 1995 showed no association with direct bird handling but rather lawn mowing and gardening in areas with high numbers of native birds.[41] Shedding of *C. psittaci* into the environment by native birds and subsequent inhalation of aerosolised dust and bird excreta was postulated as the mechanism of human infection.

Q fever

Case definition – Q fever

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

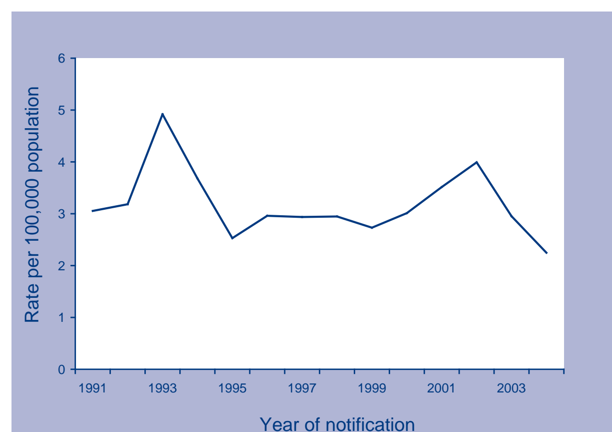
***Confirmed case:** Requires detection of* Coxiella burnetii *by nucleic acid testing, OR seroconversion or significant increase in antibody level to Phase II antigen in paired sera tested in parallel in absence of recent Q fever vaccination, OR detection of* C. burnetii *by culture (note this practice 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 2004, 440 cases of Q fever were notified to the NNDSS, a decrease of 24.5 per cent on 2003. This number of cases is relatively low compared to the count of previous years and the national rate (2.2 cases per 100,000 population) is the lowest recorded since 1991 (Figure 60). The highest rates of notifications were from Queensland (137 notifications, 3.5 cases per 100,000 population), New South Wales (223 notifications, 3.3 cases per 100,000 population) and South Australia (38 notifications, 2.5 cases per 100,000 population). The highest reporting rates were in the 40–44 year age group for males (6.8 cases per 100,000 population), and in the 55–59 year age group for females (2.3 cases per 100,000 population). Few cases were reported from children or the elderly. The male to female ratio was 3.3:1.

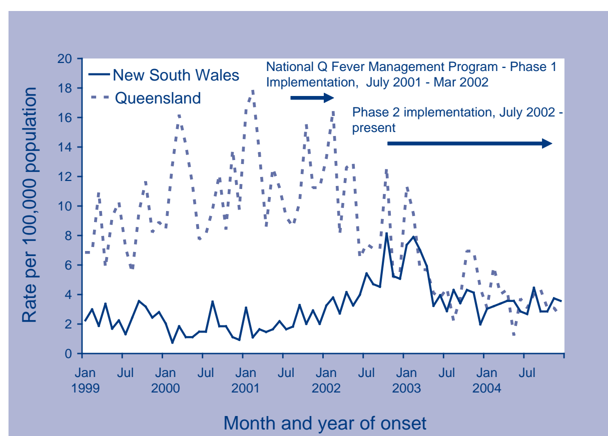
Figure 60. Trends in notification rates of Q fever, Australia, 1991 to 2004



An outbreak of Q fever occurred among persons attending sheep saleyards in rural South Australia during October and November 2004. In total, 25 persons were linked to this outbreak. A case-control study identified a statistically significant association between human illness and attendance at the saleyard. Intervention strategies including vaccination and dust control were implemented. Many of the cases were unvaccinated sheep and grain farmers.

Q fever has long been associated with work in the Australian stock industry and abattoir workers are an occupational group 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 due for completion on 30 June 2004. Several jurisdictions have completed the Program, however, Victoria and South Australia have extended the Program until 30 June 2006 and Queensland has extended it until 30 June 2007.

Figure 61. Notification rates of Q fever, Queensland and New South Wales, January 1999 to December 2004, by month of onset*



Other emerging zoonotic disease in 2004

Bat-associated emerging zoonoses (Hendra and Nipah virus activity 2004)

Surveillance of flying foxes (*Pteropus* spp.) and associated research continued to focus on henipaviruses in 2004. Hendra virus is a viral infection associated with flying foxes. Sporadic infections may occur in horses that come in close contact with infected flying foxes or their body fluids. A horse from Cairns examined by a veterinarian in early December 2004 and subsequently euthanised, tested positive to Hendra virus.[39] The veterinary doctor involved in autopsy of the horse developed a Hendra-related illness soon after and recovered. This was an isolated case. Hendra was also suspected in a horse that died south of Cairns in October 2004. These cases are consistent with previous findings and do not reflect a change in the known distribution or epidemiology of Hendra virus in Australia.[39] The timing of incidents suggests a seasonal pattern of outbreaks possibly related to the seasonality of fruit bat birthing, as Hendra virus has been isolated from foetal tissues and fluids.[42]

This report of the re-emergence of Hendra virus, and repeated outbreaks of Nipah virus-associated encephalitis in humans in Bangladesh underline our still-limited understanding of the ecology of these agents, and the need to maintain surveillance and research efforts.[39]

Other bacterial infections

Legionellosis, leprosy, meningococcal infection and tuberculosis were notifiable in all states and territories in 2004 and classified as 'other bacterial infections' in NNDSS. A total of 1,799 notifications were included in this group in 2004, which accounted for 1.6 per cent of all the notifications to NNDSS, a similar total and proportion as in 2003 (1,826 notifications and 1.7% 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.

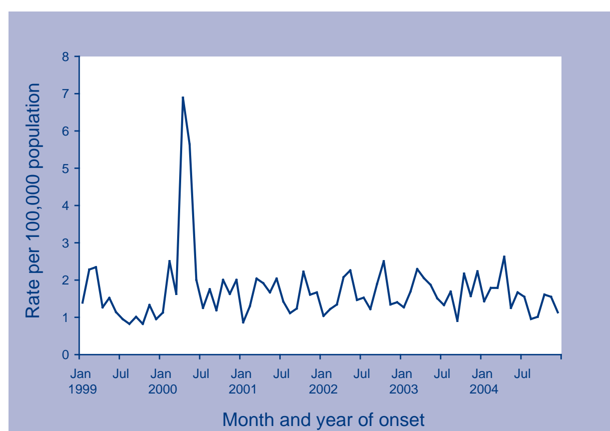
***Probable case:** Single high titre antibody titre to Legionella, OR detection of Legionella by nucleic acid testing, OR detection of Legionella by direct fluorescence assay.*

AND Fever or cough or pneumonia.

Legionellosis includes notifications of infections caused by all *Legionella* species. There were 310 notifications of legionellosis reported in 2004, giving a national rate of 1.6 cases per 100,000 population. Two hundred and thirty-four (75%) cases were confirmed, and 74 (24%) had a probable diagnosis.

In 2004, the highest rates of legionellosis were reported in South Australia (2.9 cases per 100,000 population, 45 cases) and Western Australia (2.5 cases per 100,000 population, 50 cases). Legionellosis notifications showed a peak in autumn and spring (Figure 62).

Figure 62. Trends in notification rate of legionellosis, Australia, 1999 to 2004, by month of onset



Rates of legionellosis have ranged between 0.8 and 2.6 cases per 100,000 population between 1999 and 2004, except in 2000, when rates reached 6.9 cases per 100,000 population as a result of the Melbourne aquarium outbreak, with 125 cases.[43]

In 2004, men accounted for 73.5 per cent of all cases of legionellosis resulting in a male to female ratio of 2.8:1. Cases occurred in all age groups except 5–14 years, with the highest rates in the 75–79 year age group for men (13.4 cases per 100,000 population) and the 75–84 year age groups for women (3.0 cases per 100,000 population) (Figure 63).

Data on the causative species were available for 294 (95%) of the legionellosis cases. Of these, 149 (51%) cases were identified as *L. pneumophilia*, 141 (45 %) were *L. longbeachae* and 4 cases (1 %) were *L. micdadei* (Table 23).

Data on the death of legionellosis cases was available in 112 (36%) notifications. There were 16 deaths due to legionellosis in Australia in 2004, giving a case fatality rate of 5 per cent. The break down of deaths by jurisdiction and infecting *Legionella* species is shown in Table 24. The case fatality rate for infections with *L. longbeachae* infections (5%) was higher than for *L. pneumophila* (4%) but this difference did not reach statistical significance.

There was an outbreak of *Legionella pneumophila* in New South Wales, involving 12 cases. In June, four cases of *Legionella pneumophila* serogroup 1 were discovered in Victoria, with links to a town in north-eastern Victoria where an outbreak of six cases occurred in 2000.

Figure 63. Notification rates of legionellosis, Australia, 2004, by age group and sex

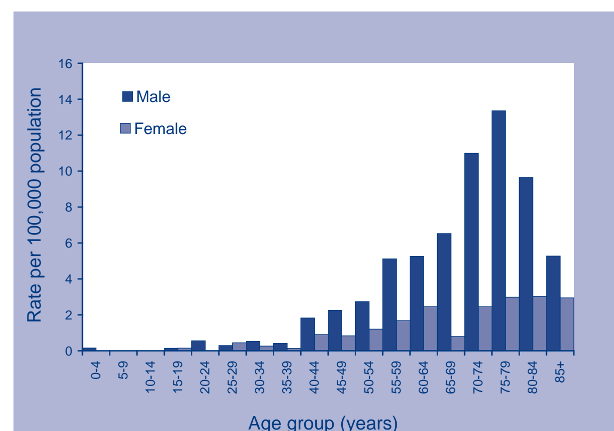


Table 23. Notifications of legionellosis, Australia, 2004, by state or territory and species

Species	State or territory								Total
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Legionella longbeachae	0	29	2	7	36	1	23	43	141
Legionella pneumophila	1	51	0	18	9	0	65	5	149
Legionella micdadei	0	1	0	0	0	0	3	0	4
Unknown species	0	1	0	6	0	0	7	2	16
Total	1	82	2	31	45	1	98	50	310

Table 24. Deaths due to legionellosis, Australia, 2004, by state or territory and species

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

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 2004, five leprosy cases were notified. This is the same number of cases as were notified in 2003. Three cases in occurred in New South Wales and one case occurred in both the Northern Territory and Queensland. Four of the five cases were female, and two cases were Indigenous Australians (one male and one female). Cases ranged in age from 30–79 years. Four cases had multibacillary leprosy and one had paucibacillary leprosy. One case had evidence of Grade 2 disability at presentation, with visible deformity or damage to hands/feet and visual impairment.[44]

The WHO has established the goal of eliminating leprosy by 2005, which is defined as a reduction in the prevalence of leprosy to less than 1 case per 10,000 population. By the end of 2001, 36 of the 37 countries and areas that make up the Western Pacific Region, including Australia, reached this target.[45]

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.*

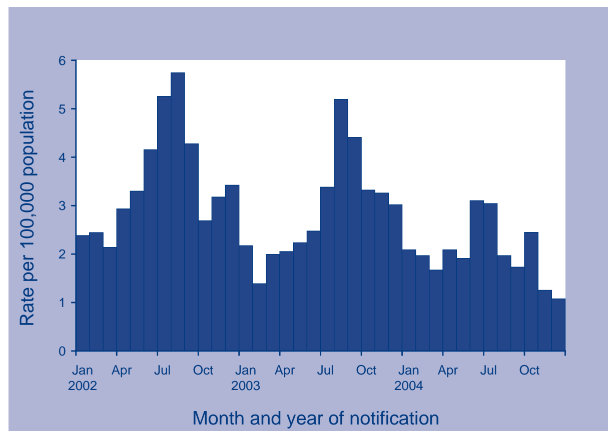
***Probable case:** Defined as the absence of evidence for other causes of clinical symptoms AND EITHER clinically compatible disease including haemorrhagic rash OR clinically compatible disease and close contact with a confirmed case within the previous 60 days.*

In Australia, serogroups B and C are the major cause of invasive meningococcal disease. In response to community concerns about increases in meningococcal disease in Australia, the Australian Government approved the National Meningococcal C Vaccination Program, which commenced in January 2003.[46]

In 2004, there were 408 notifications of invasive meningococcal disease in Australia, 170 cases fewer than in 2003 and a decrease of 29 per cent. The total in 2004 was the lowest since 1996 and is below the historical range (the 5 year mean by minus two standard deviations.) The national notification rate in 2004 was 2.2 cases per 100,000 population. Three hundred and seventy-three cases (91%) were confirmed, and 35 (8%) had a probable diagnosis.

The highest rates were reported from the Northern Territory (6.0 cases per 100,000 population, 12 cases), Tasmania (3.7 cases per 100,000 population, 18 cases) and the Australian Capital Territory (3.4 cases per 100,000 population, 11 cases). There was a small excess of cases among males (male to female ratio 1.2:1). The largest number of cases occurred in winter and spring (Figure 64).

Figure 64. Trends in notification rates of meningococcal infection, Australia, 2002 to 2004, by month of notification

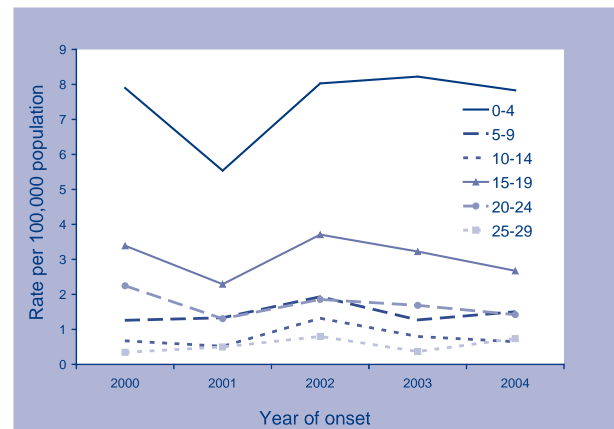


Of the 408 meningococcal notifications in 2004, 342 (84%) were serogrouped. Of these 248 (73%) were serogroup B, 75 (22%) were serogroup C, and 19 (6%) were infections with serogroup Y, serogroup W135 or serogroup A (Table 25). In 2003, of 465 serogrouped notifications, 289 (62%) were serogroup B, and 158 (34%) were serogroup C.

Overall, the highest age specific rate was in children aged 0–4 years with a rate of 10.4 cases per 100,000 population. Of these cases, 99 (75%), were serogroup B infection. In the 15–19 year age group, the overall rate of meningococcal infection was 4.8 cases per 100,000 population, 56 per cent (37 cases) of which were serogroup C.

The highest age-specific rates for serogroup B infection have persisted in the 0–4 years age group since 2000. In 2004, the rate for this age group was 6.5 cases per 100,000 population, (82 cases), while in the 15–19 years age group, the rate was 2.7 cases per 100,000 infections (37 cases) (Figure 65).

Figure 65. Notification rates of meningococcal B infection, Australia, 2000 to 2004, by age group



Between 2002 and 2004, rates of meningococcal serogroup C infection decreased in all age groups. There was a marked decrease in infection rates during 2003, the year the National Meningococcal C Vaccination Program was introduced. General practitioner based vaccination of 1–5-year-olds was completed at the end of 2004 in all jurisdictions. School based vaccination programs, first targeting 15–19-year-olds, then 6–14-year-olds, were complete in all jurisdictions, except South Australia by December 2004.

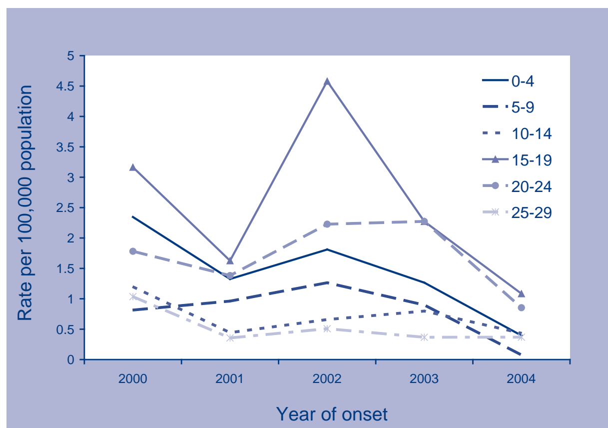
The decrease in rates of serogroup C infection was greatest in the 15–19 year age group. In 2002, the serogroup C infection rate in the 15–19 year age group, was 4.6 cases per 100,000 population (63 cases). The rate in this age group decreased to 1.1 cases per 100,000 population (15 cases) in 2004. In the 0–4 year age group, the rate decreased from 1.8 to 0.4 cases per 100,000 population from 2002 to 2004. There were similar declines in the 5–9 and 20–24 year age groups (Figure 66).

Table 25. Notifications of meningococcal infection Australia, 2004, by state or territory and serogroup

Species	State or territory								Total
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Serogroup B	4	82	8	49	11	7	55	32	248
Serogroup C	7	24	1	19	1	5	12	6	75
Other serogroups*	0	8	1	4	0	1	4	1	19
Unknown serogroup	0	39	2	9	1	5	8	2	66
Total	11	153	12	81	13	18	79	41	408

* Other includes serogroups A, Y and W135.

Figure 66. Notification rates of meningococcal C infection, Australia, 2000 to 2004, by age group



Data on deaths from meningococcal infection were available for 172 (42%) cases. There were 20 deaths due to meningococcal infection in 2004 giving a crude case fatality rate of 5 per cent. The breakdown of deaths by jurisdiction and serogroup are shown in Table 26. The case fatality rate of 5.4 per cent for infections with meningococcal group C was the same as that for meningococcal group B infections. In 2003, the case fatality rate for infections with meningococcal group C was more than three times higher than for meningococcal group B infections.[47]

Laboratory based meningococcal surveillance

The Australian Meningococcal Surveillance Programme was established in 1994 for the purpose of monitoring and analysing 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 2004 have recently been published.[48]

In 2004, a total of 361 isolates of *N. meningitidis* were analysed by the program, a 27 per cent decrease from the 494 isolates analysed in the previous year.

Consistent with routine surveillance data, serogroup B continued to be the predominant strain for the disease (243 isolates, 67%) nationally, followed by serogroup C (71 isolates, 20%). Serogroup B strains predominated in all jurisdictions except the Australian Capital Territory where 8 of 11 isolates were serogroup C.

The pattern of age distribution for meningococcal infection varied by phenotype. Serogroup B was more frequently reported in the 5–9 year (90.5%) and 0–4 year (87.4%) age groups, while the largest proportions of serogroup C occurred in the 25–44 year (35.7%), and 20–24 year (31.4%) age groups. This represents a shift in the age distribution of both serogroups from 2003 when most infections with serogroup B occurred in the 0–4 year age group, and serogroup C infections were reported most frequently in the 15–19 year age group.

In 2004, 147 of the 238 isolates (62%) tested showed decreased susceptibility to the penicillin group of antibiotics (MIC 0.06–0.5 mg/L). All isolates tested were susceptible to third generation cephalosporins and the prophylactic antibiotics, ciprofloxacin and rifampicin.

Tuberculosis

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.*

Table 26. Deaths due to meningococcal infection, Australia, 2004, by state or territory and serogroup

Species	State or territory								Total
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Serogroup B	0	4	0	1	1	1	5	1	13
Serogroup C	0	1	0	1	0	1	1	0	4
Other serogroups*	0	0	0	1	0	0	0	1	2
Unknown serogroup	0	1	0	0	0	0	0	0	1
Total	0	6	0	3	1	2	6	2	20

* Other includes serogroups A, Y and W135.

While Australia has one of the lowest rates of tuberculosis (TB) in the world, the disease remains a public health problem in the overseas-born and Indigenous communities. In 2004, 1,076 TB notifications were received by NNDSS, a rate of 5.4 cases per 100,000 population. There was an 8 per cent increase in the number of notifications in 2004 compared to 2003. The notification rates of TB were higher than the national average in the Northern Territory (14 cases per 100,000 population), and the lowest rate occurred in Tasmania (2.3 cases per 100,000 population).

The highest incidence was reported in people born overseas (21.7 cases per 100,000 population) and Indigenous Australians (8.1 cases per 100,000 population). By contrast the rate in the non-Indigenous Australian-born population was 1.2 cases per 100,000 population. For more details see the tuberculosis 2004 annual report in this issue of *Communicable Diseases Intelligence*.[49]

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. 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 2004, a total of 12 laboratories reported 26,218 infectious agents to LabVISE. This represents a 14 per cent increase in the number of reports received in 2004 compared to 2003 (Table 27). Most of the reports were from South Australia (30%), Queensland (27%) and Western Australia (16%) (Table 27).

Sixty per cent (n=15,608) of all reports received by LabVISE were viral infectious agents, and the remaining 40 per cent (n=10,610) were bacterial or other infectious agents. Among viruses, herpes viruses (33.5%; 5,268) were the most commonly reported followed by ortho/paramyxoviruses (27%; 4,124) which includes influenza, parainfluenza and respiratory syncytial viruses (Figure 67). Among non-viral infectious agents, *Chlamydia trachomatis*

Figure 67. Reports of viral infections to the Laboratory Virology and Serology Reporting Scheme, 2004, by viral group

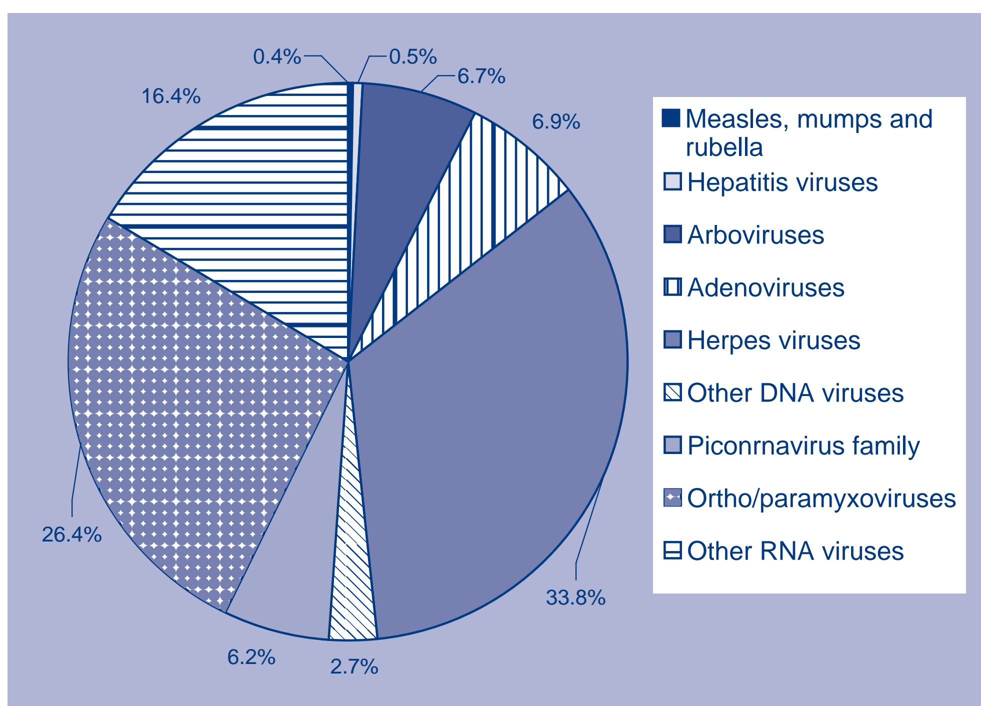


Table 27. Infectious agents reported to the Laboratory Virology and Serology Reporting Scheme, 2004, by state or territory

Organism	State or territory								Total 2004	Total 2003
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Measles virus	0	3	1	5	6	0	12	8	35	71
Mumps virus	0	0	0	1	2	0	1	2	6	10
Rubella virus	0	3	0	8	2	0	2	5	20	26
Hepatitis A virus	0	7	3	16	6	0	5	14	51	87
Hepatitis D virus	0	1	0	2	2	0	1	2	8	19
Hepatitis E virus	0	0	0	0	0	0	12	2	14	–
Ross River virus	0	19	16	608	44	3	19	34	743	1,239
Barmah Forest virus	1	14	2	153	14	0	2	9	195	408
Alphavirus (unspecified)	0	0	1	0	0	0	0	0	1	–
Dengue	0	0	5	0	1	0	0	6	12	35
Flavivirus (unspecified)	0	1	7	81	0	0	12	1	102	122
Adenovirus type 40	0	0	1	0	0	0	0	30	31	32
Adenovirus not typed/pending	10	247	3	78	438	5	165	105	1,051	928
Herpes virus type 6	0	2	0	0	0	0	4	0	6	5
Cytomegalovirus	8	374	6	108	226	17	94	1	834	859
Varicella-zoster virus	1	161	23	928	469	9	73	397	2,061	1715
Epstein-Barr virus	0	93	60	771	1,119	3	41	280	2,367	1,719
Other DNA viruses	0	15	0	111	33	3	67	194	423	279
Picornavirus family	7	502	5	21	105	3	83	238	964	805
Ortho/paramyxoviruses	5	1,329	13	330	1,255	60	403	729	4,124	4,568
Other RNA viruses	0	294	29	2	457	119	855	804	2,560	1,801
Chlamydia trachomatis	39	691	13	1,929	1,689	36	61	801	5,259	4,298
Chlamydia pneumoniae	0	1	0	0	1	0	5	2	9	15
Chlamydia psittaci	2	3	0	2	6	0	159	1	173	118
Chlamydia species (untyped)	0	3	0	0	0	0	1	1	5	2
Mycoplasma pneumoniae	1	111	23	475	381	15	321	47	1,374	1,146
Mycoplasma hominis	0	4	0	0	1	0	0	0	5	9
Coxiella burnetii (Q fever)	1	3	3	30	115	0	17	4	173	178
Rickettsia prowazeki	0	0	0	0	102	0	0	1	103	3
Rickettsia tsutsugamushi	0	0	0	0	64	0	1	2	67	4
Rickettsia – spotted fever group	0	0	0	0	136	3	0	0	139	2
Streptococcus group A	0	7	1	320	0	0	139	0	467	490
Yersinia enterocolitica	0	8	0	0	0	0	0	0	8	12
Brucella abortus	0	0	0	0	2	0	4	0	6	5
Brucella species	0	4	0	5	0	0	0	0	9	7
Bordetella pertussis	6	68	0	170	549	2	270	293	1,358	520
Bordetella parapertussis	0	0	0	0	0	0	1	0	1	–
Legionella pneumophila	0	9	0	0	8	0	59	1	77	132
Legionella longbeachae	0	3	0	0	26	1	25	21	76	84
Legionella species	0	4	0	1	0	0	10	0	15	18
Cryptococcus species	0	2	0	7	29	0	0	0	38	26
Leptospira species	0	0	0	20	3	0	0	0	23	24
Borrelia burgdorferi	0	0	0	0	0	0	0	1	1	–
Treponema pallidum	1	159	0	535	447	0	3	9	1,154	1,168
Entamoeba histolytica	0	0	0	2	0	1	10	1	14	14
Toxoplasma gondii	0	11	0	4	11	2	10	3	41	41
Echinococcus granulosus	0	0	0	0	13	0	2	0	15	21
Total	82	4,156	215	6,723	7,762	282	2,949	4,049	26,218	23,065

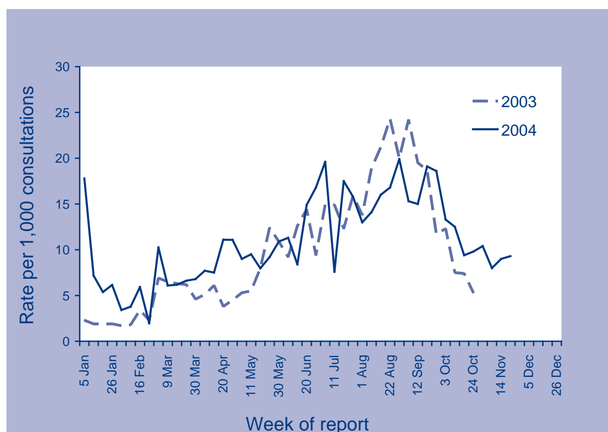
(52%; 5,259), *Mycoplasma pneumoniae* (13%; 1,374) and *Bordetella pertussis* (13%; 1,358) were the most commonly reported pathogens.

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. The data provide an indicator of the burden of disease in the primary care setting and allows trends in consultation rates to be detected.

In 2004, influenza-like illnesses (ILI), gastroenteritis, and varicella infections (chickenpox and shingles) were the communicable diseases reported to ASPREN. Each week an average of 28 general practitioners (range 10 to 40) provided information from an average of 2,913 (range 1,047–4,219) consultations per week.

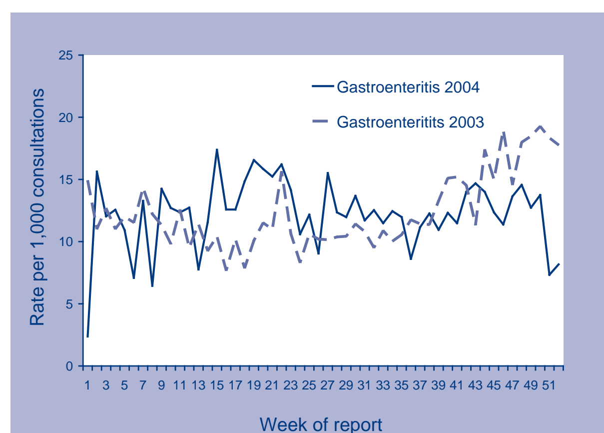
Figure 68. Consultation rates for influenza-like illness, ASPREN 2004 compared with 2003, by week of report



Influenza-like illness reports (Figure 68) showed atypical seasonal pattern with two peaks, in mid-July (20.3 ILI per 1,000 consultations), and in mid-September (18.3 ILI per 1,000 consultations). This may reflect the different peak times of ILI in different jurisdictions (Figure 68).

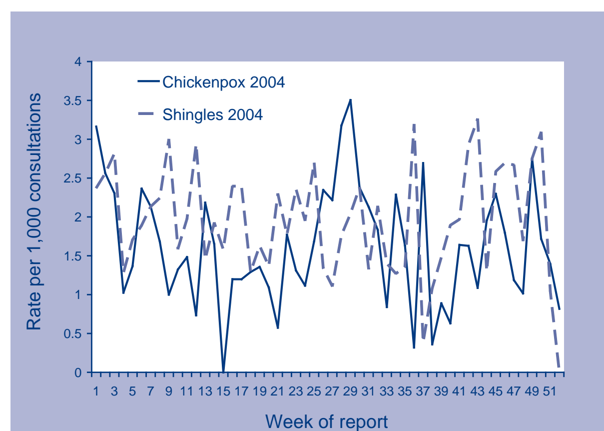
Consultations for gastroenteritis were not stable to show seasonality, they fluctuated between 6 to 17 cases per consultations. (Figure 69).

Figure 69. Consultation rates for gastroenteritis, ASPREN, 2004 compared with 2003, by week of report



Reports of varicella infections continue to be reported at a lower rate by ASPREN. Rates of shingles exceeded those for chickenpox in most weeks but there was no recognisable seasonal pattern (Figure 70).

Figure 70. Consultation rates for varicella infections, ASPREN, 2004, by week of report



Appendices

Appendix 1. Mid-year estimate of Australian population 2004, by state or territory

	State or territory								
	NSW	Vic	Qld	SA	WA	Tas	NT	ACT	Aus*
Male	3,346,616	2,453,147	1,937,822	759,773	992,452	237,918	105,173	160,253	9,994,541
Female	3,384,679	2,519,632	1,944,215	774,477	989,752	244,210	94,740	163,768	101,16,756
Total	6,731,295	4,972,779	3,882,037	1,534,250	1,982,204	482,128	199,913	324,021	20,111,297

* Includes other territories.

Appendix 2. Mid-year estimate of Australian population 2004, by state or territory and age group

	State or territory								
Age	NSW	Vic	Qld	SA	WA	Tas	NT	ACT	Aus*
0–4	425,944	306,301	250,159	88,793	124,789	30,187	17,608	20,238	1,264,281
5–9	441,816	320,014	267,610	96,154	133,574	32,477	16,640	20,973	1,329,497
10–14	458,629	333,257	280,137	100,885	141,273	34,438	16,312	21,976	1,387,173
15–19	453,556	334,947	273,665	103,547	144,666	34,291	14,652	23,849	1,383,383
20–24	459,158	352,290	277,286	102,512	141,180	30,279	15,875	28,299	1,407,023
25–29	458,261	342,422	259,821	94,003	133,213	26,614	16,756	25,423	1,356,644
30–34	513,433	384,853	291,434	106,109	147,840	31,210	18,390	25,663	1,519,131
35–39	483,197	370,477	278,701	107,976	146,408	32,182	16,670	24,075	1,459,880
40–44	515,181	378,256	296,364	117,404	155,199	36,895	16,202	24,828	1,540,561
45–49	475,223	351,708	274,081	111,200	146,141	35,685	13,823	23,647	1,431,734
50–54	435,991	321,813	254,906	104,905	134,095	33,651	12,540	22,610	1,320,721
55–59	399,113	291,829	236,244	97,358	117,462	31,060	9,424	19,509	1,202,129
60–64	304,469	221,047	176,404	72,637	86,307	24,373	6,216	12,701	904,255
65–69	255,300	186,285	139,010	61,792	69,772	20,023	3,583	9,420	745,247
70–74	217,986	159,775	112,285	53,877	56,105	16,647	2,218	7,198	626,124
75–79	192,815	141,151	95,601	50,597	46,619	14,275	1,530	6,238	548,837
80–84	136,137	99,585	66,824	36,171	32,354	10,084	847	4,363	386,374
85–89	68,889	49,048	33,922	18,224	15,819	5,180	375	2,001	193,465
90–94	27,841	21,174	13,675	7,798	7,099	2,019	151	788	80,549
95–99	6,864	5,380	3,245	1,889	1,863	465	63	191	19,960
100+	1,492	1,167	663	419	426	93	38	31	4,329
Total	6,731,295	4,972,779	3,882,037	1,534,250	1,982,204	482,128	199,913	324,021	20,111,297

* Includes other territories.

Appendix 3. Completeness of National Notifiable Diseases Surveillance System data, received from states and territories, 2004

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aus
Total notifications	1,656	31,021	5,199	25,249	7,472	1,965	24,032	14,336	110,929
Sex									
Unknown/missing	1	97	1	5	0	1	168	7	280
Per cent complete*	99.9	99.7	100.0	100.0	100.0	99.9	99.3	100.0	99.7
Age									
Unknown/missing	1	63	22		2	7	108	11	214
Per cent complete*	99.9	99.8	99.6	100.0	100.0	99.6	99.6	99.9	99.8
Indigenous status†									
Not stated/missing	1,603	23,163	404	16,475	804	1,471	11,648	4,829	60,397
Per cent complete*	3.2	25.3	92.2	34.7	89.2	25.1	51.5	66.3	45.6

* 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.

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Invasive pneumococcal disease in Australia, 2004

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Abstract

There were 2,375 cases of invasive pneumococcal disease (IPD) notified to the National Notifiable Diseases Surveillance System in Australia in 2004; a notification rate of 11.8 cases per 100,000 population. The rate varied between states and territories and by geographical region with the highest rates in the Northern Territory. Invasive pneumococcal disease was reported most frequently in children aged less than 5 years (55.4 cases per 100,000 population). Enhanced surveillance for IPD was carried out in all states and territories, in 2004, providing additional data on 2,023 (85%) cases. The overall rate of IPD in Indigenous Australians was 3.2 times the rate in non-Indigenous Australians. There were 154 deaths attributed to IPD resulting in an overall case fatality rate of 7.6 per cent. Rates of IPD in the Indigenous and non-Indigenous under 2-year-old population were similar in 2004 (91.5 and 93.6 cases per 100,000 population, respectively) following a targeted introduction of the 7-valent pneumococcal conjugate vaccine (7vPCV) in mid-2001 for Indigenous infants and children. Serotypes of isolates were identified from 80 per cent of all notified cases, with 72 per cent of isolates belonging to serotypes represented in the 7vPCV and 91 per cent in the 23-valent polysaccharide pneumococcal vaccine (23vPPV). Comparison of serotypes in the 7vPCV target population showed that the rate of IPD due to 7vPCV serotypes decreased by 74 per cent between 2001–02 and 2003–04. Of 216 isolates with reduced penicillin susceptibility, 83 per cent belonged to pneumococcal serotypes in the 7vPCV and 95 per cent in the 23vPPV. *Commun Dis Intell* 2006;30:80–92.

Keywords: disease surveillance, pneumococcal disease, Streptococcus pneumoniae

Introduction

Streptococcus pneumoniae is a leading cause worldwide of otitis media, pneumonia, bacteraemia and meningitis. Invasive pneumococcal disease (IPD) in Australia is generally a disease of young children and older adults. The incidence of IPD in Indigenous Australians has been much higher than that in non-Indigenous Australians.

More than 90 serotypes of *S. pneumoniae* identified by the polysaccharide composition of their capsule have been described. Immunity to pneumococcal infection is thought to be serotype-specific. Vaccines containing pneumococcal polysaccharides from different numbers of serotypes have been available for many years, with a 23-valent polysaccharide vaccine (23vPPV) being used in Australia from 1986 (Table 1). Polysaccharide pneumococcal vaccines are poorly immunogenic in young children. A vaccine in which polysaccharides from seven serotypes are conjugated with a protein carrier (mutated diphtheria toxoid) was developed to provide an effective pneumococcal vaccine for children. In a trial in the United States of America (USA) in infants aged 2 to 15 months the conjugate vaccine had a protective

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efficacy of 93.9 per cent.[1] The conjugate vaccine (7vPCV) was licensed for use in Australia in January 2001 and a nationally funded vaccination program for children at high risk commenced in June 2001 (Table 1).

IPD was made a notifiable disease in all Australian states and territories in 2001 and surveillance data are reported to the National Notifiable Diseases Surveillance System (NNDSS). Additional enhanced surveillance data on IPD have also been collected since 2001 and annual reports have been published.[2,3,4] In this report, the impact of the 7vPCV vaccine on IPD in vaccine eligible children has been evaluated with respect to overall rates of disease, disease caused by vaccine and non-vaccine serotypes and levels of antimicrobial resistance.

Methods and materials

Case definition

A case of invasive pneumococcal disease was defined as the isolation from or the detection by nucleic acid test in blood, cerebrospinal fluid 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 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 2004 comprised core data, which is a set of data collected on all cases of all notifiable diseases, as well as data specific for IPD.[4]

Clinical presentation

Clinical presentations were coded as pneumonia, meningitis, bacteraemia, other or unknown. Pneumonia was defined as blood culture or nucleic acid test positive for *S. pneumoniae* with clinical and/or radiological signs of pneumonia. Meningitis was defined as the detection of *S. pneumoniae* in the cerebrospinal fluid 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.

Table 1. Recommendations for pneumococcal vaccination, Australia, 2004

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
Date implemented	1998	June 2001
Target populations	All individuals aged 65 years and over. Aboriginal and Torres Strait Islander people aged 50 years and over. Children aged over 5 years who have underlying chronic illnesses predisposing to invasive pneumococcal disease (including asplenia and immunocompromise). Immunocompetent individuals with chronic illness including chronic cardiac, renal or pulmonary disease, diabetes and alcohol-related problems. Individuals aged over 5 years 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 with underlying medical conditions and Aboriginal children aged under 5 years residing in Central Australia. Aboriginal and Torres Strait Islander children under the age of 2 years residing elsewhere in the Northern Territory (i.e. other than in Central Australia), Western Australia, South Australia and Queensland. Aboriginal and Torres Strait Islander children under the age of 2 years residing in New South Wales, the Australian Capital Territory, Victoria and Tasmania, and all non-Indigenous children without underlying medical conditions.
Data source	NHMRC Immunisation Handbook 8th edition, 2003	NHMRC Immunisation Handbook 8th edition, 2003

Vaccination

The definitions of vaccination status, vaccination confirmation and vaccine failure are shown in Table 2.

Populations under surveillance

There were differences in populations under surveillance between jurisdictions in the collection of enhanced IPD data. The age groups for whom enhanced data were collected for 2004 are shown in Table 3.

Data were analysed by date of diagnosis which was the earliest date recorded of date of onset, specimen date, notification date, or notification received date.

Table 3. Enhanced invasive pneumococcal disease surveillance data collection, 2004, by state or territory

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

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 2004, was used as the denominator in rate calculations.

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 at aged 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. 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.

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 was calculated using the Chi-square test with Yates correction.

Results

There were 2,375 notifications of IPD to NNDSS in 2004; a 9.2 per cent increase over the number of notifications in 2003. The number of notifications and notification rate per 100,000 population are shown in Table 4.

The notification rates for IPD varied between 7.8 and 17 cases per 100,000 population except in the Northern Territory where the notification rate was 46.5 cases per 100,000 population. The number of notifications in 2004 was fewer in Victoria compared with the total in 2003, but increased in all other jurisdictions.

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 the winter months with the peak number of notifications in August (342 notifications). The effect of season was more evident in the distribution of cases aged five years or more compared with younger children (Figure 1).

Figure 1. Notifications of invasive pneumococcal disease, Australia, 2004, by month of report and age group

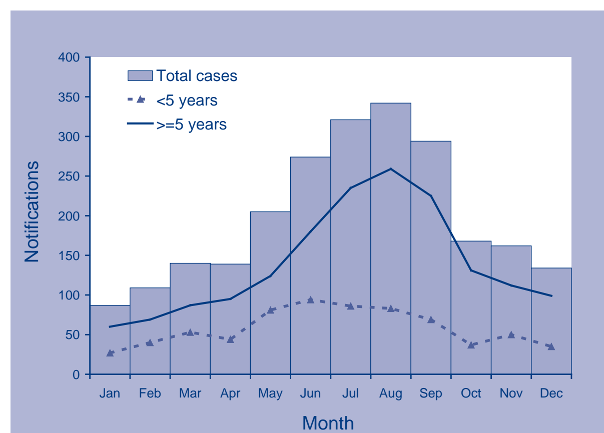


Figure 2. Notification rates of invasive pneumococcal disease, Australia, 2004, by age group and sex

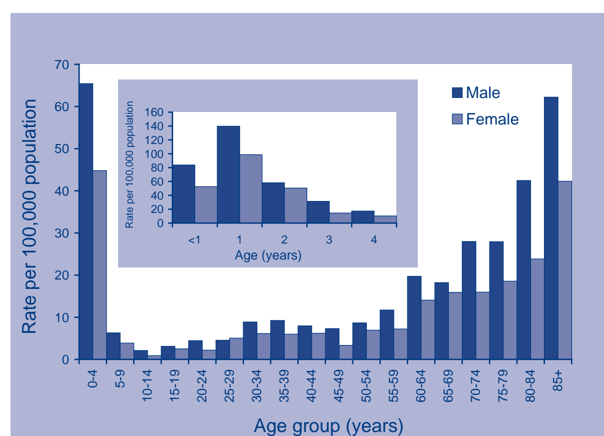
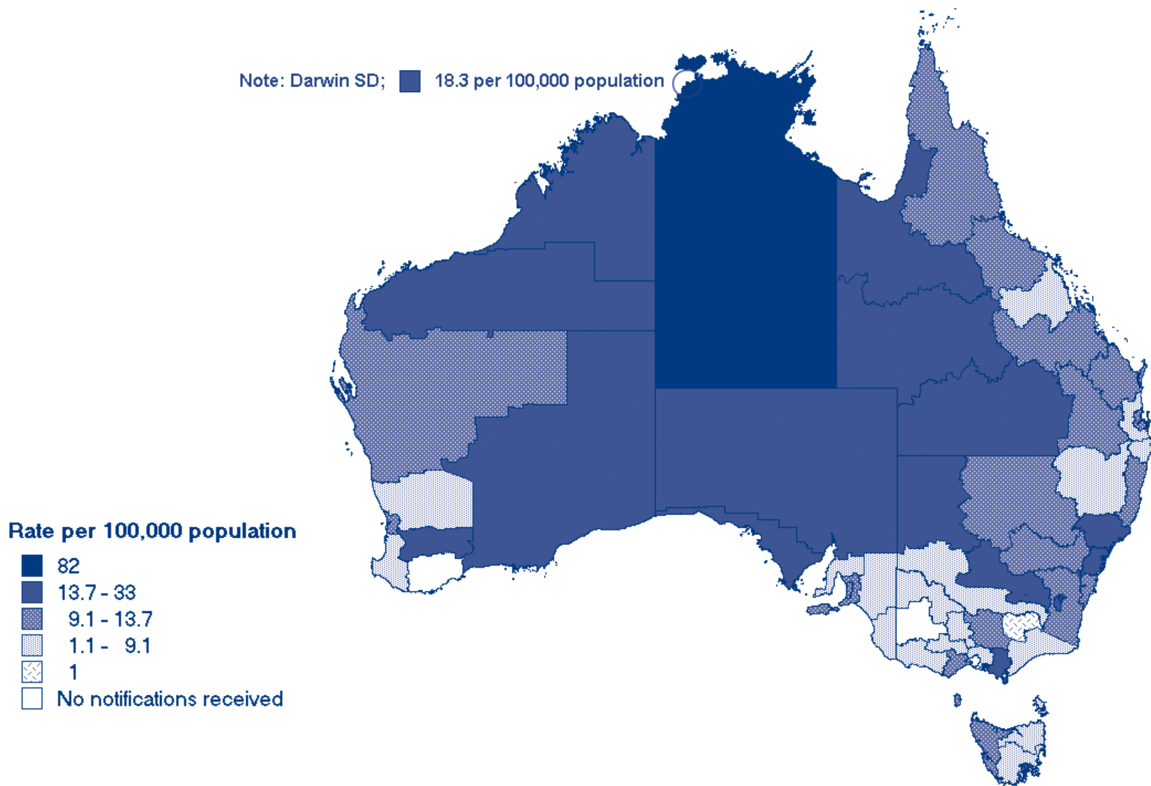


Table 4. Notifications, rates and demographics of invasive pneumococcal disease cases, Australia, 2004, by state and territory

	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Notifications	55	908	93	477	198	56	389	199	2,375
Rate/100,000	17.0	13.5	46.5	12.3	12.9	11.6	7.8	10.0	11.8
Sex									
Male:female ratio	3.2:1	1.3:1	1.2:1	1.4:1	1.5:1	1.2:1	1.4:1	1.3:1	1.4:1
Age									
<5 years	20	270	15	153	76	9	111	47	701
5 to 64 years	21	356	74	212	56	34	165	102	1,020
≥65 years	14	282	4	112	66	13	113	50	654
Indigenous status									
Indigenous	0	14	80	33	8	1	7	31	174
Non-Indigenous	3	612	13	336	188	48	354	164	1,718
Unknown	52	282	0	108	2	7	28	4	483
Enhanced surveillance cases (% of total)	26 (47%)	585 (64%)	93 (100%)	477 (100%)	198 100%)	56 (100%)	389 (99%)	199 (100%)	2,023 (85%)

Map. Notification rates of invasive pneumococcal disease, Australia, 2004, by Statistical Division of residence

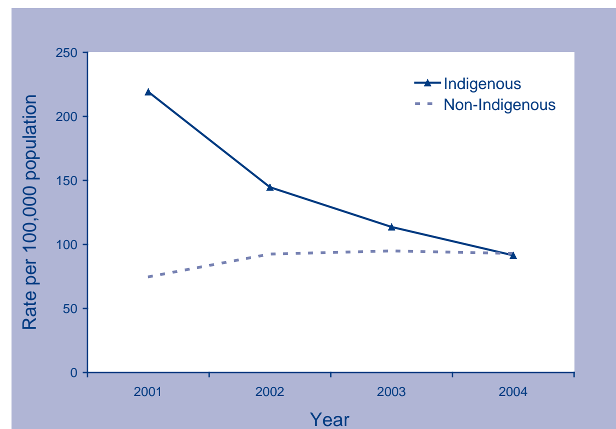


The highest rates of disease were found in children aged less than 5 years (55.4 cases per 100,000 population) and adults aged 85 years or more (48.6 cases per 100,000 population). Among children aged less than 5 years, the highest rates of IPD were recorded in children aged one year (119 cases per 100,000 population). There were 472 cases in children aged less than 2 years. In all age groups there were more male than female cases (overall male to female ratio 1.4:1)

There were 174 cases of IPD among Indigenous people (6.2% of all cases). This represents a rate of 35.9 cases per 100,000 population compared with a rate of 11.2 cases per 100,000 population in non-Indigenous people. The rates were highest in Indigenous people in the Northern Territory (134 cases per 100,000 population, 80 cases).

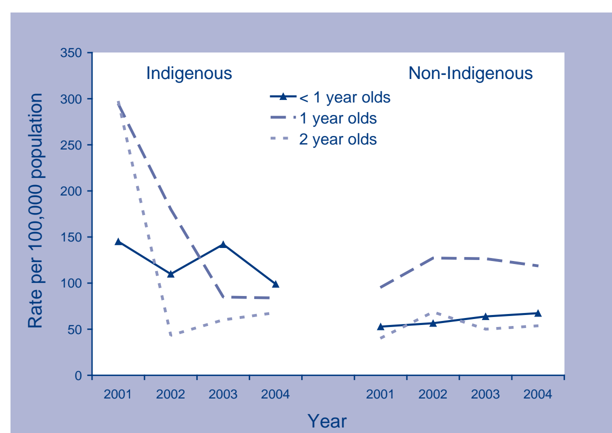
Since 2001, a 7vPCV vaccination program has provided free vaccination to Indigenous children less than 2 years of age (Table 1). The disparity in the rates of IPD between Indigenous and non-Indigenous children aged under 2 years has dropped from 2.9-fold (219.2 and 74.7 cases per 100,000 population, respectively) in 2001 to parity (91.5 and 93.6 cases per 100,000 population, respectively) in 2004 (Figure 3).

Figure 3. Notification rates of invasive pneumococcal disease in Indigenous and non-Indigenous children aged less than 2 years, Australia, 2001 to 2004



Between 2001 and 2004, the rate of IPD in Indigenous children aged one year (12 to 23 months) fell from 294 to 84 cases per 100,000 population (34 cases in 2001 to 10 cases in 2004). Similarly, the rate of IPD in Indigenous children aged 2 years (24 to 35 months) fell from 297 to 68 cases per 100,000 population (34 cases in 2001 to 8 cases in 2004, Figure 4).

Figure 4. Rates of invasive pneumococcal disease in children aged 2 years and under, 2001 to 2004, by Indigenous status and single year age group



Enhanced surveillance including data on clinical presentation and risk factors were available on 2,023 (85%) cases. Clinical presentation was reported for 1,219 (60%) enhanced notifications. Of these, 672 (55%) were pneumonia, 429 (35%) were bacteraemia, 75 (6%) were meningitis and the remainder were other presentations (n=43).

As in previous years there were significantly larger proportion of IPD cases with pneumonia among Indigenous children aged less than 5 years (45%) compared with non-Indigenous children in the same age group (22%, $p<0.01$). The proportion of IPD cases with bacteraemia was significantly larger in non-Indigenous (65%), than in Indigenous (45%, $p<0.05$) children, aged less than 5 years.

There were 154 deaths recorded among IPD cases in Australia in 2004, a case fatality rate of 7.6 per cent (Table 5). The case fatality rate in those aged 65 years and older (16%) was significantly higher than in children aged less than 5 years (2.3%, $p<0.0001$). The case fatality rate was not significantly different in Indigenous (4.8%) and non-Indigenous cases (7.6%). Of the 16 children under 5 years of age who died, 13 were under 2 years.

Risk factors for pneumococcal disease

The national surveillance working party defined risk factor categories for IPD. Other risk factors defined by jurisdictions were also collected. More than one risk factor could be recorded for each case. Recognised risk factors were collected in 686 (34%) enhanced cases. The most commonly reported risk factor was chronic disease (376 cases, 54.8%) which included chronic respiratory, cardiac and renal disease.

The frequency of risk factors for IPD in Indigenous and non-Indigenous people are shown in Table 6. Premature birth was a significantly more common risk factor in non-Indigenous children compared with Indigenous children. Immunocompromising conditions were recognised as a more common risk factor in older non-Indigenous children and adults than in Indigenous cases in the same age range.

Pneumococcal serotypes causing disease in Australia

Pneumococcal serotypes were identified for isolates from 1,915 (80%) of all notified cases in 2004. Of these, 72 per cent (1,373) were serotypes in the 7vPCV and 91 per cent (1,750) were serotypes in the 23vPPV (Table 7).

Table 5. Case fatality rates* for invasive pneumococcal disease, Australia, 2004, by age, Indigenous status and state or territory

	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Cases	26	585	93	477	198	56	389	199	2,023
Deaths	0	84	4	9	7	6	28	16	154
Case fatality rate (%)	0.0	14.3	4.3	1.9	3.5	10.7	7.2	8.0	7.6
Deaths in < 5 years	0	7	0	2	2	0	4	1	16
Case fatality rate in <5 years	0.0	2.6	0.0	1.3	2.6	0.0	3.6	2.1	2.3
Deaths in >65 years	0	56	2	4	4	5	17	6	94
Case fatality rate >65 years	0.0	24.3	50.0	3.6	6.1	38.5	15.0	12.0	16.0
Deaths in Indigenous people		1	3	1	0	0	0	3	8
Case fatality rate Indigenous	0	16.7	3.8	3.0	0.0	0.0	0.0	9.7	4.8
Deaths in non-Indigenous people	0	77	1	8	5	6	28	13	138
Case fatality rate non-Indigenous	0.0	13.7	7.7	1.8	2.6	10.9	7.3	7.7	7.6

* From enhanced invasive pneumococcal disease surveillance data.

Table 6. The frequency of risk factors for invasive pneumococcal disease, Australia, 2004, by age group and Indigenous status

Risk factor	Cases aged less than 5 years			Cases aged 5 years or more		
	Indigenous n=14	Non Indigenous n=85	Significance of difference	Indigenous n=98	Non-Indigenous n=489	Significance of difference
Premature birth	1 (7%)	30 (35%)	p<0.05	0	0	–
Congenital abnormality	1 (7%)	13 (15%)	ns	0	2 (0.4%)	–
Asplenia	0	2 (2%)	–	2 (2%)	10 (2%)	ns
Immunocompromised	0	9 (11%)	–	10 (10%)	152 (31%)	p<0.0001
Chronic illness	4 (28%)	16 (19%)	ns	56 (56%)	300 (61%)	ns

ns Not significant

Table 7. Proportion of pneumococcal serotypes in cases of invasive pneumococcal disease covered by the 7-valent and 23-valent pneumococcal vaccines,* Australia, 2004, by state or territory

	State or territory								Total
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
7v	33/46	432/565	21/88	332/454	136/187	32/46	266/356	121/173	1,373/1,915
	72%	77%	24%	73%	73%	70%	75%	70%	72%
23v	40/46	529/565	60/88	420/454	167/187	41/46	334/356	159/173	1,750/1,915
	87%	94%	68%	93%	89%	89%	84%	92%	91%

* As a proportion of serotyped isolates.

The distributions of serotypes in cases aged less than 5 years and 65 years or more, in 2004, are shown in Figure 5. Eighty-four per cent of isolates from cases of IPD aged less than 5 years were serotypes in the 7vPCV and 93 per cent were serotypes in the 23vPCV. Ninety per cent of isolates from cases of IPD aged 65 years or more were serotypes in the 23vPPV.

Figure 5a. Serotypes responsible for invasive pneumococcal disease in cases aged less than 5 years, Australia, 2004

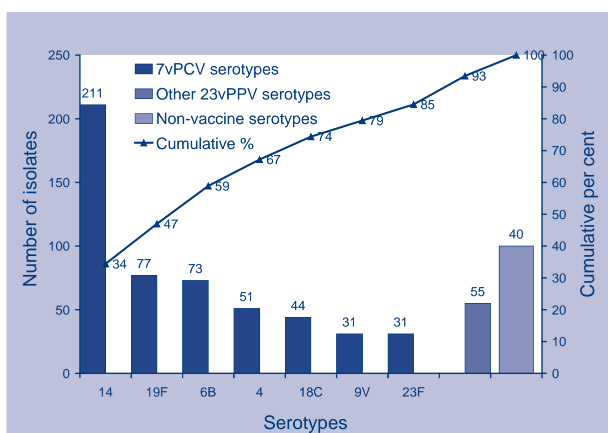
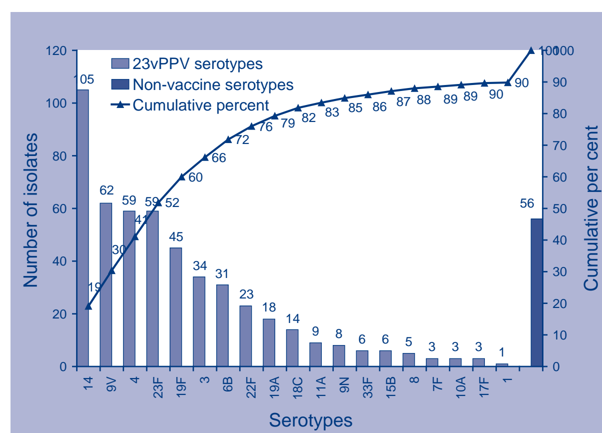


Figure 5b. Serotypes responsible for invasive pneumococcal disease in cases aged 65 years or more, Australia, 2004



The proportion of 7vPCV serotypes was significantly lower in Indigenous (18%) than in non-Indigenous (74%, $p<0.0001$) children, aged less than 2 years. Similarly, the proportion of 23-valent polysaccharide vaccine serotypes in Indigenous cases was significantly lower (65%) than in non-Indigenous cases (73%, $p<0.05$) aged 2 years and above (Table 8).

Trends in the number of 7vPCV and non-7vPCV serotypes in Indigenous and non-Indigenous cases aged under 2 years between 2002 and 2004 are shown in Figures 6a and 6b. There was a decline in the proportion of 7vPCV serotypes in Indigenous children (from 38% in 2002 to 18% in 2004) while the proportion of 7vPCV serotypes remained stable in non-Indigenous children.

The change in the rates of IPD in Indigenous children aged less than 2 years due to 7vPCV and non-7vPCV serotypes between 2001–02 and 2003–04 is shown in Table 9. Rates of disease caused by 7vPCV serotypes fell significantly (74.2%) while the increase (11.6%) in disease caused by non-7vPCV was not significant.

Vaccination status of invasive pneumococcal disease cases

Data on vaccination status was available for 1,517/2,375 (64%) cases in 2004. Of the 1,517 cases with a vaccination history, the majority (1,107, 73%) were reported as unvaccinated. IPD was reported in 15 cases who had been fully vaccinated with the 7vPCV and in 158 cases aged more than 15 years who had been fully vaccinated with the 23vPPV.

Further investigation of the 15 cases of IPD fully vaccinated with the 7vPCV showed that only three cases had evidence of vaccine failure. The three apparent vaccine failures had all received three doses of the 7vPCV, had disease caused by a 7vPCV serotype and no pre-disposing risk factors for IPD. Two of the three cases were Indigenous children.

Of the 158 cases of IPD fully vaccinated with the 23vPPV, 133 had disease caused by serotypes in the 23vPPV. These vaccine failures occurred in 69 males and 64 females aged between 17 and 95 years. Nineteen were Indigenous adults and 114 were non-Indigenous adults and all jurisdictions except the Australian Capital Territory reported vaccine failures. Of the 133 vaccine failures, 97 had predisposing risk factors for pneumococcal disease recorded.

Figure 6a. Number of 7-valent and non-7-valent vaccine serotypes causing cases of invasive pneumococcal disease in Indigenous children aged less than 2 years, 2002 to 2004

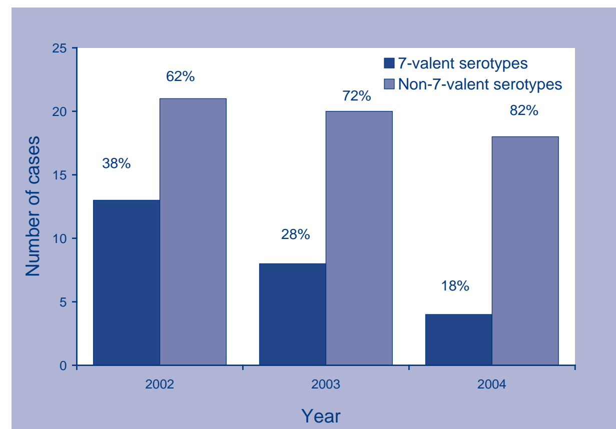


Figure 6b. Number of 7-valent and non-7-valent vaccine serotypes causing cases of invasive pneumococcal disease in non-Indigenous children aged less than 2 years, 2002 to 2004

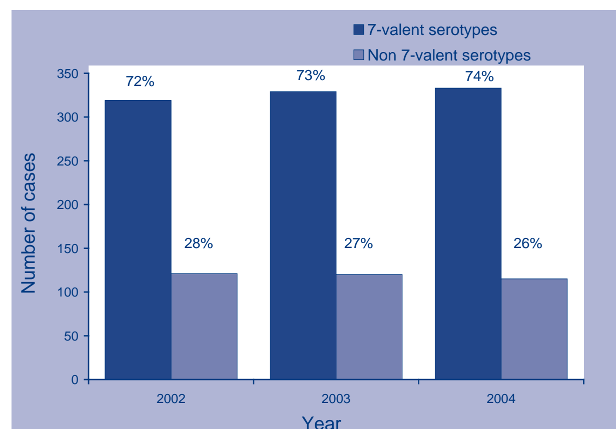


Table 8. The proportion of pneumococcal serotypes isolated from cases of invasive pneumococcal disease, which were serotypes in the 7-valent and 23-valent pneumococcal vaccines, Australia, 2004, by age and Indigenous status

	Cases aged less than 2 years with serotypes in 7-valent conjugate vaccine					Cases aged 2 years or more with serotypes in 23-valent vaccine				
	Indigenous		Non-Indigenous		Significance of difference	Indigenous		Non-Indigenous		Significance of difference
	n	%	n	%		n	%	n	%	
Total	4/22	18	333/448	74	p<0.0001	98/151	65	1,275/ 1,754	73	p=0.05

Table 9. Changes in estimated rates of invasive pneumococcal disease in Indigenous children under 2 years of age, 2001–02 and 2003–04, by serotype

Serotype	2001–02		2003–04		% change in rate	P value*
	Number of cases n=74	Rate per 100,000	Number of cases n=46	Rate per 100,000		
7vPCV serotypes	45	192.4	12	49.6	– 74.2	p<0.001
4	5	21.4	1	4.1	– 80.7	
14	14	59.9	2	8.3	– 86.2	
18C	3	12.8	1	4.1	– 67.8	
19F	4	17.1	6	24.8	+44.9	
23F	4	17.1	0	0.0	–100.0	
6B	11	47.0	2	8.3	– 82.4	
9V	4	17.1	0	0.0	– 100.0	
Non-vaccine serotypes	29	124.0	34	140.4	+11.6	NS

* Significance of difference in proportions between two time periods tested by Chi-square test.

NS Not significant.

Table 10. *Streptococcus pneumoniae* susceptibility to penicillin and ceftriaxone/cefotaxime, Australia, 2004, by state or territory*

Antibiotic	Description	State or territory							Total
		NSW	NT	Qld	SA	Tas	Vic†	WA	
Penicillin	Penicillin resistant	39	0	32	2	0	2	7	82
	Penicillin intermediate	45	10	43	19	2	27	22	168
	Penicillin susceptible	483	82	366	157	53	310	152	1,603
	Total tested	567	92	441	178	55	339	181	1,853
	% reduced susceptibility	14.8	10.9	17.0	11.8	3.6	8.6	16.0	13.5
Ceftriaxone	Ceftriaxone/cefotaxime resistant	NT	0	10	1	0	0	0	11
	Ceftriaxone/cefotaxime Intermediate	NT	3	15	2	0	9	2	31
	Ceftriaxone/cefotaxime susceptible	NT	71	416	56	55	305	179	1,082
	Total tested	NT	74	441	59	55	314	181	1,124
	% reduced susceptibility		4.1	5.7	5.1	0.0	2.9	1.1	3.7

* No data available from the Australian Capital Territory.

† Data from Victorian Hospitals Pathogen Surveillance System participating laboratories.

NT Not tested.

Antibiotic resistance in pneumococcal cases

The penicillin susceptibility was tested in 1,853 isolates and ceftriaxone/cefotaxime susceptibility was tested in 1,124 isolates (Table 10).

A total of 250 (13.5%) tested isolates had reduced susceptibility to penicillin which was an increase on the number and rate of isolates with reduced penicillin susceptibility in 2003 (142 isolates, 11.9%). Forty-two isolates (3.7%) had reduced susceptibility to ceftriaxone/cefotaxime in 2004, which was an increase in the number and rate compared to 2003 (9 isolates, 1.3%).

The serotypes of isolates with reduced penicillin susceptibility were examined (Table 11). Of the 250 isolates, 216 were serotyped. One hundred and eighty

Table 11. Proportions of pneumococcal isolates with reduced penicillin susceptibility, Australia, 2004, by age group and serotype

Serotype	Total	Children aged less than 5 years		Adults aged 65 years and over		Significance of difference
		n	%	n	%	
14	34	21	24.4	5	7.0	p<0.0001
11A	1	0	0.0	0	0.0	
19A	23	11	12.8	6	8.5	p<0.01
19F	45	17	19.8	13	18.3	ns
22F	1	0	0.0	1	1.4	
23F	5	1	1.2	2	2.8	
6A	2	1	1.2	1	1.4	
6B	19	14	16.3	4	5.6	ns
9V	77	17	19.8	35	49.3	p<0.005
Not typed	9	4	4.7	4	5.6	
Total	216	86	100.0	71	100.0	

ns Not significant

(83%) isolates with reduced penicillin susceptibility were serotypes in the 7vPCV and 205 (95%) were serotypes in the 23vPPV. There was no significant difference in the rate of penicillin resistance between children aged less than 5 years and adults aged 65 years or more.

When the prevalence of serotypes with reduced penicillin susceptibility was examined by age group, differences were noted between children aged less than 5 years and adults aged 65 years and above. There were a significantly higher proportion of penicillin insensitive serotypes 14 and 19A in children compared with adults and a higher proportion of penicillin insensitive serotype 9V in adults compared with children (Table 11). This pattern of penicillin resistant serotypes was different from that seen in 2003[5] when the proportions of penicillin resistant serotypes 19F and 14 were higher in older adults than young children.

Discussion

In 2004, IPD continued to have a significant impact on the health of young and old Australians. Serotypes causing IPD in 2004 were predominately vaccine serotypes in the 7vPCV in children aged less than 5 years and in the 23vPPV in the 65 years and older age group. All children under two years of age and all adults aged 65 years and older have been offered free vaccination with pneumococcal vaccines from January 2005.

This report details the impact of the Indigenous 7vPCV vaccine program in reducing the disease burden of IPD among Indigenous children. Rates of IPD in Indigenous children in the 1990s were among the highest recorded in the world. In 2004, the rates in Indigenous children aged less than 2 years had fallen to that of their non-Indigenous peers. IPD disease caused by 7vPCV serotypes in these Indigenous children under 2 years fell by 74 per cent in the period 2001–02 to 2003–04 with no significant increase in disease caused by non 7vPCV serotypes.

Despite the availability of the 23vPPV for Indigenous adults, through the National Indigenous Pneumococcal and Influenza Immunisation program, reductions in IPD in Indigenous adults have not been seen. A recent (2004) study estimated that the vaccine coverage was 25 per cent.[6] Although Indigenous adults were more likely to have disease caused by non-23vPCV serotypes than their non-Indigenous peers, two-thirds of cases reported in Indigenous adults in 2004 would have been potentially preventable by 23vPPV vaccination.

Reduced susceptibility to both penicillin and ceftriaxone/cefotaxime was evident in isolates from all age groups and jurisdictions in 2004. There was further evidence of specific penicillin resistant serotypes circulating among children and older adults. However the great majority of non-susceptible strains were 7vPCV serotypes and a significant reduction in the prevalence of antibiotic resistant IPD can be expected with the implementation of universal 7vPCV vaccination from 2005.[7]

In the USA the impact of the 7vPCV vaccine on IPD has recently been assessed.[8] Since the licensure of the vaccine in 2000, a reduction in the incidence of IPD in the vaccinated age groups has continued. In addition, it has been estimated that the vaccine has prevented twice as many cases indirectly through reductions in pneumococcal transmission

via increased herd immunity. Although increases in disease caused by non-7vPCV serotypes have been seen, these have been small relative to the declines in 7vPCV serotype disease. It has been recently estimated that the universal 7vPCV will prevent more than 80 per cent of childhood IPD and associated mortality in Australia. 7vPCV may also prevent 6 per cent of all pneumonia, 18 per cent of radiographically-defined pneumonia, 6 per cent of otitis media and 20–40 per cent of tympanostomy procedures in children under 5 years.[9] A reduction of 80 per cent may be a slight over-estimation, since IPD due to 7vPCV serotypes has accounted for only 72–74 per cent of disease in children aged under 2 years in recent years; nevertheless a significant reduction is anticipated. An analysis of the impact of the first year of the universal 7vPCV vaccination program on IPD in Australia will be provided in the next report.

Recent studies have revealed high-risk groups for IPD who could benefit from vaccination. A case control study in the USA identified asthma in persons aged 2–49 years as an independent risk factor for IPD.[10] Another USA study estimated the increased risk of IPD for specific chronic diseases, controlling for age and race.[11] Relative risks (compared to healthy adults) were 5.8 for diabetes, 6.9 for chronic lung disease, 10.4 for chronic heart disease, 11.5 for alcohol abuse, 32.2 for solid cancer, 48.8 for HIV/AIDS and 52.2 for haematological cancers. These observations support the recommendations in the *Australian Immunisation Handbook* that such high-risk groups receive the 23vPPV.[12]

The changing epidemiology of IPD in the era of pneumococcal conjugate vaccines is the subject of continuing research. Changes in serotypes causing IPD (‘serotype replacement’) are being measured through on-going laboratory surveillance. Despite increased prevalence of non-7vPCV serotypes in Indigenous children between 2001 and 2005, the overall rate of IPD continues to decline. Some concern has been raised about non-7vPCV serotypes causing unusual or severe presentations of IPD such as para-pneumonic empyema.[13] However, a recent review of apparent epidemiological differences between serotypes concluded that 7vPCV serotypes are the most prevalent in children aged 6 months to 2 years and in the immunocompromised and elderly adults. Continued epidemiological surveillance is required to determine whether increases in the prevalence of some non-vaccine serotypes are more significant than others.[14]

The use of 7vPCV in Indigenous children in Australia over the past three years has successfully reduced the rate of IPD to that of non-Indigenous children. There is, to date, no evidence of significant non-7vPCV serotype ‘replacement’ disease. Rates of pneumococcal resistance to penicillin are modest and resistance to ceftriaxone/cefotaxime remains rare. The introduction of the 7vPCV to the universal vaccination schedule in Australia in 2005 will further lower the disease burden of IPD among children and may contribute to reduction in other age groups. Continued enhanced IPD surveillance will be critical to assessing the impact of the expanding pneumococcal vaccine strategies.

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Communicable Diseases Unit, Brisbane

South Australia

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Northern Hospital (Epping) Pathology Service

St John of God Health Care Ballarat Pathology Service

Geelong Hospital Pathology Service (Pathcare)

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Tuberculosis notifications in Australia, 2004

Paul W Roche[1] and the National Tuberculosis Advisory Committee (Ral Antic – Chair, Ivan Bastian, Lynne Brown, Amanda Christensen, Mark Hurwitz, Anastasios Konstantinos, Vicki L Krause, Moira McKinnon, Avner Misrachi, Justin Waring) for the Communicable Diseases Network Australia

Abstract

The National Notifiable Disease Surveillance System received 1,076 tuberculosis (TB) notifications in 2004, of which 1,043 were new cases and 33 were relapses. The incidence of TB in Australia has remained at a stable rate since 1985 and was 5.4 cases per 100,000 population in 2004. The high-incidence groups remain people born overseas and Indigenous Australians at 21.7 and 8.1 cases per 100,000 population, respectively. By contrast, the incidence of TB in the non-Indigenous Australian-born population was 1.0 cases per 100,000 population. Comparison of the 2004 TB notification data against the performance indicators set by National Tuberculosis Advisory Committee highlights that enhanced TB control measures should be considered among these high-risk groups. *Commun Dis Intell* 2006;30:93–101.

Keywords: disease surveillance, tuberculosis

Introduction

Australia has one of the lowest incidence rates of tuberculosis (TB) in the world with rates remaining stable at 5–6 cases per 100,000 population since the mid–1980s.[1] By contrast, approximately 60 per cent of the 8.8 million TB cases occurring globally in 2003 live in Australia's neighbouring countries in South East Asia and the Western Pacific.[2] Australia's migrant intake includes people from countries with high prevalence of TB and those born overseas have accounted for a large proportion of Australia's TB cases over the last decade.

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 2004. The data are compared against the National Tuberculosis Performance Indicators set by the National TB Advisory Committee in the *National Strategic Plan for TB Control in Australia beyond 2000.*[3] Information about drug susceptibility is published by the Australian Mycobacterium Laboratory Reference Network in an accompanying report.

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 2004 was collated by jurisdictions and sent electronically to 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 the Communicable Diseases Network Australia, was responsible for determining the data set collected in 2004 and for its transmission to NNDSS. Data fields in the enhanced TB data set that are analysed in this report are listed in Table 1 with a brief description of each variable.

Data processing and quality control

Data on all TB notifications reported in 2004 were received by September 2005. Updated information on the outcomes of treatment of patients notified in 2003 was received by December 2005. 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.

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Table 1. Description of some of the data fields in the enhanced tuberculosis data set of the National Notifiable Diseases Surveillance System*

Data field	Description
Country of birth	Country in which the notified case was born.
Extrapulmonary site	Details of any extrapulmonary site involved.
New or relapse case	Options include: new case (without known previous treatment); relapse of disease following full treatment in Australia; relapse of disease following partial treatment in Australia; relapse of disease following full treatment overseas; relapse of disease following partial treatment overseas.
TB outcomes	Options include: cured (bacteriologically confirmed); completed treatment; 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); transferred out of Australia during treatment phase.
Age	Age of notified case at diagnosis.
Indigenous status	Whether notified case is self-identified Indigenous (Aboriginal and/or Torres Strait Islander) Australian or not.
Selected risk factors	Options include: close contact with a TB patient; currently/recently residing in a correctional facility; currently/recently residing in an aged care facility; currently/previously employed in an institution; currently/previously employed in the health industry; HIV status (positive or negative); past residence (3 months or more) in a high risk country.

* Other data collected on each case included diagnosis details, therapy and susceptibility. These were analysed in the accompanying TB laboratory report pp. 102–108.

Most 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 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.

Population estimates for 2004

The rates presented in this report were calculated using population data produced by the Australian Bureau of Statistics (ABS). The estimated resident population as at 30 June 2004, 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 ABS calculated the projections based on assumptions about future births, deaths and migrations in the Indigenous population and a 'low' and 'high' estimate were provided. For the purpose of this report, the 'low' estimate has been used, which is consistent with previous annual reports for TB notifications in Australia.

The 2001 census data were used to calculate incidence rates of TB in people born overseas. The estimated resident population of overseas-born people (total and by country of birth) in 2001 was used as the denominator in calculating rates, with additional data on recent arrivals from some countries supplied by the Australian Government Department of Immigration and Multicultural and Indigenous Affairs.

To estimate the non-Indigenous Australian-born population, the Indigenous population estimate and the overseas-born population estimate were subtracted from the total Australian population. Since some of the TB notifications in the report may include non-permanent residents of Australia in 2004, the rates may be overestimated.

Results

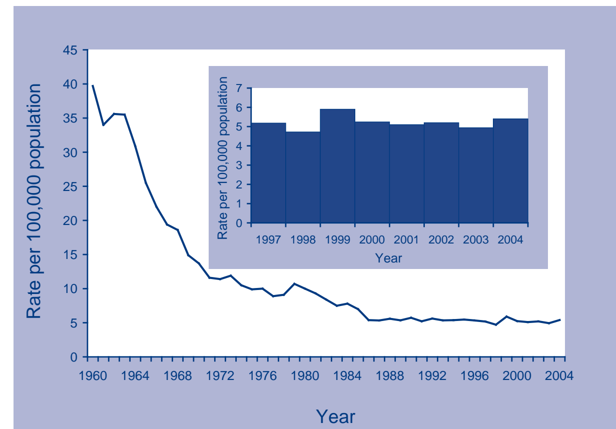
Data quality

The majority of data fields were well reported. Information on age (1,075/1,076) and sex (1,074/1,076) for all notifications were almost complete. Indigenous status was reported for 187 of the 191 (98%) people born in Australia and country of birth was recorded for 1,063 (98.7%) of the total TB notifications. The site(s) of TB disease was reported for 1,052 (97.7%) cases and whether the case was new or relapse was also reported for 1,075 (99.9%) cases. HIV status was not well reported (34%).

Tuberculosis notification rates

The total number of cases reported across Australia in 2004 was 1,076 (5.4 cases per 100,000 population). This is higher than in 2003 (982 cases, 4.9 cases per 100,000 population), and similar to the rate in 2002 (5.2 cases per 100,000 population, Figure 1).

Figure 1. Incidence rates for tuberculosis notifications, Australia, 1960 to 2004



Tuberculosis notifications by jurisdiction

New South Wales reported the largest number of TB cases (431) however the highest rate was recorded in the Northern Territory (14 cases per 100,000 population, Table 2). Of the 33 relapsed cases, 11 were identified following full treatment in Australia, one following partial treatment in Australia, 12 following full treatment overseas and 9 following partial treatment overseas.

Figure 2 presents the TB notifications rates by jurisdiction for 2002 to 2004. 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, 2002 to 2004, by state or territory

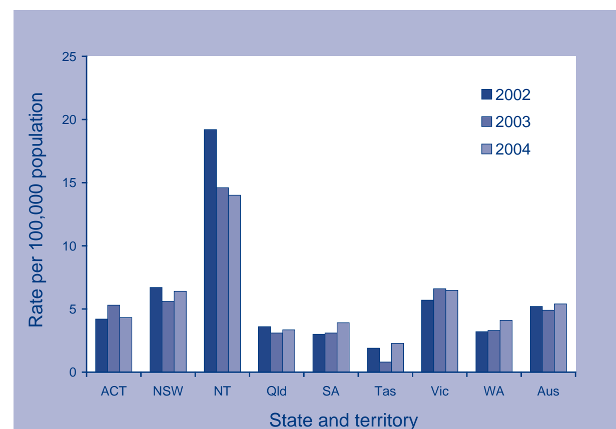


Table 2. New and relapsed cases and rates per 100,000 population, Australia, 2004, by state or territory

	New cases	New cases rate	Relapse cases	Relapse case rate	Total notifications	Total rate
ACT	14	4.3	0	0.0	14	4.3
NSW	412	6.1	19	0.3	431	6.4
NT	27	13.5	1	0.5	28	14.0
Qld	122	3.1	7	0.2	129	3.3
SA	60	3.9	0	0.0	60	3.9
Tas*	10	2.1	0	0.0	11	2.3
Vic	317	6.4	5	0.1	322	6.5
WA	80	4.0	1	0.1	81	4.1
Australia	1,042	5.2	33	0.2	1,076	5.4

* One case from Tasmania was unknown relapse/new status.

Tuberculosis notifications in the Australian-born population

In 2004, 191 (17.7%) cases of TB occurred in the Australian-born population, of whom 152 (79%) were non-Indigenous, and 39 (21%) were Indigenous Australians (Table 3).

The TB incidence rate in the non-Indigenous Australian-born population (1.0 cases per 100,000 population) has remained stable over the past 13 years. The incidence of TB in Indigenous Australians for 2004 was 8.1 cases per 100,000 population, the lowest rate reported for this population since 1991. However, the TB incidence was still 8 times the rate in non-Indigenous Australian-born people. Nineteen of the 39 cases in Indigenous Australians were reported from the Northern Territory, a jurisdiction where 28 per cent of the population are Indigenous Australians as compared to 2 per cent nation wide.

Tuberculosis notifications in the overseas-born population

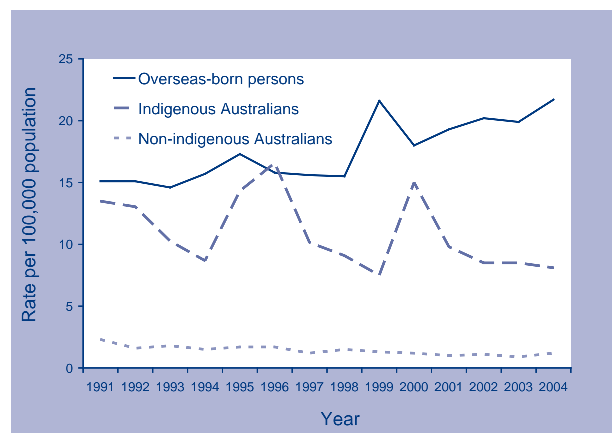
In 2004, 885 cases (82.3%) were overseas-born. The rate of notification, 21.7 cases per 100,000 population was similar to rates in this population in the previous two years (19.1 and 20.2 cases per 100,000 population in 2003 and 2002 respectively, Figure 3).

Amongst people born overseas in the Australian population, the largest number of cases was in those born in India, Viet Nam, 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 estimated resident population.

Table 3. Tuberculosis notifications and incidence rates in all Australian-born persons, Australia, 2004, by state or territory

	Indigenous	Indigenous rate	Non Indigenous Australian born	Non-Indigenous rate	Total Australian born	Total rate
ACT	0	0.0	2	0.8	2	0.8
NSW	7	4.9	54	1.1	61	1.2
NT	19	31.9	0	0.0	19	11.1
Qld	9	6.7	31	1.0	40	1.2
SA	2	7.4	10	0.8	12	1.0
Tas	0	0.0	4	1.0	4	0.9
Vic	0	0.0	40	1.0	40	1.0
WA	2	2.9	11	0.8	13	0.9
Australia	39	8.1	152	1.0	191	1.2

Figure 3. Tuberculosis incidence rates by Indigenous status and country of birth, Australia, 1991 to 2004



Tuberculosis notifications by age and sex

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 38 children aged less than 15 years. These were 15 Australian-born non-Indigenous children and 23 children born overseas. There were no Indigenous children reported with TB in 2004. The overall notification rate for the less than 15 year age group was 0.9 cases per 100,000 population (target of less than 0.1 cases per 100,000 population for all groups). The rate was highest in overseas-born children (11.4 cases per 100,000 population) and remained low in the non Indigenous Australian-born children (0.4 cases per 100,000 population, Table 5).

Table 4. Notifications of tuberculosis and estimated rate per 100,000 population for selected countries of birth, Australia, 2004

Country of birth	New	Relapse	Total cases	ERP x COB 2001*	Rate per 100,000 population in Australia 2004	WHO incidence rate per 100,000 2003†
India	123	0	123	95,455	128.9	101
Viet Nam	112	2	114	154,833	73.6	114
Philippines	73	0	73	103,942	70.2	168
China‡	67	1	68	142,778	47.6	47
Sudan	37	0	36	17,133	210.1	NA
Indonesia	26	2	28	47,156	59.4	81
Hong Kong (SAR of China)	26	0	26	67,121	38.7	80
Somalia	26	0	26	4,285	606.8	NA
Papua New Guinea	20	2	22	23,618	93.1	322
Ethiopia	18	0	18	5,777	311.6	166
Malaysia	16	0	16	78,858	20.3	64
Thailand	15	0	15	23,599	63.6	87
England	14	0	14	847,365	1.7	2
New Zealand	14	0	14	355,765	3.9	3
Others	270	22	292	2,135,821	13.7	
Overseas	856	29	885	4,087,928	21.7	
Australia§	186	4	190	16,023,372	1.2	
Total	1,042	33	1,076	20,111,300	5.4	

* Country of birth for denominator is the estimated resident population (ERP) from the 2001 census. ERP totals were updated for Somalia, Sudan and Ethiopia with data from the Australian Government Department of Immigration and Multicultural and Indigenous Affairs as of November 2005 because of recognised shift of intake from these countries.

† Rates from the World Health Organization 2005 Global Tuberculosis Report.

‡ China excludes Hong Kong SAR and Taiwan.

§ For one Australian-born case the new/relapse status was unknown.

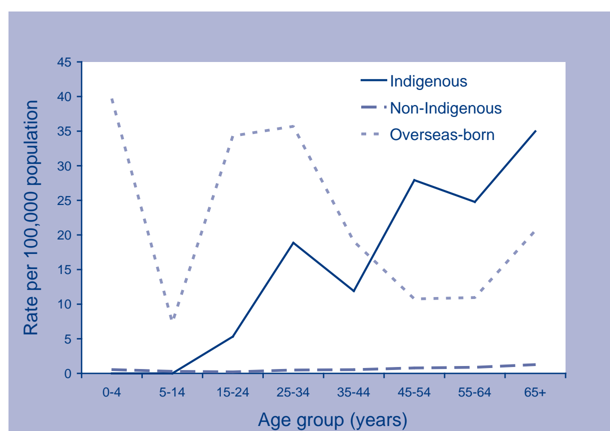
Table 5. Tuberculosis notifications and estimated incidence rate, Australia, 2004, by age group, Indigenous status and country of birth

Age group	Indigenous		Non-Indigenous		Overseas-born	
	n	Rate	n	Rate	n	Rate
0–4	0	0	7	0.6	10	39.7
5–14	0	0	8	0.4	13	7.3
Subtotal - < 15 years	0	0	15	0.4	23	11.4
15–24	4	5.3	6	0.2	135	34.3
25–34	12	18.9	14	0.5	216	35.7
35–44	6	11.9	16	0.5	165	19.1
45–54	9	27.9	21	0.8	94	10.8
55–64	4	24.8	17	0.9	76	11.0
65+	4	35.0	63	1.3	175	20.6

The male to female ratio for TB notifications was 1.6:1 in non-Indigenous Australian-born TB cases, 1.4:1 in Indigenous cases and 1.2:1 in overseas-born cases.

The age-group incidence rates for TB in overseas-born, Indigenous Australian-born and non-Indigenous Australian-born populations are shown in Figure 4. The TB incidence in the overseas-born population showed two peaks: one among infants aged less than 5 years and a second among young adults (15 to 34 years). In the non-Indigenous Australian-born there was a small but gradual increase in TB rates up to 65 years. TB rates among Indigenous Australians also showed an increase in rates with increasing age.

Figure 4. Tuberculosis incidence in Australian-born and overseas-born, 2004, by age



Tuberculosis and selected risk factors

Information on risk factors for TB disease excluding HIV were reported for 1,020 of the 1,076 cases. Where risk factors were reported, the majority (520 cases) identified as having previously resided for three or more months in high risk countries as defined by the Australian Government Department of Immigration and Multicultural and Indigenous Affairs. Among these 520 cases, 25 were Australian-born and 495 were overseas-born. An additional 105 cases were household members or close contacts of TB cases, six cases either resided or had recently resided in a correctional service and 13 cases either resided or recently resided in an aged care facility. For individuals working in high risk settings, three cases were employed or had been recently employed in institutions such as correctional facilities or aged care facilities and 23 cases were employed or had been recently employed in the health industry. Among these 23 cases, three were Australian-born and 20 were overseas-born.

Tuberculosis and HIV status

Information on HIV status was reported in 352 (34%) cases. Sixteen people were identified with HIV infection at the time of diagnosis with TB; 5 Australian-born and 11 overseas-born. The National Strategic Plan recommends that HIV status of all TB cases be reported. In 2004, reporting of HIV status was equivalent to that in 2003.

Anatomical site of disease

The anatomical site of tuberculosis infection was recorded in 1,052 cases. Of these 548 (52%) cases of notified cases had pulmonary disease only, a further 92 (8.7%) cases had pulmonary disease along with disease at an extrapulmonary site. Pulmonary TB was reported in 65 per cent of the Australian-born cases and 57 per cent of the overseas-born cases.

412 (39%) cases had extrapulmonary disease only. The sites of disease in new and relapse cases are shown in Table 6.

Treatment outcomes of 2003 tuberculosis patient cohort

Treatment outcomes were reported for 962 of the 982 TB notifications in 2003 (98%) by December 2005. Treatment success including those with bacteriologically confirmed cure and those who completed treatment without bacteriological evidence of cure were reported for 797 (95%) of 838 with assessable outcomes (Table 7). There was no treatment failure recorded. Eighteen (2.2%) cases were reported as defaulting treatment. The proportion of cases cured or who completed treatment were 97 per cent among Indigenous Australians, 97 per cent among non-Indigenous Australian born and 96 per cent among overseas born.

Death from TB is rare in Australia. In the 2003 patient cohort there were 12 deaths due to TB reported and the case fatality rate was 1.2 per cent. A number of these cases were identified on post-mortem. The following treatment outcomes were excluded from analysis: deaths from other causes (39), cases transferred out of Australia (62), and cases still undergoing treatment at the time of reporting (23).

Table 6. New and relapsed tuberculosis cases, Australia, 2004, by site of disease

Site	New cases	Relapse cases	Total case	Per cent of cases
Pulmonary only	527	21	548	52.1
Pulmonary plus other sites	91	1	92	8.7
Extrapulmonary only	405	7	412	39.2
Pleural	81	2	83	7.9
Lymph nodes	166	3	169	16.1
Bone/joint	56		56	5.3
Genito/urinary	31		31	2.9
Miliary	14		14	1.3
Meningeal	18		18	1.7
Peritoneal	5	1	6	0.6
Other	39		39	3.7

Table 7. TB treatment outcomes, Australia, 2003, by population group

Treatment outcomes	Indigenous	Non-Indigenous Australian-born	Overseas-born	All	Per cent of cases
Treatment success	34	94	669	797	95.1
– Cured*	22	14	46	82	9.8
– Completed treatment	12	80	623	715	85.3
Died of TB	1	7	4	12	1.4
Interrupted treatment†	0	0	2	2	0.2
Defaulted‡	1	2	15	18	2.1
Failed§	0	0	0	0	0
Missing	0	1	8	9	1.1
Total	36	104	698	838	100.0

* 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.

Note: The following treatment outcomes were excluded from analysis: deaths from other causes (39), cases transferred out of Australia (62) and cases still undergoing treatment at the time of reporting (23).

Table 8. National tuberculosis performance indicators, performance criteria and the current status of tuberculosis in Australia, 2003 and 2004

National TB Performance Indicator	Performance criteria	2003	2004
Annual incidence of TB (per 100,000 population)			
Crude incidence			
Indigenous Australians	<1	8.7	8.1
Non-Indigenous Australian-born	<1	0.9	1.2
Overseas-born persons*	†	10.2	10.4
Relapse cases initially treated in Australia	<2% of total treated cases	1.1	1.0
Incidence in children <15 years, by risk group			
Indigenous Australian children	<0.1	5.6	0
Non-Indigenous Australian-born children	<0.1	0.4	0.4
Overseas-born children*	†	0.0	0.0
Collection of HIV status in TB cases (% of cases with data collected)	100% over next 3 years	32.2	34
Treatment outcome measures (%)			
Cases evaluated for outcomes‡	100	98.0	NA
Cases that have treatment completed and are cured	>90	95.1	NA
Cases recorded as treatment failures	<2	0.0	NA

* The performance criteria for overseas born are applied to people who have been living in Australia for more than 5 years.

† Performance criteria currently under review.

‡ Evaluation of outcomes of 2003 patient cohort re-assessed in December 2005.

NA Assessment of outcomes of 2004 patient cohort – not available.

National Performance Indicators

The performance criteria for the National Performance Indicators were set by the National Tuberculosis Advisory Committee in 2002 and reviewed in 2003 (Table 8). As in previous TB annual reports, the performance criteria for people born overseas applies to people who have been living in Australia for more than 5 years. Of the 886 cases born overseas, 426 (48%) had been living in Australia for more than five years. The TB incidence rate for people born overseas who have been living in Australia for more than 5 years was 10.4 cases per 100,000 population.

There were no cases of TB in Indigenous children aged less than 15 years in 2004. This compares with nine cases (5.6 cases per 100,000) in 2003 and seven cases (4.3 cases per 100,000 population) in 2002.

Discussion

The incidence of TB in Australia has remained between five and six cases per 100,000 population since the mid-1980s, and represents one of the lowest incidence rates in the world. [2] Tuberculosis control in low-incidence countries faces specific problems and challenges, such as the reduced awareness of TB among healthcare professionals, the increasing importance of imported TB among migrants, and sub-groups at high risk of TB such as Indigenous Australians.

Doctors and other healthcare professionals in Australia must maintain an index of suspicion for TB, particularly when caring for migrants, Indigenous Australians, and elderly non-Indigenous Australian-born patients. This depends on adequate undergraduate and postgraduate training in TB epidemiology, diagnosis, management and control measures for doctors, nurses, laboratory staff and migrant health workers.

The overseas-born population represented 82 per cent of new TB cases in 2004. People born in countries with a high incidence of TB are likely to have acquired latent infection prior to migration. Refugees who have been living in camps where overcrowding, poor sanitation and malnutrition are at increased risk of progressing to active disease. Resettlement conditions which are socioeconomically stressful to migrants, may contribute to the progression of latent TB to active TB. Social contact with other migrants from high incidence countries may also increase the risk of exposure to TB.

Australian TB services continue to support pre-migration screening for active TB and to participate in post-migration follow-up programs in coopera-

tion with the Australian Government Department of Immigration, Multicultural and Indigenous Affairs and other organisations. Migrants need ready access to cost-free, non-threatening and culturally-appropriate TB assessment and treatment. People from Sudan, Somalia and Ethiopia were reported as high-incidence sub-populations in Australia in 2004, reflecting changes in the composition of Australia's migrant intake. Tuberculosis clinics need to adapt to the specific cultural and social needs of these new patient populations. Community leaders in the new migrant populations must be identified and encouraged to assist with TB control efforts. These TB control measures have proved successful in other migrant populations in the past and are likely to succeed again. As Australia and other low-incidence countries move towards TB elimination, the overseas-born population will continue to account for a large proportion of incident cases. Additional measures, such as active case finding and increased detection and treatment of latent TB infection, should be considered in migrant populations with a high incidence of TB.

Indigenous Australians are at increased risk of TB with incidence rates 8 times higher than among non-Indigenous Australian-born people in 2004. This disparity has remained evident for the last decade despite the efforts of TB control programs. Some of the known risk factors that explain the high incidence of TB in the Indigenous Australians are socioeconomic disadvantage), co-morbidities (such as diabetes and renal diseases), smoking, alcohol abuse and poor nutrition.[4]

The comparison of the 2004 TB notification data against the National TB Performance Indicators demonstrates some progress toward achieving the goals of the *National Strategic Plan for TB Control*. The low incidence of TB among non-Indigenous Australian-born children (0.4 per 100,000 population) and absence of any reported cases among Indigenous Australian children are approaching the target incidence of less than 1 case per million. The success of TB treatment programs in Australia are reflected in the declining proportion of relapsed cases initially treated in Australia (1%), and the absence of any cases recorded as treatment failures (Table 8).

Further action is required to reduce the incidence of TB in Indigenous communities to the same low rate as the non-Indigenous Australian-born population. The reporting of HIV status for TB cases remains at an unacceptable low level (34% in 2004). Despite incomplete reporting, 16 cases of HIV/TB co-infection were recognised in Australia in 2004 of whom 11 were overseas-born. Australian migrant intake includes people who come from countries where HIV and TB are prevalent. Privacy laws in some states confound efforts to collect information on the HIV status of TB patients. Alternative acceptable strategies must be found to obtain this essential public health information.

In 2004, 23 TB cases occurred among health care workers of whom 20 were overseas-born. Health services in Australia are increasingly reliant upon attracting medical and nursing staff from overseas, including from countries where TB is prevalent. State TB services and staff induction programs should be aware of this trend and ensure that new employees are screened and followed-up appropriately for TB.

In conclusion, easy access to effective TB treatment programs, contact tracing, and provision of health education in appropriate languages remain the essential elements for TB control. Australia also needs to remain alert to the growing global threat of TB and to contribute to TB control efforts in Southeast Asia and the Pacific region.

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Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2004

A Report of the Australian Mycobacterium Reference Laboratory Network

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Abstract

The Australian Mycobacterium Reference Laboratory Network collected and analysed laboratory data on new cases of disease caused by *Mycobacterium tuberculosis* complex in the year 2004. A total of 787 cases were identified by bacteriology, representing an annual reporting rate of 3.9 cases per 100,000 population. Almost all isolates were identified as *M. tuberculosis* (n=785), the remaining isolates being one each of *Mycobacterium africanum* and *Mycobacterium canettii.* Seven children under 10 years of age (female n=5, male n=2) had bacteriologically confirmed tuberculosis (gastric aspirate n=4, lymph node n=1, pleural n=1, thigh wound n=1). Results of *in vitro* drug susceptibility testing were available for all 787 isolates for isoniazid (H), rifampicin (R), ethambutol (E), and pyrazinamide (Z). A total of 71 (9.0%) isolates of *M. tuberculosis* were resistant to at least one of these anti-tuberculosis agents. Resistance to at least both H and R (defined as multidrug resistance) was detected in 12 (1.5%) isolates; 10 were from the respiratory tract (sputum n= 7, bronchoscopy n= 3). The country of birth was known for 68/71 (95.8%) cases with a drug resistant strain; eight were Australian, 60 were overseas born, and three were unknown. Of the 60 migrants with drug resistant disease, 37 (61.7%) were from three countries; Viet Nam (n=20), China (n=9) and India (n=8). *Commun Dis Intell* 2006;30:102–108.

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

Introduction

Australia continues to have one of the lowest incidence rates of tuberculosis (TB) in the world. These rates have remained stable since the mid-1980s.[1] Tuberculosis remains a major health problem globally, and particularly in the World Health Organization regions of South East Asia and the Western Pacific.[2] In 2003, there were an estimated 9.7 million prevalent cases of TB, equivalent to 291 cases per 100,000 population, of which some 5 million were new cases. China and India accounted for 63 per cent of the incident cases for the two regions. Overall, the Western Pacific Region reported a notification rate of 57 cases per 100,000 population in 2003.

There are two sources of TB-related data for Australia. Since 1991, the National Notifiable Diseases Surveillance System (NNDSS) has provided statistics on cases of tuberculosis reported to public health authorities in Australia's states and territories. The second source, 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 will have a proportion of 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 2004.

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

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comprising the AMRLN for specific 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, were included in the laboratory data as laboratories are generally unable to differentiate relapse cases from new cases.[3] Temporary visitors to Australia were included as were illegal aliens within correctional services facilities and asylum seekers located in detention centres or on temporary visas within Australia.

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: species of mycobacterium and results of drug susceptibility testing;
- nucleic acid amplification testing: results of testing; and
- if the isolate was drug resistant: 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 scheme 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 2004 supplied by the Australian Bureau of Statistics.[4]

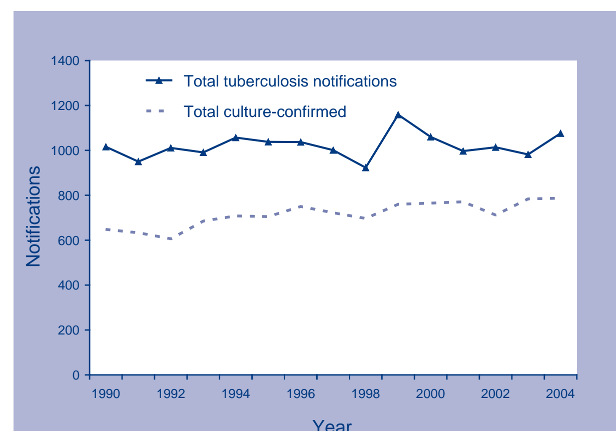
For each case, the nature of the first clinical specimen that yielded an isolate of MTBC was used to record the nominal site of disease. Culture-positive specimens collected at bronchoscopy or by gastric lavage were considered to indicate pulmonary disease. Cases with multi-site isolations, provided a sputum or bronchoscopy specimen was culture-positive, were listed as having pulmonary disease, the most important category for public health purposes. Cases for which there were multiple-site isolations were not categorised as having miliary or disseminated disease as differentiation is based on clinical findings that are generally not available to the reporting laboratories.

Drug resistance among new cases (as a proxy for primary resistance) was defined as the presence of resistant isolates of *M. tuberculosis* in patients, who in response to direct questioning, deny having had any prior anti-TB treatment (for as much as one month) and, in countries where adequate documentation is available, for whom there is no evidence of treatment. Drug resistance among previously treated cases (as a proxy for acquired resistance) was defined as the presence of resistant isolates of *M. tuberculosis* 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 no evidence of such a treatment.[5]

Results

There were 787 bacteriologically confirmed cases of tuberculosis in 2004 (Figure 1), representing an annual rate of 3.9 cases per 100,000 population. State-specific reporting rates ranged from 1.7 to 10.5 cases per 100,000 population in Tasmania and the Northern Territory respectively (Table 1).

Figure 1. Comparison between tuberculosis notifications and laboratory data, Australia, 1990 to 2004



Causative organism

Almost all isolates were identified as *M. tuberculosis* (n=785), the remaining isolates being one each of *Mycobacterium africanum* and *Mycobacterium canettii.* No isolates of *Mycobacterium bovis* were reported for 2004.

Distribution by gender, age and site of disease

Complete information for gender and age were submitted for 785/787 (99.8%) of all patients, due to additional information provided by state and territory tuberculosis centres. Of the 787 MTBC isolates, 369 (46.9%) were from females, 417 (53.0%) were from males, and gender was unknown for a single case. Seven children aged under 10 years (female n=5, male n=2) had bacteriologically confirmed tuberculosis (gastric aspirate n=4, lymph node n=1, pleural n=1, thigh wound n=1). The rela-

Table 1. Bacteriologically confirmed cases of tuberculosis in Australia, 1994 and 2002–2004, cases and rate per 100,000 population, by state or territory

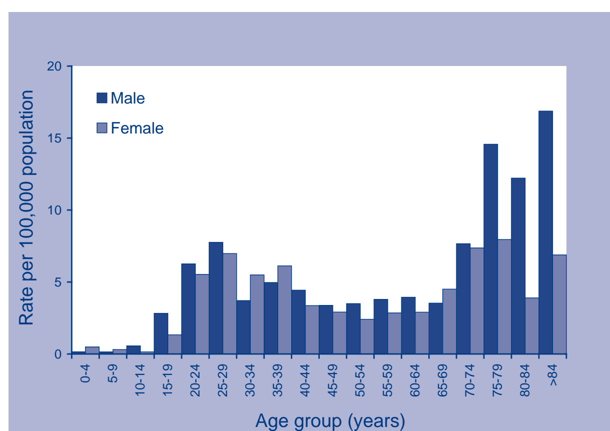
State or territory	2004		2003*		2002*		1994*	
	n	Rate	n	Rate	n	Rate	n	Rate
New South Wales†	308	4.4	325	4.6	301	4.3	278	4.4
Northern Territory	21	10.5	20	10.1	26	13.0	21	12.3
Queensland	88	2.3	91	2.4	97	2.6	88	2.8
South Australia	43	2.8	36	2.4	26	1.7	41	2.8
Tasmania	8	1.7	4	0.8	8	1.7	10	2.1
Victoria	262	5.3	254	5.2	208	4.3	217	4.8
Western Australia	57	2.9	54	2.8	46	2.4	53	3.1
Total	787	3.9	784	3.9	712	3.6	708	4.0

* 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.

tionship of tuberculosis to age and gender is shown in Figure 2. The site of disease was dependent upon age and gender. The overall male:female ratio was 1.1:1. For males, there were two distinct age groups: a rise to 7.8 cases of TB per 100,000 population at 25–29 years and in the elderly male greater than 75 years (>10 cases of TB per 100,000 population). The age groupings for females was similar with 7.0 and >8 TB cases per 100,000 population for the 25–29 and >70–74 year age ranges respectively. For respiratory isolates, the male:female ratio was 1.4:1. The median age group for patients with bacteriologically confirmed disease was 20–24 years for males and 25–29 years for females. For lymph tissue, the male:female ratio was 1:1.6. For males the median age was 25–29 years with 2.0 TB cases per 100,000 population; the remaining age groups were all less than 1.0 per 100,000 population. In contrast, the median age range for females was 35–39 years, peaking at 2.7 TB cases per 100,000 population and 2.1 TB cases per 100,000 population in the 25–29 year age group. The predominant culture-positive specimen type was sputum (n=366, 46.5%); a further 100 (12.7%) were obtained via bronchoscopy, and five were from tissue/biopsies. Thirty-seven pleural specimens (28 fluid, 9 biopsy/tissue) were culture-positive; only two fluids were smear positive.

Figure 2. Laboratory confirmation of *Mycobacterium tuberculosis* complex disease, Australia, 2004, by age and sex



The most commonly encountered extrapulmonary culture-positive specimen was lymph tissue (n=172, 21.9%) followed by peritoneal (n=25, 3.2%), bone/joint (n=22, 2.8%), and genitorurinary tract (n=17, 2.2%).

Association with HIV

The AMRLN database recorded the HIV status of only 62 (7.9%) patients. No patient was identified as HIV seropositive.

Microscopy

Results of microscopy were available for 773 of 787 (98.2%) specimens; microscopy was not performed on 12 specimens and no results were provided for the remaining two specimens. Smears were positive for 202 of 366 (55.2%) sputum and for 43 of 100 (43%) bronchoscopy specimens respectively (Table 2). A total of 37 pleural specimens (9 biopsy and 28 fluids) were culture positive for *M. tuberculosis* with two fluid specimens smear positive (5.4%) only. Lymph node specimens were smear positive for only 33 of 172 (19.2%) cases.

Drug susceptibility testing

Results of *in vitro* drug susceptibility testing were available for all 787 isolates for isoniazid (H), rifampicin (R), ethambutol (E), and pyrazinamide (Z). A total of 71 (9.0%) isolates of *M. tuberculosis* were resistant to at least one of the above anti-tuberculosis agents.

Table 2. Site of specimens smear- and culture-positive for *Mycobacterium tuberculosis* complex, 2004

	n	Smear positive (%)*
Sputum	366	202 (55.2)
Bronchoscopy	100	46 (46.0)
Lymph node	172	33 (19.2)
Pleural	37	2 (5.4)
Genito-urinary	17	ND
Bone/joint	22	ND
Peritoneal	25	ND
Skin	8	ND
Cerebrospinal fluid	5	ND

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

ND The percentage of specimens smear positive was not calculated due to small numbers.

Results of testing for streptomycin (S) were available for 221 (28.1%) of 787 isolates with nine demonstrating S mono-resistance and another 10 resistant to S + H. Resistance to at least both H and R (defined as multi-drug resistance) was detected in 12 (1.5%) isolates. All of the MDR isolates were *M. tuberculosis* (Table 3). Of the 12 MDRTB isolates, 10 were from the respiratory tract (sputum n=7, bronchoscopy n=3); the remaining isolates were from a neck abscess and a peritoneal biopsy. Four of the MDRTB-positive sputum specimens were smear positive, as was the neck abscess. None of the bronchoscopy specimens were smear positive.

Mono-resistance to isoniazid, rifampicin, ethambutol, and pyrazinamide was detected in 31, 2, 4, and 5 isolates, respectively. There were 53 isolates that demonstrated resistance to H at a concentration of 0.1 mg/L. Of these, 38 (71.7%) demonstrated resistance to H at the higher level of 0.4 mg/L. For MDRTB strains, 11/12 (91.6%) demonstrated resistance at the higher concentration. Thirty-seven of 73 (50.7%) specimens culture-positive for drug resistant *M. tuberculosis*, including 28 of 48 (58.3%) sputum or bronchoscopy specimens, were smear-positive for acid-fast bacteria.

New case or previously treated, and country of birth

There were 71 *M. tuberculosis* isolates resistant to at least one of the standard drugs (H, R, E, Z). Of these, 45/52 (86.5%) were classified as being new cases, seven were considered as previously treated, and no information was available for 19 cases. The country of birth was known for 68/71 (95.8%) cases; eight were Australian, 60 were overseas born, and three were unknown. Of the 60 migrants with drug resistant disease, 37 (61.7%) were from three countries; Viet Nam (n=20), China (n=9) and India (n=8). The 12 patients with MDR-TB were from China (n=3), India (n=2), Viet Nam (n=2) with a single case each from Australia, Eritrea, Pakistan, South Africa and the Philippines.

Use of nucleic acid amplification tests

Nucleic acid amplification testing (NAAT) was performed on 173 of 787 (22.0%) specimens, all of which subsequently grew MTBC on culture. Of these, 103 specimens were of respiratory origin (sputum n=77; bronchoscopy n=22; tissue n=3; swab n=1), and 100 (97.1%) were NAAT positive. For smear positive respiratory specimens, 81 of 82 (98.8%) were NAAT positive whilst 19 of 21 (90.5%) smear negative respiratory specimens were NAAT positive.

Table 3. Drug resistance patterns in multi-drug resistant strains of *Mycobacterium tuberculosis* complex strains, Australia, 1994 to 2004

Resistance pattern (standard drugs)*	2004	2003	2002	2001	2000	1999	1998	1997	1996	1995	1994
H+R only	7	4	8	8	3	2	2	6	10	3	2
H+R+E	2	2	1	1	1	1	1	1	1	1	0
H+R+Z	1	1	1	3	3	1	2	5	4	1	0
H+R+E+Z	2	0	2	0	1	0	1	2	0	0	0
Total (%)	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)	2 (0.3)

* The streptomycin result was not considered for this table.

H = Isoniazid, R = rifampicin, E = ethambutol, Z = pyrazinamide

There were 70 specimens of non-respiratory origin (tissue n=44; fluids n=14; aspirate n=9; pus n=3) and 44 (62.9%) were NAAT positive. For smear positive non-respiratory specimens, 16 of 18 (88.9%) were NAAT positive and 28 of 50 (56.0%) of smear negative non-respiratory specimens were NAAT positive. One specimen did not record a smear result, and one pleural biopsy yielded a non-interpretable result due to inhibitors.

Discussion

The finding of 787 cases of bacteriologically confirmed tuberculosis representing 3.9 cases per 100,000 population in 2004 was the same as for 2003 and is consistent with the results of previous AMRLN reports dating back to 1986. Similarly, distribution by gender, age and site of disease was consistent with previous reports.[6–17]

Once again, almost all isolates were *M. tuberculosis*, with only a single *M. africanum* and one case of the recently described *M. canettii*. Traditionally, the MTBC has contained four species: *M. tuberculosis*, *M. africanum*, *M. bovis* including the vaccination strain *M. bovis* (Bacille Calmette-Guérin) and *Mycobacterium microti*. More recent additions to the complex include *Mycobacterium canettii*, a rarely encountered human pathogen,[18] *Mycobacterium caprae* isolated from lymph node and lung of tuberculous goats,[19] and *Mycobacterium pinnipedii*, the causative agent of disease in seals from Australia, New Zealand, South America and Great Britain, and occasionally in human beings.[20,21] The possible evolutionary scenario for the emergence of the members of the MTBC has been discussed by Brosch, *et al*.[22]

M. canettii is an exotic member of the MTBC. Other than the initial Canetti strain, a further example of the species was cultured in 1993 from the lymph node of a 2-year-old Somali child. The peculiarity of this species is its abundant growth on solid media producing white, glossy colonies within six days of incubation. The Somali isolate was susceptible to H, R, and E but resistant to streptomycin. No result was reported for pryazinamide.[18] The *M. canettii* reported here was a lymph node isolate from a 36-year-old female.

The 'seal bacillus' associated with tuberculosis was isolated initially from three dead seals washed ashore in southern Western Australia. Comparison with other isolates cultured previously from seals and a trainer who worked with the infected seals found similar genetic profiles suggesting that the organism occupied a unique position within the MTBC.[20] It has been isolated from seals on four continents and is now recognised as *M. pinnepedii*, the causative agent of tuberculosis in seals, and occasionally humans. Transmission to humans is believed to be incidental, presumably by droplet aerosols.[21]

A longstanding member of the MTBC, *M. microti* is a causative agent of disease in wild voles or field mice but not in human beings. However, that view changed when two DNA fingerprinting methods unexpectedly found that *M. microti* had unique fingerprinting and spoligotyping profiles.[23] Similar profiles were subsequently identified in four human cases resident in The Netherlands; three of the four cases were immunocompromised (renal transplantation n=2; HIV n=1), the other case occurred in a 34-year-old immunocompetent male. From the clinical perspective, three patients were treated successfully with standard antituberculous therapy; the fourth died from overwhelming infection shortly after diagnosis. However, one patient with heavy smear positive lung disease was highly likely to have transmitted the organism to friends and a close relative. Another case of *M. microti* was confirmed from a resident of the United Kingdom although no other details were recorded.[24] The frequency of human disease caused by *M. microti* is unknown as the organism is difficult to cultivate, taking months rather than weeks to grow, and traditional phenotypic tests are unreliable.

For bacteriologically confirmed cases of tuberculosis in Australia, the most likely causative agent remains *M. tuberculosis* with the occasional *M. bovis* or *M. africanum.*[17] The most recent members of the MTBC are encountered infrequently by Australian laboratories, and indeed globally.

The level of acquired resistance in Australia remains low. Most cases with drug resistant strains occurred in the overseas born and reflects previous data.[14–17] These findings reflect more upon the performance of the TB program from their country of origin rather than the clinical management of these patients in Australia. Therefore, as a measure of performance of Australia's TB control program, the national drug resistance data has limited usefulness. For 2004, the proportion of isolates that were MDRTB was 1.5 per cent, and the number of isolates resistant to at least one of the first line anti-TB drugs was 71 (9.0%); these findings are consistent with previous reports.[6–17] In Australia, rifampicin resistance is relatively rare and is considered a useful surrogate marker for MDRTB. In the years 2000–2004, there were 3,819 bacteriologically confirmed cases of TB.[14–17] Only 58 (1.5%) demonstrated resistance to rifampicin and 51/58 (87.9%) of these isolates were MDRTB. Interestingly, 35/51 (68.6%) of the MDRTB isolates were from patients who had given a history of no previous TB treatment. For 2000–2004, the MDRTB cases were from 17 countries and of these, 11 countries were from the South East Asia and Western Pacific Regions.

In conclusion, the 2004 laboratory data for culture confirmed cases of TB demonstrates a steady-state situation for the number of cases reported, level of smear positivity for respiratory specimens, and drug resistance.

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, The Prince Charles Hospital, Chermside, Queensland

Victorian Infectious Diseases Reference Laboratory, North Melbourne, Victoria

PathWest, The Queen Elizabeth II Medical Centre, Nedlands, Western Australia

Institute of Clinical Pathology and Medical Research, Westmead Hospital, Westmead, New South Wales

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The BCG vaccine: information and recommendations for use in Australia

National Tuberculosis Advisory Committee

Executive summary

The Bacille Calmette-Guérin (BCG) vaccine since its first use in 1921 has been the subject of much controversy as to its effectiveness and applicability. BCG vaccination is still considered an important strategy in the National Tuberculosis Programs of countries with a high burden of tuberculosis (TB) because of its benefit to infants but its effect on the control of TB has been limited. By contrast, in countries with a low prevalence of TB, significant policy differences exist both within and between countries.

BCG vaccination does not prevent transmission of infection to the individual. In immune-competent neonates and infants it is accepted that BCG reduces the likelihood of TB infection progressing to disease or if disease occurs, substantially lessens the chance of a severe outcome. The benefit in older age groups is less clear.

In the Australian health worker, the BCG strategy is no longer recommended as the primary means of health care worker (HCW) protection. The preferred strategy is appropriate infection control measures, staff education and a tuberculin skin testing program that identifies and treats the at-risk infected HCW. The emergence of multi-drug resistant strains has however renewed interest in BCG in the HCW.

This document provides recommendations for use of the BCG vaccine in the Australian community based on the best available evidence and consensus opinion. State and Territory TB Control Units should be consulted with regard to their BCG vaccination guidelines.

Recommendations

BCG vaccination is not recommended for general use in the Australian population.

BCG is recommended for:

1. Aboriginal neonates in areas of high incidence of TB (e.g. Northern Territory, Far North Queensland, northern areas of Western Australia and South Australia);
2. neonates and children 5 years and under who will be travelling or living in countries or areas with a high prevalence of TB for extended periods;
3. neonates born to parents with leprosy or a family history of leprosy;

In addition to these recommendations BCG may be considered in the following:

4. children over 5 years who will be travelling or living in countries or areas with a high prevalence of TB for extended periods;
5. HCWs who may be at high risk of exposure to drug resistant cases.

Introduction

Mass BCG vaccination in populations with a low prevalence of tuberculosis disease is no longer considered necessary.[1] Rather, such an intervention should be directed at well -defined, high-risk groups principally because of its direct effect in reducing the serious consequences from primary infection. The indirect population effect of mass vaccination in terms of reducing the number of infectious cases and hence limiting future transmission to the uninfected population is considered minimal in low prevalence countries.[2]

In Australia, the broad-based BCG vaccination program originated at a time when the epidemiological circumstances of tuberculosis (TB) were quite different. Initially in 1948, vaccination targeted health workers, Aboriginal people and close contacts of active cases, especially children. In the 1950s the program was expanded to include all Australian school children except those from New South Wales and the Australian Capital Territory. This policy was discontinued in the mid-1980s (1991 in the Northern Territory) in favour of a more selective approach. The change occurred because of the low prevalence of TB in our community and concerns about the balance between the benefits and the risks.

Sweden, which prior to 1975 vaccinated all newborns, is one of the few countries to have closely studied the implications of this. The observed incidence of TB in unvaccinated Swedish children from a low risk background remains low, and importantly, the risk of serious TB is still rare.[3]

Further, the similarities in TB disease trends between Australia and countries where universal BCG vaccination has never been practised (USA, Netherlands) suggest that the incidence of TB in a community is determined by the combined effect of all TB control measures rather than BCG vaccination alone.

BCG vaccination does not prevent the transmission of infection to an individual. Its direct effect for which it was introduced appears to be in limiting the spread of primary infection in an infected individual. Varying reports suggest levels of protection anywhere from 0 to 80 per cent.[4,5,6,7] The differences possibly relate to use of different BCG strains, methodological factors, the influence of environmental mycobacteria and age, immune or genetic factors.[8]

Recent meta-analyses have been helpful in summarising the variable findings from several studies on BCG efficacy. The key conclusions were that it is about 50 per cent effective in preventing disease and that the most important protective benefits are in minimising the risk of death, meningitis and miliary disease in neonates and young children.[9,10]

Although the use of BCG in health workers has waned considerably there has been renewed interest related to multi-drug resistant TB.[7] The benefit of BCG vaccination over TST screening may be enhanced for the health worker in such a setting.[11,12,13] It offers some protection irrespective of drug susceptibility status, whereas the benefit of preventive therapy is unproven in those infected with an MDR strain.[14,15] This dilemma highlights the importance of appropriate infection control measures in health care settings.

Risk groups

For the majority of Australian born now, the risk of acquiring TB infection and developing disease is very low. However certain groups in our community are considered at increased risk.[16–18]

The National Health and Medical Research Council (NHMRC) consensus statement has defined high risk as referring to those subgroups of the population who have an annual notification rate above 25 cases per 100,000 population.[19] This provides a useful criterion for determining groups who may benefit from a BCG policy.

The following groups have been assessed as falling into the high-risk category but significant debate continues as to how extensive BCG vaccination programs should be within them.

Aboriginals

Aboriginal people are at greater risk for developing active TB than non-Aboriginal Australian born and this likely reflects socioeconomic, nutritional and health factors.[20–22] While the number of cases of active TB recorded is small, their rate of disease is estimated to be about 15–20 times higher than for the non-Aboriginal Australian born.[16–18] The rate appears to be higher in the rural and traditional communities compared to the urban groups.

The recommendation that at risk Aboriginal neonates be BCG vaccinated shortly after birth is based on the premise that in high risk populations, infants and children have a greater potential for exposure to an active case of tuberculosis. Infection in this age group has a significantly higher risk for producing the severe manifestations of TB, including meningitis, rapid dissemination and death.

Migrants

The most important factor contributing to the change in the epidemiology of TB in Australia has been the increased migration from countries with a high incidence of TB. Their rates of TB remain similar to those of their country of origin, particularly in the first 5 years after arrival.[16–18]

The rate of TB in children, particularly those aged less than 5 years, is an important indicator of TB control. The overall rates of TB for non-Indigenous children born in Australia remain very low. While the rates are higher in overseas-born children the actual numbers reported are small.[16–18] Further, data from Australian prevalence surveys indicate that the rate of TB infection in children born in Australia of overseas-born parents is as low as that of children of Australian-born parents.[23–26]

Hence it is now recommended that BCG vaccination in neonates and infants of migrant parents should be based on a careful assessment of the individual situation.

Health care workers

Health care workers are at variable risk of being exposed to patients with active TB. This will be dependent on the specific occupation and likelihood of contact with certain groups.

Two strategies have been advocated to control TB in HCWs. Namely, BCG vaccination or regular tuberculin skin testing (TST) and the use of preventive therapy in ‘converters’. The role of BCG vaccination in HCWs is unclear and the uncertainty has led to divergent policies in the States and Territories and overseas. The main issues are the lack of evidence

supporting a protective benefit from BCG in the adult and the fact that it renders future interpretation of the post-exposure TST imprecise.[7]

The TST policy is theoretically sound but weakened by the reluctance of many HCWs to comply with the recommended measures. Further, with the emergence of multi-drug resistant disease, the benefit of preventive treatment for infected contacts is uncertain.[14,15] Although the number of cases reported to date in Australia is small, multi-drug resistant TB is nevertheless a major concern because of the poor cure rate, high mortality and potential implications for exposed HCWs.

In addition, irrespective of the HCW strategy, it is important to ensure that both the individual and the institution in which they are working are adequately informed about TB and that appropriate infection control measures are in place to minimise the risk of transmission.

HCWs who are at significant risk of exposure to TB cases or potentially infected laboratory material should be recommended to have regular TST screening. This includes:

- medical and nursing staff working in Respiratory Units and at Chest Clinics;
- bronchoscopy theatre staff;
- laboratory personnel involved in handling tuberculous material; and
- staff involved in post-mortems.

BCG should not be recommended as the primary means of HCW protection. The use of BCG vaccination should be assessed according to individual circumstances. It should be considered in those who may be at high risk of exposure to drug resistant cases e.g. the HCW moving to an overseas country to work in an area with a known or suspected drug resistance problem.

The use of BCG vaccination for HCWs in low risk settings is not recommended.

Overseas travel

The number of cases of TB reported in Australians who have travelled or lived overseas for significant periods is small.

Vaccination is not considered necessary in those undertaking brief holidays to well known tourist destinations. However in neonates and children 5 years and under who will be staying in countries where the incidence of TB is high for extended periods, vaccination is recommended. Each individual's situation needs to be carefully assessed. The protective benefit of vaccination in older age groups is less certain.[7] BCG should be given 2 to 3 months prior to departure.

Other groups

There are additional groups in our community based on overseas experience that may be at increased risk of TB and these include the homeless, prison residents and injecting drug users. BCG vaccination is not recommended for these persons.

Recommendations

BCG vaccination is not recommended for general use in the Australian population based on the low incidence of tuberculosis.

BCG is recommended for:

1. Aboriginal neonates in areas of high incidence of TB (e.g. the Northern Territory, Far North Queensland, northern areas of Western Australia and South Australia);
2. neonates and children 5 years and under who will be travelling or living in countries or areas with a high prevalence of TB for extended periods;
3. neonates born to parents with leprosy or a family history of leprosy;

In addition to these recommendations BCG may be considered in the following:

4. children over 5 years who will be travelling or living in countries or areas with a high prevalence of TB for extended periods;
5. HCWs who may be at high risk of exposure to drug resistant cases.

State and Territory TB Control Units should be consulted with regard to their BCG vaccination guidelines.

Important notes

All individuals should be tuberculin skin-tested prior to BCG vaccination except in infants less than 6 months of age where a history of TB contact has been excluded.

BCG should not be given to an individual with a tuberculin reading of 5 mm or more.

BCG vaccine should not be administered unless consent has been obtained following a full explanation of the benefits and risks associated with the vaccination.

No more than one BCG is to be given, regardless of TST reaction.

Contraindications

The use of BCG vaccine is contraindicated in the following:

- persons immuno-compromised by HIV infection, corticosteroids or other immuno-suppressive agents and malignancies involving bone marrow or lymphoid systems (because of the risk of disseminated BCG infection);
- individuals with a high risk of HIV infection where HIV antibody status is unknown;
- individuals with any serious illness including the malnourished;
- individuals with generalised septic skin diseases and skin conditions such as eczema, dermatitis and psoriasis;
- pregnant women—BCG has not been shown to cause foetal damage but the use of a live vaccine in pregnancy is generally contraindicated;
- individuals who have previously had tuberculosis or a large tuberculin (Mantoux) reaction.

BCG should be deferred in the following:

- individuals with a significant febrile illness (administer 1 month from the time of recovery);
- neonates with a birth weight less than 2.5 kg or in those who may be relatively undernourished. It should not be offered to neonates of mothers who are HIV positive;
- a 4 week interval should be allowed following administration of another live vaccine unless given concurrently e.g. MMR, yellow fever (although there is no evidence that the immune response could be impaired).

NB: Care should be taken in those with a history of keloid scarring or an increased risk of developing it e.g. Aboriginals, Melanesians. The likelihood of this occurring can be minimised if the injection is given into the skin over the region of the deltoid muscle insertion.

It is recommended that a list of exclusion criteria be given to the patient to allow self-exclusion with complete anonymity regarding the specific risk factor.

Vaccination

The vaccine

- The BCG (Bacille Calmette-Guérin) vaccine[1] is a suspension of living organisms of an attenuated strain of *Mycobacterium bovis*. It is available as a freeze-dried powder for intradermal use in a 10-dose vial and should be stored at 2–8° C with protection from light. Exposure to heat and light both before and after reconstitution may result in a loss of potency. The expiry date should be checked prior to administration.
- The vaccine is reconstituted using 1.5 ml of the sterile saline supplied. It should be gently and thoroughly mixed then used strictly within a 4–6 hour period. Store at 2–8° C.
- As the vaccine does not contain a bacteriostatic agent, extreme care is required to avoid contamination. A new 26–27-gauge needle and 1 ml syringe should be used for each dose and the remaining vaccine discarded as per procedures recommended for biohazardous substances.
- Providing a strictly aseptic technique is adhered to in accordance with approved infection control guidelines, the use of a multi-dose vial is an accepted practice.

Vaccination procedure

The NHMRC recommends that administration of the BCG vaccine be carried out by an accredited health-worker to limit the risk of adverse events.

The BCG dose is:

- Adults and children over 12 months – 0.1 ml
- Infants 12 months and under – 0.05 ml

Vaccination should be deferred in premature or small-for-dates babies less than 2.5 kg.

- A tuberculin skin test (Mantoux) should be done prior to vaccination except in infants less than 6 months (exclude history of TB contact). BCG can be administered to those with a reaction size less than 5 mm providing no contraindications exist.

1 The manufacture of BCG vaccine in Australia has been discontinued. The Aventis Pasteur BCG vaccine (Toronto, Ontario, Canada) has been approved for use by the Therapeutic Goods Administration and is distributed by CSL Limited (Parkville, Victoria).

- The site of injection into the skin is very important in order to minimise the risk of keloid formation. The position normally recommended is at the level of insertion of the deltoid muscle into the humerus. While it can be given into the middle third of the antero-lateral aspect of the thigh, many prefer not to for cosmetic reasons.
- The injection must be given strictly intradermally—needle bevel uppermost, until its opening is just visible through the epidermis.
- A blanched weal should be raised. If little resistance is felt, then this may mean that the needle is in the subcutaneous tissue and therefore should be withdrawn. The injection should then be given at an alternative site. Inadvertent subcutaneous injection is likely to cause an excessive reaction.

BCG reaction

Initially a small red papule forms within a 2–3 week period followed by softening and ulceration. Healing usually occurs after several weeks with a resultant small scar. An accelerated reaction begins within 24–48 hours with induration followed by pustule formation in 5–7 days and healing within 10–15 days.

BCG aftercare

Information both verbal and written should be provided to the vaccinee or carer on what to expect and how to care for the resultant local reaction. The importance of reporting promptly any suspected problems should be stressed.

Adverse effects

Serious complications from BCG vaccination including anaphylactoid reactions are rare.[27–29]

Adverse effects include:

- regional lymphadenitis – this is the commonest adverse reaction;
- subcutaneous abscess;
- accelerated local reactions;
- osteitis;
- keloid scarring;
- disseminated infection.

Correct assessment and technique is essential to minimise these risks.

Immuno-compromised individuals can develop disseminated infection from BCG, e.g. malnourished children and the HIV positive person.

Reactions of an untoward nature may require antituberculous treatment.

Adverse events following vaccination should be notified to the relevant State Health Authority.

BCG revaccination

In many developing countries systematic revaccination has been accepted practice because of doubts about the persistence capacity of the vaccine when given in the early neonatal period.[30] However, such an approach is not supported by scientific evidence.

The effectiveness of repeat BCG to the individual remains in question.[31,32,33] Previously, the finding of a negative tuberculin skin test response was considered to indicate the need for revaccination. It was argued that revaccination may increase the rate of tuberculin conversion and result in more sustained reactivity over time. However the tuberculin response is not a correlate of the protective benefit derived from BCG vaccination and there is no evidence that a waning of tuberculin sensitivity with time equates to a loss of TB specific immunity.[7, 34]

Based on the information available, BCG revaccination is not recommended in any person.[34]

BCG alternative

BCG remains the only available vaccine against TB. However it only offers partial and variable protection to the uninfected for a relatively short period.

Several new vaccine candidates are under investigation. These include recombinant vaccines, sub-unit vaccines and DNA-based vaccines. Novel adjuvants are also currently being tested with experimental sub-unit vaccines.[35–37] The improved safety of the latter over live-attenuated vaccines offers potential benefit to HIV- infected persons.

The relatively short-lived efficacy of BCG for only 10–20 years appears accepted.[38] A vaccine that both has the ability to boost immunity in those vaccinated in childhood to protect against the risk from primary infection or if already infected prevent reactivation of latent infection would be a substantial advance in the control of TB globally. Despite significant ongoing research the prospect of such a vaccine remains distant.[37]

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Guidelines for Australian Mycobacteriology Laboratories

National Tuberculosis Advisory Committee

Executive summary

Guidelines for Australian laboratories performing tuberculosis (TB) microscopy and culture have been developed through extensive consultation with expert groups. The aims of these guidelines are:

1. to provide consensus recommendations on the infrastructure, equipment and work practices required by mycobacteriology laboratories;
2. to inform laboratory administrations and governments of the necessary level of investment required to maintain modern mycobacteriology facilities; and
3. to provide informal measures for reviewers inspecting mycobacteriology facilities.

These guidelines include safety recommendations based largely on the Australian/New Zealand Standard 2243.3 *Safety in laboratories – Microbiological aspects and containment facilities.* However, these standards have been rationalised and PC2 facilities with additional processes and precautions in place are recommended for the majority of TB investigations. Guidelines are also provided on staff training, education, health screening and vaccination. Certain procedures and work practices are recommended for mycobacteriology laboratories to guarantee safety, high-quality results, and prompt turnaround times. These guidelines will be reviewed each 1–2 years and feedback from expert groups and individuals is welcomed.

Introduction

Tuberculosis (TB) represents an increasing threat to global health with at least 2 million deaths and 8 million new cases annually. Unlike the United States of America (USA) and some European countries where TB epidemics again occurred in the late 1980s–1990s, Australia has maintained a very low incidence of TB with just 1,159 active cases reported in 1999 (i.e. 6.1 cases per 100,000 population).[1] Australia has maintained these low rates despite migration from TB–endemic countries, because of the continued efforts of clinical, laboratory and public health personnel involved in TB control. However, Australia must continue these efforts and must also support TB control efforts in our region because over 60 per cent of the global TB burden occurs in South East Asia and the Pacific.

Following the USA TB epidemic in the early 1990s, the US Centers for Disease Control and Prevention (CDC) set demanding criteria for mycobacteriology laboratory performance:[2,3]

- acid-fast examinations to be reported within 24 hours of specimen+ collection;
- identification of *Mycobacterium tuberculosis* complex (MTBC) within an average of 10–14 days; and
- reporting of drug susceptibility results within an average of 15–30 days.

These 'rapid' turnaround times (TATs) can only be met by increasing investment in mycobacteriology staff and by the introduction of new expensive broth-based culture systems. A 1991 review of USA laboratory practices revealed that only 29 per cent and 20 per cent were using the BACTEC radiometric system for culture and susceptibility testing respectively, resulting in substantial delays in the reporting of results.[4] A 1994 follow-up review of laboratory practices found a marked improvement in performance with laboratories reporting microscopy results within 24 hours having risen from 52.1 to 77.6 per cent, the reporting of MTBC within 21 days increasing by 22.1 per cent to 72.9 per cent, and susceptibility testing being completed within 28 days rising from 16.7 to 48.9 per cent.[5] These results were associated with the introduction of new technologies. The percentage of laboratories using fluorescence microscopy, BACTEC for primary culture, and BACTEC for susceptibility testing having risen from 27.1 to 79.6 per cent, 74.5 to 100 per cent, and 26.2 to 73.3 per cent respectively.[5]

Mycobacteriology laboratory services in Australia

Despite having low incidences of TB in Australia and other industrialised countries, their mycobacteriology laboratories are increasingly expected to meet the USA standards. Australian laboratories have reported 700–760 isolates of *M. tuberculosis* complex per year between 1996–1999.[6] Only approximately 75–85 Australian laboratories perform TB cultures and the majority of this work is performed

in the five Mycobacterium Reference Laboratories (MRLs) and other large centres. Smaller laboratories therefore may not have had the necessary workload to maintain expertise or to justify investment in the new technologies.

These problems are perhaps best expressed in the results of the Royal College of Pathologists of Australasia (RCPA) national quality assurance program (QAP). Since 1996, smears containing between 1–9 acid-fast bacilli (AFB)/high power field (i.e. 2+) have been called negative by 1.5–6.0 per cent of laboratories. Of equivalent concern is that a similar number of laboratories (2.4–5%) are reporting false-positive results for QAP smears negative for AFB.

Of the 80 or so laboratories performing culture, up to 16 per cent of laboratories have failed to recover MTBC from QAP samples that on occasion have contained +++ AFB. The majority of culture errors have occurred in laboratories which process fewer than 1,000 specimens per year. In a 1999 item (RCPA 99:6:3), two samples were sent for mycobacterial culture. For the sample containing approximately 500 colony forming units of *M. bovis* (BCG)/sample, 80/83 (96%) of laboratories successfully isolated mycobacteria, but for the other sample containing approximately 50 colony forming units BCG/sample, only 63/83 (76%) were able to recover mycobacteria. Three of 83 laboratories failed to recover mycobacteria from either sample. These results suggest that a minority of laboratories are failing in one or more areas of specimen processing, media quality, culture performance, or culture interpretation. Additionally, the number of false-positive reports has risen from 0 per cent in the 1995 and 1996 RCPA-QAP to a high of 5.3 per cent in 1999. Although the number of laboratories performing culture has remained steady, there is an apparent ‘ebb and flow’ with some laboratories ceasing to perform culture whilst others have begun to do so.

Rationale for national guidelines

These guidelines have been produced to assist several groups directly and indirectly involved in maintaining the high quality of mycobacteriology investigations performed in Australia.

Consensus guidelines for a mycobacteriology laboratory

These guidelines aim to document the infrastructure, equipment, staffing and work practice requirements for a modern mycobacteriology laboratory. These guidelines have been developed through extensive consultation with the Mycobacterium Reference Laboratories, the Mycobacterium Special Interest Group (SIG) of the Australian Society for Microbiology (ASM), the Public Health Laboratory Network (PHLN), the Royal College of Pathologists of Australasia (RCPA), and other interested parties. Mycobacteriology laboratory staff can therefore use these guidelines as a benchmark tool for assessing their own laboratory performance.

Laboratory administration

Laboratories must balance the increasing investment required to provide a modern high-quality mycobacteriology service against the expected income from a limited number of TB and other mycobacteriology requests. These guidelines attempt to provide some guidance on the minimum workload, staffing, equipment and infrastructure required to provide an acceptable service. Laboratory administrators can then decide whether their workload justifies the costs of providing these services.

Laboratory reviewers

The reviewers for the National Association of Testing Authorities (NATA)/RCPA often do not have extensive expertise in mycobacteriology. These guidelines aim to provide a tool for assessing a mycobacteriology laboratory. However, while the safety requirements are obviously mandatory, it must be emphasised that reviewers should not consider any other single element as mandatory. Rather, a laboratory should be assessed across the spectrum of infrastructure, equipment, staffing, work practices and workload requirements, and must not be failed on any one deficiency. For example, a high-quality laboratory may fulfil all requirements but may not have an ‘adequate’ workload to maintain expertise. Such a laboratory could adequately compensate by additional training for staff and demonstrated close liaison with the relevant MRL or other major laboratory. In contrast, an inadequate laboratory is likely to be deficient in several aspects.

Government authorities and the general public

Australia has one of the lowest TB rates in the world. However, continued funding is required to maintain this enviable position. The experience in New York City in the 1980s–1990s demonstrates the alternative outcome.[7] Reduced TB funding in the late 1970s–early 1980s resulted in degraded TB services including a deterioration in the TB laboratory system. An epidemic followed with more than 20,000 excess cases including outbreaks of multidrug-resistant disease. Failure of the under-resourced laboratories to detect TB promptly and to perform drug susceptibility tests (DST) expeditiously were contributing factors to this TB epidemic.[2,7] Over US$1 billion has been spent bringing TB back under control in New York City.[7] Some of these funds have been spent renovating and improving the TB laboratories.

This document therefore aims to inform government authorities of the requirements for effective TB laboratory services so that adequate funds are available to meet these needs. The Australian public can also be assured that high-quality mycobacteriology services are continuing to be provided throughout Australia.

Formulation of the TB laboratory guidelines

These guidelines reaffirm and reiterate the biosafety requirements for Australian mycobacteriology laboratories as outlined in the latest Australian/New Zealand Standard 2243.3 *Safety in laboratories – Microbiological aspects and containment facilities.*[8] Laboratories must also comply with the National Pathology Accreditation Advisory Council (NPAAC) Standards for Pathology Laboratories[9] and relevant NATA requirements.

The guidelines specific for TB laboratories have been developed through extensive consultation with MRL staff, the ASM Mycobacterium SIG, PHLN members, RCPA, and other interested parties. Similar guidelines for USA laboratories have also been referenced.[2,3] The New Zealand Ministry of Health have published a large compendium entitled *Guidelines for tuberculosis control in New Zealand,* which includes a chapter for the mycobacteriology laboratory (available from: http://www.moh.govt.nz/49ba80c00757b8804c25667300d47d0/4760df3580a6f5b5cc256c86006ed394?OpenDocument). There are no matching documents produced by European or British authorities (FA Drobniewski, PHLS Mycobacterium Reference Unit, King's College Hospital, London, personal communication). These Australian guidelines will be reviewed each 1–2 years and feedback from expert groups is welcomed.

Risk group classification of Mycobacterium tuberculosis

Australian/New Zealand Standard 2243.3 *Safety in laboratories – Microbiological aspects and containment facilities* recognises *M. tuberculosis* as a Risk Group 2 organism; multidrug-resistant strains are considered to be Risk Group 3 organisms. This classification leaves mycobacteriology laboratories with an interesting dilemma. Clinical specimens will be processed, cultures performed, and susceptibility tests completed before laboratory staff will know whether they should have undertaken these tests in a Physical Containment Level 2 (PC2) or PC3 facility.

One approach would be to treat all specimens as containing MDRTB organisms until proven otherwise and hence require all TB investigations be performed in a PC3 facility. While providing the highest level of staff protection, this approach is arguably excessive because Australia has a very low rate of MDRTB (0.5–0.9% in 1998–1999,[6]) and only 6–7 laboratories are undertaking DSTs. Many Australian mycobacteriology laboratories do not meet all of the requirements of a PC3 facility. Mandatory use of PC3 facilities would therefore require major infrastructure investment or would result in an excessive workload concentrating on the limited number of PC3 laboratories.

A reasonable compromise position could be for all mycobacteriology investigations to be undertaken in PC2 facilities with additional processes and precautions in place (see below). Laboratories undertaking more than 5,000 cultures per year, performing DSTs, or knowingly handling MDRTB strains should have PC3 facilities or have building plans to acquire PC3 facilities by 2007.

Guidelines for a laboratory performing smear microscopy

Approximately 80 laboratories in Australia perform smear microscopy then forward the specimen to another laboratory for mycobacterial culture. The microscopy-only laboratories almost universally perform direct Ziehl-Neelsen (ZN) smears. While opening sputum containers and making smears may produce aerosols, there is no epidemiological evidence in low-income countries associating smear preparation with any measurable increased risk of acquiring TB infection. The World Health Organization (WHO) and the International Union Against TB and Lung Disease (IUATLD) therefore consider smear preparation a low-risk procedure.[10] Nonetheless, all suitable safety measures must be provided in a high-income country such as Australia with a low incidence of TB.

General laboratory facilities, equipment and work practices

Laboratories performing smear microscopy must comply with the requirements of a PC2 facility.[8] The following (additional) requirements must be emphasised:

1. The smear preparation procedure must be performed in a Class I or Class II biosafety cabinet (BSC).

2. The operator must wear gloves and a long-sleeved gown.

3. Any manipulation involving shaking, mixing or sonication must be performed in the BSC and a period of at least 5 minutes elapse before the container is opened in the BSC.

4. In the unlikely event that a concentrated smear is being prepared, a centrifuge with sealed rotors or safety cups must be used and must be capable of attaining 3,000 *g*.

5. Access to the laboratory must be limited to personnel and persons specified by the laboratory management.

6. Packaging of specimens for shipment by a public carrier to the culture laboratory must comply with International Air Transport Association (IATA) regulations (summarised in AS/NZS 2243.3,[8]). A specific Australian standard on the shipment of biological materials is also in preparation.

Requirements specific to a laboratory performing TB smear microscopy

The following work practices are recommended for laboratories performing TB smear microscopy:

1. Smear results should be available within 24 hours of specimen reception. On weekends, the requesting doctor should discuss urgent requests for TB smear microscopy with the clinical microbiologist. Results should be available within 24 hours even on weekends for specimens considered urgent; results for non-urgent routine requests should be available on the following Monday. The treating doctor and the laboratory director/clinical microbiologist should liaise to decide whether such weekend specimens are urgent or non-urgent.

2. Specimens for cultures should be transported to the relevant laboratory within 24 hours.

3. A positive- and a negative-control smear should be included with each batch of smears.

4. Positive results should be quantified using the IUATLD/WHO scale:[10]

negative	No acid-fast bacilli (AFB) in at least 100 high power fields (HPFs)
exact figure/100	1–9 AFB per 100 HPFs
+	10–99 AFB per 100 HPFs
++	1–10 AFB per field in at least 50 HPFs
+++	More than 10 AFB per field in at least 20 HPFs

5. A laboratory performing TB smear microscopy should process a minimum of 10 requests per week to maintain expertise. A technician should process and read no more than 20 ZN smears per day on average. More smears (2–3-fold) can be read per day if a fluorochrome stain is used.

6. The staining reagents must be labelled with their identity, concentration, preparation date, expiration date, initials of the technician who prepared the reagent, and any relevant safety symbols.

7. The staining method should be clearly described in the laboratory method manual, which should also list the remedial actions if the positive or negative control slide fails.

8. Larger laboratories that process many specimens (and perform cultures) may use a fluorochrome stain. All fluorochrome-positive slides from new smear-positive patients must be checked by ZN stain.

9. The laboratory should have the ability to monitor the number of specimens collected per year, the number of patients from whom 1, 2 or 3 sputa are collected, and the number of smear-positive results in TB suspects and patients on treatment.

10. The laboratory must participate in an external quality assurance program. The RCPA program sends 8–10 AFB smears per year. Quantitation errors are of minor significance.[10] Similarly, scanty false-negative results are understandable if the QAP sends a slide with 1–9 AFB/100 fields. In contrast, >1 false-positive or high false-negative result in any one year should trigger remedial action.

Requirements for a sputum collection area

Some laboratories may be responsible for collecting TB sputum specimens. The laboratory must therefore ensure that a high-quality specimen is collected, suitably labelled, and that the collection is performed safely. Whereas smear preparation is a low-risk procedure, sputum collection from a smear-positive patient is a high-risk procedure and must be performed in the correct setting.[10]

1. The laboratory should provide an instruction form to the patient describing the method of producing a good sputum specimen, the timing of the collection, and the handling of the specimen (e.g. refrigeration at 4° C).

2. Appropriate containers should be provided to the patient.

3. Sputum specimens should be collected in well-ventilated areas away from other patients. Patients at high-risk of having TB should be referred to hospitals where they can be evaluated and managed appropriately.

4. For patients at high-risk of having TB, sputum collection and cough-inducing procedures should be performed in negative-pressure ventilation rooms. Health-care workers (HCWs) should wear respiratory protection (i.e. a properly fit-tested high-efficiency N95-standard mask or powered air-purifying respirator – PAPR) when present in rooms or enclosures in which cough-inducing procedures are being performed on patients who may have infectious TB. These high-risk patients should also be managed appropriately before and after the specimen collection to limit cross-infection to other patients and to HCWs. The US Centers for Disease Control and Prevention have released extensive guidelines on reducing TB transmission in the health-care setting.[11]

Guidelines for laboratories performing mycobacterial cultures

As discussed earlier, a PC2 laboratory with additional equipment and work practices would appear to be an appropriate facility for performing the large majority of TB cultures in Australia. Alternatively, these laboratories could be considered PC3 laboratories without some of the building and engineering requirements. Nonetheless, laboratories undertaking more than 5,000 cultures per year, performing susceptibility tests, or knowingly handling MDRTB strains should have PC3 facilities or have building plans to acquire PC3 facilities by 2007.

General laboratory facilities, equipment and work practices

1. The TB culture laboratory must be in a self-contained room physically separated from other areas.

2. Access to the TB laboratory must be limited to staff trained to work in the area. Access should be restricted by lockable doors.

3. A pressure steam steriliser must be available for decontaminating laboratory waste, preferably within the laboratory.

4. A directional air flow shall be maintained by extracting room air. Recirculation is permitted but not into areas outside the PC2-PC3 facility.

5. All procedures must be performed in a Class I or Class II biosafety cabinet.

6. The operator must wear gloves and a long-sleeved gown. These personal protection items must not be worn outside of the TB laboratory. N-95 HEPA masks should be provided for staff to clean laboratory spills or if other accidents occur.

7. Any manipulation involving shaking, mixing or sonication must be performed in the BSC and a period of at least 5 minutes elapse before the container is opened in the BSC.

8. A centrifuge with sealed rotors or safety cups must be used. This centrifuge should attain 3,000 *g* to reliably sediment AFB.

Requirements specific to a laboratory performing TB culture

1. A scientist with a university degree (or equivalent training and experience) should be responsible for the TB laboratory. All staff working in the TB laboratory should have been suitably trained and have evidence of on-going training. A clinical microbiologist should have active input into the laboratory planning, procedures, and supervision, and should be available to communicate any positive culture results, where necessary.

2. A TB culture facility should process 20 or more specimens for culture per week.

3. Ideally, specimens should be processed on each day of the working week. Smaller laboratories culturing 20–50 specimens per week may choose to process cultures 3–4 times per week. In these circumstances, any smear-positive specimen should be cultured on the day of smear preparation.

4. All specimens should be inoculated in a broth-based culture system +/– onto solid media.

   The BACTEC and newer non-radiometric systems have revolutionised mycobacteriology providing TATs appreciably faster than those achieved by culture on solid media.[2–5,12] Broth-based culture systems should therefore be used by default. USA authorities recommend that each specimen should also be inoculated onto solid media to detect strains that may not grow in broth. Growth on solid media only in comparative studies may be due to the 'splitting' of samples with low AFB counts across multiple media and may not be a major problem if all of the sediment is inoculated into the broth. Processing of multiple specimens from each TB suspect also increases the sensitivity of culture. Furthermore, the requirement for solid media adds to the cost and workload of a laboratory. Selective use of solid media may therefore be acceptable (e.g. on all sterile site specimens such as tissues or CSF, and on any smear-positive specimen).

   Specimens from skin, lymph nodes and abscesses that may contain pathogenic non-tuberculous mycobacteria (NTM) should also be inoculated onto/into additional media for incubation at 30° C.

5. Specimens from sterile sites not requiring decontamination (e.g. cerebrospinal fluid, biopsies) should be directly inoculated into the broth- and solid-media systems.

6. The inclusion of positive- and negative-culture controls with every batch of specimens for culture is not necessary. Positive controls represent a potential source of contamination and should only be included when a new batch of media is used. Negative-controls will only reliably detect gross contamination that will be self-evident. Low-level contamination will be inconsistent and may not be detected in negative-control vials. Recording of background bacterial contamination rates and recovery rates of NTM is far more important.

7. Contamination rates should be recorded. Bacterial contamination rates less than 8 per cent are acceptable representing the best balance between excessive contamination and overly stringent decontamination (that risks false-negative culture results).

8. Laboratories must be alert to cross-contamination between specimens resulting in false-positive results. Laboratory cross-contamination should be considered in the following circumstances:[13]

   - a single smear-negative *M. tuberculosis*-culture-positive specimen when other samples from the patient are smear- and culture-negative;
   - the patient's clinical presentation or course is inconsistent with TB;
   - unusual clustering of positive-culture results processed on the same day;
   - isolates with unusual DST profiles processed on the same day;
   - 5 colonies grow on solid media, or time to growth detection is >30 days in automated broth cultures, or discordant results are obtained when solid- and broth-based are inoculated with the same specimen.

   Suspicions of laboratory cross-contamination events should be investigated by:

   - reviewing the laboratory logbook for other culture-positive specimens processed at the same time;
   - reviewing the patient's history, radiological investigations, clinical course, and response to therapy; and
   - genotyping of the suspicious isolates which may demonstrate identical profiles to laboratory control strains (e.g. H37Rv) or to isolates from epidemiologically-unrelated patients processed on the same day;
   - reviewing the laboratory procedures.

9. Non-automated broth-based cultures should be read every 2–3 days for weeks 1–3, and weekly thereafter for 6–12 weeks (depending on the specimen type and smear result). Solid media should be read twice weekly for weeks 1–4 then weekly thereafter.

10. All positive broth-based cultures must be: ZN-stained, sub-cultured to solid media (to detect mixed mycobacterial growths), and sub-cultured to blood agar (to detect bacterial contamination). The initial mycobacterial isolate from a patient must be identified as *M. tuberculosis* complex, *M. avium* complex, or another NTM. The indications for performing further susceptibility testing on *M. tuberculosis* isolates are listed below and the indication for referring NTM are provided in the section headed 'Referral of non-tuberculosis mycobacteria cultures'.

11. Laboratories performing cultures but referring isolates for identification and DST must send positive cultures to the reference laboratory within 48 hours of culture positivity. The isolate must be accompanied by documentation of all relevant clinical and laboratory information (e.g. patient details, original specimen type, AFB smear result, associated histological investigations that may have been performed on the same specimen).

12. A rapid (molecular) test is preferred for confirming growth of MTBC in ZN-positive cultures. The AccuProbe nucleic acid hybridisation test (Gen Probe, San Diego, CA) or an MTBC-specific nucleic acid amplification test (NAAT) are recommended. HPLC may be a reasonable alternative in laboratories with this equipment and expertise. The BACTEC NAP test is no longer considered acceptable because mixed cultures, growth temperatures and phases, and certain NTM can all produce anomalous results and time delays.

13. Laboratories should perform DSTs or refer isolates to reference laboratories for DSTs in the following circumstances:

   - all initial isolates of *M. tuberculosis;*
   - isolates from patients who remain culture-positive after 3 months of treatment;

- isolates from patients who are clinically failing treatment; or
- an initial isolate from a patient relapsing after previously successful TB treatment.[14,15]

The minimum DSTs that should be performed are for isoniazid (high- and low-level concentrations as appropriate), rifampicin, ethambutol, +/– streptomycin.

14. All positive culture and DST results that will affect patient management should be phoned and faxed to the treating doctor and the responsible TB control unit as soon as the results are available. For example, the initial results on all new patients, relapses and failure cases must be phoned and faxed directly to the treating doctor. Repeat results on subsequent specimens from the same episode can be sent in printed form.
15. Laboratories should aim to report positive MTBC cultures within an average of 14–21 days from time of specimen reception. These TATs are achievable using modern broth-based culture systems.
16. All MTBC isolates should be retained for at least six months by the referring laboratory and for at least three years by the reference laboratory.
17. Reference laboratories should also provide directly or through collaborative agreements, access to molecular epidemiological tools (e.g. restriction fragment length polymorphism – RFLP, spoligotyping, variable number tandem repeat – VNTR) so that outbreak strains and laboratory cross-contamination episodes can be recognised.
18. Microbiological laboratories performing TB cultures should ensure that they, or the reference laboratory to which their cultures are referred, include all positive culture results in the national figures collated through the MRL network.
19. Laboratories performing TB cultures must participate in a recognised QAP program. The RCPA QAP program distributes 8–10 specimens for mycobacterial culture per year. A review of laboratory procedures should be instituted if more than one false-positive or false-negative QAP culture result occurs per year.
20. Laboratories performing TB cultures should liaise closely with their state MRL. This liaison may be demonstrated by consultation over positive cultures, attendance at clinical meetings, and/or staff visits to the MRL. Such liaison is particularly important if the laboratory does not have the minimum recommended workload or is not fulfilling QAP or other requirements.
21. The ASM Special Interest Groups for Media Quality Control and Mycobacteriology are developing guidelines for assuring the quality of solid media used in mycobacteriology laboratories.[16] Laboratories must comply with this document particularly when the final version is published.

Referral of non-tuberculous mycobacteria cultures

With the low incidence of TB in Australia, the culture, identification and susceptibility testing of non-tuberculous mycobacteria represents an increasing proportion of the workload for the MRL network. These investigations should not be performed on every NTM isolate (as many represent colonisation or contamination) but only when clinically relevant. Diagnostic criteria[17] have been described for determining the significance of a pulmonary NTM isolate, particularly *M. avium* complex (MAC) and *M. abscessus*. These criteria should be applied when deciding which NTM to refer for identification:

1. If three sputum/bronchial washings are available in the previous 12 months: three smear-negative culture-positive results, or two culture-positive results of which one is also smear-positive;
2. If only one bronchial wash is available, the smear and/or culture show a heavy burden (2+–4+) of NTM;
3. If the above investigations are non-diagnostic, a lung biopsy yields a NTM or shows granulomatous inflammation and/or AFB.

Mycobacterial culture laboratories must therefore consider these diagnostic criteria and liaise with the requesting clinician before forwarding a NTM isolate to an MRL for further identification.

Susceptibility testing of NTM is a controversial issue. There are no data to show that DST results predict clinical outcome for many NTM infections. Furthermore, NCCLS has only recently released recommendations to standardise the performance of NTM DST.[14,15] Hence, mycobacterial culture laboratories should only expect an MRL to provide DST results in the following circumstances:[14,15]

Clarithromycin susceptibility testing for MAC

1. Clinically significant isolate from a patient who has received previous macrolide therapy (i.e. clarithromycin or azithromycin);
2. patients who have developed MAC bacteraemia on macrolide preventative therapy;
3. patients failing or relapsing on macrolide therapy; and

4. baseline isolates from significant MAC infections may also be tested (or stored and tested retrospectively if the patient does not respond to treatment).

Mycobacterium kansasii

1. All initial isolates of *M. kansasii* should be tested against rifampicin;
2. for patients failing or relapsing on treatment; and
3. for rifampicin-resistant isolates, the following antibiotics should be tested: isoniazid, ethambutol, rifabutin, clarithromycin, ciprofloxacin, streptomycin, and co-trimoxazole.

Rapidly growing non-tuberculous mycobacteria

All clinically significant rapid growers should be subjected to testing against: amikacin, cefoxitin, ciprofloxacin, clarithromycin, doxycycline, imipenem, and a sulphonamide. Tobramycin should also be tested for *M. chelonae* isolates only.

Susceptibility testing in other circumstances may be performed following close communication between the treating clinician, the mycobacterial culture laboratory, and the MRL, and with reference to the published guidelines on NTM DST.[14,15]

Guidelines for laboratories performing susceptibility tests

Laboratories performing mycobacterial drug susceptibility testing must meet the requirements (i.e. facilities, equipment and work practices) for laboratories performing mycobacterial cultures. Laboratories performing susceptibility tests should have PC3 facilities or have building plans to acquire PC3 facilities by 2007.

Drug susceptibility testing for *Mycobacterium tuberculosis*

The DSTs must be performed using a broth-based culture system so that results are available promptly. Using these methods, laboratories should aim to report MTBC DST results within an average of 15–30 days from the time of the original specimen reception.[2,3] The DSTs themselves can generally be completed within 7–14 days of obtaining the initial *M. tuberculosis* isolate from the primary cultures.

Drug susceptibility tests must be performed in the following circumstances:

- all initial isolates of *M. tuberculosis;*
- isolates from patients who remain culture-positive after 3 months of treatment;
- isolates from patients who are clinically failing treatment; or
- an initial isolate from a patient relapsing after previously successful TB treatment.[14,15]

The minimum DSTs that should be performed are for isoniazid (high- and low-level concentrations as appropriate), rifampicin, ethambutol, +/– streptomycin. The critical concentrations to be employed for these antibiotics in the BACTEC radiometric method are listed in Table 1. Revised guidelines on breakpoint concentrations may be required when the BACTEC system is superseded by non-radiometric methods (e.g. the MGIT 960 has received FDA approval for TB DST).

Supplemental tests to determine low-level resistance should be performed for isoniazid and may also be performed for ethambutol (Table 1). For isolates demonstrating isoniazid resistance at the critical concentration but susceptible at the higher concentration, USA authorities recommend adding the following comment to the report: 'These test results indicate low-level resistance to isoniazid. Some experts believe that patients infected with strains exhibiting this level of INH resistance may benefit from continuing therapy with INH. A specialist in the treatment of tuberculosis should be consulted regarding the appropriate therapeutic regimen and dosages'.[14,15] Australian laboratories could consider adding a similar comment in these circumstances after discussion with their TB Chest Clinic specialists.

Table 1. Critical concentrations for first- and second-line drug susceptibility testing of *Mycobacterium tuberculosis* using the radiometric BACTEC technique

Drug	Critical concentration (μg/ml)	Supplemental tests (μg/ml)
Isoniazid	0.1	0.4
Rifampicin	2.0	
Ethambutol	2.5	7.5
Streptomycin	2.0	6.0
Capreomycin	1.25	
Ethionamide	1.25	
Kanamycin	5.0	
Amikacin	1.0	
Clofazimine	0.5	
Ofloxacin	2.0	
Rifabutin	0.5	

Second-line drug susceptibility tests should be performed on:

- all MDRTB isolates (i.e. isolates demonstrating isoniazid and rifampicin resistance);
- all isolates demonstrating resistance to ≥ 2 first-line drugs; and
- isolates from patients experiencing severe adverse reactions to first-line agents.

The critical concentrations to be employed for second-line agents in the BACTEC radiometric method are also listed in Table 1. These breakpoints were determined in a multi-centre validation of second-line drug susceptibility testing by the radiometric BACTEC 460 technique.[18] No critical concentration could be recommended for cycloserine because of inconsistent results. The clinical significance of rifabutin 'susceptibility' in the setting of rifampicin resistance remains uncertain. Some clinicians argue that rifampicin resistance implies clinical resistance to all rifamycins and that rifabutin-susceptible rifampicin-resistant isolates merely reflect the use of an incorrect breakpoint for rifabutin.

Pyrazinamide susceptibility testing remains controversial and difficult. The majority of Australian reference laboratories use Wayne's pyrazinamidase (PZase) method to infer pyrazinamide susceptibility or resistance. This method is technically demanding particularly with drug-resistant strains and can give false-susceptible results if resistance is conferred by a mechanism other than PZase mutation. Only one Australian laboratory uses the expensive BACTEC pyrazinamide vials. USA recommendations suggest that, if pyrazinamide resistance rates are low, pyrazinamide susceptibility testing need only be performed as a second-line test on multi- or poly-resistant strains.[14,15] In 2000, only 8 (1.0%) of 768 *M. tuberculosis* isolates in Australia were reported as pyrazinamide resistant. Formal pyrazinamide testing in Australia could therefore be considered a second-line test. The informal PZase surveillance performed by MRLs should provide continued justification for this recommendation.

Drug susceptibility testing for other slow-growing mycobacteria

Clarithromycin and azithromycin are the only drugs where a correlation has been demonstrated between *in vitro* DST results and clinical outcome for MAC disease. Hence, MAC DSTs should only be performed for this drug class. The BACTEC radiometric system provides accurate and reliable results for MAC DST. Laboratories are referred to the NCCLS recommendations and manufacturer's instructions for performing these tests.[14,15]

The NCCLS recommendations also provides guidelines for performing DSTs for *M. kansasii* using the radiometric BACTEC system.[14,15] In general, susceptibility testing for other slow-growing mycobacteria have not been properly standardised or validated, and should only be performed in rare instances with close consultation between the treating physician and the laboratory.

Drug susceptibility testing for rapid-growing mycobacteria

Drug susceptibility testing for rapid-growing mycobacteria (RGM) can be performed by: broth microdilution, E-test, agar disc elution, and agar disc diffusion. The NCCLS recommends the broth microdilution based on a multi-centre study that evaluated the inter-laboratory reproducibility of broth microdilution for commonly encountered pathogenic RGM.[15,19] However, broth microdilution remains problematic: requiring expertise with the recommended method, requiring knowledge of the expected susceptibility patterns of different RGM, and difficulties with trailing endpoints.[19]

Agar disk diffusion is based on the Kirby-Bauer technique using commercially available antibiotic disks. Its major advantages are cost and ease of use, plus the ability to view colonial morphology. Unfortunately, the disadvantages are substantial, particularly with the interpretation of partial zones of inhibition when the disk concentration is close to the MIC breakpoint. Furthermore, many of the newer drugs (e.g. fluoroquinolones, clarithromycin, imipenem) have not had disk susceptibility validated against an MIC method.[19]

Although broth microdilution is recommended by the NCCLS for the susceptibility testing of RGM, the inherent technical and interpretive difficulties have discouraged the widespread uptake of this method by reference laboratories around the world. The AMRLN laboratories continue to use the agar disc diffusion method complemented by identification to species level. The AMRLN laboratories will continue to review these alternative DST methods for RGM (including E-test) and will adopt the preferred practical methodology when an international consensus is reached.

Guidelines for nucleic acid amplification tests

General requirements for a microbiology nucleic acid amplification facility

The National Pathology Accreditation Advisory Council has published standards and guidelines for laboratories performing NAAT.[20] The NPAAC document addresses specimen collection, transportation, reagent preparation, nucleic acid extraction, amplifi-

cation, product detection, data recording, reporting, sample storage and quality assurance. Laboratories performing NAAT for TB diagnosis must comply with these NPAAC recommendations. Some of the standards and guidelines of particular relevance to TB NAAT are highlighted below.

1. Samples that have been used for other tests prior to NAAT are at increased risk of cross-contamination. Wherever possible, NAAT should be performed on dedicated samples or on aliquots taken before other tests are performed.
2. The efficiency and quality of DNA extraction impacts greatly on the final test result. The extraction methods performed on various specimen types must be documented in the laboratory manuals and validated.
3. All NAAT methods must be properly validated before routine use. When a commercial test is used according to the manufacturer's instructions, no re-validation is required. Modified commercial tests and 'in house' methodologies must be validated as outlined in the NPAAC publication *Requirements for the validation of in-house* in vitro *diagnostic devices (IVDs).*
4. NAATs are capable of detecting very small quantities of nucleic acid and are therefore liable to false-positive results due to contamination events. Staff competence, laboratory design and routine use of controls limit and detect these contamination events. Three physically-separated areas are required in a NAAT laboratory for: DNA extraction, reagent preparation, and amplification/product detection. The movement of specimens and equipment shall be unidirectional from pre- to post-amplification areas. At least one negative control and a weak positive control must be subject to the whole test process including DNA extraction.

External quality assurance programs in the USA have demonstrated that laboratories performing TB NAAT but not conforming to these basic requirements have higher rates of false-positive reactions despite using FDA-approved commercial assays.[21]

Special considerations for a tuberculosis nucleic acid amplification facility

Brief literature review

Nucleic acid amplification tests for *M. tuberculosis* have the potential to rapidly determine whether a patient has TB, whether TB treatment is necessary, and whether infection control and contact tracing investigations are required. The features of some commercial NAAT are summarised in Table 2. Though theoretically able to detect a single copy of TB DNA with high specificity, NAAT has proven to have variable sensitivity compared with culture, particularly when investigating smear-negative respiratory specimens (Table 3). The predictive values of NAAT and the pre-test probability of TB in the patient population must therefore be considered when order-

Table 2. Description of some commercial nucleic acid amplification tests.*

NAAT	Method	Target	Sample volume (µl)	Detection	Assay time (hours)	Automation	IAC	FDA approval
AMTD2	TMA	16S RNA	450	Chemiluminescence	2.5	No	No	Yes
AMPLICOR	PCR	16S DNA	100	Colorimetric	6	Yes	Yes	Yes
DTB	SDA	IS6110	500	Fluorimetric	3	Yes	Yes	No

* AMTD2, Amplified *M. tuberculosis* Direct assay, Gen-Probe Inc, San Diego, Calif.; AMPLICOR *M. tuberculosis* assay, Roche Molecular System, Branchburg, NJ; DTB, BD ProbeTec energy transfer (ET) system, Becton Dickinson Biosciences Microbiology Products, Sparks, Md; TMA, transcription mediated amplification; SDA, strand displacement amplification; IAC, internal amplification control. Modified from Relevance of commercial amplification methods for direct detection of *Mycobacterium tuberculosis* complex in clinical samples.22

Table 3. Performance characteristics of some commercial nucleic acid amplification tests*

NAAT	Sensitivity (smear-positive, %)	Sensitivity (smear-negative, %)	Specificity (%)
AMTD2	90–100	63.6–100	92.1–100
AMPLICOR	87.5–100	17.2–71.7	91.3–100
DTB	98.5–100	33.3–100	96–99.8

* Modified from Relevance of commercial amplification methods for direct detection of *Mycobacterium tuberculosis* complex in clinical samples.22

ing and interpreting NAAT tests. Furthermore, the performance of NAAT on non-respiratory specimens has not been properly measured or validated.

Recognising the performance limitations of TB NAAT, the American Thoracic Society conducted a workshop to determine the appropriate use of these tests based on various clinical, laboratory and public health considerations.[23] These issues include:

- the expense of performing NAAT;
- the laboratory preparedness to perform NAAT on a regular basis;
- the cost-benefit of NAAT testing; and
- the interpretation of discrepant smear and NAAT results, particularly AFB-positive NAAT-negative results (i.e. such results may more likely represent the presence of NTM or a false-negative TB result depending on the patient's pre-test probability).

A model was constructed determining when the result of the NAAT test would produce a clinical or public health action. This model relied upon the pre-test clinical suspicion of TB, the AFB microscopy result, and the NAAT result. Treatment, isolation and contact tracing decisions were largely unaffected by NAAT results in smear-positive patients considered at high risk of TB and in smear-negative patients at low-risk of TB. Hence, NAAT testing in these circumstances may represent an inappropriate use of healthcare resources. In contrast, NAAT testing did affect clinical and public health decision-making in smear-negative patients at risk of TB and in smear-positive patients considered at low risk.

The Centers for Disease Control and Prevention updated their guidelines for TB NAAT testing and result interpretation.[24] These guidelines stated that the appropriate number of specimens to test with NAAT will vary depending on the clinical situation, the prevalence of TB, the prevalence of NTM, and laboratory proficiency. An algorithm was presented suggesting which specimens to test, when to perform tests for PCR inhibition, and the interpretation of discrepant smear- and NAAT results.

Basic principles about NAAT testing

The above preamble demonstrates that NAAT testing is only indicated in particular circumstances (e.g. smear-negative patients considered at high risk of TB), that these circumstances are the exact instances where NAAT performance is imperfect (e.g. NAAT detects only one-half to two-thirds of smear-negative TB patients), and that further clinical, public health, and economic research is required to determine the proper indications for TB NAAT testing. In the meantime, clinicians and laboratory staff must recognise the following principles:

1. NAAT is a supplemental test and does not replace smear microscopy or mycobacterial culture.
2. NAAT should not be performed automatically on every TB specimen or TB suspect.
3. As with all mycobacterial investigations, the decision to perform NAAT and the result interpretation requires close liaison between the clinician and laboratory staff.
4. Clinical material (e.g. cerebrospinal fluid) should not be preserved for NAAT if this compromises the ability to perform established tests of better diagnostic utility (e.g. culture).[24]
5. Experience is limited with NAAT on non-respiratory specimens and such testing has not been approved by the FDA.[23,24] Again, close clinical and laboratory consultation is required before deciding to perform NAAT on a non-respiratory specimen and particularly when interpreting the result.

A proposed algorithm for NAAT testing of respiratory specimens

Each mycobacteriology laboratory will need to develop a NAAT testing algorithm based on the above principles but also considering the characteristics of their patient population, the prevalence of TB and NTM cases in their locale, the potential sample load, and the laboratory size and resources.

One Australian MRL has developed the following guidelines that others could consider and modify depending on their local circumstances.

The use of NAAT for screening specimens from patients with suspected TB should be limited to:

- respiratory smear-positive specimens where the result is likely to influence clinical (treatment) and/or public health (isolation, contact investigation) decisions;
- respiratory smear-negative specimens from a patient with a high probability of TB, when prompt management and public health decisions are required; and
- selected non-respiratory specimens (e.g. meningeal, some tissue biopsies) where a prompt management decision is necessary (recognised that such tests have not been validated or approved).

The use of NAAT is considered inappropriate in the following instances:

- when a patient is respiratory smear-negative and has a low probability of TB;
- when a patient is respiratory smear-positive and has a very high probability of TB; and
- paucibacillary non-respiratory specimens (e.g. pleural fluid, ascitic fluid).

Staff screening and health care

Safety in the laboratory is the responsibility of management, the biosafety committee (BC), appointed safety officers, the laboratory supervisor, and the laboratory personnel. The Australian/New Zealand Standard 2243.3 *Safety in laboratories – Microbiological aspects and containment facilities* describes the detailed responsibilities of each of these groups.[8]

Personnel working in mycobacteriology laboratories require:

1. thorough initial training in TB laboratory procedures and safety measures;
2. on-going education; and
3. additional health checks.

All new staff should have a two-step tuberculin skin test (TST) performed. An initial positive result must be followed-up by chest X-ray (CXR) and a medical consultation. TST-negative staff members should be required to have annual skin tests; any TST conversion must be followed by CXR, medical examination, and consideration of chemoprophylaxis. Similar investigations should be instituted following a laboratory accident or known exposure event.

These annual screenings may fortuitously detect a recent TB infection. It is far more important that laboratory personnel are educated about the risks of TB, the likely presenting symptoms (e.g. chronic cough, weight loss, fever), and the need to inform their treating doctor that they work in a TB laboratory.

Laboratory personnel must also be informed of the medical conditions that increase the risk of progression to active TB disease (i.e. HIV infection, organ transplantation, steroid use, malignancy, chronic renal failure, diabetes). Personnel with these conditions can then be encouraged to discuss their situation with their treating physician and laboratory administration, and to find an alternative work environment within the microbiology laboratory.

Finally, the Australian/New Zealand Standard 2243.3 recommends vaccination as an additional safety measure for personnel working with *M. tuberculosis.*[8] The efficacy of BCG remains controversial with reported protection levels varying between 0–80 per cent.[25,26] Efficacy of vaccination in adulthood is even more controversial. Despite these uncertainties, interest in BCG vaccination has increased with the advent of MDRTB. BCG vaccination has negligible side effects and may provide some protection irrespective of the drug susceptibility status of the infecting strain. However, BCG vaccination confounds the alternative strategy of performing regular TSTs on HCWs and offering preventative therapy to 'converters'. Many HCWs do not comply with TST screening and preventative therapy for MDRTB-exposed individuals is problematic. In these uncertain circumstances, the following recommendations seem reasonable:

1. No benefit is to be gained from re-vaccinating laboratory personnel who have received BCG previously. This recommendation is true irrespective of the person's TST status.
2. Laboratory personnel should be required to participate in a TST and health screening program.
3. Non-vaccinated laboratory personnel at increased risk of MDRTB exposure (e.g. those working in laboratories performing DSTs) should be offered BCG after counselling about the advantages and disadvantages of the vaccination.

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Surveillance of antibiotic resistance in *Neisseria gonorrhoeae* in the WHO Western Pacific Region, 2004

The WHO Western Pacific Gonococcal Antimicrobial Surveillance Programme

Abstract

The World Health Organization Western Pacific Region Gonococcal Antimicrobial Surveillance Programme (WHO WPR GASP) examined approximately 10,000 isolates of *Neisseria gonorrhoeae* from 15 countries for resistance to antibiotics in 2004. Treatment options for gonorrhoea in the Region are limited by persisting high rates of resistance to penicillins and quinolones. There were infrequent instances of spectinomycin resistance and the presence of gonococci with decreased susceptibility to third generation cephalosporins was again noted in several centres. *Commun Dis Intell* 2006;30:129–132.

Keywords: antimicrobial resistance, disease surveillance, gonococcal, Neisseria gonorrhoeae, *World Health Organization Western Pacific Region*

Introduction

The World Health Organization (WHO) Western Pacific Region (WPR) has continuing and increasing problems with antimicrobial resistance in *Neisseria gonorrhoeae*. This has been documented by its Gonococcal Antimicrobial Surveillance Programme (WPR GASP). Penicillin resistance emerged and spread in the 1970s with the appearance of penicillinase-producing *N. gonorrhoeae* (PPNG) and gonococci resistant to penicillins by chromosomally mediated resistance (CMRNG). Since its inception in 1994, the WPR GASP has reported on the progressive increase in quinolone resistance in gonococci in the Region.[1,2] The rates of resistance to both of these antibiotic groups have been so high for so long that they should now only be used in programmatic treatments for infections acquired in this Region in specific situations where their efficacy is clearly demonstrated. Other established treatments for gonorrhoea have also suffered a loss of efficacy at different periods. Gonococci with high-level plasmid-mediated resistance to tetracyclines (TRNG) are frequently encountered in many centres,[2] spectinomycin resistance emerged rapidly when used widely in Korea in the 1980s[3] and more recently, there have been reports of the spread of gonococci with decreased susceptibility to third generation cephalosporins in Japan.[4]

Laboratory assessment of *in vitro* resistance to antibiotics in *N. gonorrhoeae* provides a reliable indication of the likely clinical efficacy of different treatment regimens. This report provides an analysis of antimicrobial resistance in *N. gonorrhoeae* in the WHO WPR derived from the results of the WHO WPR GASP surveillance for 2004.

Methods

The methods used by the WHO WPR GASP have been published[1] and provide full details of the source of isolates, sample populations, laboratory test methods and quality assurance programs used to generate data. These methods were unaltered in 2004. As a guide to the interpretation of the following data, a WHO expert committee has recommended that treatment regimens be altered once resistance to a particular antibiotic reaches 5 per cent.[5,6]

Results

Just over 10,000 gonococcal isolates were examined for susceptibility to one or more antibiotics in 15 participating countries in 2004.

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Quinolone antibiotics

Table 1 shows the distribution of quinolone-resistant *N. gonorrhoeae* (QRNG) in 13 countries that examined a total of 9,470 isolates in 2004. The proportion of QRNG found in isolates tested ranged from 2 per cent in New Caledonia and Papua New Guinea to nearly 100 per cent in the Hong Kong SAR and China. QRNG represented about 20 per cent of all gonococci tested in Australia and New Zealand, about 50 per cent were QRNG in Brunei, the Philippines and Singapore and 85 per cent or more in Japan, Korea, Laos and Viet Nam. These rates were in general higher than in previous years although decreases were noted in New Caledonia, the Philippines and Brunei when comparisons were made with 2003 data. Most of the resistance was at the higher level MICs (ciprofloxacin MIC ≥ 1 mg/L) that are associated with high rates of treatment failure.

Cephalosporins

Strains with some decrease in susceptibility to third generation cephalosporins were again detected in isolates from Australia, Brunei, China, and Papua New Guinea in 2004. Because of some methodological differences in testing, MIC values are not directly comparable between centres, but values ranged up to 0.25 mg/L.

Spectinomycin

A small number of spectinomycin resistant strains were reported from China. Only very small numbers of spectinomycin resistant gonococci have been reported in recent years in WPR GASP surveys.

Penicillins

Resistance to penicillins has been widespread and at high levels for many years in the WPR, and may be the result of penicillinase production or a combination of chromosomally mediated mechanisms. Table 2 shows the penicillin susceptibility of 9,983 gonococci in 15 WHO WPR centres. Little change was seen in 2004 from the generally high levels seen in previous years. There was an increase of note in PPNG in Brunei from 55 per cent in 2003 to 85 per cent, and a decrease in the Philippines from 78 per cent in 2003 to 37 per cent. The proportion of PPNG in Fiji increased to 6.4 per cent from the 3 per cent detected in 2003.

Tetracyclines

Tetracycline antibiotics are still widely available in the WPR. About 6,300 isolates were examined for one particular form of resistance, namely, that high-level plasmid-mediated form referred to as TRNG, in 10 countries in 2004 (Table 3). Again, rates of resistance, expressed as a percentage of all isolates tested, were similar to those found in 2003, except for increases in New Zealand where the proportion doubled in 2004 to 17.9 per cent, and Singapore (58% in 2003 and 72% in 2004), and a decrease in the Philippines from 29 per cent to 8 per cent. Low proportions of TRNG (< 10%) were found in Japan, Korea, New Caledonia, Papua New Guinea and the Philippines. The proportions of TRNG were from 14 to 34 per cent in isolates from Australia, China, New Zealand and Viet Nam while in Singapore TRNG were 72 per cent of 160 isolates tested.

Table 1. Quinolone resistance in strains of *Neisseria gonorrhoeae* isolated in 13 countries in the World Health Organization Western Pacific Region, 2004

Country	Tested	Less susceptible		Resistant		All QRNG	
	n	n	%	n	%	n	%
Australia	3,542	68	1.9	757	21.4	825	23.3
Brunei	113	15	13.3	46	40.7	61	54.0
China	1,203	60	4.9	1,135	94.3	1,195	99.2
Hong Kong SAR	2,811	144	5.1	2,647	94.2	2,627	99.3
Japan	261	16	6.1	213	81.6	239	91.6
Korea	93	17	18.2	65	70.0	82	88.2
Lao PDR	48	4	8.0	42	88.0	46	96.0
New Caledonia	43	0	0.0			1	2.3
New Zealand	773	14	1.8	148	19.1	162	20.9
Papua New Guinea	92	1	1.0	1	1.0	2	2.0
Philippines	175	2	1.1	83	47.4	85	48.5
Singapore	160	10	6.2	80	50.0	90	56.2
Viet Nam	156	49	31.4	82	52.5	131	83.9

QRNG Quinolone-resistant *Neisseria gonorrhoeae.*

Table 2. Penicillin resistance in 9983 strains of *Neiserria gonorrhoeae* isolated in 15 countries in the World Health Organization Western Pacific Region, 2004

Country	Tested	PPNG		CMRNG		All Pen R	
	n	n	%	n	%	n	%
Australia	3,542	393	11.1	377	10.6	770	21.7
Brunei	111	95	85.6	0		95	85.6
China	1,002 for PPNG	489	48.8		26.4		75.2
Fiji	606	39	6.4				
Hong Kong SAR	2,811	857	30.5	646	23.0	1,503	53.5
Japan	261	8	3.0	60	23.0	68	26.0
Korea	93	23	24.7	49	52.7	72	77.4
Lao PDR	48	40	83.0	8	17.0	48	100
New Caledonia	43					3	7.0
New Zealand	773	29	3.7	16	2.0	45	5.8
Papua New Guinea*	52 for PPNG 92 for all pen R	27	51.9			45	48.9
Philippines	175	65	37.1			90	51.4
Singapore	160	78	48.7	3	1.8	81	50.5
Tonga	110	3	2.7	6	5.4	9	8.1
Viet Nam	156	47	30.1	2	1.3	49	31.4

Papua New Guinea tested 52 of 92 isolates for lactamase production.

PPNG Penicillinase–producing *Neisseria gonorrhoeae*.

CMRNG Chromosome–mediated resistance *Neisseria gonorrhoeae*.

Table 3. High-level tetracycline resistance in strains of *Neisseria gonorrhoeae* isolated in 10 countries in the World Health Organization Western Pacific Region, 2004

Country	Number tested	TRNG n	TRNG %
Australia	3,542	490	13.8
China	1,202	411	34.2
Japan	261	6	2.3
Korea	93	2	2.1
New Caledonia	43	1	2.3
New Zealand	584	105	17.9
Papua New Guinea	92	4	4.3
Philippines	175	14	8.0
Singapore	160	115	71.9
Viet Nam	150	30	20.0

TRNG Tetracycline resistant *Neisseria gonorrhoeae*.

Discussion

Attempts to treat and control gonorrhoea are compromised by the emergence and spread of antibiotic-resistant *N. gonorrhoeae*. The data from 2004 indicate that the problems of providing efficacious treatment for gonorrhoea in the WHO WPR continue. The WHO recommends that standard treatment schedules should be changed when resistance to an antibiotic reaches a level of 5 per cent or more.[6] Resistance rates for the recommended cheaper oral agents such as the penicillins or quinolones remained well above this 5 per cent level in many centres and show no signs of decreasing. It is highly unlikely that effective newer derivatives from these antibiotic families will be developed.[7,8]

Alternative treatments are available but these either require intramuscular injection or else are more expensive than traditional agents. One group of antibiotics now widely used is the third generation cephalosporins, either as an oral preparation such as cefixime or cefdinir or the injectable ceftriaxone. The slow spread of gonococci with decreased susceptibility to third generation cephalosporins continues in the WHO WPR. After first reports from Japan, from 2000 onwards a small number of isolates with altered susceptibility to third generation cephalosporins has been reported each year in WHO WPR surveys in various countries. At different times Australia, Cambodia, Brunei, China, Japan, Korea, Malaysia, New Zealand, Papua New Guinea and Singapore have reported their presence. The reduced susceptibility is associated with the presence of a number of mosaic *penA* genes[4] and these gonococci are often

multi-resistant due to the aggregation of different resistance mechanisms.[9] These strains have now spread beyond the WHO WPR.[10,11]

Over-reliance on antibiotic treatment as a principal mechanism of gonococcal disease control in the absence of other important measures to decrease disease rates has undoubtedly contributed to the problem of antimicrobial resistance in *N. gonorrhoeae.*[12] The combination of high gonococcal disease rates and general problems of antibiotic resistance in the WHO WPR will continue without concerted efforts that simultaneously address the linked, but separate, issues of control of sexually transmitted diseases and containment of antimicrobial resistance.[8,12] Surveillance of antimicrobial resistance is an essential component of local, regional and international efforts for control of gonorrhoeae.

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National Rotavirus Surveillance Program annual report, 2004-05

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Abstract

The National Rotavirus Reference Centre together with collaborating laboratories Australia-wide has conducted rotavirus surveillance since June 1999. This report describes the serotypes of rotavirus strains responsible for the hospitalisation of children with acute gastroenteritis during the period 1 July 2004 to 30 June 2005. Six hundred and twelve faecal samples from across Australia were examined using monoclonal antibody immunoassays, reverse transcription-polymerase chain reaction and polyacrylamide gel analysis. Serotype G1 was the dominant serotype nationally, representing 48.3 per cent of all strains, followed by serotype G3 (36.7%) and serotype G9 (6.9%). As in previous years, there was substantial geographic variation in the prevalence of rotavirus serotypes. *Commun Dis Intell* 2006;30:133–136.

Keywords: rotavirus, annual report, disease surveillance

Introduction

Group A rotaviruses are the single most important cause of severe gastroenteritis in young children worldwide. An estimated 500,000 children die annually of severe diarrhoea, however, few of these deaths occur in developed countries.[1] Rotavirus induced disease accounts for up to 50 per cent of childhood hospitalisations for diarrhoea, with 10,000 Australian children hospitalised each year,[2] costing an estimated $26 million in direct costs. There is wide acceptance of the need for a vaccine to prevent rotavirus disease in children under 5 years of age throughout the world, as a component of the United Nations Millennium Development Goal 4 ('Reduce childhood mortality'). The first oral rotavirus vaccine was shown to be highly efficacious for the prevention of severe diarrhoea and hospitalisation due to rotavirus infection. A major setback was an apparent association with intussusception, a form of bowel obstruction in infants, which forced a withdrawal of the vaccine 9 months after introduction.[3] Two new rotavirus vaccines (Rotarix®, GlaxoSmithKline; and Rotateq®, Merck) have been developed, and are nearing licensure in many countries. National rotavirus surveillance is an important component in decisions about rotavirus vaccine implementation.

The previous rotavirus surveillance report from the National Rotavirus Surveillance Program, covering the period 1 July 2003 to 30 June 2004, documented the re-emergence of serotype G1 as the major serotype in Australia.[4] Prior to this, serotype G9 had been dominant in 2002/03, representing 74.7 per cent of samples nationally at that time.[5]

The surveillance and characterisation of rotavirus strains causing annual epidemics of severe diarrhoea in young children in Australia continues to be undertaken by the National Rotavirus Reference Centre in Melbourne, together with eight collaborating centres. In this report we describe the results for the period 1 July 2004 to 30 June 2005, and identify the geographic distribution of the predominant rotavirus serotypes.

Methods

Rotavirus positive specimens detected by enzyme immunoassay (EIA) or latex agglutination in collaborating laboratories were collected, stored frozen and forwarded to Melbourne, together with relevant age and sex details of patients. Specimens were then serotyped using an in-house monoclonal antibody (MAb) based serotyping EIA. The EIA employed a

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panel of MAbs specific for the major glycoprotein VP7 of the outer capsid of the five major group A human rotavirus serotypes (G1, G2, G3, G4 and G9).[6] Strains which could not be assigned a G serotype were genotyped by reverse transcription/polymerase chain reaction (RT/PCR), using serotype specific oligonucleotide primers.[7] Polyacrylamide gel electrophoresis (PAGE) was used to classify rotavirus strains genetically into electropherotypes, and to examine the extent of sharing of the same electropherotype between collaborating centres.

Results

Number of isolates

A total of 612 specimens were received for analysis from Melbourne and the collaborating centres in Western Australia, the Northern Territory, New South Wales, and South Australia. Five hundred and sixty-eight specimens were confirmed as rotavirus positive using our in-house EIA assay. Specimens containing insufficient specimen for testing, or specimens that were not confirmed to be positive for rotavirus (n=44) were not analysed further.

Age distribution

The overall age distribution of children with acute rotavirus gastroenteritis is depicted in the Figure. In the reporting period, 42.5 per cent of cases were from infants aged 12 months or less, 31 per cent were from patients 13–24 months of age, and 11.2 per cent were from patients 25–36 months of age. Overall, 84.7 per cent of samples were from aged children three years or less, and 90.8 per cent were from aged children five years or less. The male to female ratio was 1 to 1.

Children under 12 months of age were more likely to have a G3 infection (51.6%) than those aged 13–24 months (29.4%). In Alice Springs and Darwin 68.7 per cent of children with a serotype G3 infection were aged 12 months or less. Other serotypes (G1, G2 or G9) were equally common in children under 12 months (38.3%) and in those aged 13–24 months (31.5%).

Serotype distribution

The rotavirus serotypes identified in Australia from 1 July 2004 to 30 June 2005 are shown in the Table. Serotype G1 was the most common, representing 48.2 per cent of all specimens. It was the dominant strain in four of the eight centres (Melbourne, Sydney (POW, Westmead), Adelaide), and was the second most common type in the remaining centres. Serotype G3 was the second most common serotype nationally, and represented 36.6 per cent

Figure. Cases of rotavirus, Australia, 1 July 2004 to 30 June 2005, by age group

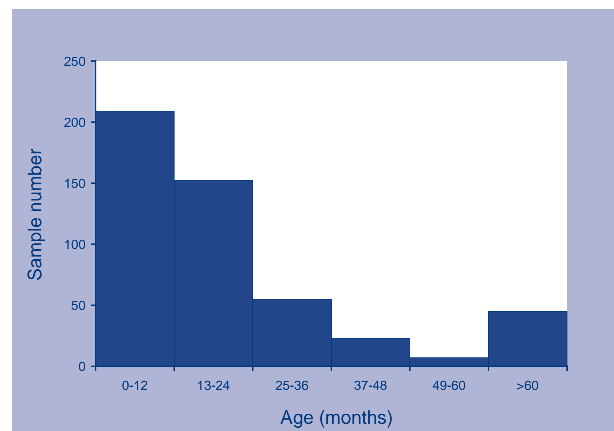

Table. Rotavirus G serotypes, Australia, 1 July 2004 to 30 June 2005

Centre	Total number	G1		G2		G3		G4		G9		Mix		NR	
		%	n	%	n	%	n	%	n	%	n	%	n	%	n
Melbourne	147	77.6	114	0.0		6.8	10	1.4	2	8.8	13	0.7	1	4.8	7
Sydney (POW)	44	56.8	25	2.3	1	0.0		0.0		38.6	17	0.0		2.3	1
Sydney (Westmead)	16	53.3	8	0.0		13.3	2	0.0		0.0		6.7	1	33.3	5
Perth[+]	98	34.7	34	2.0	2	58.2	57	0.0		5.1	5	0.0		0.0	
PathWest[†]	142	40.1	57	0.0		40.1	57	1.4	2	2.1	3	0.7	1	15.5	22
Alice Springs	55	1.8	1	0.0		98.2	54	0.0		0.0		0.0		0.0	
Darwin	46	30.4	14	4.3	2	60.9	28	0.0		0.0		4.3	2	0.0	
Adelaide	21	100	21	0.0		0.0		0.0		0.0		0.0		0.0	
Total	568	48.2	274	0.9	5	36.6	208	0.7	4	6.7	38	0.9	5	6.0	34

* An additional 44 specimens were omitted from analysis due to insufficient sample or specimen was not confirmed to be rotavirus positive.

† The two Western Australia centres represent different geographic areas, one urban (Perth) and one remote (PathWest).

of specimens overall. It was identified in six centres and was the dominant type in Western Australia and the Northern Territory. Serotype G9 was the third most common, but represented only 6.7 per cent of all specimens. It was identified in four centres, and was the second most common type in two centres (Melbourne and Sydney POW). Serotype G2 and G4 were each identified in three centres during the study, but each represented less than one per cent of the total strains identified.

Less than one per cent of the rotavirus samples contained multiple serotypes, and in 6.0 per cent of the samples a serotype was not identified. The latter could be samples with virus numbers below the detection limits of our assays, or could have contained inhibitors present in extracted RNA that prevent the function of the enzymes used in RT and/or PCR steps. It is unlikely that these represent unusual serotypes not identified using standard methods, since none of the non-typeable isolates exhibited unusual PAGE patterns. Future studies will include further characterisation of the genes encoding the outer capsid proteins of these strains.

Discussion

National rotavirus surveillance from 1 July 2004 to 30 June 2005 highlighted G1 as the dominant serotype nationally. Thus G1 has been the dominant type for the last two years.[4] It was identified in all centres; was dominant along the Eastern seaboard, in Melbourne and Sydney and Adelaide; and was the second most common serotype from Perth and PathWest. Previously, serotype G1 was dominant from 1999 to 2001.[8,9] The emergence of serotype G9 during 2002–2003, replaced G1 as the dominant serotype in Australia for a short time. Epidemiological studies conducted throughout the world continue to identify serotype G1 as the dominant serotype.[10,11]

G3 was the second most common serotype during this survey, continuing its emergence as a significant cause of acute gastroenteritis in Australia. A slight increase of G3 was seen in this survey, rising from a prevalence of 25.7 per cent during 2003/04 to 36.6 per cent in 2004/05. Of more significance was the finding that serotype G3 was dominant in both Western Australia and the Northern Territory. These serotype G3 strains may move eastward to Sydney and Melbourne, as was earlier seen with serotype G9. The initial major impact of G9 was seen in Western Australia, then the Northern Territory in following seasons, before becoming the dominant type in the eastern states.

The decline in prevalence of serotype G9 has been as dramatic as its emergence. G9 was first identified during Australia-wide surveillance in 1997,[12] and became the dominant strain nationally in 2001/02, comprising 40 per cent of the strains[13] and 74.7 per cent in 2002/03.[5] However, during the current survey, G9 was present in only four centres, and represented only 6.7 per cent of all strains. Thus serotype G9 has waned to become a minor cause of rotaviral disease in Australian children.

During the previous 2003/04 survey,[4] the serotype G3 strains seen in Western Australia infected children aged 13–24 months more frequently than children aged 12 months or less (P<0.001). In contrast, serotype G3 identified during the 2004/05 season has been associated with younger infants. Over 50 per cent of the children infected with a G3 strain were aged 12 months or less. However, these occurred mainly in the Northern Territory, where rotavirus infection in general appears to cause disease in a younger age group than in the rest of Australia.

The rotavirus serotyping results from this survey, together with those of previous years, highlight continuing changes in the prevalence and emergence of rotavirus serotypes. Multi-centre surveillance of rotavirus is important to continue to monitor strains in Australia, since state-to-state variation continues to be evident. This information is relevant to strategies about implementation of rotavirus vaccines.

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Influenza surveillance in Victoria, 2005

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Abstract

Influenza activity remained within normal seasonal activity with a well-defined peak at week 29 (beginning 18 July) during the Victorian influenza season from May to September 2005. Surveillance was based on sentinel general practice influenza-like illness (ILI) notifications with laboratory confirmation, medical locum service ILI notifications and laboratory notification of influenza detections. One thousand and eighty-seven consultations for ILI were reported from 38 general practices, while medical practitioners from the locum service reported 317 consultations for ILI. The average weekly rate of ILI from sentinel surveillance was 7.3 per 1,000 consultations. Similar numbers of influenza A subtypes H1N1 and H3N2 were detected; 45 per cent of which were A/California/7/2004-like (H3), 44 per cent were A/New Caledonia/20/99-like (H1) and 11 per cent were A/Wellington/1/2004 (H3). Of the influenza B samples, 67 per cent were B/Hong Kong/330/2001-like and 33 per cent were B/Shanghai/361/2002-like. The influenza vaccine for 2005 contained: A/New Caledonia/20/99(H1N1)-like virus, A/Wellington/1/2004(H3N2)-like virus, and B/Shanghai/361/2002-like virus. Although the predominant H3 and B circulating strains were not included in the vaccine, there was reasonable serological cross protection between vaccine and circulating strains. *Commun Dis Intell* 2006;30:137–143.

Keywords: disease surveillance, epidemiology, influenza

Introduction

Influenza surveillance in Victoria is conducted by the Victorian Infectious Diseases Reference Laboratory (VIDRL) and the Department of Human Services (DHS). Surveillance comprises notifications of laboratory confirmed influenza, 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 objectives of the influenza surveillance system are to:

- monitor the epidemiology of laboratory confirmed influenza in Victoria;
- identify the onset, duration and magnitude of annual influenza seasons in Victoria;
- 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; and
- provide a role in early recognition of new influenza viruses and new or emerging respiratory diseases.

Laboratory confirmed influenza in Victoria is a group B notifiable disease in accordance with the Health (Infectious Diseases) Regulations 2001. This report describes the results from influenza surveillance in Victoria for 2005 and comparison with previous years.

Methods

General Practice Sentinel Surveillance (coordinated by VIDRL)

In 2005, 74 general practitioners (GPs) from 23 metropolitan and 15 rural practices were recruited for sentinel ILI surveillance (Figures 1a and 1b), aiming to achieve a coverage of approximately one practice per 200,000 population in metropolitan Melbourne and one practice per 100,000 population in rural areas.[1] Participating GPs were provided with an incentive of Continuing Professional Development points from the Royal Australian College of General Practitioners or the Australian College of Rural and Remote Medicine. GPs were required to submit weekly reports on the total number of consultations; report the age, sex and vaccination status of patients presenting with ILI (defined as fever/history of feverishness, cough and fatigue/malaise); take a nose and throat swab from at least five patients with an ILI for respiratory virus testing at VIDRL; and complete an evaluation questionnaire.

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Figure 1a. Distribution of sentinel surveillance sites in metropolitan Victoria

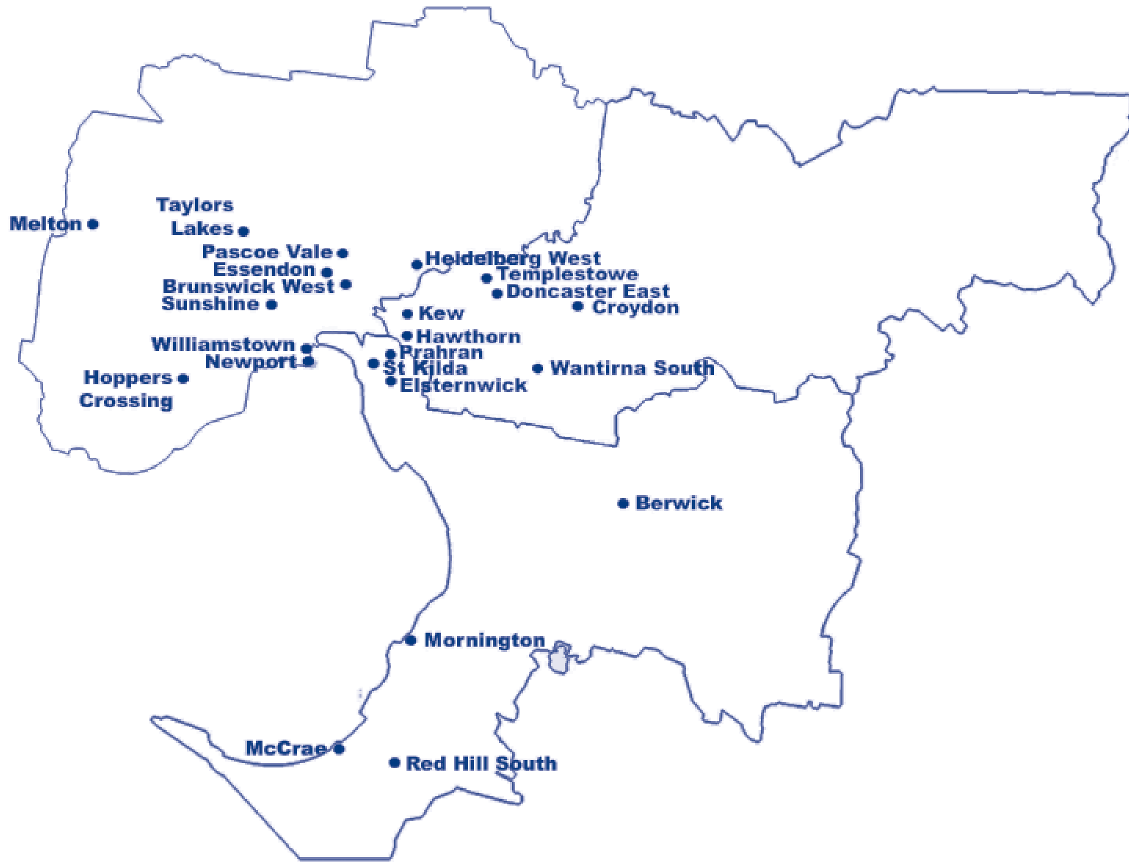
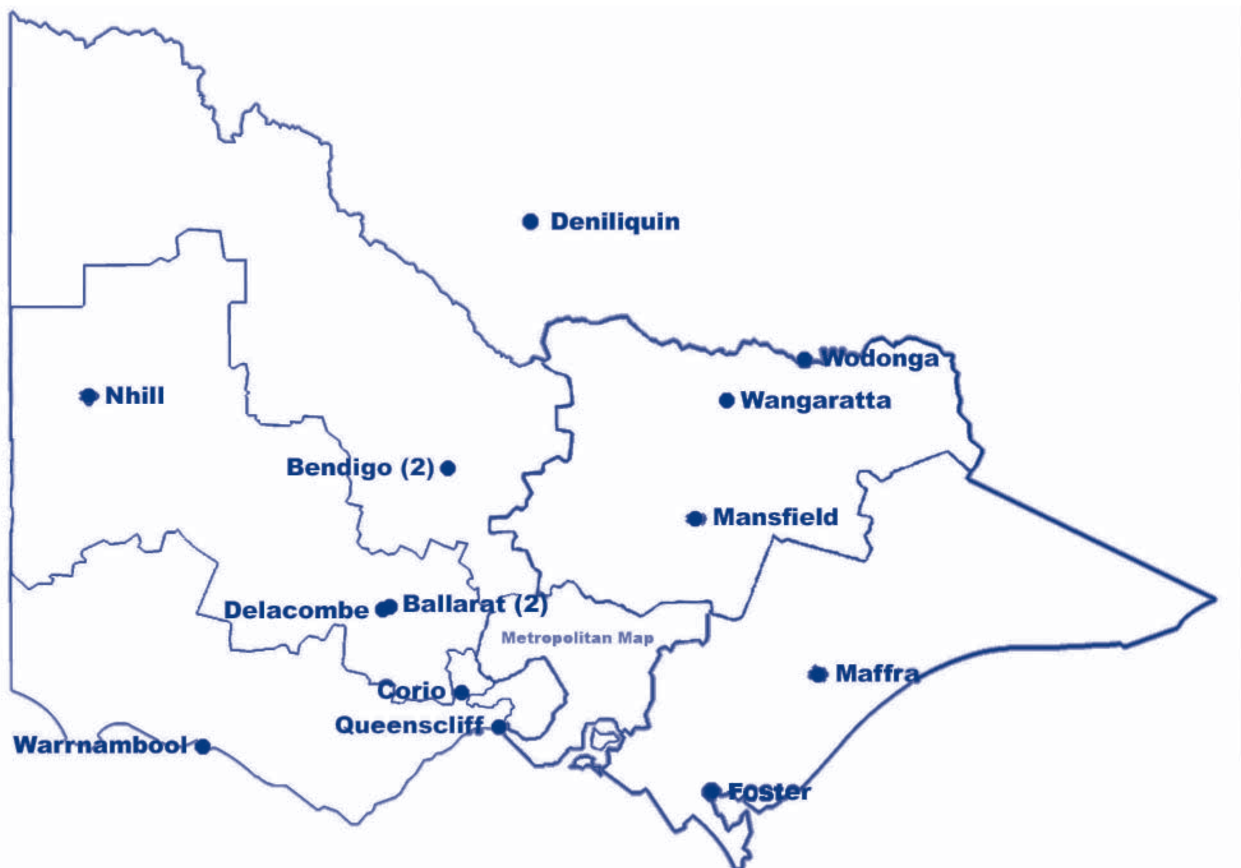


Figure 1b. Distribution of sentinel surveillance sites in rural Victoria



General practice sentinel surveillance was conducted for 22 weeks between 2 May and 2 October 2005 (weeks 18–39 inclusive). ILI activity for the year 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 2.5–15, higher than expected >15–35, and epidemic activity above 35.[2]

GPs were asked to collect swabs from patients within three days of onset of ILI symptoms and forward them in viral transport medium to VIDRL with data on: the patient's age; vaccine status; date of illness onset; and the GP's 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. Aliquots of all specimens positive for influenza were forwarded to the WHO Collaborating Centre for Reference and Research on Influenza, Parkville, Melbourne, for virus strain identification.

Melbourne Medical Locum Service Surveillance (coordinated by VIDRL)

ILI surveillance using MMLS data commenced in 2003. The MMLS provides a 24-hour, seven days a week medical locum service to patients within an approximate 35 kilometre radius of metropolitan Melbourne. Data were collected on cases with a final diagnosis reference to 'flu' or 'influenza'. ILI rates were calculated per 1,000 call-outs per week. Data from MMLS are collected all year.

Notifications of laboratory confirmed influenza (coordinated by DHS)

Cases of laboratory confirmed influenza are notified to the Department of Human Services under the Health (Infectious Diseases) Regulations 2001. In addition to VIDRL, which provided about half of all notifications, 11 other laboratories notified laboratory confirmed influenza. Notifications were extracted by date of notification from the Notifiable Infectious Diseases Surveillance database at DHS.

Data collation and reporting

All data from sentinel surveillance and MMLS were collected and collated weekly. ILI surveillance data were forwarded to the Australian Government Department of Health and Ageing weekly, and summary reports were prepared fortnightly and on an annual basis. Reports were distributed to interested health professionals, participating GPs and state and commonwealth departments of health. They were also posted on the VIDRL website (http://www.vidrl.org.au). Summary reports of laboratory confirmed influenza notifications made to the Victorian Department of Human Services were updated daily and posted on the Communicable Diseases Section website (http://www.health.vic.gov.au/ideas/surveillance/daily.htm).

Other influenza related studies

Two other studies were completed in 2005. The first study evaluated influenza surveillance in Victoria for the years 2002–04 using the evaluation framework for public health surveillance systems from the US Centers for Disease Control and Prevention.[4] The second study compared five available data sources for influenza and ILI in Victoria for timeliness and information provided. In addition to the three sources reported here, that is, notification of laboratory confirmed influenza, GP sentinel surveillance and MMLS surveillance, the comparison included data on emergency department admissions and hospitalisations for influenza and ILI.

Results

Participating sentinel practices

On average, 31 sentinel practices (82% of participating sites) reported each week from the 38 participating sentinel practices. There were 149,018 consultations of which 1,087 (0.7%) were for ILI during the reporting period, 2 May to 2 October 2005. The average weekly ILI consultation rate for the season was 7.3 per 1,000 cases: 7.5 per 1,000 cases in metropolitan sites and 6.4 per 1,000 cases in rural sites (Figure 2). The increase in ILI rates from the GP sentinel surveillance and the MMLS preceded the increase in notifications of laboratory confirmed influenza by two and four weeks respectively.

Figure 3 compares the ILI consultation rate in 2005 with rates from 1997 onwards. ILI in 2005 quickly rose to normal seasonal activity at week 19 (beginning 9 May). The consultation rate peaked at 13.1 per 1,000 cases, in the range of normal seasonal activity, similar to the 1999 season. Where gender was recorded, the male to female ratio for ILI in 2005 was 1:1.2 (502 and 582 consultations respectively).

Figure 2. Weekly reporting of notified cases of laboratory confirmed influenza and influenza-like illness from the Melbourne Medical Locum Service, metropolitan and rural sentinel sites, weeks 18 to 39, 2005

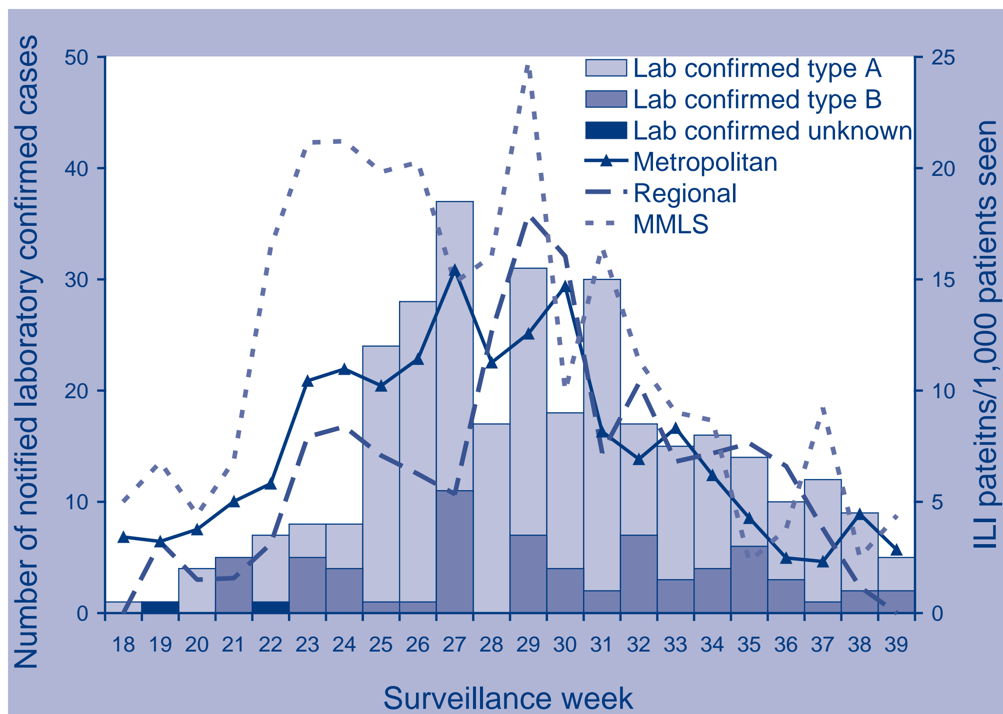
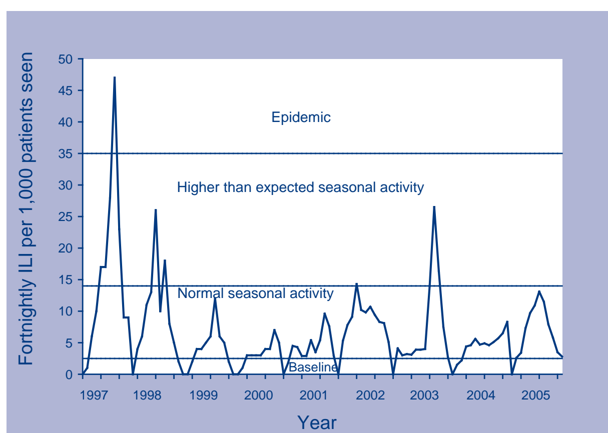


Figure 3. Fortnightly consultation rates for influenza-like illness, Victoria, 1997 to 2005



Sentinel surveillance GPs sent a total of 420 specimens for testing by PCR. Nineteen were inhibitory to the assay and were not included in the analysis. Of the 401 specimens remaining, influenza A was detected in 156 (38.9%) and influenza B in 26 (6.5%) specimens.

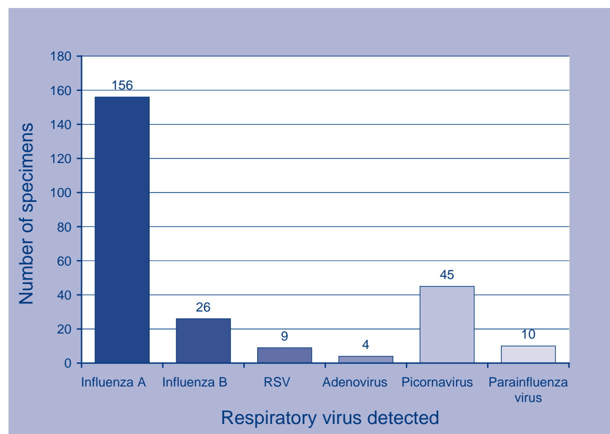
Aliquots of the 182 positive influenza samples were sent to the WHO Collaborating Centre for Reference and Research on Influenza for virus strain typing. Fifty-four per cent of these isolates were recovered: 88 per cent were influenza A and 12 per cent were influenza B. Of the influenza A samples 45 per cent were A/California/7/2004-like (H3), 44 per cent were A/New Caledonia/20/99-like (H1) and 11 per cent were A/Wellington/1/2004 (H3). Of the influenza B samples 67 per cent were B/Hong Kong/330/2001-like and 33 per cent were B/Shanghai/361/2002-like. The influenza vaccine for 2005 contained the following: A/New Caledonia/20/99(H1N1)-like virus, A/ Wellington/1/2004(H3N2)-like virus, and B/Shanghai/361/2002-like virus.[5] Although the predominant H3 and B circulating strains were not included in the vaccine, there was reasonable serological cross protection between vaccine and circulating strains.

Of the specimens received from sentinel surveillance 68 (17%) were positive for other respiratory viruses, the most common of which were picornavirus and parainfluenza virus (Figure 4).

Laboratory surveillance

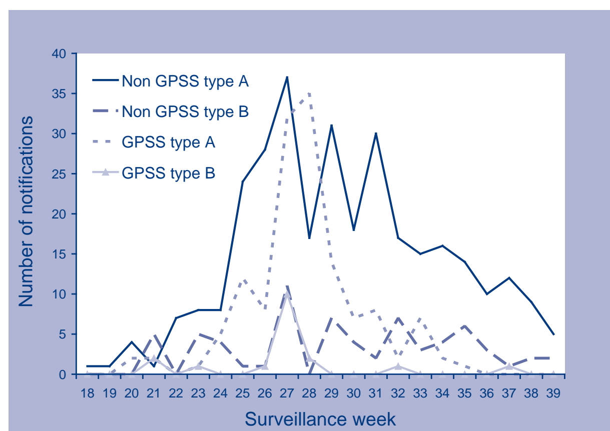
A total of 537 cases of laboratory confirmed influenza were notified to the Department of Human Services in the sentinel surveillance period between 2 May and 2 October. Of these, 156 (29%) were notified from positive specimens collected from the General Practitioner Sentinel Surveillance Program with the remainder from all other sources. There were 451 (84%) type A and 86 (16%) type B notifications. A slightly higher proportion of notifications from GP sentinel surveillance was type A (88% vs 82%). The female to male ratio was 1:1.1 with no significant difference by notification source.

Figure 4. Respiratory viruses detected from sentinel patients with influenza-like illness, Victoria, 2005



There was a sharper and later peak in influenza type A notifications from the general practitioner sentinel surveillance compared to other notifications (Figure 5). Relatively few notifications of laboratory confirmed influenza were received outside the GP sentinel surveillance period; 27 cases from 1 January to 1 May and 13 cases from 3 October to 30 November. Only one outbreak of influenza was notified in the year up to 30 November 2005. Reported in January, it occurred in an aged care facility with 19 people ill, of whom seven had laboratory confirmed influenza.

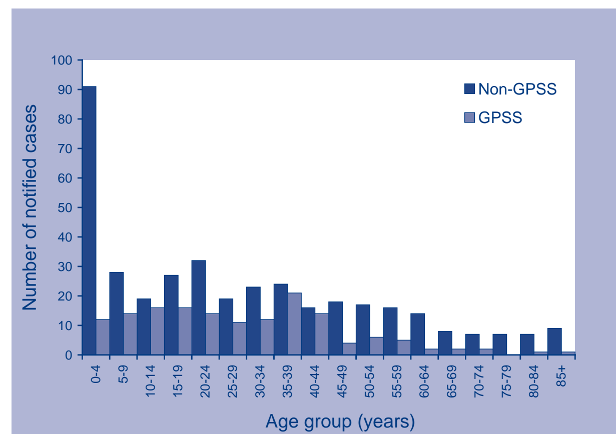
Figure 5. Laboratory confirmed influenza, Victoria, 2005, by type and notification source



GPSS General practice sentinel surveillance.

There was a marked difference in the age distribution of laboratory confirmed influenza notifications from general practitioner sentinel surveillance compared to other notification sources (Figure 6). Nearly one in four cases notified from non-sentinel practice surveillance was aged less than five years and there was a higher proportion of cases aged 65 years or older among non-sentinel surveillance notifications.

Figure 6. Laboratory confirmed influenza, Victoria, 2005, by age group and notification source



GPSS General practice sentinel surveillance.

Melbourne Medical Locum Service

During the surveillance period from week 18 to 39, MMLS recorded a total of 26,775 consultations of which 317 (1.2%) were for an ILI. The average ILI per 1,000 consultations was 11.8. Figure 2 shows a comparison of ILI rates for the influenza season between metropolitan and rural sentinel surveillance and MMLS data. MMLS surveillance continues all year and demonstrated a decline in ILI rates to below 2.0 ILI per 1,000 consultations by week 41.

Other influenza related studies

The evaluation of influenza surveillance was completed in 2005 and concluded with five recommendations summarised in the Box.

The review of relevant available emergency department and hospital admission data concluded that, while these data accurately reflected ILI activity in the community, they were not available in a timely fashion as currently collected, reviewed and made available. Each of the five data sources had different utilities. All contributed to describing inter-pandemic influenza seasonal activity but MMLS surveillance was the most efficient and could be managed with minimal extra resources, although it did not provide specimens for viral testing.[6]

Discussion

As measured by ILI rates from the GP Sentinel Surveillance Scheme, influenza activity in 2005 remained within the normal seasonal level with a well-defined peak of 13.1 ILI per 1,000 consultations during July. Although the *initial increase* in ILI activity occurred earlier for both sentinel and MMLS surveillance than for laboratory notifications, the

Box. Summary of recommendations arising from the evaluation of influenza surveillance in Victoria

1. Improve coordination between VIDRL and DHS, including weekly sharing of data between institutions and production of a single annual surveillance report.
2. Improve the quality of data for notifications of influenza held in the DHS database, including: follow-up of cases; ensuring notification of all laboratory-confirmed cases occurs; collation of strain information by DHS; adherence to serological case definitions for influenza, and; distinction in analysis and reports between the sources of laboratory-confirmed influenza notifications, namely GP sentinel surveillance and hospital laboratories.
3. All GP sentinel surveillance spatial data should be reported according to the geographic boundaries defined by DHS.
4. A review of the utility of available data on the impact of influenza on morbidity and mortality in Victoria should be conducted.
5. ILI data should be collected from Victorian emergency departments to provide an early warning system for epidemic influenza. Work in DHS on electronic syndromic surveillance may provide a simple and timely method of capturing these data.

peak in laboratory notifications occurred two weeks earlier than it did in the community surveillance systems. This may reflect a delay in notification of the increase in ILI rates observed at the start of the season. It is not clear why the onset of seasonal activity was seen earlier in locum service, compared with sentinel surveillance, but may represent earlier contact with the medical system by patients who are more acutely ill and who require after-hours care. However the early rise in ILI activity detected by community surveillance reinforces the importance of these systems in detecting increased influenza activity in the community.

The GP Sentinel Surveillance Scheme also serves an important function in notifiable disease surveillance by making a substantial contribution to the total laboratory confirmed influenza cases (nearly 30%) notified during the influenza season. However, given the differing age distributions of these patients, sentinel patients may not be representative of all patients with influenza in the community. Similarly, cases identified as part of outbreaks may skew data describing the epidemiology of community-acquired influenza.

Both influenza A sub-types, H3N2 and H1N1, circulated throughout the season. Most influenza A viruses were reported as either A/California/7/2004-like (H3) or A/New Caledonia/20/99-like (H1), both of which have been included in the 2006 vaccine. A/Wellington also circulated in smaller numbers throughout the whole season. Both A/New Caledonia and A/Wellington were covered by the 2005 vaccine. Circulating influenza B was either B/Shanghai/3611/2002-like or B/HongKong/330/2001-like. B/Shanghai was covered by the 2005 vaccine.

The vaccine for the 2006 season will contain the following:[7]

- A/New Caledonia/20/99 (H1N1)-like virus
- A/California/7/2004 (H3N2)-like virus
- B/Malaysia/2506/2004-like virus

The data received from MMLS reflected that of metropolitan surveillance. Following analysis and confirmation that MMLS is an appropriate data source for ILI surveillance, MMLS has been adopted as an integral component of influenza surveillance in Victoria.[8] MMLS data are collected all year round and will continue to supplement the sentinel surveillance data in 2006, including during the Melbourne Commonwealth Games. In light of the findings from the evaluation, coordination of surveillance activities between DHS and VIDRL has been improved.

Acknowledgements

The sentinel influenza surveillance program would not be possible without the ongoing support of the participating general practitioners and their practice staff. The Viral Identification Laboratory at VIDRL is responsible for the laboratory diagnosis for surveillance cases. We thank other laboratories for notification of influenza to DHS and the WHO Collaborating Centre for Reference and Research on Influenza for provision of influenza strain identification. Dr Ian Barr of the WHO Collaborating Centre provided advice on the serological match of vaccine and circulating influenza virus strains. Our thanks also go to the Melbourne Medical Locum Services for sharing their data with us. We also acknowledge the private pathology providers who facilitate transport of respiratory specimens from rural and regional general practices. Albert Tiong was responsible for the evaluation of the influenza surveillance system in Victoria. VIDRL

receives a contribution for its influenza surveillance program from the Department of Human Services, Victoria.

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Creutzfeldt-Jakob disease: Australian surveillance update to December 2005

Genevieve M Klug, Alison Boyd, Victoria Lewis, Samantha L Douglass, Rebecca Argent, James S Lee, Colin L Masters, Steven J Collins
Australian National Creutzfeldt-Jakob Disease Registry

Abstract

Australia-wide prospective surveillance of human transmissible spongiform encephalopathies (TSEs) has been conducted by the Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR) since October 1993. In addition, the Registry retrospectively ascertained TSE cases within Australia from 1970. Referrals of all suspect cases of human prion diseases or TSEs are investigated by the ANCJDR and include Creutzfeldt-Jakob disease (CJD), Gerstmann-Sträussler-Scheinker syndrome, fatal familial insomnia and variant CJD. This semi-annual progressive update presents epidemiological findings of the ANCJDR based on Australian data obtained for the period 1970 to 31 December 2005. *Commun Dis Intell* 2006;30:144–147.

Keywords: disease surveillance, Creutzfeldt-Jakob disease, transmissible spongiform encephalopathies

Surveillance summary to 31 December 2005

From 1 October 1993 to 31 December 2005, the ANCJDR has been notified of 1,061 suspect transmissible spongiform encephalopathy (TSE) cases in Australia whose disease onset and death occurred after 1 January 1970. Of these, 447 cases have been excluded as non-TSE cases. Based on the EUROCJD diagnostic criteria,[1] 504 cases have been classified as definite (311) or probable TSE (193) and a further eight cases have been classified as possible (Table 1). The majority of the definite and probable cases are sporadic (90.4%), while the remainder are familial (8%) and iatrogenic cases (1.6%). The eight possible cases have been classified as sporadic (7) and iatrogenic (1). One-hundred and two cases are currently under investigation with 55 of these cases still alive. During 2005, 66 new suspect cases have been evaluated by the ANCJDR. Nine of these cases have been excluded from the register, 44 remain under review, and 13 have been classified as definite cases after neuropathological examination. As of 31 December 2005, no variant CJD cases and no further iatrogenic CJD cases have been identified in Australia.

Table 1. Classification of ANCJDR cases, 1 January 1970 to 31 December 2005

Classification	Sporadic	Familial	Iatrogenic	Variant CJD	Unclassified	Total	Cases classified during 2005*
Definite	275	31	5†	0	0	311	+18
Probable	180	9	4	0	0	193	+7
Possible	7	0	1	0	0	8	+1
Incomplete	0	0	0	0	102‡	102	+16
Total	462	40	10	0	102	614	+42

* Describes the classifications made during the 2005 surveillance year (includes cases notified in 2005 or previous years).

† Includes one definite iatrogenic case who received pituitary hormone treatment in Australia but disease onset and death occurred while a resident of the United Kingdom. This case is not included in statistical analysis since morbidity and mortality did not occur within Australia.

‡ Includes 55 living cases.

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For the prospective ascertainment period of 1993 to 2005, an average of 82 suspect cases per year have been notified to the ANCJDR for investigation. During this period, annual notifications have varied, which may reflect in part the varied ascertainment methods employed by the ANCJDR during particular time periods as previously described[3] (Figure 1). Since 2000, the annual number of notifications has been relatively constant (approximately 60 suspect cases notified per year). A comparison of the notifications by state and territory between 1993 to 2005 shows that the number of notifications has been relatively stable in the larger states, while in more recent years, notifications have slightly declined in several of the states and territories with smaller populations (Figure 1, Table 2).

Figure 1. Notifications of suspect cases to the ANCJDR, 1993 to 2005, by state or territory

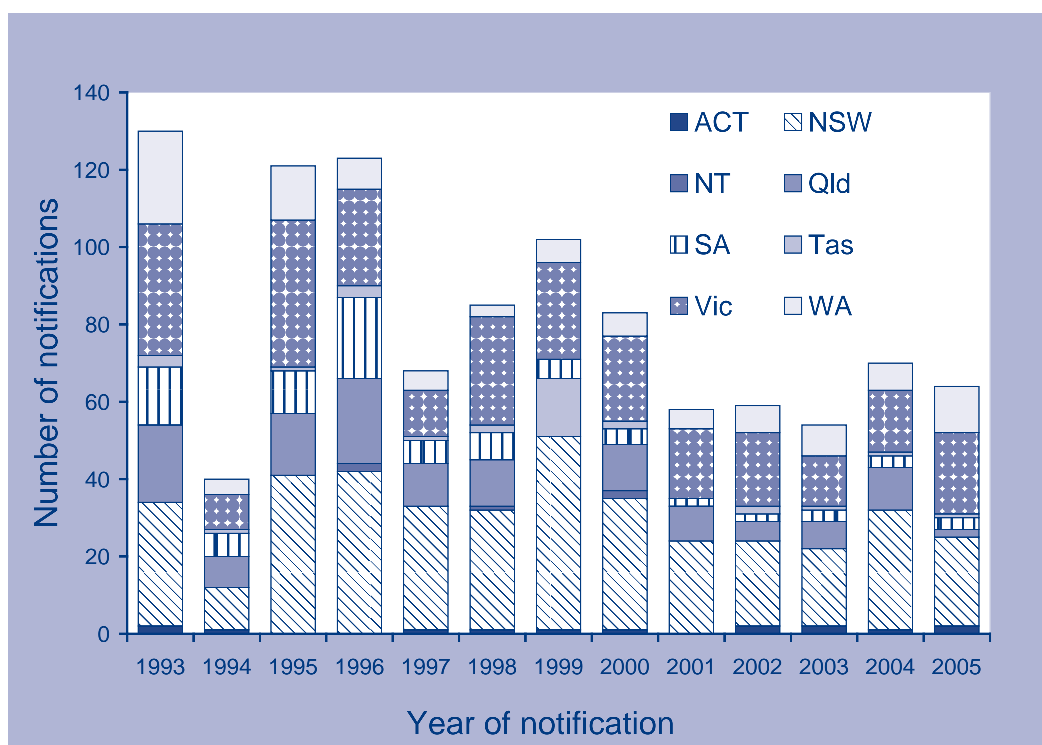


Table 2. Number of suspect cases notified with or without post mortem examinations, 1998 to 2005, by state or territory

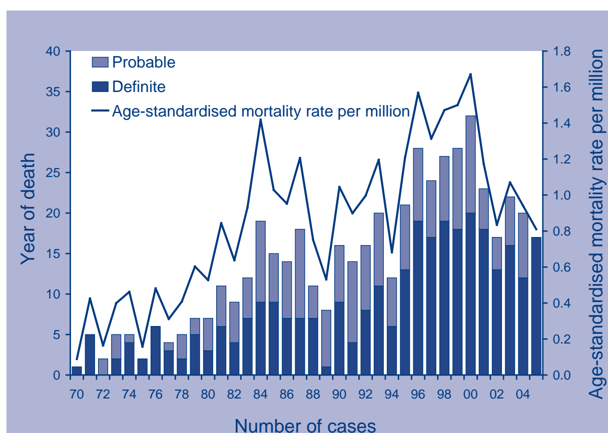
Death year	ACT		NSW		NT		Qld		SA		Tas		Vic		WA	
	State or territory of suspect cases*															
	n	PM	n	PM	n	PM	n	PM	n	PM	n	PM	n	PM	n	PM
1998	1	0	17	8	1	0	4	3	4	3	0		10	6	3	2
1999	1	0	20	11	0		9	6	5	4	0		9	5	1	1
2000	0		21	13	1	0	9	5	2	2	2	0	13	9	5	4
2001	1	0	22	12	0		8	7	2	1	0		13	6	3	1
2002	2	0	17	9	0		5	4	2	2	3	2	15	5	5	3
2003	0		18	13	0		5	2	3	2	0		11	6	8	2
2004	1	0	20	9	0		5	2	2	1	0	1	8	6	2	1
2005	0		17	9	0		1	0	0		1		13	10	6	2
Total	5	0	152	84	2	0	4	29	20	15	6	3	92	0	38	16

* Where death is known to have occurred.

PM Post mortem

A clear increase in the annual incidence of Australian TSEs was observed during the period of 1970 to 2000 (Figure 2). This increase has also been observed in international CJD surveillance units and probably reflects case ascertainment bias associated with heightened recognition and case notification as well as improved investigation and case confirmation.[4] Since 2000, the number of TSE deaths has declined and stabilised at approximately 20 cases per year. In particular, a decline in the number of probable cases has been observed and this probably relates to a number of issues, including broadened surveillance responsibilities and difficulties encountered following changes to privacy legislation. For the period of 1970 to 2005, the average annual age-adjusted mortality rate was 0.85 deaths per million per year. During the prospective period of ANCJDR surveillance from 1993 to 2005, the average annual rate of mortality

Figure 2. Number and age-standardised mortality rate of ANCJDR definite and probable cases, 1970 to 2005



Mortality rates were calculated using the Australian Bureau of Statistics 2000 resident population estimates for Australia.

was 1.19 deaths per million persons which is similar to the rates reported by other countries undertaking prospective ascertainment.[4]

Of the 455 sporadic cases, 53 per cent were female and 47 per cent were male. This ratio has been consistently observed and suggests no sex predilection. The median age of death is 67 years (females, 68 years; males, 66 years) with a range of 25-89 years. The median illness duration from onset to death of sporadic CJD cases is four months (females, 4 months; males, 3 months); however duration ranges from 0.9 to 60 months. Overall, the 70-74 year age group had the highest mortality rate from sporadic CJD with 4.2 cases per million per annum. In females, the mortality rate peaks in the 65-69 year age group with 4.8 cases per million per annum, whereas in males, the maximum rate is 4.1 cases per million per annum in the 70-74 year age group.

Familial CJD similarly shows no clear sex bias. Of the 40 cases, 55 per cent were female and 45 per cent were male. Typically, the duration of disease and age at death of familial cases is longer and younger respectively, when compared with sporadic CJD.[4] The median duration of Australian familial cases was found to be seven months (range, 1.5-192 months) which is significantly longer than the duration of sporadic CJD cases ($p<0.0001$ by Log Rank Test). Median age at death was 59 years but has been as young as 20 years or as old as 82 years. When comparing male to female cases, death occurred at an earlier age in males (median, 51 years; range, 20-82 years). For females, median age at death was 62.5 years (range, 38-82 years). Mortality rates in familial cases peaked in the 65-69 year age group for both males and females and overall, the rate of death was 0.33 familial cases per million per annum in this age group.

A total of nine iatrogenic cases of CJD have been identified in Australia. These comprise five dura mater graft-related cases (2 definite, 3 probable), three human-derived pituitary gonadotrophin-related cases (2 definite, 1 probable) and one possible human-derived pituitary growth hormone case. One additional case of gonadotrophin-related CJD received treatment in Australia, but disease onset and death occurred while a resident in the United Kingdom and thus is not included in the analysis of Australian cases. Of the dura mater cases, 80 per cent were male and the median age at death was 46.5 years (range, 27-62 years) with median duration 2.5 months (range, 2-10 months). In the remaining female case, death occurred at 26 years after a duration of disease of 3.5 months. In the three female gonadotrophin-related cases, the median death age was 41 years (range, 37-50) and duration was longer than the dura mater-related cases at 10 months (range, 3-25 months). As of 31 December 2005, no further cases of iatrogenic CJD have been detected since the last identified dura mater-related CJD case in 2000.

Analysis of the distribution of CJD cases according to state and territory shows an alignment with the relative population distributions (Table 3). No significant increase in mortality from sporadic CJD has been observed for a particular state or territory when compared to the national rate, indicating no increased or decreased risk. A comparison of the temporal pattern of CJD case numbers and average annual crude incidence for each state or territory between 1993 to 2005 (Table 3) suggests that in a number of states or territories of Australia, there is a decline in the ascertainment of CJD cases in

Table 3. Australian transmissible spongiform encephalopathy (TSE) deaths, 1993 to 2005, by state or territory

State or territory	TSE cases by year of death													Total	Mean crude mortality rate (deaths/million/yr)
	93	94	95	96	97	98	99	00	01	02	03	04	05		
ACT		1					1			1		1		4	0.98
NSW	2	3	7	6	10	10	13	12	9	4	7	11	5	99	1.18
NT						1								1	0.41
Qld	5	2	5	6	3	3	7	7	3	3	3			47	1.05
SA	1	3	2	3	3	1	3	2			1	1		20	1.04
Tas				1						2			1	4	0.65
Vic	10		4	8	5	9	3	9	10	5	8	5	9	86	1.40
WA	2	3	3	4	3	3	1	2	1	2	2	2	2	30	1.27
Total	20	12	21	28	24	27	28	32	23	17	21	20	17	291	1.18

more recent years. In some regions this coincides with lower notifications to the ANCJDR and fewer post-mortems of suspect cases (Table 2). In the more populous states and territories, the number of CJD cases and those suspect cases that are investigated by post-mortem examination have remained constant. Overall, post-mortem examination has been performed on 56 per cent of all Australian CJD cases and 55 per cent of all suspect cases notified to the ANCJDR where death is known to have occurred since 1993.

Since May 2003, six Australian states and territories have included CJD as a notifiable disease. To date, no clear increase of suspect case notifications has been observed. In contrast, notifications to the ANCJDR have slightly decreased in regions where CJD has not been scheduled as a notifiable disease. The ANCJDR will continue to evaluate the influence of compulsory notification on CJD notifications.

Acknowledgements

The ANCJDR wishes to thank families, medical practitioners and associated staff for their generous support of Australian CJD surveillance. The ANCJDR also thanks Dr Handan Wand, Dr Matthew Law and Professor John Kaldor (National Centre in HIV Epidemiology and Clinical Research at the University of New South Wales) for their expert epidemiological and statistical support.

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OzFoodNet: enhancing foodborne disease surveillance across Australia: Quarterly report, 1 October to 31 December 2005

The OzFoodNet Working Group

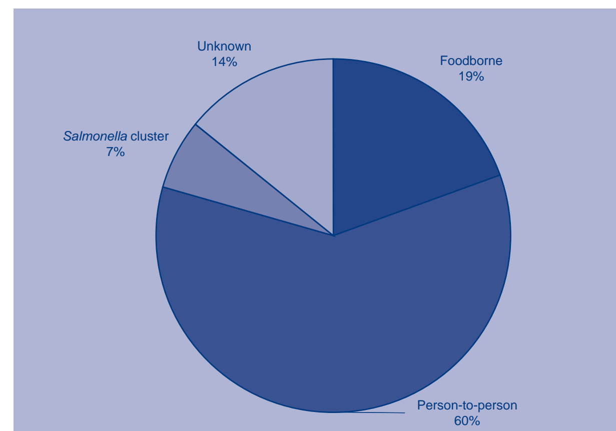
Introduction

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 around the country between 1 October and 31 December 2005.

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 results of outbreak investigations can take months to finalise. We would like to thank 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.

During the fourth quarter of 2005, OzFoodNet sites reported 185 outbreaks of enteric illness (including foodborne outbreaks). 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 more than 3,692 people and resulted in 142 persons being admitted to hospital. Five deaths were reported. As has been the case in previous reports, the majority (60%, n=111) of outbreaks resulted from infections suspected to be spread by person-to-person transmission (Figure 1). Fifty-three per cent of these person-to-person outbreaks occurred in aged care facilities, 16 per cent in hospitals and 16 per cent in child-care centres.

Figure 1. Mode of transmission for outbreaks of gastrointestinal illness reported, Australia, 1 October to 31 December 2005



Foodborne disease outbreaks

There were 36 outbreaks of illness where consumption of contaminated food was suspected or proven to be the primary mode of transmission. These outbreaks affected 571 people. This compares with 26 outbreaks for the fourth quarter of 2004 and 30 outbreaks in the third quarter of 2005.

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All data are reported using the date the report was received by the health agency.

Salmonella was responsible for 13 outbreaks during the quarter, with *Salmonella* Typhimurium being the most common serovar. *S.* Typhimurium 44 and *S.* Typhimurium 135a were responsible for four outbreaks each; *S.* Typhimurium 197 and *S.* Typhimurium 170/108 were each responsible for one outbreak. Other *Salmonella* serovars causing single outbreaks were *S.* Potsdam, *S.* Birkenhead and a combined outbreak of *S.* Saintpaul and *S.* Chester. *Campylobacter* was responsible for five outbreaks. *Clostridium perfringens*, ciguatera fish poisoning and norovirus were each responsible for two outbreaks. Scombroid poisoning and *Listeria monocytogenes* serotype O1 also each caused an outbreak during the fourth quarter of 2005. No aetiological agent was identified for the remaining 28 per cent (10/36) of outbreaks.

Eleven of the outbreaks reported in the quarter were associated with meals served in restaurants, five from food prepared in private residences (including two instances that can be attributed to contaminated fish), three in bakeries and two each in aged care facilities, takeaway outlets, nationally franchised fast food outlets, commercial caterers and at camps. Single outbreaks were associated with food prepared in a child-care centre, an institution, a hospital, a school and a grocery store/deli and an unknown setting. One outbreak was caused by contaminated town drinking water. Eleven of the outbreaks occurred in October, seventeen in November and eight in December.

To investigate these outbreaks, sites conducted 10 cohort studies and two case control studies. For 20 outbreaks, only descriptive data were collected. Individual patient data was not collected for four outbreaks. Investigators obtained microbiological evidence linking a food vehicle to illness in three outbreaks, microbiological evidence and analytical epidemiological evidence in three outbreaks and analytical epidemiological evidence alone in four outbreaks. For the remaining outbreaks, investigators obtained descriptive epidemiological evidence implicating the food vehicle or suggesting foodborne transmission.

In New South Wales there were 10 outbreaks of foodborne illness reported during the quarter. The aetiological agent was identified in seven of these outbreaks. *S.* Typhimurium 197 affected 33 children and staff at a child-care centre. The food vehicle was suspected to be contaminated fruit and sandwiches prepared at the centre. Twenty-three people were ill in a community outbreak of *Clostridium perfringens* infection following a meal of fried rice. Eight people were infected with *S.* Typhimurium 44 from Caesar salad dressing made with raw eggs and served alone or on chicken wraps at a restaurant. *Campylobacter* affected five people after they had eaten takeaway food such as sandwiches and salads prepared at a hospital kiosk by an ill food handler. A second outbreak of *Campylobacter*, in which three people were ill, was thought to have been caused by the consumption of takeaway chicken from a nationally franchised food company. Four people were ill with symptoms consistent with histamine poisoning after eating tuna steaks prepared at home. One sample of tuna from the retailer showed high levels of histamines. Three residents of a residential facility were infected with *Salmonella* Birkenhead, one of whom died. Pureed food was thought to be the source of infection. An aetiological agent was not identified in the remaining three outbreaks.

Queensland reported 11 outbreaks of foodborne illness for the fourth quarter. Twenty-six people were affected in an outbreak of *Salmonella* Chester and *Salmonella* Saintpaul associated with contaminated drinking water. A case control study showed that cases were more likely to have consumed unboiled or filtered tap water (Odds Ratio 11.1, 95 per cent Confidence Interval 1.4–90, p=0.007). *Salmonella* Chester was isolated from unchlorinated drinking water and *Salmonella* Chester and *Salmonella* Saintpaul were isolated from effluent samples collected from a nearby abattoir. Two outbreaks in Queensland were caused by ciguatera fish poisoning following meals of barracuda bought at a retail outlet and then cooked at home and yellowtail kingfish caught off Fraser Island by a camping group. There were three outbreaks caused by *S.* Typhimurium 44, two of which occurred in private homes. The first affected 23 people and was caused by cold prawn soup prepared by ill food handlers. The second outbreak affected three people after eating rolls containing bacon and runny eggs. The third outbreak involved two children at a camp, with anecdotal evidence of further cases, although no food vehicle was identified. An outbreak of *Clostridium perfringens* infection affected 14 people after a restaurant meal of lamb or chicken guvec (Turkish casserole). Low to moderate counts of *C. perfringens* were detected in leftover chicken guvec from a different night and low counts were detected in cooked chicken meat and lamb meat. Six residents from four aged care facilities with a common supplier of meals were ill in an outbreak of *Salmonella* Potsdam. No food source was identified and two cases died. Raw chicken kebabs purchased from a grocery store and cooked at a picnic were thought to be the source of a *Campylobacter* outbreak affecting four people. An aetiological agent was not identified in the remaining two outbreaks, but both were suspected to be caused by toxins.

Victoria reported six outbreaks of foodborne disease for the quarter. Roast baby pigs supplied to a function by two separate restaurants was suspected as the cause of an outbreak of *S.* Typhimurium 170 affecting 20 people. A cohort study of 120 people

was unable to identify the food vehicle, as only 50 per cent of the cohort was interviewed and most interviewees were unable to identify which pig they had consumed. Vanilla slices purchased from a bakery were the cause of an outbreak of norovirus in a workplace. The proprietor of the cake shop prepared the cakes while ill with gastroenteritis, which was later confirmed as norovirus. No food vehicle was identified in an outbreak of *Campylobacter* affecting five people in one work place. An aetiological agent was not identified in the remaining three Victorian outbreaks.

Tasmania reported four outbreaks of foodborne disease for the quarter, all of which were part of a larger state-wide outbreak of *S.* Typhimurium 135a. Two of these outbreaks followed the consumption of food from bakeries and affected 107 and 6 people, respectively. The other two outbreaks involved food prepared in restaurant settings and affected 77 and 11 people respectively. The outbreaks were all thought to be related to foods containing raw or undercooked eggs or foods cross contaminated by eggs. *S.* Typhimurium 135a was isolated from the cream in cakes and piping bags from one bakery and raw egg mayonnaise and raw egg tartare sauce from a restaurant. Several epidemiological studies of the state-wide *S.* Typhimurium 135a outbreak were conducted and traceback revealed that eggs purchased by the bakeries and restaurants had a common supplier. *S.* Typhimurium 135a was isolated from drag swabs taken from laying sheds on the egg farm.

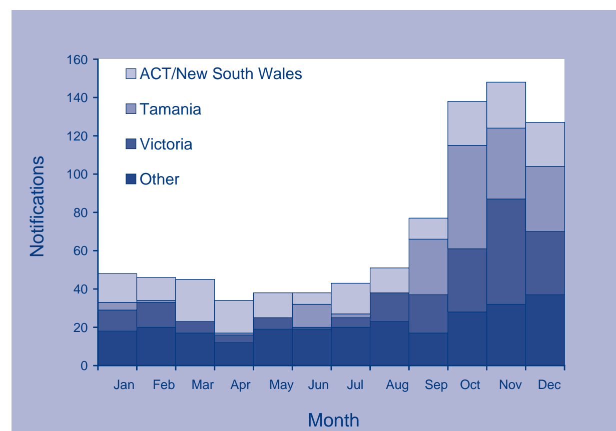
South Australia reported three foodborne outbreaks for the fourth quarter. Cold meats were responsible for an outbreak of *Listeria monocytogenes* O1 in a hospital. Two of the cases had admissions at a common hospital and had identical *Listeria* isolates by pulsed field gel electrophoresis (PFGE). One of the cases who had complications from diabetes, died. Cold cooked corned beef slices and mixed meat and salad sandwiches from the hospital kitchen were positive for *Listeria monocytogenes* serotype O1 and had the same PFGE as the patients' sample isolate. Food samples from the meat manufacturer that supplied cold meat to the hospital was also positive for *Listeria monocytogenes*. Two other cases each had isolates of *L. monocytogenes* with unique PFGE patterns that were different from the other cases and to isolates from food samples. A smallgoods company issued a consumer level recall for a range of ready-to-eat products. Dips were believed responsible for an outbreak of norovirus which affected 21 people from five different groups who ate at, or purchased dips from a restaurant over a period of three days. No food vehicle was identified in an outbreak of *Campylobacter* at a college boarding house where 36 people were ill.

Western Australia reported two foodborne outbreaks for the quarter. Both occurred in restaurant settings, were of unknown aetiology and no food vehicles were identified. The Australian Capital Territory and the Northern Territory did not report any foodborne outbreaks for the fourth quarter of 2005.

Clusters and multi-state investigations

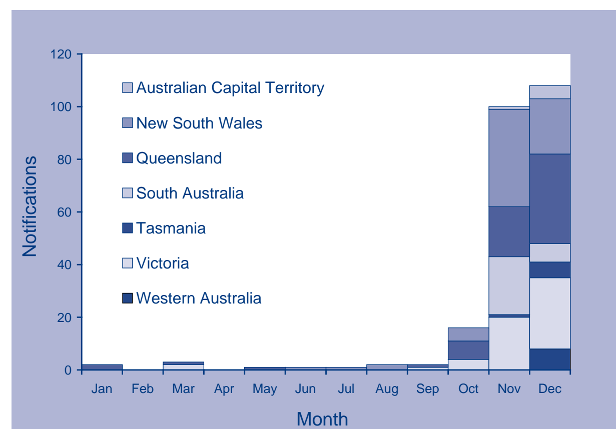
During the quarter, OzFoodNet investigated a multi-state outbreak of two phage types of *S.* Typhimurium: 44 and 135a (Figures 2 and 3). The outbreak of *S.* Typhimurium 44 affected all jurisdictions, except the Northern Territory. *S.* Typhimurium 44 cases commonly reported eating raw or undercooked eggs and 62 per cent (8/13) of *S.* Typhimurium 44 clusters that were part of the larger investigation

Figure 2. *Salmonella* Typhimurium 135/135a notifications in Australian states and territories, 2005, by month of diagnosis



Source: National Notifiable Diseases Surveillance System. Analysed on 9 February 2006.

Figure 3. *Salmonella* Typhimurium 44 notifications, Australia, 2005, by month of diagnosis and state or territory



Source: National Notifiable Diseases Surveillance System. Analysed on 9 February 2006.

were due to raw or undercooked eggs. The outbreak of *S.* Typhimurium 135a centred on Victoria, with increased numbers of cases in Tasmania and New South Wales. *S.* Typhimurium 135a is a very common phage type and cases were reported from all jurisdictions. Hypothesis generating interviews suggested that cases of *S.* Typhimurium 135a commonly purchased foods from a major retail chain, and ate chicken, meat and some fresh produce. To explore sources of both phage types, the outbreak investigation team instituted a multi-state case control study, which is currently being analysed.

During the quarter, OzFoodNet also investigated a small multi-state cluster of *Salmonella* Havana, although no source was identified. Western Australia investigated a large increase in *Salmonella* Oranienberg affecting approximately 78 people in the fourth quarter 2005. Cases continued to occur in the first months of 2006. Cases were predominantly women. Western Australia is conducting a case control study to examine potential sources of infection for the outbreak. There were several other state-based investigations into various *Salmonella*, including: *S.* Paratyphi B Java and *S.* Typhimurium 12a in the Northern Territory; *S.* Typhimurium 9 in Queensland; *S.* Typhimurium u307, *S.* Typhimurium 186, *S.* Typhimurium 12, and *S.* Typhimurium 6 var 1 in Victoria; *S.* Chester and *S.* Infantis in South Australia; and *S.* Bovismorbicans and *S.* Virchow in Western Australia.

There were also State-based investigations into increases of various other pathogens, including Shiga toxin-producing *Escherichia coli* infections in South Australia, hepatitis A in the Northern Territory, and *Shigella sonnei* biotype G in New South Wales. Cryptosporidiosis reports were elevated in several jurisdictions during the quarter.

Discussion

During the fourth quarter of 2005, there were several outbreaks linked to eggs. The outbreak of *S.* Typhimurium 135a in Tasmania was the largest outbreak of foodborne illness in the State for several years. The outbreak highlighted the relationship between bakery-associated outbreaks and raw eggs.[1,2,3] As a result of the outbreaks, in November 2005 the implementation sub-committee of the Food Regulation Standing Committee discussed the safety of bakery products. Foods prepared in bakeries are at higher risk of contamination due to the nature and handling of bakery products, as they are:

- commonly handled following cooking;
- made of ingredients facilitating bacterial growth;
- commonly filled using single use piping bags that are re-used;
- left unrefrigerated for long periods of time; and
- prepared in premises where *Salmonella* contaminated raw meat and eggs are not properly separated from ready-to-eat products.

Between 2001 and September 2005, OzFoodNet recorded 11 outbreaks associated with the consumption of food from bakeries (OzFoodNet Unpublished).[1] These 11 outbreaks affected at least 243 people with 23 (9%) people hospitalised. One death occurred in association with these outbreaks. The median size of the outbreaks was 16 cases (range 5–70). *Salmonella* Typhimurium caused 82 per cent (9/11) of outbreaks, with norovirus and *Clostridium perfringens* each causing one outbreak. Seventy-three per cent (8/11) of outbreaks involved cream or custard filled cakes or tarts.

There were several *Salmonella* outbreaks with significant links to eggs during the quarter. In particular, the outbreaks in Tasmania resulted in public warnings about the safety of raw and undercooked eggs. Several outbreaks of *S.* Typhimurium 44 implicated foods containing raw or undercooked eggs. This phage type of *S.* Typhimurium is relatively uncommon, but had been reported from the environment of chicken layer flocks in 2005 (personal communication, D Davos, December 2005). In Australia, the commonly held belief is that it is safe to eat eggs that haven't been cooked properly. This results in outbreaks of gastroenteritis despite a low overall prevalence of *Salmonella* on eggs.[4] Food Standards Australia New Zealand is currently assessing the need for a Primary Production Standard for egg production.

Outbreaks of *Listeria* in Australia are rare, although small clusters occasionally occur in hospitals.[4,5] The importance of molecular typing to compare the relatedness of strains of *L. monocytogenes* was highlighted in the recent outbreak in South Australia. In the outbreak, investigators, using PFGE, were able to identify a common strain infecting two patients which was associated with cold meats served at the hospital. Some jurisdictions are increasingly using molecular testing to compare relatedness of strains for a variety of pathogens, but usually only test isolates within the particular State. In this report, Western Australia was able to rapidly identify clusters of *S.* Bovismorbificans and *S.* Virchow using PFGE (personal communication, Minda Sarna, January 2006). Ideally, laboratories across Australia could compare molecular patterns of *Listeria* and other organisms to complement existing subtyping

1 Excluding outbreaks associated with Vietnamese pork rolls, which are prepared in bakeries. Outbreaks associated with these products were excluded as they are ethnic speciality foods.

schemes and improve detection of multi-state outbreaks, which has proven very successful in other countries.[6]

References

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Table. Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 October to 31 December 2005

State	Month of outbreak	Setting prepared	Infection	Number affected	Evidence	Responsible vehicle
NSW	October	Institution	*Salmonella* Birkenhead	3	D	Suspected pureed food
		Child care	*Salmonella* Typhimurium 197	33	A	Suspected hand cut fruit and sandwiches
		Takeaway	*Campylobacter*	5	D	Ready to eat food such as sandwiches & salads
		Takeaway	Unknown	4	D	Roast beef and gravy
	November	Restaurant	Unknown	2	D	Suspected freshly squeezed blood orange juice crush
		Home	Histamine poisoning	4	D	Tuna steak
		Caterer	*Clostridium perfringens*	23	A	Suspected fried rice
		Takeaway	*Campylobacter*	3	D	Suspected chicken
		Restaurant	*Salmonella* Typhimurium 44	8	D	Caesar salad dressing made with raw eggs
	December	Takeaway	Unknown	2	D	Suspected chicken and bacon burgers
Qld	October	Caterer	*Salmonella* Potsdam	6	D	Unknown
		Other	*Salmonella* Saintpaul/ *Salmonella* Chester	26	AM	Town drinking water
	November	Restaurant	Unknown	5	D	Unknown
		Restaurant	Unknown	18	A	Seafood mornay & rice
		Restaurant	*Clostridium perfringens*	14	M	Chicken and/or lamb guvec
		Home	*Salmonella* Typhimurium 44	3	D	Egg and bacon roll
		Camp	*Salmonella* Typhimurium 44	2	D	Unknown
	December	Camp	Ciguatoxin	8	D	Yellowtail kingfish
		Home	*Salmonella* Typhimurium 44	23	D	Prawn soup
		Grocery store/deli	*Campylobacter*	4	D	Chicken kebabs
		Home	Ciguatoxin	10	D	Barracuda

Table. Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 October to 31 December 2005, *continued*

State	Month of outbreak	Setting prepared	Infection	Number affected	Evidence	Responsible vehicle
SA	November	Hospital	*Listeria monocytogenes* serotype O1	3	M	Cold meats
		School	*Campylobacter*	36	A	Unknown
	December	Restaurant	Norovirus	21	D	Dips
Tas	October	Bakery	*Salmonella* Typhimurium 135a	107	AM	Cream cake and icing (piping bags)
		Restaurant	*Salmonella* Typhimurium 135a	11	D	Suspect sauces/dressings containing raw egg, undercooked hamburger patties with raw egg binder
	November	Bakery	*Salmonella* Typhimurium 135a	6	D	Salad rolls/sandwiches
	December	Restaurant	*Salmonella* Typhimurium 135a	77	AM	Mustard seed mayonnaise containing raw egg, tartare sauce containing raw egg, avocado spread (softened with raw egg)
Vic	October	Aged care facility	Unknown	6	D	Suspect roast pork and meat pie
	November	Unknown	*Campylobacter*	5	D	Unknown
		Bakery	Norovirus	9	D	Vanilla slices
		Home	Unknown	16	D	Chicken, cashew and mayonnaise sandwiches
		Aged care facility	Unknown	12	D	Unknown
	December	Restaurant	*Salmonella* Typhimurium 170	20	D	Suspected roast pork
WA	October	Restaurant	Unknown	21	D	Unknown
		Restaurant	Unknown	15	D	Unknown

* No foodborne outbreaks reported in the Northern Territory 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.

A report from the Communicable Diseases Network Australia 1 October to 31 December 2005

The Communicable Diseases Network Australia (CDNA) consists of communicable disease authorities from various Australian Government agencies and state and territory health authorities, in addition to expert bodies and individuals in specific areas of communicable disease epidemiology, clinical management, disease control and la-boratory diagnosis. The CDNA provides national public health leadership and co-ordination on communicable disease surveillance, prevention, and control, and offers strategic advice to governments and other key bodies on public health actions to minimise the impact of communicable diseases in Australia and the region.

Refugee health

CDNA endorsed the *Pre-Departure Health Screening Protocols For Refugees Arriving From Africa* (February 2006). The Department of Immigration and Multicultural Affairs had been trialling an interim protocol since April 2005 which provided valuable feedback used to refine and finalise the protocol. The protocol can be viewed on the Communicable Diseases Australia website at: http://www.health.gov.au/cdna

Guidelines for the Prevention and Control of Influenza Outbreaks in Residential Care Facilities in Australia

This document is to provide national best practice guidelines for staff of public health units and aged care facilities for preventing, defining and managing outbreaks of influenza in residential care facilities in Australia during interpandemic periods. CDNA members endorsed the guidelines which are to be published on the Australian Government Department of Health and Ageing Communicable Diseases Australia website with limited copies to be printed and distributed to jurisdictional health authorities.

Tuberculosis treatment and management

CDNA endorsed the two papers developed by the National Tuberculosis Advisory Committee—a CDNA sub-committee:

- *BCG Vaccine: Information and Recommendations for Use in Australia* – provides recommendations for the use of the Bacille Calmette-Guérin vaccine in the Australian community.
- *Guidelines for Australian Mycobacteriology Laboratories* – practical guidelines for Australian laboratories performing tuberculosis microscopy and culture.

Both of these documents have been published in this issue of *Communicable Diseases Intelligence* pp 109–115 and pp 116–128. These will also be available on the Communicable Diseases Australia website.

Therapeutic Guidelines: Antibiotic version 13

CDNA endorsed the Guidelines making artemether + lumefantrine the first line of treatment for uncomplicated *Plasmodium falciparum* malaria, where previously the first line treatment was quinine.

Treatment of severe malaria

CDNA resolved to encourage the Therapeutic Goods Administration to make parenteral Artesenate available for the treatment of severe malaria in Australia.

Pandemic influenza

Jurisdictional representatives from CDNA continue to be involved in planning for possible pandemic influenza. National Influenza Pandemic Action Committee CDNA jurisdictional representatives have met frequently via teleconference to consider issues relating to communication strategies, antiviral availability and registers, social distancing, border screening and other issues which will be directly relevant at the jurisdictional level. The continued involvement of CDNA jurisdictional members is anticipated.

Exercise Eleusis

CDNA played an active part in Exercise Eleusis ’05, a desktop exercise run by the Australian Government Department of Agriculture, Fisheries and Forestry which included simulated human H5N1 cases. The objective of the exercise was to test the preparedness of Australia’s animal and human health agencies should pandemic influenza enter Australia. CDNA played a pivotal role in the exercise and demonstrated the systems and processes are in place for CDNA to effectively respond during a pandemic.

Biosecurity Surveillance System

CDNA continues to provide policy and implementation advice to the Biosecurity Surveillance System project team and the Australian Government Department of Health and Ageing. Issues surrounding surveillance, privacy, outbreak case reporting and user testing are focus points for CDNA members, in particular the CDNA jurisdictional representatives.

2006

In 2006 CDNA will continue to meet via teleconference on a fortnightly basis, with face-to-face meetings to be conducted in May and in the latter half of 2006.

Erratum

The article Salmonella *Typhimurium phage type 170 in a tertiary paediatric hospital with person-to-person transmission implicated* published in the last issue of *Communicable Diseases Intelligence (Commun Dis Intell* 2005;29:374–378) contained an error.

In the first paragraph of the introduction the sentence ‘The most commonly reported phage type (PT) among *Salmonella* Typhimurium is PT170’ is incorrect. It was only 2004 that PT 170 was the major phage type replacing the most dominated PT135 for the first time since the National Enteric Pathogens Surveillance System records began (Dr. Diane Lightfoot, personal communication, 24 January 2006).

Therefore, the first introduction should read:

‘*Salmonella* species are a common cause of gastrointestinal illness in humans. *Salmonella enterica* serotype Typhimurium (STm) is the most frequently notified serotype in Australia. One of the most commonly reported phage types (PT) among *S.* Typhimurium is PT170. Notifications of STm 170 have increased in eastern Australia since 2002 and comprised 16 per cent of all salmonellosis notifications in New South Wales in 2004.[1]’

Surveillance systems reported in *CDI*, 2006

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 surveillance schemes aim for complete case ascertainment, others include only a proportion of all cases of the conditions under surveillance, and these samples are subject to systematic and other biases. Results generated from surveillance schemes must be interpreted with caution, particularly when comparing results between schemes, between different geographical areas or jurisdictions and over time. Surveillance data may also differ from data on communicable diseases gathered in other settings.

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

Other surveillance schemes for which *CDI* publishes annual reports include tuberculosis notifications (*Commun Dis Intell* 2006; this issue), the Australian Mycobacterium Reference Laboratory Network (*Commun Dis Intell* 2006; this issue), invasive pneumococcal disease surveillance (*Commun Dis Intell* 2006 this issue), and the Australian Rotavirus Surveillance Program (*Commun Dis Intell* 2006 this issue).

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 includes 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 every fortnight (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 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 from the HIC to the ACIR. This constitutes a nearly complete population register, as approximately 99 per cent 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 the HIC 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, the HIC 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.

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

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

Australian Gonococcal Surveillance Programme

The Australian Gonococcal Surveillance Programme (AGSP) is a continuing program to monitor antimicrobial resistance in *Neisseria gonorrhoeae* and includes the reference laboratories in all states and territories. These laboratories report data on sensitivity to an agreed core group of antimicrobial agents on a quarterly basis and provide an expanded analysis as an annual report in *CDI* (*Commun Dis Intell* 2005;29:137). The antibiotics which 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 five per cent 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 is 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* 2005;29:150).

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; congenital cytomegalovirus infection; congenital rubella; perinatal exposure to HIV, HIV infection and AIDS; neonatal herpes simplex virus infection; hepatitis C virus infection and B Group *Streptococcus* sepsis. Studies on Varicella virus infection – neonatal, congenital and severe complications of varicella in children aged 1 to 15 years are planned for 2006.

The primary objectives of the APSU are to document the number of Australian children 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 2004, over 1,100 clinicians participated in the surveillance of 12 conditions through the APSU, with an overall monthly response rate of 91 per cent. 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 NHMRC Enabling Grant 402784. For further information please contact the APSU Director, Professor Elizabeth Elliott on telephone: +61 2 9845 2200, facsimile +61 2 9845 3005 or email: apsu@chw.edu.au

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 2006, six conditions are being monitored; four are related to communicable disease issues. These include influenza, gastroenteritis, varicella 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

There are two definitions for influenza in 2004. A patient may be coded once or twice depending on their symptoms. The definition for influenza 1 will include more individuals.

Influenza 1

Must have the following: cough, fatigue and fever. (Note there is no time frame to these symptoms).

Influenza 2

(a) Viral culture or serological evidence of influenza virus infection; or

(b) influenza epidemic, plus four of the criteria in (c); or

(c) six of the following:

1. sudden onset (within 12 hours);
2. cough;
3. rigors or chills;
4. fever;
5. prostration and weakness;
6. myalgia, widespread aches and pains;
7. no significant respiratory physical signs other than redness of nasal mucous membrane and throat;
8. influenza in close contacts.

Gastroenteritis

Intestinal disease – presumed or proven to be infective in origin.

Varicella/chickenpox

Any consultation at which varicella/chickenpox is diagnosed on clinical or other grounds.

Shingles

Any consultation at which shingles is diagnosed on clinical or other grounds.

In 2006 ASPREN will be expanded and strengthened to enable earlier identification of any influenza outbreak. The current distribution of reporters will be reviewed and a larger more representative group recruited. Consultation with GP organizations and other stakeholders, including other GP networks, will occur and workshops for potential recorders organised in every state. Finally the current paper based system for recording information will be replaced by a web/email based system ensuring the timely analysis and distribution of data to interested groups and individuals.

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.

Laboratory Virology and Serology Reporting Scheme

The Virology and Serology Laboratory 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).

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 that have been sero-typed and phage typed by the six *Salmonella* typing laboratories in Australia. *Salmonella* isolates are submitted to these laboratories for typing by primary diagnostic laboratories throughout Australia.

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

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 and reported in the National Notifiable Diseases Surveillance System (see above).
- In 2006, 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 Virology and Serology Laboratory Reporting Scheme laboratory reports of influenza diagnoses including virus type.

The results of each of the schemes are published together fortnightly throughout the year on the *Communicable Diseases Australia* website as the National Influenza Surveillance Scheme.

Annual reports on influenza in Australia are published in *CDI* each year (*Commun Dis Intell* 2005; 29:125–136). These reports include the above data as well as absenteeism data from a major national employer, hospitalisation and mortality data and influenza typing data from the WHO Collaborating Centre for Influenza Reference and Research.

OzFoodNet: enhanced foodborne disease surveillance

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

OzFoodNet reports quarterly on investigations of gastroenteritis outbreaks and clusters of disease potentially related to food. Annual reports have been produced and published in *CDI* since 2002. Data are reported from all Australian jurisdictions.

Sentinel Chicken Surveillance Programme

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

These viruses are enzootic in parts of the north-east Kimberley region of Western Australia and the Top End of the Northern Territory but are epizootic in other areas of the Kimberley, Pilbara, Gascoyne Murchison and Mid-west regions of Western Australia, in north Queensland and in Central Australia. MVEV is also responsible for occasional epidemics of encephalitis in eastern Australia. Since 1974, a number of sentinel chicken flocks have been established in Australia to provide an early warning of increased MVEV activity. These programs are supported by individual state health departments. Each State has a contingency plan which 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 2005; 29:341-357)*.

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.

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 about the epidemiology and control 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. Structured abstracts are not acceptable . Manuscripts submitted as articles must be 3,000 words or less and are peer-reviewed. Occasionally, reports of urgent public health importance may be published immediately, at the discretion of the Editor.

Short reports

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

Surveillance summaries

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

Outbreak reports

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

Case reports

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

Letters to the Editor

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

Document preparation

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

In addition:

- Arial font is preferred but if not available use Times New Roman.
- Abstracts should not exceed 250 words. Do not cite references in abstracts.
- Include up to 10 keywords.
- Avoid too many abbreviations. Use standard abbreviations, do not make up 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 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 7791 or by email to the address above.

Communicable diseases surveillance

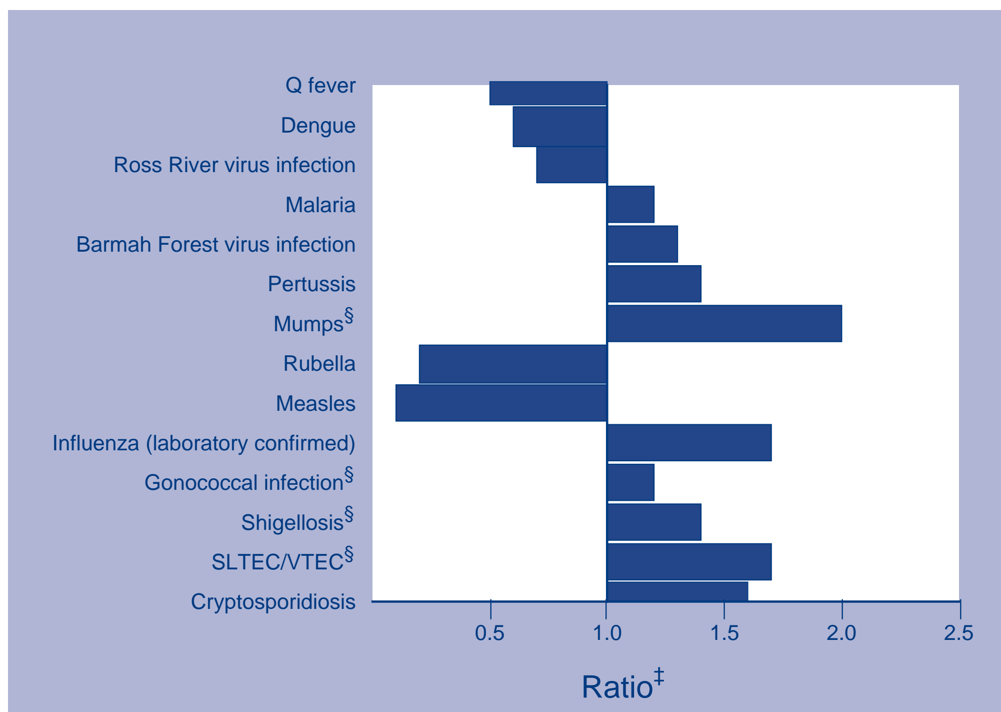
Highlights for 4th quarter, 2005

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

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia. NNDSS collates data on notifiable communicable diseases from State or 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', and those from ASPREN are referred to as 'consultations' or 'encounters' while data from the LabVISE scheme are referred to as 'laboratory reports'.

Figure 1 shows the changes in select disease notifications with an onset in the fourth quarter of 2005 compared with a five year mean for the same period. These diseases were above the five year mean for the same period and exceeded two standard deviations from the five year mean: hepatitis E, Shiga-like toxin-producing *Escherichia coli*/verotoxin-producing *E. coli* (SLTEC\VTEC), shigellosis, gonococcal infections, syphilis and mumps. Diseases for which the number of notifications were below the five year

Figure 1. Selected* diseases from the National Notifiable Diseases Surveillance System,† comparison of provisional totals for the period 1 October to 31 December 2005 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.

† Some Victorian data for the period may be incomplete.

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

§ Notifications above or below the 5-year mean plus two standard deviations for the same period.

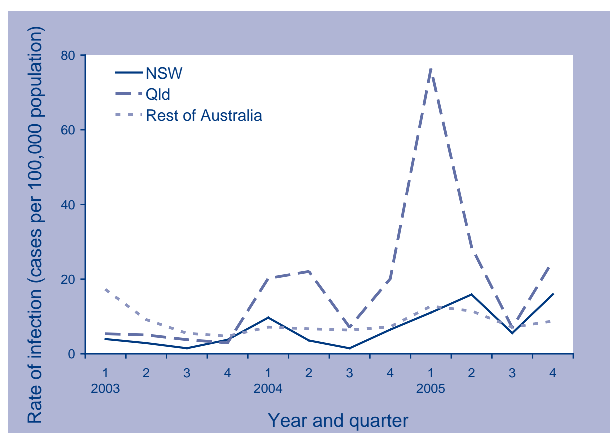
mean for the same period include measles, rubella, dengue, Q fever and Ross River virus infection. The number of notifications reported from Victoria this quarter was less than usual due to data processing difficulties.

Gastrointestinal illnesses

Cryptosporidiosis

There were 727 notifications of cryptosporidiosis during the quarter which was 1.6 times the five year mean for the same period. All jurisdictions reported cases but the majority were from New South Wales and Queensland, with 270 and 247 cases respectively. Four hundred and fifty-eight (63%) notifications were identified as *Cryptosporidium parvum* infection; there was no species information provided for the remaining 37 per cent of cases. Figure 2 shows that the rate of infection in Queensland is often higher that the rest of Australia and that the infection rate was also higher in New South Wales this year.

Figure 2. Notification rates of cryptosporidiosis, New South Wales and Queensland compared to the rest of Australia, 1 January 2003 to 31 December 2005



In the fourth quarter, the rise in cases in New South Wales has been most notable in regional and rural areas where there have been reports of increases and outbreaks of cryptosporidiosis in calves, and 'massive' numbers of oocysts in the faeces of sick calves (NSW Health, personal communication). Cryptosporidiosis has been recognised worldwide primarily in neonatal calves as well as in other neonatal farm animals.[1]

A state-wide outbreak investigation resulted in a hypothesis that the rise in the number of cryptosporidiosis cases in New South Wales was due to infection after contact with sick cattle, or after drinking or swimming in contaminated water. Water may have been contaminated by sick cattle, heavy rain or inadequate chlorination and filtration. It is thought that those who initially contract cryptosporidiosis from calves may then spread the disease via person-to-person transmission, and may possibly also contaminate swimming pools. There have been three small clusters of cases linked to three swimming pools (n=1, n=3 and n=7) which have since been subject to an environmental assessment. Another cluster (n=6) was identified in recruits from a military training facility. Appropriate public health action was undertaken in all cases (NSW Health, personal communication).

Shiga-like toxin producing *Escherichia coli*/ verotoxin producing *E. coli*

Twenty-one notifications of SLTEC/VTEC were received during the quarter, which was 1.7 times the five year mean for the same period. South Australia notified 12 cases, New South Wales reported seven cases and Queensland and Tasmania reported one case each. Of the two cases with serotype information, one was *E. Coli* serotype O111 and one was O157:H–.

Shigellosis

There were 156 notifications of shigellosis during the quarter which was 1.4 times the five year mean for the same period. This was 126 cases above the five year average plus two standard deviations. Notifications were from all jurisdictions except Tasmania with 40 cases from the Northern Territory, 34 from Western Australia, 21 each from New South Wales and South Australia, 20 from Queensland, 19 from Victoria and 1 from the Australian Capital Territory.

There was one death attributed to shigellosis infection in the quarter. Seventy (45%) of the 156 infections were notified in Indigenous Australians. Twenty per cent of infections were in non-Indigenous people, and Indigenous status was unknown for 35 per cent of cases. Of the 156 notifications, there were 68 (44%) notifications of *Shigella sonnei,* and 74 (47%) of *Shigella flexneri.* There was a single case of *Shigella dysenteriae* 2c. Fourteen cases (9%) did not have subtyping information.

Sexually transmissible infections

Gonococcal infection

During the quarter there were 1,726 notifications of gonococcal infection received from all jurisdictions, which was 1.2 times the five year mean for the same period and 240 cases above the five year average plus two standard deviations. The majority of these

notifications were reported by Queensland (369), New South Wales (337), Western Australia (355) and Victoria (308).

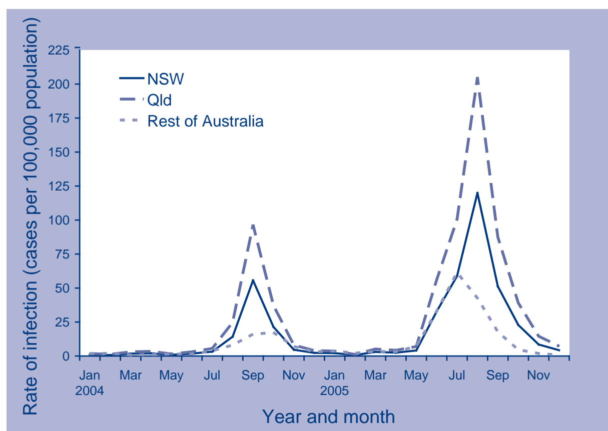
Forty per cent (697/1726) of the notifications were in the 15–24 year age group. The highest rate of gonococcal infection in females (92.7 cases per 100,000 population), was in the 15–19 year age group. In males, the 20–24 year age group had the highest gonococcal infection rate (139.1 cases per 100,000 population).

Vaccine preventable diseases

Influenza (laboratory confirmed)

There were 411 cases of laboratory-confirmed influenza in the fourth quarter of 2005 compared to the five year average of 345 cases for this quarter. Queensland reported 201 cases, and 152 reports were received from New South Wales. Sixty-five per cent of cases (267/411) were type A, 29 per cent (267/411) type B, and 1.5 per cent (6/411) were of unknown type. The number of notifications has remained higher in the fourth quarter in New South Wales and Queensland compared to the rest of Australia after the seasonal peak in the third quarter 2005 (Figure 3).

Figure 3. Notification rates of laboratory confirmed influenza, New South Wales and Queensland compared to the rest of Australia, 1 January 2004 to 31 December 2005

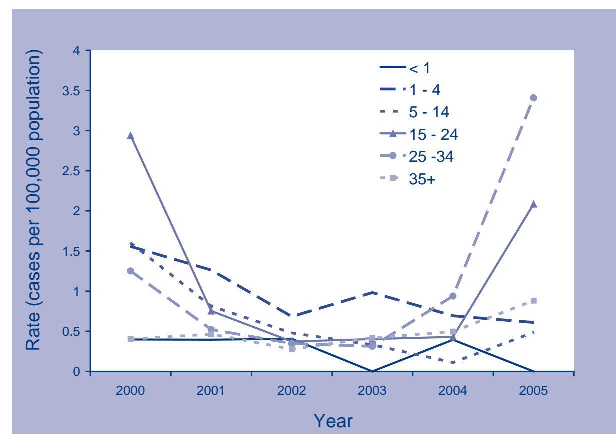


Mumps

There were 34 notifications of mumps in the quarter, which was double the five year mean for the same period. Nineteen cases (56%) were reported from New South Wales; there were also five notifications received from Victoria, three from Queensland and South Australia, and two from Western Australia. Of the 34 cases, 13 (38%) cases were reported from the 20–34 year age range, with a male to female ratio of 0.3:1.

Vaccination status data were available for 102 of 208 (49%) mumps notifications analysed from 2005. In cases where data were available, 34 of 37 (92%) cases in the 25–34 year age range were unvaccinated, and 86 per cent in the 15–24 year age range cases. The high rate of mumps was in the 25–34 year age range and to a lesser extent in the 15–24 year age range, which probably represents a susceptible cohort of individuals who have not been immunised (Figure 4).

Figure 4. Notification rates of mumps, Australia, 1 January 2000 to 31 December 2005, by age group



Mumps vaccine was made available in Australia in 1980 for use at 12–15 months and was combined with the measles vaccine in 1982. Therefore, no childhood doses of mumps vaccine were available to individuals currently in the 25–34 year age range and the uptake of vaccine in older individuals from the 15–24 year age range group was likely to be moderate. A similar situation has recently been reported in England and Wales, with high mumps notification rates in young adults born just before the introduction of routine vaccination, who were probably not infected as children because of reduced virus circulation following the introduction of vaccination in younger cohorts.[2]

Pertussis

For the fourth quarter, 2,559 pertussis notifications were received, which was 1.6 times the five year mean for the same period. Of the total number of notifications, 1,207 (47%) were reported by New South Wales and 518 (20%) were from Queensland. Three per cent (86 cases) of the 2,559 notifications

were reported in infants aged less than one year. The only pertussis death for 2005 was reported this quarter from Queensland in an unvaccinated one-month-old male. The highest rate of infection in females, 100 cases per 100,000 population occurred in the 50–54 year age range. The highest rate in males was 73 cases per 100,000 population in the 65–69 year age group.

Vectorborne diseases

Barmah Forest virus infection

There were 264 cases of Barmah Forest virus infection in the fourth quarter which was 1.3 times the five year mean for the same period. The majority of cases were from Queensland with 119 cases, New South Wales with 91 cases and Western Australia with 34 cases. This represents an early peak in infection rates compared to previous years.

Nationally, the infection rate was 5.2 cases per 100,000 population, but it was higher in the Northern Territory at 16 cases per 100,000 population (8 cases) and Queensland with 12 cases per 100,000 population.

Malaria

There were 129 cases of malaria in the fourth quarter, which was 1.2 times the five year mean for this quarter. There were 46 cases reported in Queensland and 24 in New South Wales. The highest rate of infection, 10 cases per 100,000 population was in the Northern Territory (5 cases). The national infection rate was 2.5 cases per 100,000 population.

Sixty-one per cent (79/129) of malaria cases were imported from overseas (19 cases notified from Queensland were imported from Papua New Guinea), while 18 per cent (23/129) were not. The import status of 28/129 (22%) cases was unknown. Thirty per cent (39/129) of notifications were *Plasmodium falciparum* and 22 per cent (29/129) were *Plasmodium vivax*. There were three *Plasmodium ovale* notifications, one *Plasmodium malariae* notification and four mixed infections. Forty-six (41%) cases did not have typing information.

There was a cluster of six malaria cases notified from Queensland in a group of 24 high school students who visited the Solomon Islands during September. All cases were on chemoprophylaxis.

Ross River virus infection

This quarter, 578 notifications of Ross River virus infection were reported compared to 239 in the same period last year. While this is indicative of an early seasonal increase, the number of cases was less than the five year mean for the same period. The majority of cases were from Western Australia (161), New South Wales (148) and Queensland (143).

Ninety-eight of Western Australia's notifications (61%) were from the Peel area south of Perth and almost all were notified in the months of November and December. The rate of infection was 24 cases per 100,000 population. Twenty-nine cases occurred in the Northern Territory, which was a rate of 58 cases per 100,000 population. South Australia and Queensland both had rate of 15 cases per 100,000 population with 59 and 143 cases respectively, which was higher than the national rate of 10.7 cases per 100,000 population.

Other bacterial infections

Brucellosis

There were 18 cases of brucellosis notified in the fourth quarter which was 1.4 times the five year mean for the same period. Seventeen cases were from Queensland, and one was from New South Wales. Fifteen of the 18 cases occurred in males. There were five *Brucellosis suis* cases and one *Brucellosis melitensis* case reported. Of the 17 cases from Queensland, six reported contact with a feral animal prior to illness.

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1. Radostits OM, Blood DC, Gay CC. *Veterinary Medicine. A Textbook of the Diseases of Cattle, Sheep, Pigs, Goats and Horses.* 1994. Eighth edition. Balliere Tindall.

2. Savage E, Ramsey M, White J, Beard S, Lawson H, Hurijan R, Brown D. Mumps outbreaks in England and Wales in 2004: observational study. *BMJ* 2005;330;1119–1120.

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Tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 28,499 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 October and 31 December 2005 (Table 2). The notification rate of diseases per 100,000 population for each State or Territory is presented in Table 3.

There were 5,223 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 October to 31 December 2005 (Tables 4 and 5).

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
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 Octeober to 31 December 2005, by date of onset*

Disease	State or territory								Total 4th quarter 2005[†]	Total 3rd quarter 2005	Total 4th quarter 2004	Last 5 years mean 4th quarter	Year to date 2005	Last 5 years YTD mean	Ratio[‡]
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis B (incident)	1	12	0	10	1	0	13	5	42	58	61	77.8	238	368.6	0.6
Hepatitis B (unspecified)	29	655	58	236	54	13	332	78	1,455	1,923	1,472	1,641.0	6,979	6,769.2	1.0
Hepatitis C (incident)	0	9	0	NN	9	0	12	21	51	86	107	123.0	318	522.0	0.6
Hepatitis C (unspecified)	43	1,395	51	737	109	33	511	242	3,121	3,859	3,023	3,752.6	14,123	16,078.4	0.9
Hepatitis D	0	1	0	2	0	0	1	2	6	16	5	3.8	30	24.6	1.2
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	1	0	0.0	3	0.5	0.0
Campylobacteriosis[§]	107	NN	64	1,253	801	217	1,382	734	4,558	3,792	4,559	4,328.8	16,044	15,104.4	1.1
Cryptosporidiosis[‖]	7	270	16	247	35	13	113	26	727	334	476	359.0	3,137	1,950.3	1.6
Haemolytic uraemic syndrome	0	1	0	0	0	0	0	0	1	3	6	5.0	12	12.6	1.0
Hepatitis A	2	19	9	9	3	0	9	2	53	85	61	109.6	305	499.4	0.6
Hepatitis E	0	1	0	0	0	0	0	0	1	5	6	3.0	30	16.0	1.9
Listeriosis	1	6	0	4	4	0	3	0	18	9	14	16.0	54	66.0	0.8
Salmonellosis (NEC)	15	527	79	689	149	171	384	231	2,245	1,322	1,955	1,743.0	8,229	7,157.2	1.1
Shigellosis	1	21	40	20	21	0	19	34	156	156	136	117.0	721	505.4	1.4
SLTEC, VTEC[¶]	0	7	0	1	12	1	0	0	21	17	14	11.8	84	49.0	1.7
Typhoid	0	2	0	3	0	0	1	1	7	11	16	14.0	50	65.8	0.8
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	0	0	0.4	3	3.4	0.8
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Tularemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 October to 31 December 2005, by date of onset,* *continued*

Disease	State or territory								Total 4th quarter 2005[†]	Total 3rd quarter 2005	Total 4th quarter 2004	Last 5 years mean 4th quarter	Year to date 2005	Last 5 years YTD mean	Ratio[‡]
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection**	160	2,450	312	1,920	586	198	1,769	1,237	8,632	10,117	9,139	6,458.0	39,978	25,596.6	1.6
Donovanosis	0	0	0	2	0	0	0	1	3	3	4	4.2	13	19.2	0.7
Gonococcal infection	10	337	282	369	56	9	308	355	1,726	1,919	1,759	1,526.0	7,742	6,491.2	1.2
Syphilis (all)	2	177	24	75	2	3	109	33	425	565	560	499.2	2,108	1,610.8	1.3
Syphilis < two years duration	0	19	6	29	0	0	32	6	92	150	164	NA	551	123.2	NA
Syphilis > two years or unspecified duration	2	157	17	46	2	3	76	27	330	411	392	NA	1,541	315.2	NA
Syphilis - congenital	0	1	1	0	0	0	1	0	3	4	4	3.6	16	14.0	1.1
Vaccine preventable disease															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.2	0.0
Haemophilus influenzae type b	0	3	0	1	0	0	1	0	5	5	2	3.4	18	22.6	0.8
Influenza (laboratory confirmed)[‖]	4	152	2	201	2	3	14	33	411	2,960	763	345.0	4,550	2,642.0	1.7
Measles	0	0	0	1	0	0	1	0	2	1	17	19.6	10	84.2	0.1
Mumps	1	19	1	3	3	0	5	2	34	76	36	24.8	234	115.0	2.0
Pertussis	131	1,207	13	518	318	5	302	65	2,559	3,596	3,355	2,345.0	10,954	6,945.0	1.6
Pneumococcal disease (invasive)[‖]	7	122	15	56	32	7	49	25	313	633	465	497.3	1,670	2,195.3	0.8
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Rubella	0	0	0	2	0	0	0	0	2	7	10	60.8	29	185.4	0.2
Rubella - congenital	0	0	0	0	0	0	0	0	0	1	0	0.2	1	1.0	1.0
Tetanus	0	0	0	0	0	2	0	0	2	0	1	1.4	2	4.8	0.4
Vectorborne diseases															
Barmah Forest virus infection	0	91	8	119	9	1	2	34	264	226	274	183.2	1,288	1,025.8	1.3
Dengue	0	19	0	11	3	0	0	5	38	33	19	58.2	208	340.8	0.6
Flavivirus infection (NEC)	0	1	0	2	0	0	1	0	4	8	5	10.0	29	69.6	0.4
Japanese encephalitis virus[‖]	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.4	0.0
Kunjin virus[‖]	0	0	1	0	0	0	0	0	1	0	2	0.8	2	9.0	0.2
Malaria	4	24	5	46	8	1	18	23	129	162	149	145.6	807	660.0	1.2
Murray Valley encephalitis virus[‖]	0	0	0	0	0	0	0	0	0	0	0	0.0	2	2.3	0.0
Ross River virus infection	1	148	29	143	59	1	36	161	578	283	239	354.8	2,334	3,392.2	0.7

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 October to 31 December 2005, by date of onset,* *continued*

Disease	State or territory								Total 4th quarter 2005†	Total 3rd quarter 2005	Total 4th quarter 2004	Last 5 years mean 4th quarter	Year to date 2005	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax‖	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Australian bat lyssavirus‖	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Brucellosis	0	1	0	17	0	0	0	0	18	8	16	9.0	41	29.4	1.4
Leptospirosis	0	8	0	6	6	0	4	1	25	25	25	38.6	125	194.6	0.6
Lyssavirus unspecified‖	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Ornithosis	0	16	0	0	0	0	8	1	25	40	53	42.4	156	174.0	0.9
Q fever	0	27	1	26	1	0	4	3	62	73	117	147.0	331	609.2	0.5
Other bacterial infections															
Legionellosis	0	18	0	11	21	0	14	23	87	91	75	87.2	339	347.8	1.0
Leprosy	0	0	0	0	0	0	0	1	1	1	1	1.0	7	6.2	1.1
Meningococcal infection††	1	29	3	14	9	4	28	12	100	141	81	141.6	391	593.4	0.7
Tuberculosis	0	69	7	36	9	2	102	9	234	183	306	285.6	893	1,006.2	0.9
Total	529	8,021	1,044	6,865	2,324	687	5,665	3,433	28,568	32,889	29,386	25,619.5	126,731	104,021	1.1

* 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.

§ 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 four 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 2005, by state or territory. (Rate per 100,000 population)

Disease*	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis B (incident)	1.2	0.7	0.0	1.0	0.3	0.0	1.0	1.0	0.8
Hepatitis B (unspecified)	35.8	38.9	116.1	24.3	14.1	10.8	26.7	15.7	28.9
Hepatitis C (incident)	0.0	0.5	0.0	NN	2.3	0.0	1.0	4.2	1.3
Hepatitis C (unspecified)	53.1	82.9	102.0	75.9	28.4	27.4	41.1	48.8	62.1
Hepatitis D	0.0	0.1	0.0	0.2	0.0	0.0	0.1	0.4	0.1
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis[†]	121.0	NN	128.1	129.1	208.8	180.0	111.2	148.1	136.0
Cryptosporidiosis	7.4	15.9	32.0	22.5	9.1	10.8	9.1	5.2	13.8
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis A	2.5	1.1	18.0	0.9	0.8	0.0	0.7	0.4	1.1
Hepatitis E	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Listeriosis	1.2	0.4	0.0	0.4	1.0	0.0	0.2	0.0	0.4
Salmonellosis (NEC)	18.5	31.3	158.1	71.0	38.8	141.9	30.9	46.6	44.7
Shigellosis	1.2	1.2	80.0	2.1	5.5	0.0	1.5	6.9	3.1
SLTEC, VTEC[‡]	0.0	0.4	0.0	0.1	3.1	0.8	0.0	0.0	0.4
Typhoid	0.0	0.1	0.0	0.3	0.0	0.0	0.1	0.2	0.1
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
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[§]	186.4	145.6	624.3	197.8	152.8	164.3	142.3	249.6	171.5
Donovanosis	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.2	0.1
Gonococcal infection	12.3	19.9	564.2	37.9	14.6	7.5	24.8	70.8	34.2
Syphilis (all)	1.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Syphilis < 2 years duration	0.0	1.1	12.0	3.0	0.0	0.0	2.6	1.2	1.8
Syphilis > 2 years or unspecified duration	1.2	9.3	34.0	4.7	0.5	2.5	6.1	5.4	6.5
Syphilis - congenital	0.0	0.1	2.0	0.0	0.0	0.0	0.1	0.0	0.1

Table 3. Notification rates of diseases, 1 October to 31 December 2005, by state or territory. (Rate per 100,000 population)*, continued*

Disease*	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
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	0.2	0.0	0.1	0.0	0.0	0.1	0.0	0.1
Influenza (laboratory confirmed)	4.9	9.0	4.0	20.5	0.5	2.5	1.1	6.7	8.1
Measles	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.1
Mumps	1.2	1.1	2.0	0.3	0.8	0.0	0.4	0.4	0.7
Pertussis	140.7	71.0	26.0	53.5	82.4	4.1	24.3	13.1	50.3
Pneumococcal disease (invasive)	8.6	7.2	30.0	5.8	8.3	5.8	3.9	5.0	6.2
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.1
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	1.7	0.0	0.0	0.1
Vectorborne diseases									
Barmah Forest virus infection	0.0	5.4	16.0	12.3	2.3	0.8	0.2	6.1	5.2
Dengue	0.0	1.1	0.0	1.1	0.8	0.0	0.0	1.0	0.8
Flavivirus infection (NEC)	0.0	0.1	0.0	0.2	0.0	0.0	0.1	0.0	0.1
Japanese encephalitis virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0	0.1
Malaria	4.9	1.4	10.0	4.6	2.1	0.8	1.4	4.6	2.5
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	2.5	8.8	58.0	14.7	15.4	0.8	2.9	24.0	10.7
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	1.4	0.0	0.0	0.0	0.0	0.3
Leptospirosis	0.0	0.5	0.0	0.6	1.6	0.0	0.3	0.2	0.5
Lyssavirus unspecified	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	1.2	1.0	0.0	0.0	0.0	0.0	0.6	0.2	0.5
Q fever	0.0	1.6	2.0	2.7	0.3	0.0	0.3	0.6	1.2
Other bacterial infections									
Legionellosis	0.0	1.1	0.0	1.1	5.5	0.0	1.1	3.0	1.6
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.1
Meningococcal infection[||]	1.2	1.7	6.0	1.4	2.3	3.3	2.3	2.4	2.0
Tuberculosis	0.0	4.1	14.0	3.7	2.3	1.7	8.2	3.8	4.6

* 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.

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 October to 31 December 2005, and total reports for the year[†]

	State or territory								This period 2005	This period 2004	Year to date 2005	Year to date 2004
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	–	–	–	2	1	–	1	–	4	14	8	35
Mumps virus	–	–	–	2	5	–	6	–	13	1	38	6
Rubella virus	–	–	–	1	–	–	–	–	1	9	12	20
Hepatitis viruses												
Hepatitis A virus	–	–	1	5	9	–	–	–	15	15	53	51
Hepatitis D virus	–	–	–	–	1	–	1	–	2	1	14	8
Hepatitis E virus	–	–	–	–	–	–	1	–	1	1	12	14
Arboviruses												
Ross River virus	–	–	10	32	55	–	7	15	119	36	452	743
Barmah Forest virus	–	3	1	14	9	–	–	–	27	27	185	195
Flavivirus (unspecified)	–	1	–	6	–	–	1	–	8	8	37	102
Adenoviruses												
Adenovirus type 1	–	1	–	–	–	–	–	–	1	–	7	–
Adenovirus not typed/pending	–	40	1	25	81	–	28	–	175	258	673	1,052
Herpesviruses												
Herpes virus type 6	–	–	–	–	–	–	1	–	1	2	2	6
Cytomegalovirus	1	55	1	23	194	4	31	–	309	220	1,038	834
Varicella-zoster virus	1	38	–	271	54	4	17	1	386	544	1,497	2,061
Epstein-Barr virus	–	15	26	229	194	3	25	113	605	512	2,148	2,367
Other DNA viruses												
Parvovirus	–	5	–	33	32	–	9	–	79	129	202	413
Picornavirus family												
Coxsackievirus A9	–	1	–	–	–	–	–	–	1	–	3	1
Coxsackievirus A16	–	1	–	–	–	–	–	–	1	–	6	5
Echovirus type 5	–	2	–	–	–	–	–	–	2	–	2	–
Echovirus type 7	–	1	–	–	–	–	–	–	1	11	8	12
Echovirus type 9	–	1	–	–	–	–	–	–	1	6	2	10
Echovirus type 18	–	1	–	–	–	–	–	–	1	14	14	19
Echovirus type 30	–	4	–	–	–	–	–	–	4	1	35	7
Poliovirus type 1 (uncharacterised)	–	2	–	–	–	–	–	–	2	3	21	18
Poliovirus type 2 (uncharacterised)	–	1	–	–	–	–	–	–	1	8	19	21
Rhinovirus (all types)	1	62	–	–	17	–	–	–	80	188	326	617
Enterovirus not typed/ pending	–	26	–	9	3	–	6	–	44	72	185	205
Ortho/paramyxoviruses												
Influenza A virus	–	4	–	8	40	–	8	–	60	137	696	492
Influenza B virus	–	7	–	–	17	–	3	–	27	89	253	219
Parainfluenza virus type 1	–	5	–	–	6	–	3	–	14	12	59	143
Parainfluenza virus type 2	–	2	–	–	1	–	–	–	3	4	49	15
Parainfluenza virus type 3	–	33	–	7	56	–	16	–	112	228	386	655
Respiratory syncytial virus	–	33	–	54	24	1	17	–	129	114	1,677	2,599

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 October to 31 December 2005, and total reports for the year,[†] *continued*

	State or territory								This period 2005	This period 2004	Year to date 2005	Year to date 2004
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other RNA viruses												
HTLV-1	–	–	–	–	3	–	–	–	3	2	9	15
Rotavirus	1	99	–	1	119	3	42	–	265	626	1,263	1,247
Astrovirus	–	–	–	–	–	–	4	–	4	–	4	–
Norwalk agent	–	–	–	–	–	–	67	–	67	168	230	659
Other												
Chlamydia trachomatis - not typed	–	113	–	626	474	14	39	1	1,267	1,242	5,045	5,257
Chlamydia pneumoniae	–	–	–	–	–	–	2	–	2	2	8	9
Chlamydia psittaci	–	–	–	–	–	–	14	–	14	35	52	173
Chlamydia species	–	–	–	–	–	–	1	–	1	–	1	3
Mycoplasma pneumoniae	–	11	8	114	108	19	65	46	371	331	1,298	1,374
Mycoplasma hominis	–	1	–	–	–	–	–	–	1	1	5	5
Coxiella burnetii (Q fever)	–	2	–	13	21	–	5	–	41	51	162	173
Rickettsia prowazeki	–	–	–	–	–	–	–	–	–	–	–	–
Rickettsia tsutsugamushi	–	–	–	–	–	–	–	–	–	–	–	–
Rickettsia - spotted fever group	–	–	–	–	58	–	–	–	58	89	236	139
Streptococcus group A	–	6	–	129	–	–	43	–	178	107	609	467
Brucella species	–	1	–	4	–	–	–	–	5	4	14	9
Bordetella pertussis	–	11	1	63	278	1	52	–	406	599	1,573	1,358
Legionella pneumophila	–	1	–	–	3	–	2	–	6	12	23	77
Legionella longbeachae	–	1	–	–	13	–	–	–	14	17	51	76
Legionella species	–	–	–	1	–	–	–	–	1	1	1	15
Cryptococcus species	–	2	–	–	10	–	–	–	12	6	41	38
Leptospira species	–	–	–	2	8	–	–	–	10	4	33	23
Treponema pallidum	–	25	–	133	92	–	1	–	251	244	1,086	1,154
Entamoeba histolytica	–	–	–	–	–	–	2	–	2	5	14	14
Toxoplasma gondii	–	2	–	6	3	–	2	–	13	14	44	41
Echinococcus granulosus	–	–	–	–	2	–	–	–	2	3	10	15
Total	4	619	49	1,813	1,991	49	522	176	5,223	6,227	21,931	25,286

* 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 2005*

State or territory	Laboratory	October 2005	November 2005	December 2005	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	61	84	108	253
	New Children's Hospital, Westmead	81	65	33	179
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	–	–	–	–
	South West Area Pathology Service, Liverpool	124	15	–	139
Queensland	Queensland Medical Laboratory, West End	667	611	606	1,884
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	757	731	501	1,989
Tasmania	Northern Tasmanian Pathology Service, Launceston	19	15	10	44
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	CSL, Melbourne	20	9	16	45
	Monash Medical Centre, Melbourne	27	12	2	41
	Royal Children's Hospital, Melbourne	99	83	43	225
	Victorian Infectious Diseases Reference Laboratory, Fairfield	102	69	43	214
Western Australia	PathCentre Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	45	103	62	210
Total		2,002	1,797	1,424	5,223

* The complete list of laboratories reporting for the 12 months, January to December 2005, 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 50 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 4,000 and 6,000 consultations are recorded each week.

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published.

In 2005, eight conditions are being monitored, four of which are related to communicable diseases. These include influenza, gastroenteritis, varicella and shingles. There are two definitions for influenza for 2005. A patient may be coded once or twice depending on their symptoms. The definition for influenza 1 will include more individuals. Definitions of these conditions were published in Commun Dis Intell *2006;30:158.*

Data from 1 January to 31 December 2005 compared with 2004 are shown as the rate per 1,000 consultations in Figures 5 and 6.

Figure 5. Consultation rates for influenza-like illness, ASPREN, 1 January to 31 December 2005, by week of report

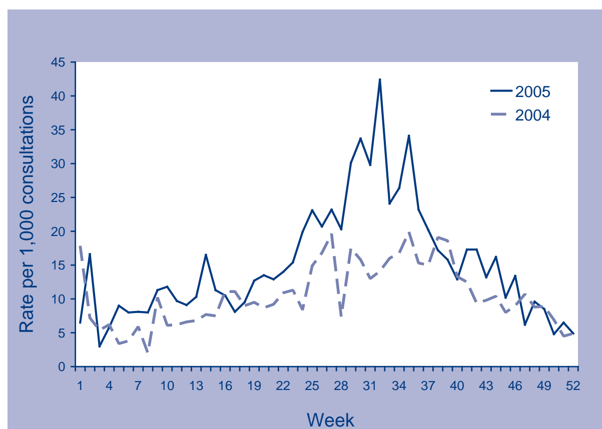
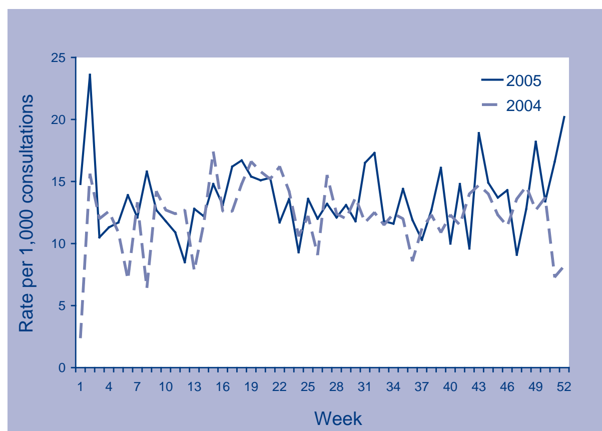


Figure 6. Consultation rates for gastroenteritis, ASPREN, 1 January to 31 December 2005, by week of report



Childhood immunisation coverage

Tables 6, 7 and 8 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR).

The data show the percentage of children fully immunised at 12 months of age for the cohort born between 1 July and 30 September 2004, at 24 months of age for the cohort born between 1 July and 30 September 2003, and at 6 years of age for the cohort born between 1 July and 30 September 1999 according to the Australian Standard Vaccination Schedule.

For information about the Australian Childhood Immunisation Register see Surveillance systems reported in CDI, *published in* Commun Dis Intell *2006;30:157 and for a full description of the methodology used by the Register see* Commun Dis Intell *1998;22:36-37.*

Commentary on the trends in ACIR data is provided by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS). For further information please contact the NCIRS at telephone: +61 2 9845 1435, Email: brynleyh@chw.edu.au.

Immunisation coverage for children 'fully immunised' at 12 months of age for Australia did not change from the last quarter, remaining at 91.0 per cent (Table 6). It has now remained at 91 per cent for three consecutive quarters. There were no significant changes in coverage in any jurisdiction for 'fully immunised' coverage or for coverage for individual vaccines.

Immunisation coverage for children 'fully immunised' at 24 months of age for Australia also did not change from the last quarter, remaining at 92.1 per cent. Similarly, there were no significant changes in coverage in any jurisdiction for 'fully immunised' coverage or for coverage for individual vaccines (Table 7).

Table 6. 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 2004; assessment date 31 December 2005

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	1,068	22,285	837	13,328	4,309	1,507	15,994	6,381	65,709
Diphtheria, tetanus, pertussis (%)	94.2	92.2	91.0	92.3	92.0	94.4	93.5	90.4	92.4
Poliomyelitis (%)	94.1	92.0	90.8	92.2	91.9	94.4	93.4	90.3	92.3
Haemophilus influenzae type b (%)	96.0	94.2	94.4	94.2	94.5	95.5	95.1	93.3	94.4
Hepatitis B (%)	96.3	95.2	94.9	94.8	94.8	95.6	94.9	93.0	94.8
Fully immunised (%)	93.7	90.7	90.1	91.1	91.2	93.4	92.0	88.8	91.0
Change in fully immunised since last quarter (%)	+0.1	+0.1	-1.6	+0.3	+0.1	+1.4	-0.1	-0.4	+0.0

Table 7. 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 2003; assessment date 31 December 2005*

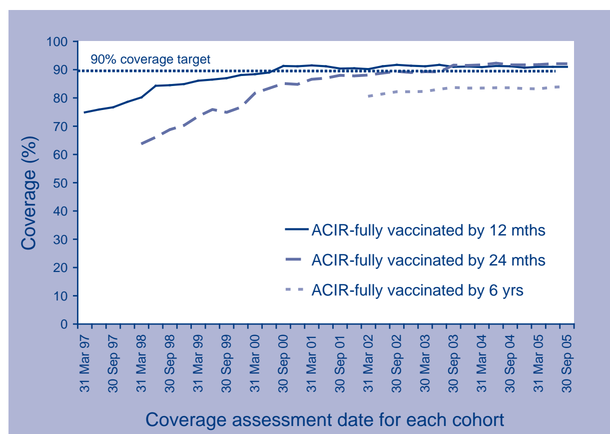
Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,060	22,667	827	13,412	4,681	1,456	16,361	6,236	66,700
Diphtheria, tetanus, pertussis (%)	97.3	95.0	96.4	94.9	95.5	96.2	95.4	95.2	95.2
Poliomyelitis (%)	97.1	94.9	96.0	94.8	95.4	96.4	95.4	95.2	95.2
Haemophilus influenzae type b (%)	95.4	93.2	94.6	93.6	94.0	95.0	93.8	93.2	93.6
Measles, mumps, rubella (%)	95.9	93.3	95.0	93.7	94.3	95.3	94.2	93.8	93.8
Hepatitis B(%)	97.2	95.8	97.5	95.4	96.1	96.9	96.1	96.2	95.9
Fully immunised (%)	94.8	91.7	93.1	91.9	92.6	94.4	92.5	91.4	92.1
Change in fully immunised since last quarter (%)	+0.6	+0.1	-1.9	-0.1	+1.3	+1.2	-0.5	+0.7	-0.0

* The 12 months age data for this cohort was published in *Commun Dis Intell* 2005;29:115.

Table 8 shows immunisation coverage estimates for children 'fully immunised' at 6 years of age and for individual vaccines for Australia by state or territory. This was largely unchanged in all jurisdictions. Coverage for vaccines assessed at 6 years is at or near 85 per cent in most jurisdictions, but Western Australia, South Australia and Queensland still remain below this. The sharp decline in coverage for this age group that occurred in Queensland in 2004–05 appears to have halted with 'fully immunised' coverage increasing for the second consecutive quarter.

Figure 7 shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and 6 years, although the rate of increase has slowed over the past 2 years for all age groups. The Figure shows that there have now been nine consecutive quarters where 'fully immunised' coverage at 24 months has been greater than 'fully immunised' coverage at 12 months, following the removal of the requirement for 18 month DTPa vaccine. However, both measures have been above 90 per cent for this 27-month period and show levels of high coverage for the vaccines included maintained over a significant period of time. Currently, coverage for the more recent vaccines, meningococcal C conjugate at 12 months and pneumococcal conjugate at 2, 4, and 6 months, are not included in the 12 or 24 months coverage data respectively.

Figure 7. Trends in vaccination coverage, Australia, 1997 to 2005, by age cohorts



Gonococcal surveillance

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

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various States and Territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5 per cent or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatment.[1] Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as TRNG. Tetracyclines are however, not a recommended therapy for gonorrhoea in Australia. Comparability of data is achieved by means of a standardised system of testing and a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. For more information see Commun Dis Intell *2006;30:157.*

Table 8. 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 1999; assessment date 31 December 2005

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,129	22,965	792	14,065	4,802	1,579	16,642	6,769	68,743
Diphtheria, tetanus, pertussis (%)	89.1	85.7	84.3	82.7	82.8	87.7	88.0	80.9	85.1
Poliomyelitis (%)	89.5	85.8	85.9	82.8	82.9	87.7	88.2	81.2	85.2
Measles, mumps, rubella (%)	89.1	85.7	85.3	82.9	83.0	87.7	88.3	81.1	85.2
Fully immunised (%)[1]	88.2	84.7	83.1	81.4	81.8	86.6	87.3	79.5	84.0
Change in fully immunised since last quarter (%)	-2.3	+0.3	+0.3	+0.9	-1.5	+1.4	+0.4	-1.0	+0.2

Reporting period 1 July to 30 September 2005

The AGSP laboratories received a total of 968 gonococcal isolates of which 939 remained viable for susceptibility testing. This was about 15 per cent more than the 829 gonococci reported for the same period in 2004. About one third of this total was from New South Wales, a quarter from Victoria, 13.5 per cent each from the Northern Territory and Queensland, 10 per cent from Western Australia, and 5 per cent from South Australia. There were five isolates each from Tasmania and the Australian Capital Territory.

Penicillins

In this quarter, 338 (36%) of the 939 isolates examined were penicillin resistant by one or more mechanisms. One hundred and twenty-four (13.2%) were penicillinase producing *Neisseria gonorrhoeae* (PPNG) and 214 (22.8%) were resistant by chromosomal mechanisms, (CMRNG). The proportion of all strains resistant to the penicillins by any mechanism ranged from 1.7 per cent in the Northern Territory to 51 per cent in New South Wales. High rates of penicillin resistance were also found in Victoria (50%), South Australia (32%) and Western Australia (21%) with a lower rate, (18.5%), in Queensland.

Figure 8 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 and territory. A high proportion of those strains classified as PPNG or else resistant by chromosomal mechanisms fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

Figure 8. Categorisation of gonococci isolated in Australia, 1 July to 30 September 2005, by penicillin susceptibility and region

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

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

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

PPNG Penicillinase producing *Neisseria gonorrhoeae.*

In New South Wales, most of the penicillin resistance was due to CMRNG (121, 37.8%) with 42 PPNG (13.1%). The proportion of CMRNG in Victoria (35%) was slightly less than in New South Wales and that of PPNG slightly higher (14.8%). In other centres, PPNG formed a higher proportion of penicillin resistant gonococci. The proportion of PPNG in Queensland and Western Australia was 17 per cent and in South Australia 18 per cent. PPNG were also present in the Australian Capital Territory and the Northern Territory (1 and 2 isolates respectively), but there were no PPNG in Tasmania. CMRNG were present in Queensland (1.5% of isolates there), South Australia (13%) and Western Australia (4.5%). There were no CMRNG reported from Tasmania or the Northern Territory and a single CMRNG from the Australian Capital Territory.

Ceftriaxone

Seven isolates with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected. Four were found in New South Wales, two in Victoria and one in Queensland. All seven isolates were penicillin resistant by chromosomal mechanisms (CMRNG) and five were also quinolone resistant (ciprofloxacin MICs 1 mg/L or more). It is emphasised that no treatment failures have been documented locally when a 250 mg IM dose of ceftriaxone has been used.

Spectinomycin

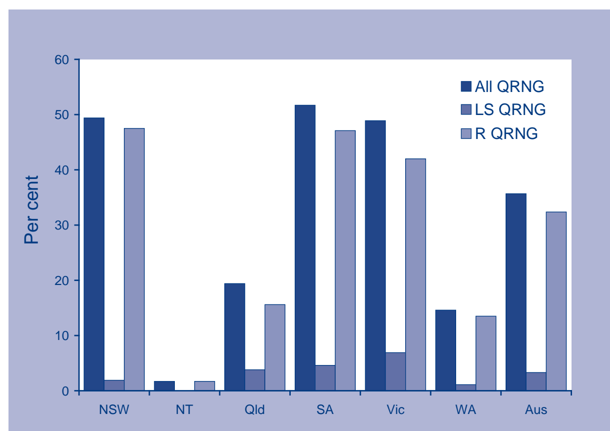
All isolates were susceptible to this injectable agent.

Quinolone antibiotics

The number (335) and proportion (35.7%) of quinolone resistant *N. gonorrhoeae* (QRNG) detected in this quarter represent the highest rates of QRNG found in this program to date. In the third quarter of 2004 there were 200 QRNG, 24 per cent of all gonococci tested. The majority of QRNG (304 of 335, 91%) exhibited higher-level resistance to ciprofloxacin of 1 mg/L or more (Figure 9). 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 again widely distributed and were detected in all states and territories with the exception of Tasmania. The highest proportion of QRNG was found in South Australia where 23 QRNG were 52 per cent of all gonococci tested. In Victoria there were 112 QRNG (49%), in New South Wales 158 QRNG (also 49% of isolates), in Queensland 25 (19%), in Western Australia 13 (14%) with two QRNG detected in both the Northern Territory and the Australian Capital Territory.

Figure 9. The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 July to 30 September 2005, by jurisdiction



LS QRNG Ciprofloxacin MICs 0.06–0.5 mg/L.

R QRNG Ciprofloxacin MICs ≥1 mg/L.

High level tetracycline resistance

The number (156) and proportion (16.6%) of high level tetracycline resistance (TRNG) detected was higher than that recorded in the 2004 (121, 14.6%) figures. TRNG were found in all states and territories except for Tasmania and the Australian Capital Territory and represented between 2.5 per cent (Northern Territory) and 26 per cent of isolates (Victoria).

Reference

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

HIV and AIDS surveillance

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

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in 'HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia, annual surveillance report'. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Internet: http://www.med.unsw.edu.au/nchecr. Telephone: +61 2 9332 4648. Facsimile: +61 2 9332 1837. For more information see Commun Dis Intell *2006;30:159.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 July to 30 September 2005, as reported to 31 December 2005, are included in this issue of Communicable Diseases Intelligence *(Tables 9 and 10).*

Table 9. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 July to 30 September 2005, 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 2005	This period 2004	YTD 2005	YTD 2004
HIV diagnoses	Female	0	12	0	1	0	0	7	4	24	16	71	86
	Male	3	61	1	37	6	0	67	12	187	187	629	578
	Not reported	0	0	0	0	0	0	0	0	0	0	0	1
	Total*	3	73	1	38	6	0	74	16	211	203	700	666
AIDS diagnoses	Female	0	2	0	2	0	0	3	1	8	2	20	13
	Male	0	13	0	3	1	0	17	0	34	30	102	113
	Total*	0	15	0	5	1	0	20	1	42	32	122	127
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	2	2	6
	Male	0	4	0	1	0	0	4	0	9	16	34	54
	Total*	0	4	0	1	0	0	4	0	9	18	36	60

* Totals include people whose sex was reported as transgender.

Table 10. Cumulative diagnoses of HIV infection, AIDS, and deaths following AIDS since the introduction of HIV antibody testing to 30 September 2005 and reported by 31 December 2005, by sex and state or territory

Sex		State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	30	812	18	241	87	8	333	179	1,708
	Male	252	12,983	124	2,553	870	90	4,926	1,140	22,938
	Not reported	0	233	0	0	0	0	22	0	255
	Total*	282	14,056	142	2,803	958	98	5,300	1,326	24,965
AIDS diagnoses	Female	9	236	2	68	31	4	105	36	491
	Male	92	5,230	41	994	391	48	1,913	415	9,124
	Total*	101	5,482	43	1,064	423	52	2,028	453	9,646
AIDS deaths	Female	6	132	1	41	20	2	59	24	285
	Male	71	3,536	26	646	272	32	1,380	291	6,254
	Total*	77	3,678	27	689	292	34	1,447	316	6,560

* Totals include people whose sex was reported as transgender.

National Enteric Pathogens Surveillance System

The National Enteric Pathogens Surveillance System (NEPSS) collects, analyses and disseminates data on human enteric bacterial infections diagnosed in Australia. Communicable Diseases Intelligence *NEPSS quarterly reports include only* Salmonella. *NEPSS receives reports of* Salmonella *isolates that have been serotyped and phage typed by the 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 2005 are included in Tables 11 and 12. Data include cases reported and entered by 20 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 *2006;30:159–160.*

Fourth quarter 2005

The total number of reports to NEPSS of human *Salmonella* infection rose to 2,198 in the fourth quarter of 2005, 82 per cent more than in the third quarter of 2005. This increase significantly exceeds the usual seasonal surge in reports in the latter months of each year. The fourth quarter count was 25 per cent more than the comparable fourth quarter of 2004 and approximately 30 per cent greater than the 10-year historical mean for this period. Much of this increase is accounted for by two phage types of *S*. Typhimurium, PT 135 and PT 44.

Reports of *S*. Typhimurium PT 135 increased markedly this quarter; some cases associated with defined outbreaks, others occurring as apparently sporadic infections. This increase was most apparent in the south-eastern States and Western Australia. The phage typing laboratories noted that the majority (approximately 85%) of isolates of *S*. Typhimurium PT 135 during this period manifested a consistent pattern of phage reactions. In the present summary report, these are all included within the *S*. Typhimurium PT 135 category.

Reports of *S*. Typhimurium PT 44 also increased this quarter, with most cases in the eastern mainland States.

Other common salmonellae with counts above their historical averages during the fourth quarter included *S*. Oranienburg (in Western Australia), *S*. Typhimurium PT 197 (in Queensland and New South Wales) and *S*. Saintpaul (in Queensland).

During the fourth quarter of 2005, the 25 most common *Salmonella* types in Australia accounted for 1,491 cases, 68 per cent of all reported human *Salmonella* infections. Nineteen of the 25 most common *Salmonella* infections in the fourth quarter of 2005 were also among the 25 most commonly reported in preceding quarter.

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 11. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 October to 31 December 2005, as reported to 20 January 2006

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total all *Salmonella* for quarter	19	550	77	608	133	197	414	200	2,198
Total contributing *Salmonella* types	11	117	33	117	50	16	97	67	236

Table 12. Top 25 *Salmonella* types identified in Australia, 1 October to 31 December 2005, by state or territory

National rank	*Salmonella* type	State or territory								Total 4th quarter 2005	Last 10 years mean 4th quarter	Year to date 2005	Year to date 2004
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
1	*S.* Typhimurium PT 135	2	71	0	46	5	149	125	27	425	137	802	564
2	*S.* Typhimurium PT 44	6	62	0	48	28	6	50	8	208	17	220	34
3	*S.* Saintpaul	0	21	7	66	3	0	9	5	111	73	433	397
4	*S.* Typhimurium PT 9	3	20	0	16	8	7	31	2	87	122	430	364
5	*S.* Typhimurium PT 170	0	35	0	8	0	2	20	0	65	56	469	579
6	*S.* Birkenhead	0	28	0	32	0	0	3	1	64	60	218	264
7	*S.* Typhimurium PT 197	0	23	0	28	1	0	3	2	57	18	547	268
8	*S.* Virchow PT 8	0	9	2	30	0	0	2	11	54	43	243	334
9	*S.* Oranienburg	0	3	0	0	5	1	2	43	54	11	87	43
10	*S.* Chester	0	9	3	20	0	1	4	2	39	36	185	190
11	*S.* Infantis	0	15	1	1	9	0	8	2	36	30	170	158
12	*S.* Muenchen	0	7	3	15	4	0	0	2	31	26	142	116
13	*S.* Aberdeen	0	4	0	24	0	0	0	0	28	21	152	134
14	*S.* Hvittingfoss	0	5	0	20	0	0	1	1	27	18	184	149
15	*S.* Potsdam	0	3	0	17	1	0	2	0	23	18	49	62
16	*S.* Waycross	0	4	0	19	0	0	0	0	23	17	113	121
17	*S.* Typhimurium RDNC	0	9	0	5	1	0	6	1	22	18	108	104
18	*S.* Typhimurium PT 12	1	5	0	0	2	0	5	8	21	14	118	233
19	*S.* Anatum	0	1	1	11	3	0	2	2	20	19	75	89
20	*S.* Mississippi	0	0	0	0	0	20	0	0	20	14	75	75
21	*S.* Stanley	1	6	0	4	1	0	4	0	16	13	67	77
22	*S.* Enteritidis PT 6a	1	6	0	1	0	0	2	6	16	6	90	72
23	*S.* Havana	0	8	0	0	1	0	1	5	15	11	38	49
24	*S.* Weltevreden	0	3	5	4	2	0	0	1	15	9	58	69
25	*S.* Agona	0	7	0	3	2	0	1	1	14	16	66	80

Overseas briefs

World Health Organization Disease Outbreak News

This material has been summarised from information provided by the World Health Organization (http://www.who.int).

Avian influenza

China

30 December 2005

The Ministry of Health in China has confirmed an additional case of human infection on the mainland with the H5N1 avian influenza virus. The case is a 41-year-old woman from the south-eastern province of Fujian. She developed symptoms of fever followed by pneumonia on 6 December, and was admitted to hospital two days later. The patient died on 21 December.

On 13 December, initial laboratory tests on samples from the patient tested negative for H5N1. But further tests on 23 December—including polymerase chain reaction tests carried out at the Chinese Center for Disease Control in Beijing—showed positive results. The virus was also isolated from the patient. Close contacts who have been placed under medical observation have not displayed any symptoms, health authorities report.

Agricultural authorities so far have not been able to confirm the presence of the H5 virus subtype in poultry in the vicinity of the patient's residence or place of work. Investigators have not been able to confirm any direct contact between the patient and poultry prior to the onset of illness. The investigation, however, is continuing and answers to these and other questions are still being sought.

This is China's seventh laboratory-confirmed human case. Of these cases, three have been fatal (including this latest case). To date, China has reported human cases in six provinces and regions: Hunan, Anhui, Guangxi, Liaoning, Jiangxi and Fujian.

Indonesia

23 December 2005

The Ministry of Health in Indonesia has confirmed two additional cases of human infection with the H5N1 avian influenza virus.

The first case occurred in an 8-year-old boy from Central Jakarta. He developed symptoms of fever and cough on 8 December, was hospitalised on 13 December, and died on 15 December.

Family members and close contacts were placed under observation and tested for possible infection; however, none have developed symptoms. Investigations have been undertaken to determine the source of the boy's exposure and samples from pigeons around his household are being tested.

The second case occurred in a 39-year-old man from East Jakarta. He first reported symptoms of fever, headache, cough and shortness of breath on 9 December. He was hospitalised on 11 December and died on 12 December.

Family members and close contacts were placed under observation. Investigations are being undertaken to determine the source of the man's exposure. While he did not keep poultry in his household, chickens and other birds were found in his neighbourhood. Samples from these birds have been taken and are undergoing tests to determine whether they may have been the source of infection.

These newly confirmed cases bring the total number in Indonesia to 16. Of these cases, 11 were fatal.

Thailand

9 December 2005

The Ministry of Public Health in Thailand has confirmed a further case of human infection with the H5N1 avian influenza virus. The case occurred in a 5-year-old boy, who developed symptoms on 25 November, was hospitalised on 5 December, and died on 7 December. The child resided in the central province of Nakhonnayok.

A thorough investigation of this case is currently under way. Early results suggest that the child may have acquired his infection from dead chickens in the neighbourhood. His family members and neighbours have been placed under medical observation. All remain healthy to date.

The child is the fifth laboratory-confirmed case in Thailand this year and the second death. Since January 2004, Thailand has reported 22 cases, of which 14 were fatal.

ProMED-mail

This material has been summarised from information provided by ProMED-mail (http://www.promedmail.org).

Chikungunya – Mauritius and Reunion Island

Source: Liberation, 30 December 2005 [translated and edited]

On this island of 760,000 inhabitants in the Indian Ocean, 6,200 people have been infected by the Chikungunya virus, and the number increases by 250 new cases per week.

Chikungunya is a mosquito-borne viral disease; *Aedes* mosquitoes such as *Ae. aegypti* and *Ae. africanus* are pertinent to the African context. Chikungunya virus is a member of the family *Togaviridae*. Many outbreaks of Chikungunya since the beginning of 2005 have been noted mainly in the west rim (the offshore islands of East Africa and Kenya), the north rim (Sri Lanka) as well as the east rim (Indonesia) of the Indian Ocean Basin. ‘Chikungunya’ is a Swahili word which means ‘curved up’: a rather poetic description for this disease which is not lethal, but causes acute articular pain and can lead to serious neurological complications.

The Department of Epidemiology of Reunion and the Regional Department of Health and Social Affairs fear the epidemic will spread during the next summer rainy season in the southern region.

West Nile virus update 2005 – Western Hemisphere

Source: USA Centres for Disease Control and Prevention, Division of Vectorborne Infectious Diseases, West Nile virus, 20 December 2005 [edited]

As of 20 December 2005, avian, animal or mosquito West Nile virus infections have been reported to CDC from a total of 48 states and one district. Human cases have been reported in a total of 42 states.

Human cases reported to CDC reflect both mild and severe human disease cases occurring between 1 January 2005 and 20 December 2005 that have been reported to ArboNet by state and local health departments.

Of the 2,799 cases, 1,168 (42%) were reported as West Nile meningitis or encephalitis (neuro-invasive disease), 1,472 (53%) were reported as West Nile fever (milder disease), and 159 (6%) were clinically unspecified at this time.

The high proportion of neuro-invasive disease cases among reported cases of West Nile virus disease reflects surveillance reporting bias. Serious cases are more likely to be reported than mild cases. Also, the surveillance system is not designed to detect asymptomatic infections. Data from population-based surveys indicate that, among all people who become infected with West Nile virus (including people with asymptomatic infections), less than one per cent will develop severe neuro-invasive disease.

Avian influenza – Eurasia – FAO, update

Source: FAO-AIDE News, issue No 37, 23 December 2005 [edited].

Latest information on avian influenza

The first case of highly pathogenic avian influenza (HPAI) H5N1 was reported from the Republic of Korea on 12 December 2003. The disease has spread from South East Asia to the north-west involving Quinghai Lake, Xinjang Province in China, Mongolia, Russia, Kazakhstan, Romania, Turkey, Croatia, and now has also been confirmed in the Ukraine.

Country situation

Europe

Ukraine

22 December 2005

Massive deaths of poultry started on 25 November 2005 on the Crimean Peninsula, and preliminary testing confirmed the presence of avian influenza virus of sub-type H5 on 8 December 2005. It was later confirmed as HPAI H5N1. The disease has so far spread to at least 27 villages on the Crimean Peninsula.

Control measures imposed include quarantine of infected properties, the creation of sanitary cordons of approximately 3 km radius and prohibition of the sale of backyard poultry and poultry products in the Crimea. Veterinarians and soldiers have culled more than 67,000 domestic fowl, including chicken, geese, ducks and turkeys in affected villages.

Romania

22 December 2005

In November, AI sub-type H5 outbreaks were reported in seven swans, a chicken and a turkey in Calarasi and Braila counties, and H5N1 was confirmed. During December, the disease was further reported in chickens and ducks another three counties near the border with the Ukraine. Romania has been conducting surveillance by the sampling of 1,200 birds every week. The Ministry of Agriculture urged villagers to keep their poultry confined to avoid contact with migrating birds. Some 53,000 poultry have been culled in Romania between 7 October 2005 and early December.

Russia

20 December 2005

Outbreaks were suspected or confirmed in Omsk, Tambov, Cheliabinsk, Altai, Kurgan Regions in November, and in Kurgan, Astrakhan and Kalmykia regions in December. In Astrakhan regions (in the Volga Delta near the border with Kazakhstan) and in Kalmykia, around 600 dead swans have been found.

Research undertaken by the Russian zoonotic infections laboratory indicated differences between viruses isolated in the Novosibirsk Region: virus isolated in the summer/autumn of 2005 was almost identical to the strain that caused an outbreak in northern China in spring 2005 but the virus found in the second outbreak was similar to virus found in Viet Nam in 2002–2003.

South, South East and East Asia

China

15 December 2005

Further outbreaks were confirmed in chickens, ducks and geese in 8 of the 30 provinces/autonomous regions during November and in one in December. The most recent case was reported in ducks in Suichuan County (Jiangxi Province) on 6 December 2005. The country decided to vaccinate all 14 billion poultry. There had been 21 outbreaks in China this year, 144,624 birds have died and 21.1 million have been culled.

Viet Nam

21 December 2005

The latest case of HPAI was found on 15 December 2005 in a flock of 140 ducks in Ninh Binh Province. Since 1 October 2005, a total of 3,702,257 poultry have been culled of which 1,245,072 were chickens and 1,980,369 ducks. As of 21 December 2005, vaccination has been implemented in 64 provinces and cities, of which two rounds of vaccination have been completed in 21 provinces. A total of 135.3 and 67.7 million doses have been vaccinated in chickens and ducks respectively.

Indonesia

30 November 2005

Outbreaks have been detected in 23 of Indonesia's 33 provinces. H5N1 virus has been discovered in Aceh Province. Birds were found infected with avian influenza virus in 7 of 20 sub-districts in Jakarta. Outbreaks were also suspected in Tangerang Province, West Java and East Nusa Tenggara Province and West Timor.

Creutzfeldt-Jakob disease (new variant) update 2005

Source: UK Department of Health, Monthly Creutzfeldt-Jakob Disease Statistics, Press release no. 2005/0438, 5 December 2005 [edited]

Definite and probable CJD cases in the United Kingdom (UK), as of 2 December 2005:

Summary of vCJD cases – deaths

Deaths from definite vCJD (confirmed): 109

Deaths from probable vCJD (without neuropathological confirmation): 43

Deaths from probable vCJD (neuropathological confirmation pending): 1

Number of deaths from definite or probable vCJD (as above): 153

Summary of vCJD cases – alive

Number of probable vCJD cases still alive: 6

Total number of definite or probable vCJD (dead and alive): 159

Since the previous monthly statistics were released, the total number of deaths from definite or probable vCJD has increased by one. Consequently, the overall total number of definite or probable vCJD cases (dead and alive) is now 159. These data are consistent with the view that the vCJD outbreak in the UK is now in decline. The number of deaths due to definite or probable vCJD in the UK during the 1st 11 months of 2005 is now 5. The peak number of deaths was 28 in the year 2000, followed by 20 in 2001, 17 in 2002, 18 in 2003, and 9 in 2004.

Marburg haemorrhagic fever – Angola

Source: Terranet plus Beirut, 7 November 2005 [edited]

Angola is officially free of Marburg haemorrhagic fever after the outbreak of the [filoviral] disease that killed 227 of the 252 people it infected over the last year.

The Angolan outbreak began in October 2004 in a hospital in Uige province but was not formally identified until March 2005, by which time the epidemic had prompted the deployment of dozens of experts from the WHO and other organisations. There is no cure for Marburg haemorrhagic fever, whose exact origin is unknown and which was first detected in 1967, when West German laboratory workers in the town of Marburg were infected by monkeys from Uganda. It spreads through contact with bodily fluids such as blood, excrement, vomit, saliva, sweat, and tears but can be contained with relatively simple hygienic precautions, according to experts. The most serious outbreak of Marburg until now had been in the Democratic Republic of Congo, where 123 people died between 1998 and 2000.

No human case of Marburg haemorrhagic fever has been reported in Angola since July 2005. The final assessment appears to be that during the course of the outbreak, there were 252 confirmed cases of infection and 227 of those died. These figures have been revised down from the 374 cases and 329 deaths reported by the WHO on 24 August 2005. The Angolan outbreak remains the most serious outbreak of Marburg haemorrhagic fever so far recorded.

Japanese encephalitis virus infection – India (Uttar Pradesh)

Source: Sify, Chennai, India, 4 November 2005 [edited]

The official death toll from Japanese encephalitis virus infection in Uttar Pradesh climbed to more than 1,300 on Friday, health officials said. The vast majority of the dead are children, mostly malnourished and under the age of 15. Some 250 people are still in state-run hospitals with encephalitis.

The encephalitis outbreak was first reported from Gorakhpur, 250 kilometres south-east of Lucknow in July and more than 4,900 cases have since been recorded.

Deaths hit a record 1,228 in 1988. The death toll for the current year has surpassed this threshold and made a new record at 1,302.

Poliomyelitis – Indonesia

Source: Jakarta Post 19 October 2005 [edited]

The government has reported the discovery of polio cases in Riau and Aceh Provinces as it prepares to embark on a third vaccination drive in November 2005 to fight the virus that has infected 269 children since it resurfaced in March 2005 after a decade-long absence.

Indonesia has staged two rounds of the polio vaccination drive so far, the first in August 2005 and the second in September 2005. The next round of the drive would likely be held on 27 November 2005.

The latest outbreak is said to be genetically linked to the virus circulating in Sudan, Saudi Arabia and Yemen. The authorities believe that it was brought to Indonesia by migrant workers, pilgrims or travellers.

The identification of the two most recent cases is another setback for interruption of transmission of wild poliovirus in Indonesia. The two provinces are geographically separated from Lampung, the most southerly province on Sumatra Island, suggesting significant spread of the wild poliovirus on the island of Sumatra.

As of 11 October 2005, a total of 1,349 cases were reported, of which 489 were reported from Nigeria, 472 from Yemen, 264 from Indonesia, 37 from India, 26 from Sudan, 18 from Pakistan, 17 from Ethiopia, eight from Angola, four from Afghanistan, four from Niger, three from Mali, three from Somalia, one from Chad, one from Eritrea, one from Cameroon, and one from Nepal.