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Australia's notifiable diseases status, 2001

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

National Centre in HIV Epidemiology and Clinical Research

National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

National Enteric Pathogens Surveillance Scheme

National Rotavirus Research Centre

Sentinel Chicken Surveillance Programme

The National Creutzfeldt-Jakob Disease Registry

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Abstract

In 2001 there were 104,187 notifications of communicable diseases in Australia reported to the National Notifiable Diseases Surveillance System (NNDSS). The number of notifications in 2001 was an increase of 16 per cent of those reported in 2000 (89,740) and the largest annual total since the NNDSS commenced in 1991. In 2001, nine new diseases were added to the list of diseases reported to NNDSS and four diseases were removed. The new diseases were cryptosporidiosis, laboratory-confirmed influenza, invasive pneumococcal disease, Japanese encephalitis, Kunjin virus infection, Murray Valley encephalitis virus infection, anthrax, Australian bat lyssavirus, and other lyssaviruses (not elsewhere classified). Bloodborne virus infections remained the most frequently notified disease (29,057 reports, 27.9% of total), followed by sexually transmitted infections (27,647, 26.5%), gastrointestinal diseases (26,086, 25%), vaccine preventable diseases (13,030 (12.5%), vectorborne diseases (5,294, 5.1%), other bacterial infections (1,978, 1.9%), zoonotic infections (1,091, 1%) and four cases of quarantinable diseases. In 2001 there were increases in the number of notifications of incident hepatitis C, chlamydial infections, pertussis, Barmah Forest virus infection and ornithosis. There were decreases in the number of notifications of hepatitis A, *Haemophilus influenzae* type b infections, measles, rubella, Ross River virus infections and brucellosis. This report also summarises data on communicable diseases from other surveillance systems including the Laboratory Virology and Serology Reporting Scheme and sentinel general practitioner schemes. In addition, this report comments on other important developments in communicable disease control in Australia in 2001. *Commun Dis Intell* 2003;27:1–78.

Keywords: surveillance, communicable diseases, epidemiology

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Abbreviations used in this report

7vPCV	7-valent conjugate pneumococcal vaccine
23vPPV	23-valent conjugate pneumococcal vaccine
AIDS	Acquired immune deficiency syndrome
ASPREN	Australian Sentinel Practice Research Network
BF	Barmah Forest virus
CDNA	Communicable Diseases Network Australia
CJD	Creutzfeldt-Jakob disease
DoHA	Department of Health and Ageing
DT	Definitive Type (of *Salmonella*)
DTP	Diphtheria-tetanus-pertussis
EAGAR	Expert Advisory Group for Antimicrobial Resistance
Hib	*Haemophilus influenzae* type b
HIV	Human immunodeficiency virus
HUS	Haemolytic uraemic syndrome
ICD10-AM	International Classification of Diseases, version 10, Australian Modification
IDU	Injecting drug use(r)
IPD	Invasive pneumococcal disease
JE	Japanese encephalitis virus
JETACAR	Joint Expert Technical Advisory Committee on Antibiotic Resistance
LabVISE	Laboratory Virology and Serology Reporting Scheme
MMR	Measles-mumps-rubella
MVE	Murray Valley encephalitis virus
NCHECR	National Centre in HIV Epidemiology and Clinical Research
NCIRS	National Centre for Immunisation Research and Surveillance
NEC	Not elsewhere classified
NN	Not notifiable
NNDSS	National Notifiable Diseases Surveillance System
OPV	Oral polio vaccine
PNG	Papua New Guinea
RR	Ross River virus
SLTEC/VTEC	Shiga-like toxin producing *Escherichia coli*, Verotoxin-producing *E. coli*
STI(s)	Sexually transmitted infection(s)
STM	*Salmonella* Typhimurium
TB	Tuberculosis
USA	United States of America
WHO	World Health Organization
WPR	Western Pacific Region

2001: the year in review

The year 2001 will be remembered for the terrorist attacks on the United States of America (USA) and the deliberate release of anthrax. A total of 22 cases of anthrax were detected and there were five deaths.[1] In response to these events, governments around the world prepared for bioterrorism by stockpiling of vaccines and antibiotics, monitoring unusual clinical presentations through 'syndromic surveillance' and strengthening laboratory capacity to test clinical and environmental samples for pathogens of biosecurity concern. The release of anthrax in the USA was followed by 'white powder incidents' in Australia and elsewhere, straining emergency, medical and laboratory services. No deliberate releases of pathogens were detected in Australia.

Improvements continued to be made in the surveillance and control of communicable diseases in Australia in 2001. Following the demonstration of high vaccine efficacy in the USA,[2] the seven-valent conjugate pneumococcal vaccine (7vPCV) was introduced in Australia in July 2001. A targeted vaccination schedule was developed to immunise children at high risk. Enhanced surveillance was introduced to measure the impact of vaccines on the serotype frequency and prevalence of antibiotic resistance in the pneumococci. Continued development of Australia's response to the transmissible spongiform encephalopathies included the introduction of a certification system for imported beef products in July 2001.[3] The publication of *Guidelines for the early clinical and public health management of meningococcal disease in Australia* by the Communicable Diseases Network Australia (CDNA) in June 2001 was timely as there were a number of highly publicised clusters of meningococcal cases in Australia later in 2001.

Internationally, cases of vaccine-derived polio causing paralytic disease caused concern about the global polio eradication program. An outbreak of 21 cases of polio in Hispaniola and three cases in the Philippines occurred in communities with relatively low vaccination rates. These outbreaks demonstrate the potential of the polio virus to evade the impact of vaccination, and underline the importance of maintaining high levels of vaccination coverage.

New molecular clues to the basis of the virulence of pandemic strains of laboratory-confirmed influenza were unravelled in 2001.[4,5] The Australian Action Plan for Pandemic Influenza, updated in 2001, established plans, levels of alerts and responsibilities for the control of an influenza pandemic, were one to occur.

The surveillance of communicable diseases in Australia was further improved in 2001 by a revision of the diseases under surveillance and through the introduction of enhanced surveillance of invasive pneumococcal disease. In their first year of operation OzFoodNet, the network of foodborne disease epidemiologists in Australia, were involved in the control of two international foodborne disease outbreaks and identified 86 domestic outbreaks. The OzFoodNet report for 2001 provides valuable additional information about the epidemiology of foodborne disease in Australia.[6]

Control of communicable diseases in Australia continues to face challenges. In 2001, these included imported cases of measles causing outbreaks among unvaccinated people. Clusters of meningococcal disease in adolescents and young adults in a series of well publicised clusters in 2001 and 2002, prompted the Commonwealth Government to commence an immunisation program with the meningococcal C vaccine.

Continued improvements will need to be made to surveillance systems to manage the changing epidemiology of communicable diseases in Australia and to provide essential data for biosecurity.

Introduction

Surveillance of communicable diseases is vital for the control of communicable diseases, to identify and assess diseases requiring control or prevention and to monitor trends over time. It is also required for the guidance of policy making.

Surveillance in Australia exists at the national, state/territory and local levels. Primary responsibility for public health action lies with the states and territories and local health authorities.

The role of surveillance at a national level includes:

- identifying national trends in disease;
- guidance for policy development at a national level and resource allocation;
- Monitoring the need for and impact of national control programs;
- coordination of 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 a Commonwealth responsibility.

The National Notifiable Diseases Surveillance System (NNDSS) is based on fortnightly reporting by the states and territories to the Commonwealth. Fifty-five communicable diseases agreed upon nationally through CDNA are reported to NNDSS (Table 1). The system is complemented by several other surveillance systems, which provide information on other particular diseases, including some that are not reported to NNDSS.

Table 1. Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2001

Disease group	Disease	Data received from:*
Bloodborne diseases	Hepatitis B (incident)	All jurisdictions
	Hepatitis B (unspecified)	All jurisdiction, except NT
	Hepatitis C (incident)	All jurisdictions except Queensland and NT
	Hepatitis C (unspecified)	All jurisdictions
	Hepatitis D	All jurisdictions
	Hepatitis (NEC)	All jurisdictions
Gastrointestinal diseases	Botulism	All jurisdictions
	Campylobacterosis	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
	Viral haemorrhagic fever	All jurisdictions
	Yellow fever	All jurisdictions
Sexually transmissible infections	Chlamydial infection	All jurisdictions
	Donovanosis	All jurisdictions except SA
	Gonococcal infection	All jurisdictions
	Syphilis	All jurisdictions
Vaccine preventable diseases	Diphtheria	All jurisdictions
	Haemophilus influenzae type b	All jurisdictions
	Laboratory-confirmed influenza	All jurisdictions
	Measles	All jurisdictions
	Mumps	All jurisdictions
	Pertussis	All jurisdictions
	Invasive pneumococcal disease	All jurisdictions
	Poliomyelitis	All jurisdictions
	Rubella	All jurisdictions
	Tetanus	All jurisdictions
Vectorborne diseases	Arbovirus infection NEC	All jurisdictions
	Barmah Forest virus infection	All jurisdictions
	Dengue	All jurisdictions
	Japanese encephalitis	All jurisdictions
	Kunjin	All jurisdictions except ACT†
	Malaria	All jurisdictions
	Murray Valley encephalitis	All jurisdictions†
	Ross River virus infection	All jurisdictions
Zoonoses	Anthrax	All jurisdictions except SA
	Australian bat lyssavirus	All jurisdictions
	Brucellosis	All jurisdictions
	Leptospirosis	All jurisdictions
	Ornithosis	All jurisdictions
	Lyssaviruses (unspecified)	All jurisdictions
	Q fever	All jurisdictions

Table 1 (continued). Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2001

Disease group	Disease	Data received from:*
Other bacterial infections	Legionellosis	All jurisdictions
	Leprosy	All jurisdictions
	Invasive meningococcal infection	All jurisdictions
	Tuberculosis	All jurisdictions

* Jurisdictions may not have reported a disease either because legislation had not yet made that disease notifiable in that jurisdiction, or because notification data for that disease were not reported to the Commonwealth in 2001.

† In the Australian Capital Territory, infections with Murray Valley encephalitis virus and Kunjin virus were combined under Murray Valley encephalitis

NEC: Not elsewhere classified

The results of communicable disease surveillance are reported through several avenues of communication. Fortnightly teleconferences of the CDNA provide the most up-to-date information on topics of immediate interest. The *Communicable Diseases Intelligence* journal, published quarterly, contains results of surveillance and research reports on the epidemiology and control of various communicable diseases. Data summaries are published on the Communicable Diseases Australia website on a fortnightly basis. The annual report of the NNDSS, Australia's notifiable diseases status, provides yearly summaries of notifications.

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 Commonwealth Department of Health and Ageing (DoHA) does not have any legislated responsibility for public health apart from human quarantine. States and territories have agreed to forward data on nationally agreed communicable diseases to DoHA for the purposes of national communicable disease surveillance.

In 2001, data were transmitted to DoHA each fortnight by the states and territories. The Commonwealth received final data sets for 2001 from the states and territories by July 2002. Apparent errors or incomplete data for some diseases, together with any queries arising from the data, were returned to the states and territories for review.

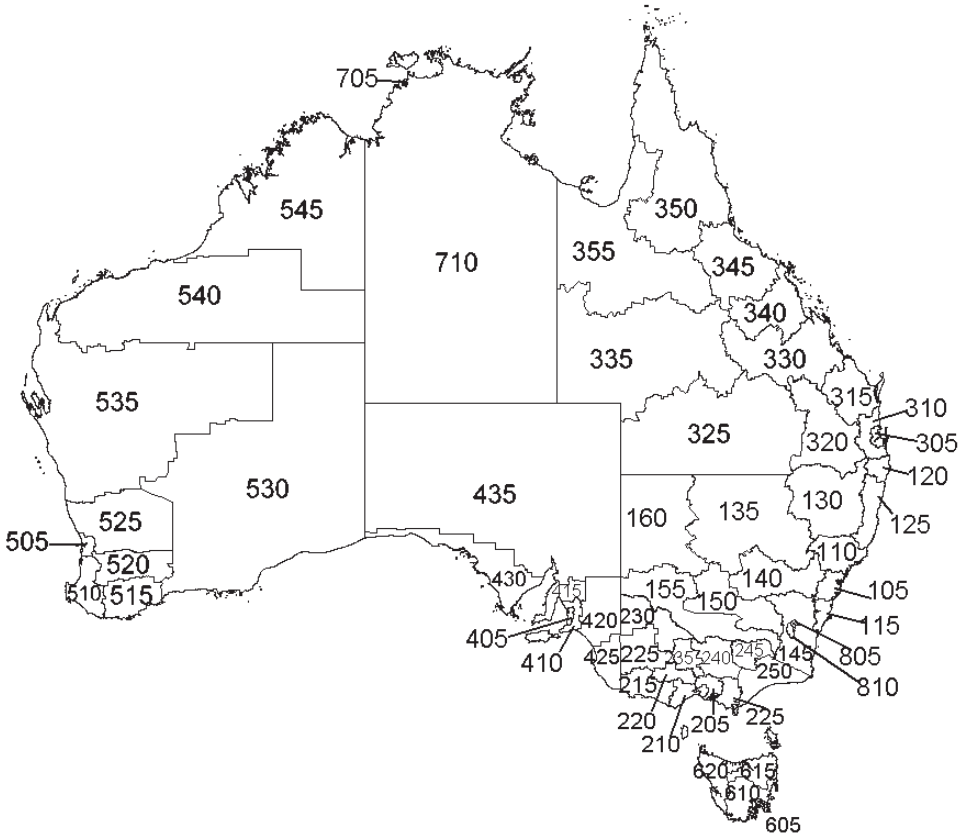
The national data set includes fields for a unique record reference number; notifying state or territory; disease code; age; sex; Indigenous status; postcode of residence; the date of onset of the disease; and the date of report to the state or territory health department. Additional information was available on the species and serogroups isolated in cases of legionellosis, invasive meningococcal disease and malaria, and on the vaccination status in cases of childhood vaccine-preventable diseases. 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.

Analyses in this report are based on date of disease onset, unless otherwise specified. For analysis of seasonal trends, notifications were reported by month of onset. Population notification rates were calculated using 2001 mid-year census-based estimates of the resident population, supplied by the Australian Bureau of Statistics (Appendix 1). Population data used in previous annual reports was based on forward projections from the 1996 census. The population calculated for the year 2001 is less than the year 2000 estimate. Comparison of rates across these years will thus be subject to slight error.

Where diseases were not notifiable in a state or territory for a particular year, adjusted rates were calculated using a denominator excluding that jurisdiction's population. The Australian Institute of Health and Welfare supplied hospital admission data for the financial year 2000–01.

Maps were generated using MapInfo, and were based on postcodes of residence, which have been allocated to Statistical Divisions by the Australian Bureau of Statistics (Map 1). The two Statistical Divisions that make up the Australian Capital Territory are combined, as the population for one division is very small. Similarly, the Darwin and 'Northern Territory – balance' Statistical Divisions have been combined to calculate rates for the Northern Territory as a whole. 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 divided into three equal-sized groups.

Map 1. Australian Bureau of Statistics Statistical Divisions



Statistical Division		Population
Australian Capital Territory		
805	Canberra	310,521
810	ACT - balance	318
New South Wales		
105	Sydney	4,085,578
110	Hunter	576,863
115	Illawarra	389,271
120	Richmond-Tweed	211,167
125	Mid-North Coast	272,966
130	Northern	173,218
135	North Western	116,895
140	Central West	172,749
145	South Eastern	182,464
150	Murrumbidgee	148,737
155	Murray	109,960
160	Far West	23,587
Northern Territory		
705	Darwin	90,011
710	NT - balance	105,452
Queensland		
305	Brisbane	1,626,865
310	Moreton	694,464
315	Wide Bay-Burnett	234,751

Statistical Division		Population
Queensland continued		
320	Darling Downs	202,352
325	South West	25,597
330	Fitzroy	181,206
335	Central West	12,135
340	Mackay	127,531
345	Northern	200,174
350	Far North	225,522
355	North West	35,760
South Australia		
405	Adelaide	1,096,102
410	Outer Adelaide	110,663
415	Yorke & Lower North	44,225
420	Murray Lands	68,497
425	South East	62,794
430	Eyre	33,493
435	Northern	81,860
Tasmania		
605	Greater Hobart	194,228
610	Southern	34,832
615	Northern	133,080
620	Mersey-Lyell	108,236

Statistical Division		Population
Victoria		
205	Melbourne	3,466,025
210	Barwon	249,067
215	Western District	98,344
220	Central Highlands	138,229
225	Wimmera	50,838
230	Mallee	88,372
235	Loddon-Campaspe	162,031
240	Goulburn	188,124
245	Ovens-Murray	90,943
250	East Gippsland	79,849
255	Gippsland	154,034
Western Australia		
505	Perth	1,381,127
510	South West	187,862
515	Lower Great Southern	52,128
520	Upper Great Southern	19,610
525	Midlands	52,986
530	South Eastern	58926
535	Central	60,253
540	Pilbara	40,429
545	Kimberley	30,539
Total Australia		19,467,896

Notes on interpretation

The notifications reported to the NNDSS may be influenced by a number of factors that should be considered when interpreting the data. Due to under-reporting, notified cases can only represent a proportion of the total number of cases that occurred (Figure 1). This proportion (the 'notified fraction') varies between diseases, between states and territories and with time.

Figure 1. The communicable disease surveillance pyramid

Adopted from the Centers for Disease Control and Prevention Website: (http://www.cdc.gov/foodnet/Surveys.htm#whatpyr)

The surveillance pyramid is a model for understanding disease reporting. This illustration shows the chain of events that must occur for an episode of illness in the population to be notified. At the bottom of the pyramid, 1) some of the general population is exposed to an organism; 2) exposed persons become ill; 3) the illness is sufficiently troubling that some persons seek care; 4) a specimen is obtained from some persons and submitted to a clinical laboratory; 5) a laboratory appropriately tests the specimen; 6) the laboratory identifies the causative organism and thereby confirms the case, or the diagnosing doctor confirms the case on clinical grounds; 7) the laboratory-confirmed or clinically-confirmed case is reported to a local or state health department, then to the Commonwealth.

Methods of surveillance can vary between states and territories, each with different requirements for notification by medical practitioners, laboratories and hospitals. In addition, the list of notifiable diseases and the case definitions may vary between states and territories.

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 records, duplication in reporting may also occur if patients move from one jurisdiction to another and were notified in both.

The completeness of data in this report is summarised in Appendix 2. The patient's sex was missing in 0.5 per cent of notifications (n=509) and patient's age missing in 0.9 per cent of notifications (n=900). The patient's Indigenous status was reported for 55,084 (52.9%) notifications nationally. The proportion of reports with missing data in these fields varied by state and territory and by disease.

The date of disease onset is uncertain for some communicable diseases and is often equivalent to the date of presentation to a medical practitioner or date of specimen collection at a laboratory. Analysis by disease onset is an attempt to estimate disease activity within a reporting period. As considerable time may have elapsed between onset and report dates for some diseases, analyses were performed by report date for hepatitis B (unspecified) and hepatitis C (unspecified).

Between May and August every year, DoHA receives a final annual dataset from all states and territories. This yearly procedure updates only the notifications reported to NNDSS during the last calendar year. States and territories may still revise notification counts for earlier years, as duplicates are removed and other data corrected. An update of historical data for 1991 to 1999 was carried out during the year 2000 to address this issue. States and territories were also surveyed on changes in surveillance and other disease control or health promotion activities during 2001.

The present report is based on 'finalised' annual data from each state and territory, from which duplicate records or erroneous data have been removed. Totals in this report may vary slightly from the cumulative totals of the numbers reported in *Communicable Diseases Intelligence*. The present report has been informed by the discussions and comments of CDNA members and state and territory epidemiologists. The state and territory data managers also met through 2001, and their contribution to the accuracy of these data is gratefully acknowledged.

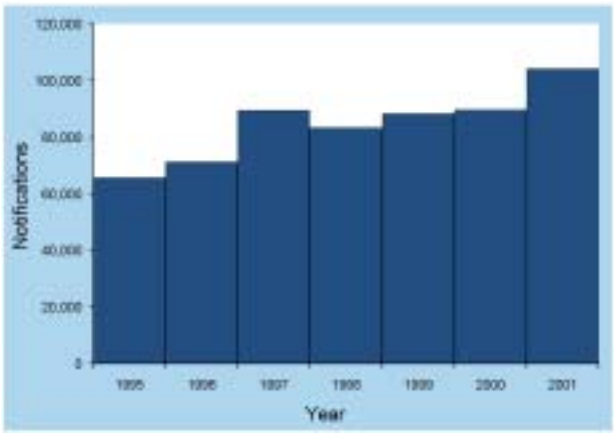
Results

Summary of 2001 data

There were 104,187 communicable disease notifications for 2001 (Table 2). Notification rates per 100,000 population for each disease by state or territory are shown in Table 3. Comparative data for the period 1997 to 2001 are shown in Table 4a. Table 4b presents details on reporting of diseases by states and territories since 1991.

The number of notifications in 2001 was an increase of 16 per cent on notifications in 2000 (89,740) and the largest number of reports received in any year since the NNDSS commenced in 1991 (Figure 2).

Figure 2. Trends in notifications to the National Notifiable Diseases Surveillance System, Australia, 1991 to 2001



In part the increase in total notifications to NNDSS in 2001 was due to changes in the number of diseases reported. In 2001, nine new diseases were added to the NNDSS and four diseases were removed. The new diseases were cryptosporidiosis, laboratory-confirmed influenza, invasive pneumococcal disease, Japanese encephalitis (JE), Kunjin virus infection, Murray Valley encephalitis virus (MVE) infection, anthrax, Australian bat lyssavirus and other lyssaviruses [not elsewhere classified (NEC)]. While there were no reports for four of these diseases, in 2001 there were 1,615 cases of cryptosporidiosis notified, 1,286 cases of laboratory-confirmed influenza, 1,681 cases of invasive pneumococcal disease, four cases of Kunjin and six cases of MVE. The four diseases removed from the NNDSS schedule in 2001 were yersiniosis, chancroid, lymphogranuloma venereum and hydatid disease, which together accounted for only 100 notifications in 2000.

In 2001, bloodborne virus infections remained the most frequently notified disease (29,057 reports, 27.9% of total), followed by sexually transmitted infections (27,647, 26.5%), gastrointestinal diseases (26,086, 25%), vaccine preventable diseases (13,030, 12.5%), vectorborne diseases (5,294, 5.1%), other bacterial infections (1,978, 1.9%), zoonotic infections (1,091, 1%) and four cases of quarantinable diseases (Figure 3).

The major changes in communicable disease notifications in 2001 are shown in Figure 4, as the ratio of notifications in 2001 compared to the mean number of notifications for the previous five years. There were increases in the number of notifications of incident hepatitis C, chlamydial infection, pertussis, Barmah Forest virus (BF) infection and ornithosis and invasive meningococcal disease. There were decreases in the number of notifications of hepatitis A, *Haemophilus influenzae* type b (Hib) infection, measles, rubella, Ross River virus (RR) infection and brucellosis.

In the financial year 2000–01, there were 89,318 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, the Australian Institute of Health and Welfare). This represents 1.5 per cent of all hospital separations in that period. A further 61,035 separations were recorded with a principal diagnosis of influenza or pneumonia (ICD10-AM J10-J18).

Figure 3. Notifications to the National Notifiable Diseases Surveillance System, Australia, 2001, by disease category

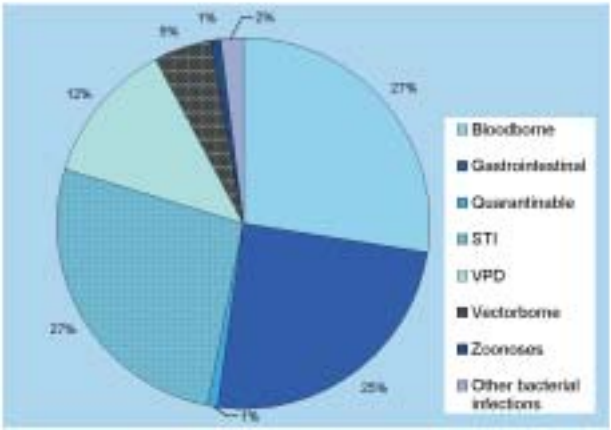


Figure 4. Selected diseases from the National Notifiable Diseases Surveillance System, comparison of total notifications for 2001 with previous five year means

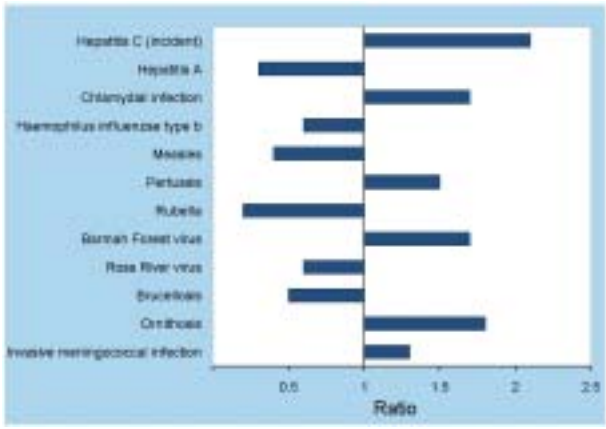


Table 2. Notifications of communicable diseases, Australia, 2001, by state or territory*

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis B (incident)	2	88	3	48	23	22	199	39	424
Hepatitis B (unspecified)†‡	54	4,710	NN	773	310	28	1,899	650	8,424
Hepatitis C (incident)	18	251	-	-	80	7	86	158	600
Hepatitis C (unspecified)†‡§	213	8,439	213	3,156	884	381	4,972	1,328	19,586
Hepatitis D	0	12	0	2	0	0	7	0	21
Hepatitis (NEC)	0	0	0	0	2	0	0	0	2
Gastrointestinal diseases									
Botulism	0	0	0	1	0	0	1	0	2
Campylobacteriosis‖	425	-	277	3,969	2,661	677	5,486	2,629	16,124
Cryptosporidiosis	10	192	248	418	66	79	436	166	1,615
Haemolytic uraemic syndrome	0	2	0	0	1	0	0	0	3
Hepatitis A	14	195	38	120	20	4	102	37	530
Hepatitis E	0	6	0	1	0	0	3	0	10
Listeriosis	1	12	0	20	6	2	10	11	62
Salmonellosis	76	1,647	373	2,201	610	163	1,085	890	7,045
Shigellosis	6	132	103	108	34	6	94	79	562
SLTEC,VTEC¶	0	1	0	14	27	0	4	3	49
Typhoid	2	32	2	10	3	1	17	17	84
Quarantinable diseases									
Cholera	0	1	0	1	1	0	1	0	4
Plague	0	0	0	0	0	0	0	0	0
Rabies	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 diseases									
Chlamydial infection	301	4,451	1,239	5,596	1,402	380	3,924	2,733	20,026
Donovanosis	0	0	13	19	NN	0	0	10	42
Gonococcal infection**	20	1,341	1,424	1,102	208	21	696	1,346	6,158
Syphilis††	11	502	427	225	20	16	15	205	1,421
Vaccine preventable diseases									
Diphtheria	0	0	1	0	0	0	0	0	1
Haemophilus influenzae type b	0	9	3	6	3	0	4	1	26
Laboratory-confirmed Influenza	14	243	92	392	135	0	177	233	1,286
Measles	0	30	0	11	2	2	83	13	141
Mumps	1	28	1	3	12	2	38	29	114
Pertussis	86	4,435	145	1,634	2,010	106	872	227	9,515
Invasive pneumococcal disease	18	434	97	425	114	61	327	205	1,681
Poliomyelitis	0	0	0	0	0	0	0	0	0
Rubella‡‡	1	58	0	134	5	2	60	3	263
Tetanus	0	0	0	0	1	1	1	0	3
Vectorborne diseases									
Arbovirus infection (NEC)	1	15	0	3	0	1	16	0	36
Barmah Forest virus infection	2	398	37	603	6	1	19	75	1,141
Dengue	11	50	43	43	7	1	6	15	176
Japanese encephalitis	0	0	0	0	0	0	0	0	0
Kunjin virus infection	-	1	2	0	0	0	0	1	4
Malaria	17	153	61	300	35	8	88	50	712
Murray Valley encephalitis	0	0	3	1	1	0	0	1	6
Ross River virus infection	9	717	223	1,569	141	13	345	202	3,219

Table 2 (continued). Notifications of communicable diseases, Australia, 2001, by state or territory*

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Zoonoses									
Anthrax	0	0	0	0	NN	0	0	0	0
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0
Brucellosis	0	0	0	17	1	0	1	0	19
Leptospirosis	0	65	4	129	3	5	37	2	245
Ornithosis	1	37	1	0	15	0	68	9	131
Lyssavirus (unspecified)	0	0	0	0	0	0	0	0	0
Q fever	2	139	0	454	16	1	65	19	696
Other bacterial infections									
Legionellosis	2	67	3	37	32	3	121	42	307
Leprosy	0	3	0	0	0	0	0	2	5
Invasive meningococcal infection	6	230	13	127	39	23	163	76	677
Tuberculosis	9	415	35	100	51	12	299	68	989
Total	1,333	29,541	5,124	23,772	8,987	2,029	21,827	11,574	104,187

* Analysis by date of onset, except for hepatitis B and hepatitis C unspecified, where analysis is by report date. Date of onset is a composite of three components: (i) the true onset date from a clinician, if available, (ii) the date the laboratory test was ordered, or (iii) the date reported to NNDSS.

† Unspecified hepatitis includes cases with hepatitis in whom the duration of illness cannot be determined.

‡ The analysis was performed by report date.

§ Includes incident hepatitis C in the Northern Territory and Queensland.

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

¶ Infections with Shiga-like toxin (verotoxin) producing *E. coli.* (SLTEC/VTEC).

** Northern Territory, Queensland, South Australia, Victoria and Western Australia: includes gonococcal neonatal ophthalmia.

†† Includes congenital syphilis.

‡‡ Includes congenital rubella.

NN Not notifiable.

NEC Not elsewhere classified.

- Elsewhere classified.

Table 3. Notification rates of communicable diseases, Australia, 2001, by state or territory (rate per 100,000 population)*

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis B (incident)	0.6	1.3	1.5	1.3	1.5	4.7	4.1	2.0	2.2
Hepatitis B (unspecified)†‡	16.7	71.3	NN	21.3	20.5	5.9	39.4	34.1	43.7
Hepatitis C (incident)	5.6	3.8	-	-	5.3	1.5	1.8	8.3	3.8
Hepatitis C (unspecified)†‡§	65.7	127.7	106.5	86.8	58.4	80.6	103.1	69.7	100.5
Hepatitis D	0.0	0.2	0.0	0.1	0.0	0.0	0.1	0.0	0.1
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis‖	131.1	-	138.5	109.2	175.7	143.1	113.8	137.9	125.2
Cryptosporidiosis	3.1	2.9	124.0	11.5	4.4	16.7	9.0	8.7	8.3
Haemolytic uraemic syndrome	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0
Hepatitis A	4.3	3.0	19.0	3.3	1.3	0.8	2.1	1.9	2.7
Hepatitis E	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Listeriosis	0.3	0.2	0.0	0.6	0.4	0.4	0.2	0.6	0.3
Salmonellosis	23.4	24.9	186.5	60.5	40.3	34.5	22.5	46.7	36.2
Shigellosis	1.9	2.0	51.5	3.0	2.2	1.3	1.9	4.1	2.9
SLTEC,VTEC¶	0.0	0.0	0.0	0.4	1.8	0.0	0.1	0.2	0.3
Typhoid	0.6	0.5	1.0	0.3	0.2	0.2	0.4	0.9	0.4
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.1	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
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 diseases									
Chlamydial infection	92.8	67.3	619.4	153.9	92.6	80.3	81.4	143.4	102.8
Donovanosis	0.0	0.0	6.5	0.5	NN	0.0	0.0	0.5	0.2
Gonococcal infection**	6.2	20.3	711.9	30.3	13.7	4.4	14.4	70.6	31.6
Syphilis††	3.4	7.6	213.5	6.2	1.3	3.4	0.3	10.8	7.3
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.5	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	1.5	0.2	0.2	0.0	0.1	0.1	0.1
Laboratory-confirmed influenza	4.3	3.7	46.0	10.8	8.9	0.0	3.7	12.2	6.6
Measles	0.0	0.5	0.0	0.3	0.1	0.4	1.7	0.7	0.7
Mumps	0.3	0.4	0.5	0.1	0.8	0.4	0.8	1.5	0.6
Pertussis	26.5	67.1	72.5	45.0	132.7	22.4	18.1	11.9	48.8
Invasive pneumococcal disease	5.6	6.6	48.5	11.7	7.5	12.9	6.8	10.8	8.6
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella‡‡	0.3	0.9	0.0	3.7	0.3	0.4	1.2	0.2	1.3
Tetanus	0.0	0.0	0.0	0.0	0.1	0.2	0.0	0.0	0.0
Vectorborne diseases									
Arbovirus infection (NEC)	0.3	0.2	0.0	0.1	0.0	0.2	0.3	0.0	0.2
Barmah Forest virus infection	0.6	6.0	18.5	16.6	0.4	0.2	0.4	3.9	5.9
Dengue	3.4	0.8	21.5	1.2	0.5	0.2	0.1	0.8	0.9
Japanese encephalitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection	-	0.0	1.0	0.0	0.0	0.0	0.0	0.1	0.0
Malaria	5.2	2.3	30.5	8.3	2.3	1.7	1.8	2.6	3.7
Murray Valley encephalitis	0.0	0.0	1.5	0.0	0.1	0.0	0.0	0.1	0.0
Ross River virus infection	2.8	10.8	111.5	43.2	9.3	2.7	7.2	10.6	16.5

Table 3 (continued). Notification rates of communicable diseases, Australia, 2001, by state or territory (rate per 100,000 population)*

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	NN	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.0	0.0	0.5	0.1	0.0	0.0	0.0	0.1
Leptospirosis	0.0	1.0	2.0	3.5	0.2	1.1	0.8	0.1	1.3
Ornithosis	0.3	0.6	0.5	0.0	1.0	0.0	1.4	0.5	0.7
Lyssavirus (unspecified)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Q fever	0.6	2.1	0.0	12.5	1.1	0.2	1.3	1.0	3.6
Other bacterial infections									
Legionellosis	0.6	1.0	1.5	1.0	2.1	0.6	2.5	2.2	1.6
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0
Invasive meningococcal infection	1.9	3.5	6.5	3.5	2.6	4.9	3.4	4.0	3.5
Tuberculosis	2.8	6.3	17.5	2.8	3.4	2.5	6.2	3.6	5.1

* Analysis by date of onset, except for hepatitis B and hepatitis C unspecified, where analysis is by report date. Date of onset is a composite of three components: (i) the true onset date from a clinician, if available, (ii) the date the laboratory test was ordered, or (iii) the date reported to NNDSS.

† Unspecified hepatitis includes cases with hepatitis in whom the duration of illness cannot be determined.

‡ The analysis was performed by report date.

§ Includes incident hepatitis C in the Northern Territory and Queensland.

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

¶ Infections with Shiga-like toxin (verotoxin) producing *E. coli.* (SLTEC/VTEC).

** Northern Territory, Queensland, South Australia, Victoria and Western Australia: includes gonococcal neonatal ophthalmia.

†† Includes congenital syphilis.

‡‡ Includes congenital rubella.

NN Not notifiable.

NEC Not elsewhere classified.

\- Elsewhere classified.

Table 4a. Notifications and notification rates of communicable diseases, Australia, 1997 to 2001, by state or territory (rate per 100,000 population)*

Disease	Notifications					Rate per 100,000 population				
	1997	1998	1999	2000	2001	1997	1998	1999	2000	2001
Bloodborne diseases										
Hepatitis B (incident)	269	265	303	395	424	1.5	1.4	1.6	2.1	2.2
Hepatitis B (unspecified)†‡	6,542	6,562	7,164	7,908	8,424	35.7	35.4	38.2	41.7	43.7
Hepatitis C (incident)	154	350	396	441	600	1.0	2.3	2.6	2.9	3.8
Hepatitis C (unspecified)†‡§	17,290	18,075	18,655	19,569	19,586	93.4	96.5	98.4	102.2	100.5
Hepatitis D	-	-	19	27	21	-	-	0.1	0.2	0.1
Hepatitis (NEC)	6	4	0	1	2	<0.1	<0.1	0.0	<0.1	<0.1
Gastrointestinal diseases										
Botulism	0	1	0	2	2	0.0	<0.1	0.0	<0.1	<0.1
Campylobacteriosis‖	11,752	13,433	12,657	13,595	16,124	95.9	108.4	100.8	107.1	125.2
Cryptosporidiosis	-	-	-	-	1,615	-	-	-	-	8.3
Haemolytic uraemic syndrome	-	-	23	15	3	-	-	0.1	0.1	<0.1
Hepatitis A	3,044	2,697	1,554	812	530	16.4	13.3	8.2	4.2	2.7
Hepatitis E	-	-	9	10	10	-	-	0.1	0.1	0.1
Listeriosis	73	55	64	67	62	0.4	0.3	0.3	0.3	0.3
Salmonellosis	7,054	7,613	7,147	6,151	7,045	38.1	40.7	37.7	32.1	36.2
Shigellosis	795	599	547	487	562	6.5	4.8	4.4	3.8	2.9
SLTEC,VTEC¶	-	-	47	33	49	-	-	0.3	0.2	0.3
Typhoid	79	60	68	58	84	0.4	0.3	0.4	0.3	0.4
Quarantinable diseases										
Cholera	2	4	3	1	4	<0.1	<0.1	<0.1	<0.1	<0.1
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
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 diseases										
Chlamydial infection	9,239	10,927	14,045	16,866	20,026	75.4	88.2	74.1	88.0	102.8
Donovanosis	49	31	17	12	42	0.5	0.3	0.2	0.1	0.2
Gonococcal infection**	4,684	5,469	5,644	5,686	6,158	25.3	29.2	29.8	29.7	31.6
Syphilis††	1,296	1,683	1,844	1,755	1,421	7.0	9.0	9.7	9.2	7.3
Vaccine preventable diseases										
Diphtheria	0	0	0	0	1	0.0	0.0	0.0	0.0	<0.1
Haemophilus influenzae type b	51	35	40	28	26	0.3	0.2	0.2	0.1	0.1
Laboratory-confirmed influenza	-	-	-	-	1,286	-	-	-	-	6.6
Measles	838	288	238	107	141	4.5	1.5	1.3	0.6	0.7
Mumps	191	182	172	212	114	1.0	1.0	1.1	1.4	0.6
Pertussis	10,825	5,791	4,417	5,942	9,515	58.4	30.9	23.3	31.0	48.8
Invasive pneumococcal disease	-	-	-	-	1,681	-	-	-	-	8.6
Poliomyelitis	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0

Table 4a (continued). Notifications and notification rates of communicable diseases, Australia, 1997 to 2001, by state or territory (rate per 100,000 population)

Disease	Notifications					Rate per 100,000 population				
	1997	1998	1999	2000	2001	1997	1998	1999	2000	2001
Rubella‡‡	1,387	753	377	322	263	7.5	4.0	2.0	1.7	1.3
Tetanus	7	8	2	6	3	<0.1	<0.1	<0.1	<0.1	<0.1
Vectorborne diseases										
Arbovirus infection (NEC)	19	88	62	69	36	0.1	0.5	0.3	0.4	0.2
Barmah Forest virus infection	691	529	638	634	1,141	3.7	2.8	3.4	3.3	5.9
Dengue	174	579	132	215	176	0.9	3.1	0.7	1.1	0.9
Japanese encephalitis	-	-	-	-	0	-	-	-	-	0.0
Kunjin virus infection	-	-	-	-	4	-	-	-	-	<0.1
Malaria	749	660	732	951	712	4.0	3.5	3.9	5.0	3.7
Murray Valley encephalitis	-	-	-	-	6	-	-	-	-	<0.1
Ross River virus infection	6,596	3,151	4,416	4,200	3,219	35.6	16.8	23.3	21.9	16.5
Zoonoses										
Anthrax	-	-	-	-	0	-	-	-	-	0.0
Australian bat lyssavirus	-	-	-	-	0	-	-	-	-	0.0
Brucellosis	39	45	52	27	19	0.2	0.2	0.3	0.1	0.1
Leptospirosis	114	202	323	243	245	0.6	1.1	1.7	1.3	1.3
Ornithosis	35	64	84	100	131	0.4	0.7	0.9	1.1	0.7
Lyssavirus (unspecified)	-	-	-	-	0	-	-	-	-	0.0
Q fever	545	560	515	573	696	2.9	3.0	2.7	3.0	3.6
Other bacterial infections										
Legionellosis	157	262	249	472	307	0.8	1.4	1.3	2.5	1.6
Leprosy	12	3	6	4	5	0.1	<0.1	<0.1	<0.1	<0.1
Invasive meningococcal infection	494	480	591	621	677	2.7	2.6	3.1	3.2	3.5
Tuberculosis	989	960	1,143	1,024	989	5.3	5.1	6.0	5.3	5.1
Total	86,241	82,468	84,395	89,641	104,187					

* Analysis by date of onset, except for hepatitis B and hepatitis C unspecified, where analysis is by report date. Date of onset is a composite of three components: (i) the true onset date from a clinician, if available, (ii) the date the laboratory test was ordered, or (iii) the date reported to NNDSS.

† Unspecified hepatitis includes cases with hepatitis in whom the duration of illness cannot be determined.

‡ The analysis was performed by report date.

§ Includes incident hepatitis C in the Northern Territory and Queensland.

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

¶ Infections with Shiga-like toxin (verotoxin) producing *E. coli.* (SLTEC/VTEC).

** Northern Territory, Queensland, South Australia, Victoria and Western Australia: includes gonococcal neonatal ophthalmia.

†† Includes congenital syphilis.

‡‡ Includes congenital rubella.

NEC Not elsewhere classified.

\- Elsewhere classified.

Table 4b. Years from which diseases became notifiable to NNDSS in different jurisdictions in Australia*

Disease	Year in which data first sent to Commonwealth								Period of national reporting	Exceptions to national reporting
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Bloodborne diseases										
Hepatitis B (incident)	1995	1993	1993	1991	1993	1993	1993	1996	1995 to present	ACT did not report 1994; WA did not report 1994-1995
Hepatitis B (unspecified)	1991	1991	NN	1991	1991	1991	1991	1991	1991 to present	NT does not report
Hepatitis C (incident)	1995	1993	-	-	1993	1995	1997	1997	1993 to present	All jurisdictions except Qld and NT
Hepatitis C (unspecified)	1991	1991	1991	1991	1994	1991	1991	1993	1995 to present	Includes reports of incident hepatitis C, 1991 to 1994
Hepatitis D	1999	1999	1999	1999	1999	1999	1999	2001	1999 to present	WA did not report 1991-2000
Hepatitis (NEC)	1991	1991	1991	1991	1991	1991	1991	2001	1991 to present	WA did not report 1991-2000
Gastrointestinal diseases										
Botulism	1992	1998	1998	1998	1993	1992	1992	2001	1992 to present	State reporting started as shown
Campylobacteriosis	1991	NN	1991	1991	1991	1991	1991	1991	1991 to present	NSW does not report
Cryptosporidiosis	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	
Haemolytic uraemic syndrome	1999	1999	1999	1999	1999	1999	1999	1999	1999 to present	
Hepatitis A	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Hepatitis E	1999	1999	1999	1999	1999	1999	1999	2001	1999 to present	WA did not report 1991-2000
Listeriosis	1991	1991	1994	1991	1992	1991	1991	1991	1991 to present	SA did not report 1991 NT did not report 1991-1993
Salmonellosis (NEC)	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Shigellosis	1991	2001	1991	1991	1991	1991	1991	1991	1991 to present	NSW did not report 1991-2000
SLTEC, VTEC	1999	1999	1999	2001	1999	1999	1999	2001	1999 to present	Qld and WA did not report 1991-2000
Typhoid[1]	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Quarantinable diseases										
Cholera	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Plague	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Rabies	1993	1997	1991	1991	1991	1991	1991	1991	1991 to present	
Viral haemorrhagic fever	1993	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Yellow fever	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	

Table 4b (continued). Years from which diseases became notifiable to NNDSS in different jurisdictions in Australia*

Disease	Year in which data first sent to Commonwealth								Period of national reporting	Exceptions to national reporting
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Sexually transmissible diseases										
Chlamydial infection	1993	1991	1991	1991	1993	1991	1991	1994	1994 to present	NSW did not report 1994 - 1998
Donovanosis	1991	2002	1991	1991	2002	1993	1991	1991	1991 to present	NSW and SA did not report 1991-2001 Tasmania did not report 1991-1992
Gonococcal infection[2]	1991	1993	1991	1991	1991	1991	1991	1991	1991 to present	
Syphilis (includes congenital syphilis)	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Vaccine preventable diseases										
Diphtheria	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Haemophilus influenzae type b	1991	1991	1991	1991	1991	1991	1991	1994	1991 to present	WA did not report 1991-1993
Laboratory-confirmed influenza	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	
Measles	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Mumps	1992	1992	1995	1997	1994	1995	1992	1994	1995 to present	Qld did not report (1995-1996 & 1999-2000)
Pertussis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Invasive pneumococcal disease	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	
Poliomyelitis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Rubella (includes congenital rubella)	1991	1991	1993	1991	1993	1995	1992	1994	1993 to present	Tasmania did not report 1993-1994
Tetanus	1991	1991	1991	1994	1991	1991	1991	1991	1991 to present	Qld did not report 1991-1993
Vectorborne diseases										
Arbovirus infection (NEC)[3]	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	Includes JE, MVE and Kunjin 1991-2000
Barmah Forest virus infection	1995	1995	1997	1995	1995	1995	1995	1996	1995 to present	
Dengue	1993	1991	1991	1991	1991	1991	1991	1991	1991 to present	ACT did not report 1991-1992
Japanese encephalitis	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	
Kunjin virus infection	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	Reported under MVE in ACT
Malaria	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Murray Valley encephalitis	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	Combined with Kunjin in ACT
Ross River virus infection	1993	1993	1991	1991	1993	1993	1991	1993	1993 to present	

Table 4b (continued). Years from which diseases became notifiable to NNDSS in different jurisdictions in Australia*

Disease	Year in which data first sent to Commonwealth								Period of national reporting	Exceptions to national reporting
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Zoonoses										
Anthrax	2001	2001	2001	2001	2002	2001	2001	2001	2001 to present	
Australian bat lyssavirus	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	
Brucellosis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Leptospirosis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Ornithosis	1991	2001	1991	1992	1991	1991	1991	1991	1991 to present	NSW did not report 1991-2000 Qld did not report 1997-2001
Lyssaviruses (unspecified)	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	
Q fever	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Other bacterial infections										
Legionellosis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Leprosy	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Invasive meningococcal infection	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Tuberculosis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	

* Data from NNDSS annual reports from 1991. First full year of reporting to Commonwealth is shown. Some diseases may have been notifiable to State or Territory Health Departments before the dates shown here.

1. Includes paratyphoid in New South Wales, Queensland and Victoria.
2. Includes neonatal ophthalmia in the Northern Territory, Queensland, South Australia, and Victoria.
3. Before 1997, includes Ross River virus, dengue and Barmah Forest virus infection.

NN Not notifiable in 2001

Bloodborne diseases

In 2001, bloodborne viruses reported to the NNDSS included hepatitis B, C, D and hepatitis (NEC). Diagnoses of infections with human immuno-deficiency virus (HIV) and acquired immune deficiency syndrome (AIDS) 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.

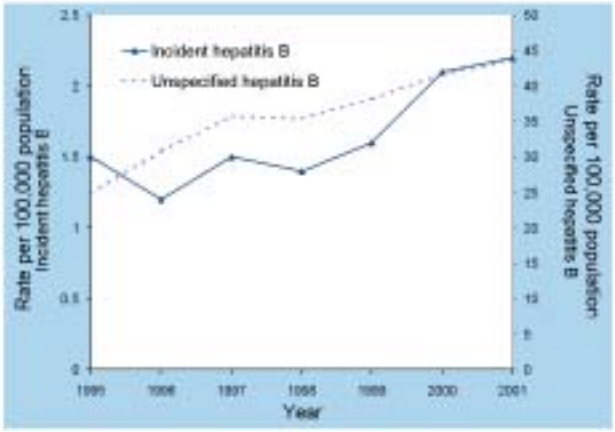
When reported to NNDSS, newly acquired hepatitis C and hepatitis B virus infections (referred to as 'incident') were differentiated from those where the timing of disease acquisition was unknown (referred to as 'unspecified'). As considerable time may have elapsed between onset and report date for chronic hepatitis infections, the analysis of unspecified hepatitis B and unspecified hepatitis C infections in the following sections is by report date, rather than by onset date.

Hepatitis B

Incident hepatitis B notifications

Since 1994, all states and territories, except the Australian Capital Territory, have reported incident cases of hepatitis B to the NNDSS. The Australian Capital Territory began reporting hepatitis B in 1995. The rate of incident hepatitis B notification between 1995 and 2000 ranges from around 1 to 2 cases per 100,000 population (Figure 5). In total, 424 incident cases were reported to the NNDSS with an onset date in 2001, giving a national notification rate of 2.2 cases per 100,000 population for the year. In 2001, the highest rates were reported from Tasmania (4.7 cases per 100,000 population) and Victoria (4.1 cases per 100,000 population).

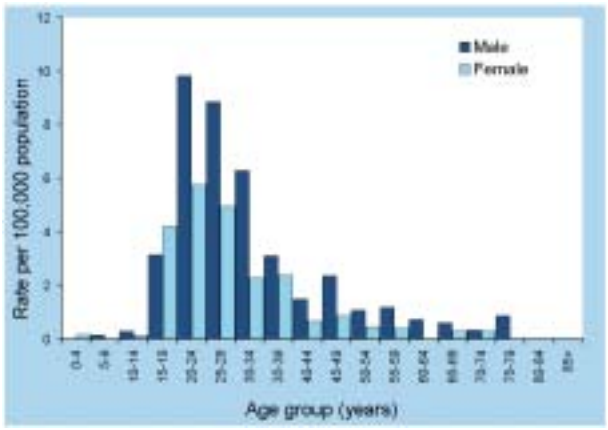
Figure 5. Trends in notification rates, incident and unspecified* hepatitis B virus infection, Australia, 1995 to 2001



* By report date.

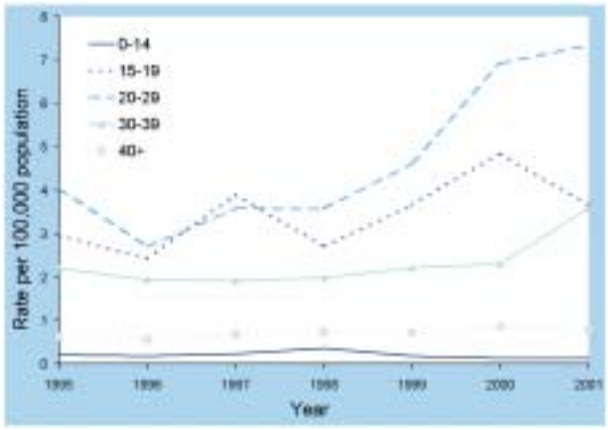
The highest rate of incident hepatitis B notifications were in the 20–24 year age group for both males and females (Figure 6). The highest notification rate for men was 9.8 cases per 100,000 population, while the highest notification rate for women was 5.8 cases per 100,000 population. Overall, there were more infections in males than in females, with a male to female ratio of 1.7:1.

Figure 6. Notification rates for incident hepatitis B virus infections, Australia, 2001, by age group and sex



Trends in the age distribution of incident hepatitis B virus infections are shown in Figure 7. Rates in children aged 0–14 years and adults over 40 years of age have remained relatively stable, while increases have been observed in the 20–39 year age range.

Figure 7. Trends in notification rates of incident hepatitis B virus infections, Australia, 1995 to 2001, by age group



Risk factor information for incident hepatitis B virus infection was available from all states and territories, except New South Wales and Queensland. The data are summarised in Table 5.

Table 5. Risk factors identified in notifications of incident hepatitis B virus infection, Australia, 2001, by reporting state or territory

Risk factor	ACT		NT		SA		Tas		Vic		WA	
	n	%	n	%	n	%	n	%	n	%	n	%
Injecting drug use*	1	50	0	–	5	21.7	15	68	94	48.0	13	33.3
Sexual contact with hepatitis B case	0	–	0	–	7	30.4	2	9	66	33.7	9	23.1
Household/other contact with hepatitis B	0	–	0	–	0	–	0	–	1	0.5	1	2.6
Overseas travel	0	–	0	–	1	4.3	0	–	0	–	2	5.1
Other	0	–	0	–	4	17.4	3	14	22	11.2		–
None identified	1	50	0	–	6	26.1	2	9	13	6.6	3	7.7
No information available	0		3	100	0	–	0	–	0	–	11	28.2
Total	2		3		23		22		196		39	

* Injecting drug users may have multiple risk factors for hepatitis B virus infection.

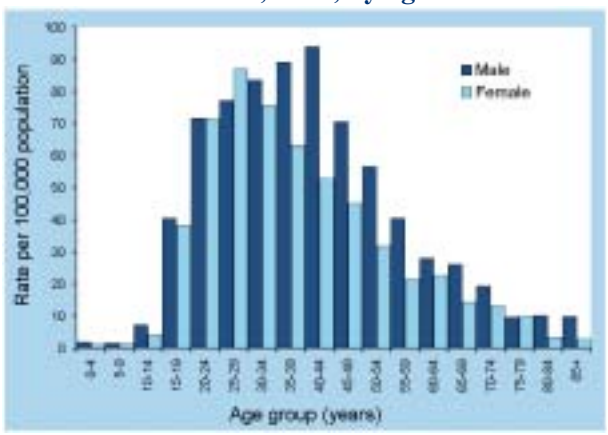
In response to an outbreak of incident hepatitis B observed in Victoria during the second quarter of the year, the Victorian Department of Human Services commenced enhanced surveillance to obtain detailed risk factor information directly from cases. A public alert was released through needle and syringe programs, and a strategy implemented to provide free hepatitis B vaccine to people known to be injecting drug users.

Unspecified hepatitis B notifications

Hepatitis B notifications have been reported to the NNDSS since 1991 by all jurisdictions except the Northern Territory, with unspecified cases separately notified from incident cases in most jurisdictions since 1994. The notification rate of unspecified hepatitis B cases ranged from 20 to 40 cases per 100,000 population between 1995 and 2001 (Figure 5). In 2001 there were 8,424 unspecified hepatitis B virus infection cases notified, at a rate of 43.7 cases per 100,000 population. The male to female ratio for unspecified hepatitis B cases was 1.3:1. By state and territory, the highest rates of notification were in New South Wales (71.3 cases per 100,000 population), Western Australia (34.1cases per 100,000 population) and Victoria (39.4 cases per 100,000 population). The highest rates were in the 40–44 year age group for men (93.9 cases per 100,000 population) and the 25–29 year age group for women (87.0 cases per 100,000 population, Figure 8).

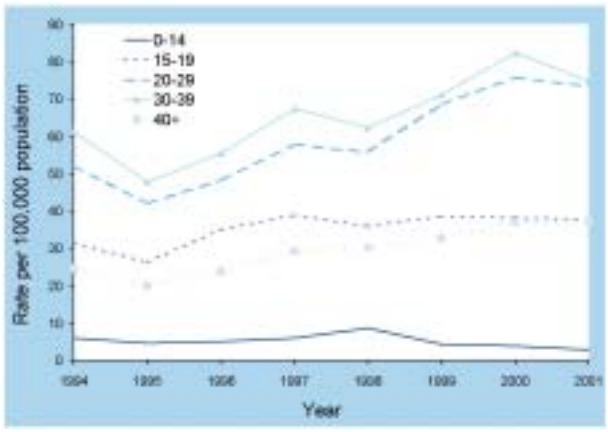
Trends in the age distribution of unspecified hepatitis B virus infections are shown in Figure 9. There have been moderate decreases in the number of reports of unspecified hepatitis B cases in the 0–14 year age range, while all other age groups have shown an upward trend in reporting rates over time.

Figure 8. Notification rates for unspecified hepatitis B virus infections, Australia, 2001, by age and sex*



* By report date.

Figure 9. Trends in notification rates of unspecified hepatitis B virus infections, Australia, 1994 to 2001, by age group



There were nine cases of unspecified hepatitis B virus infection in children in the 0–4 year age group reported from New South Wales, South Australia, Queensland and Western Australia. No unspecified hepatitis B cases were identified in children aged 0–4 years in the Australian Capital Territory, the Northern Territory, Tasmania or Victoria. Infant hepatitis B immunisation for Indigenous infants was introduced in the Northern Territory in 1988 and then expanded to all infants in this jurisdiction in 1990. Universal infant hepatitis B immunisation was introduced in the rest of Australia in May 2000. The effect of vaccination may take a number of years to become observable in childhood rates of the disease. Data on vaccination coverage, provided by the Australian Childhood Immunisation Register, indicates approximately 95 per cent of infants are currently receiving hepatitis B vaccination in Australia.

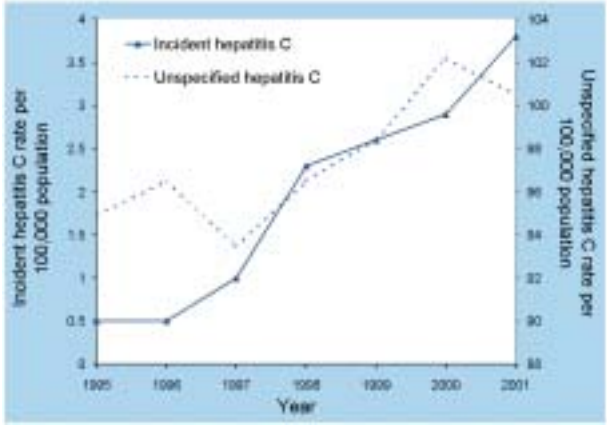
Hepatitis C

Unspecified hepatitis C notifications

Hepatitis C infection has been notifiable in all Australian states and territories since 1995. While the rate of unspecified hepatitis C notifications has ranged from 1.5 to 3 cases per 100,000 population in 1997 and 2000 respectively, (Figure 10), 2001 is the first year since 1997 where the number of notifications has decreased. Improved surveillance practice, such as better classification of incident cases and increased duplicate checking may account for some of the decrease in unspecified hepatitis C notifications. Whether the decrease represents a smaller pool of infected individuals previously undiagnosed will only become apparent in coming years.

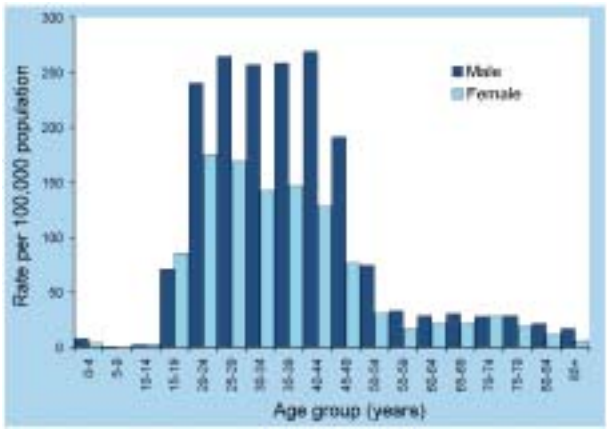
In 2001 there were 19,586 unspecified hepatitis C infections reported to NNDSS, a notification rate of 100.5 cases per 100,000 population. Of the total notifications of unspecified hepatitis C, 43 per cent of the notifications were from New South Wales. The highest notification rates were from the Northern Territory (106.5 cases per 100,000 population) and Victoria (103.1 cases per 100,000 population). The male to female ratio was 1.7:1. The highest reporting rate was in the 40–44 year age group for males (269.1 cases per 100,000 population), although there was little variation across the 25–44 year age range, from 240 to 269.1 cases per 100,000 population. The highest notification rate for females (175.1 cases per 100,000 population) was in the 20–24 year age group (Figure 11), while again there was little variation across the 20–44 year age range.

Figure 10. Trends in notification rates, incident and unspecified* hepatitis C infection, Australia, 1995 to 2001



* By report date.

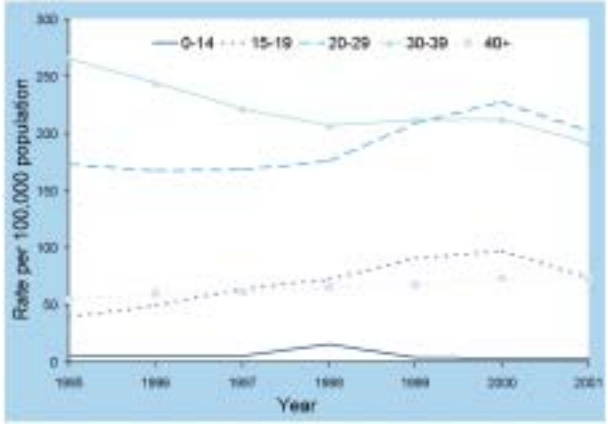
Figure 11. Notification rates for unspecified hepatitis C infections, Australia, 2001, by age group and sex*



* By report date.

Trends in the age distribution of unspecified hepatitis C infections are shown in Figure 12. Overall, the highest rates are in the 20–39 year age range. The most notable trends are the increase in notification rates in the 15–24 year age range and a decrease in the 30–39 year age group between 1998 and 2000. Between 2000 and 2001 there were decreases in all groups in the 15–39 year age range.

Figure 12. Trends in notification rates of unspecified hepatitis C infections, Australia, 1995 to 2001, by age group*



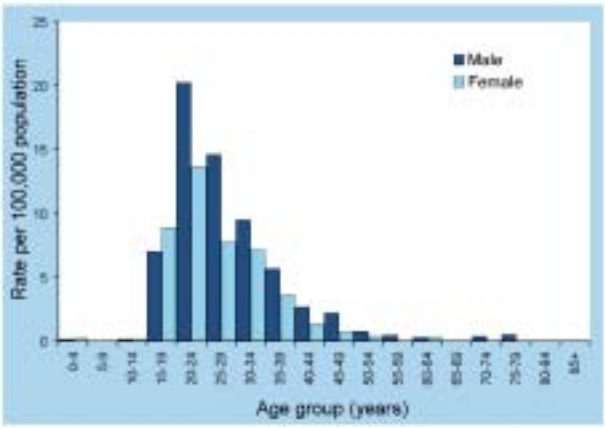
* By report date.

Incident hepatitis C notifications

Reporting of incident hepatitis C notifications from New South Wales and Western Australia commenced in 1993, from the Australian Capital Territory in 1994, from South Australia and Tasmania in 1995 and from Victoria in 1997. Incident hepatitis C cases are not differentiated from unspecified hepatitis C cases in Queensland or the Northern Territory. For the purposes of this report, only incident hepatitis C cases from 1997 onwards were analysed.

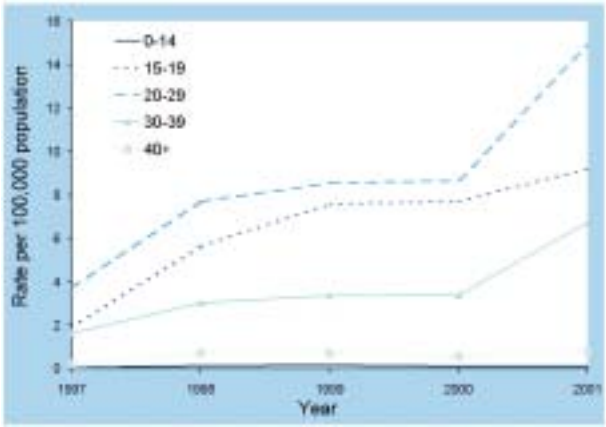
In total there were 600 incident cases of hepatitis C reported with an onset date in 2001, giving a rate of 3.8 cases per 100,000 population. The proportion of all hepatitis C infection notifications that were identified as incident cases was three per cent in 2001, which continues the upward trend of this proportion since 1997, when the proportion was 0.9 per cent. The highest rates of incident hepatitis C infection in 2001 were reported from Western Australia (8.3 cases per 100,000 population), the Australian Capital Territory (5.6 cases per 100,000 population and South Australia (5.3 cases per 100,000 population). The majority of incident hepatitis C notifications were in the 20–24 year age group for both males and females, with rates of 20.3 and 13.7 cases per 100,000 population, respectively (Figure 13). Overall, the male to female ratio was 1.5:1.

Figure 13. Notification rates for incident hepatitis C infections, Australia, 2001, by age group and sex



Trends in the age distribution of incident hepatitis C infections are shown in Figure 14. While rates in the 0–14 year and over 40 year age groups have remained stable, increases were observed in the 15–39 year age range, with steep increases in the 20–39 year age range between 2000 and 2001.

Figure 14. Trends in notification rates of incident hepatitis C infections, Australia, 1997 to 2001, by age group



Enhanced surveillance for incident hepatitis C infection notifications

In 1998 CDNA established the Hepatitis C Surveillance Committee. The committee was given the responsibility for improving the national capacity to monitor the occurrence of the infection and its consequences, by the development and implementation of a national hepatitis C surveillance strategy.[7] In 2001, its terms of reference were extended to include the development of national surveillance for hepatitis B virus infection, and the name was changed to the CDNA Viral Hepatitis Surveillance Committee.

In reviewing existing procedures in the course of developing the surveillance strategy, the committee identified the lack of standard case definitions across jurisdictions, and the absence of information on risk factors for hepatitis C as key weaknesses in national surveillance. The committee endorsed standard case definitions, and a set of categories that would be used to classify exposure for all cases determined to be incident. Despite competing priorities and resource limitations, in 2001 some states and territories were able to introduce enhanced surveillance for incident hepatitis C infections.

Surveillance of incident hepatitis C infection cases is difficult due to the asymptomatic nature of the disease and the need to collect paired sera to diagnose recent infection by seroconversion. Detection of incident cases prior to 2001 was on the basis of seroconversion or clinical illness. In recognition that cases of transmission from mother to child would not usually be detected by either seroconversion or clinical illness, in 2001 perinatal cases were included as incident infections. Enhanced surveillance, where all hepatitis C notifications are further investigated to ascertain the likely time of infection, is time and labour intensive, due to the large number of notifications. Trends in the number of incident cases are affected

by surveillance practice, and it is recognised that the number of hepatitis C notifications may vastly underestimate the true incidence of hepatitis C in Australia. The increase in incident hepatitis C notifications to the NNDSS should not necessarily be interpreted as evidence of increasing transmission in the Australian community. Instead the increase in the number of notifications may be a product of improved surveillance, increased awareness, and more widespread testing.

Incident hepatitis C cases have been separately reported by all jurisdictions except Queensland and the Northern Territory since 1997. In 2001, Western Australia, South Australia, Victoria, New South Wales and Tasmania undertook enhanced surveillance for incident cases. Enhanced surveillance has operated in South Australia and Tasmania for several years. Western Australia commenced enhanced surveillance for incident hepatitis C infections in 2001, incorporating new nationally agreed variables from the hepatitis C surveillance strategy. Data collection forms were sent to:

- doctors who notify cases as incident;
- doctors of patients identified by the major public laboratory as seroconverting within the past two years;
- a 30 per cent sample of doctors who notified unspecified hepatitis C infection cases.

All Public Health Units in New South Wales introduced enhanced surveillance in 2001. All hepatitis C notifications were followed up in a two phase process. Firstly, a form was sent to all doctors notifying a hepatitis C positive case, asking the doctor to indicate if the case was an incident infection. If a positive response was received, either the doctor or the case (with consent of the diagnosing doctor) were contacted, for collection of additional risk factor information. Efforts were taken to improve data quality by coordination and cleaning of the data at the New South Wales Health Department Central Office.

In Victoria, enhanced hepatitis C surveillance commenced in February 2001. In this populous state with a centralised reporting system, a 10 per cent random sample of all hepatitis C notifications were followed up with the diagnosing physician to determine if they were incident infections.

In 2001, additional data collected on incident hepatitis C infections were available from the Australian Capital Territory, South Australia, Tasmania and Victoria. The following analyses refer only to incident hepatitis C cases reported in these jurisdictions in 2001, thus the figures reported below may vary from the analysis by onset date. In total there were 209 cases: 18 cases from both the Australian Capital Territory and Tasmania, 86 cases from South Australia and 87 cases from Victoria. Most incident hepatitis C infections (165 of 209, 79%) were diagnosed by seroconversion alone (Table 6). Some cases were diagnosed both clinically and by seroconversion. One perinatal case was identified in South Australia.

The majority (176/209, 84%) of cases of incident hepatitis C were associated with injecting drug use (IDU) (Table 7). Further analysis of exposure in people who did not report injecting drug use are shown in Table 8. Multiple exposures were recorded. Cases not reported to be associated with injecting drug use include transmission via blood transfusion (n=1), needle stick injuries in a healthcare worker (n=2), surgery (n=1), perinatal transmission (n=1), tattoos (n=1), ear or body piercing (n=1), imprisonment (n=5), and sexual partner with hepatitis C infection (n=4). One case with an exposure identified as 'other' was a victim of domestic violence by a partner with hepatitis C. In total, an exposure could not be identified for 16 non-IDU cases.

There may be selection bias in the analysis of exposure for incident cases, as injecting drug users are more likely to undergo regular testing, due to the recognised risk in this group. The most likely route of infection is difficult to determine when multiple possible exposures are recorded.

Projections of hepatitis C in Australia

It is recognised that notifications of hepatitis C infection do not provide an accurate estimate of the number of people in Australia living with hepatitis C infection. To plan an appropriate public health response to the epidemic, accurate estimates of incidence and prevalence, and projections of the long-term sequelae of infection, are required.

In 2001 the Hepatitis C Virus Projections Working Group undertook mathematical modelling of the epidemiology and natural history of hepatitis C infection in Australia, in order to estimate hepatitis C infection incidence and prevalence rates in Australia up to the end of 2001. Future trends in the long-term sequelae of hepatitis C infection were also modelled.[8]

It was estimated that in Australia in 2001 there would be 16,000 incident cases of hepatitis C infection, and that 210,000 (range 157,000–252,000) people will have antibodies to the virus. It was estimated that in 2001, 6,500 people were living with hepatitis C related cirrhosis, that 175 people developed hepatitis C associated liver failure, and that 50 people developed hepatitis C related hepatocellular carcinoma. Finally, it was estimated that 22,500 quality adjusted life years will have been lost in Australia in 2001 due to chronic hepatitis C infection, the majority (77%) in people with early (stage 0/1) liver disease. These models suggest that by 2020 the prevalence of hepatitis C related cirrhosis and the incidence of hepatitis C related liver failure and hepatocellular carcinoma will more than triple in Australia.

Table 6. Method of diagnosis, incident hepatitis C cases, the Australian Capital Territory, South Australia, Tasmania and Victoria, 2001

Method of diagnosis	State or territory				Total
	ACT	SA	Tas	Vic	
Seroconversion	18	71	14	62	165
Clinical	0	6	4	14	24
Seroconversion and clinical	0	8	0	11	19
Perinatal	0	1	0	0	1
Total	18	86	18	87	209

Table 7. Assessment of injecting drug use, incident hepatitis C cases, Australian Capital Territory, South Australia, Tasmania and Victoria, 2001

Injecting drug use	State or territory			
	ACT	SA	Tas	Vic
Only in previous 2 years	0	79	0	15
More than 2 years ago	0	0	0	50
IDU, but time not specified	14	0	18	0
No history of IDU	0	7	0	8
IDU status unknown	4	0	0	14
Total	18	86	18	87

IDU Injecting drug use

Table 8. Exposure assessment, incident hepatitis C cases, Australian Capital Territory, South Australia, Tasmania and Victoria, 2001

Risk factor	State or territory						
	ACT		SA		Tas	Vic	
	All cases (n=18)	Non-IDU* (n=4)	All cases (n=86)	Non-IDU* (n=7)	All cases (n=18)	All cases (n=87)	Non-IDU* (n=22)
Injecting drug use (IDU)	14	na	79	na	18	65	na
Household contact with hepatitis C	1	0	0	0	0	0	0
Received blood product in Australia	0	0	1	1	0	0	0
Needlestick injury, healthcare worker	0	0	2	2	0	0	0
Surgical work	0	0	0	0	0	4	1
Perinatal	0	0	1	1	0	0	0
Tattoos	0	0	0	0	4	6	1
Ear/body piercing	0	0	0	0	2	4	1
Sexual partner with hepatitis C	1	0	0	0	4	14	4
Imprisonment	0	0	0	0	3	16	5
Household contact with hepatitis C	0	0	0	0	1	0	0
Other risk identified	0	0	1	1	0	0	0
Non-IDU risk identified, but not in past 2 years	0	0	0	0	0	1	0
Unable to determine risk	4	4	2	2	0	10	10

Note: Some people may have more than one exposure

Hepatitis D

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

In Australia in 2001, there were 21 notifications of hepatitis D to the NNDSS, a notification rate of 0.1 cases per 100,000 population. Of the 21 notifications, 12 were reported from New South Wales, seven from Victoria, and two from Queensland. The majority (16/21, 76%) of cases were for males, with the highest rate reported in the 35–39 year age group (0.5 cases per 100,000 population).

Gastrointestinal diseases

Gastrointestinal diseases are a major cause of illness in Australia. Recently, incidence of gastroenteritis in Australia has been estimated at approximately one episode per person per year.[9] If 35 per cent of gastroenteritis is due to contaminated food, then there may be significantly more than the previously estimated four million annual cases of foodborne disease in Australia each year.[10] Since the majority of gastroenteritis is mild and self-limiting, only a small proportion of cases present to medical practitioners, an even smaller number are investigated, and fewer yet are notified to health departments for transmission to the NNDSS.

In 2001, notifications of gastroenteritis increased to 26,086, which was 25 per cent of all notifications to NNDSS. This represents a 22 per cent increase from notifications in 2000. The overall increase in notifications was due to the changes in gastrointestinal diseases that were notifiable in Australia in 2001. Diseases notified are botulism, campylobacteriosis, cryptosporidiosis, haemolytic uraemic syndrome (HUS), hepatitis A and E, listeriosis, salmonellosis, shigellosis, shiga-like toxin producing *E. Coli*/verotoxigenic *E. coli* (SLTEC/VTEC) and typhoid.

Cryptosporidiosis was made a notifiable disease from 2001. Although the reporting of cryptosporidiosis was incomplete in 2001, the relatively large number of cases notified, accounts for some of the increase in total notifications of gastrointestinal disease in 2001. Other reasons for the increase in notifications include an 18 per cent increase in campylobacteriosis. In 2001, New South Wales reported shigellosis for the first time and Western Australia began reporting botulism, hepatitis E and SLTEC/VTEC.

Yersiniosis was removed from the list of gastrointestinal diseases notifiable in 2001. Notifications of this disease had declined from 370 cases in 1993 to 73 cases in 2000 (a decline from 3.2 to 0.6 cases per 100,000 population). This disease is rare in Australia and the USA, but common in Europe, where it is frequently associated with the consumption of undercooked pork.[11]

In 2001, OzFoodNet a network of foodborne disease epidemiologists began work to enhance the surveillance of foodborne disease in Australia. The annual report of OzFoodNet activities in 2001[6] contains additional information on gastrointestinal disease, which complements data in this report.

Botulism

Botulism is a notifiable disease in all Australian states and territories. No cases of classic foodborne botulism have been reported since notification commenced. Infant (or intestinal) botulism cases arise from ingestion of *Clostridium botulinum* spores, which germinate in the intestine. Sources of spores are multiple, and include dust and foods such as honey.[12] There have been five cases of infant intestinal botulism reported since 1996, including two cases reported in 2001.

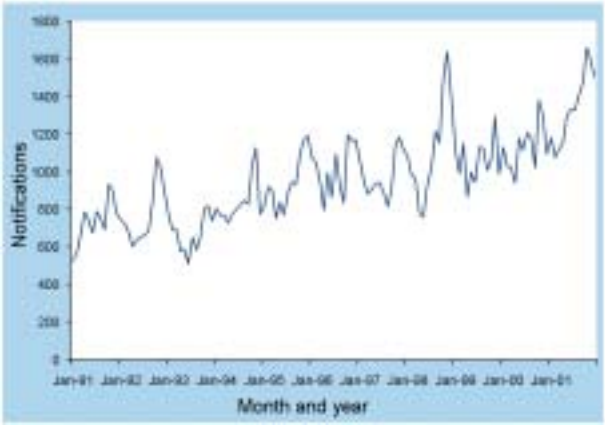
Of these two cases, one was from Victoria and the other from Queensland, and both occurred in infants aged less than one year. The first case was a five-month-old infant hospitalised after a three-day history of poor feeding, constipation, ptosis, difficulty in swallowing, weakness and loss of head control. Although there were various potential environmental exposures, including dust, no source for the child's infection could be determined.

The second case was a 10-week-old infant who presented with acute flaccid paralysis (prominent bulbar weakness). Subsequently, *Clostridium botulinum* type B was isolated from the faeces. The infant had a history of probable consumption of honey within the two weeks prior to onset of the disease. Parents are advised not to feed honey to infants or to dip pacifiers in honey, because of the risk of botulism.[13]

Campylobacteriosis

There were 16,124 notifications of campylobacteriosis in Australia in 2001. This represents an increase of 18 per cent on the 13,595 cases reported in 2000 and continues a trend of increasing notifications of campylobacteriosis in Australia (Figure 15). The national rate of campylobacteriosis reported to NNDSS (125.2 cases per 100,000 population) makes this disease the most commonly reported disease in Australia and it exceeds that of *Salmonella* more than threefold. Data from the United Kingdom suggest that this disease may be under-reported by a factor of eight times.[14] *Campylobacter jejuni* is now the most common bacterial cause of foodborne disease in industrialised countries.[15]

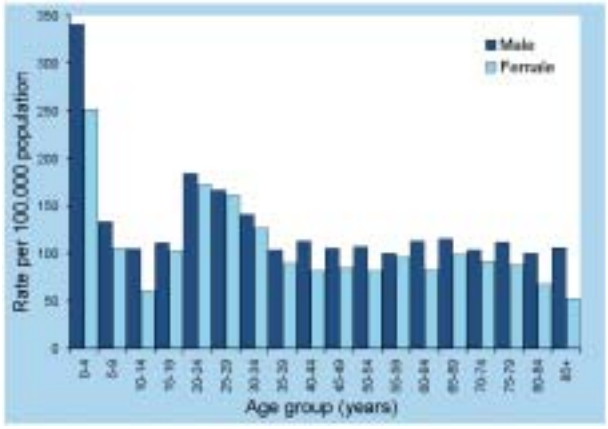
Figure 15. Trends in notifications of campylobacteriosis, Australia, 1991 to 2001, by month of onset



Reports were received from all states and territories except New South Wales, where cases are included in the categories 'foodborne disease' or 'gastroenteritis in an institution.' The highest rates of campylobacteriosis were in South Australia (175.7 cases per 100,000 population) and the lowest in Queensland (109.2 cases per 100,000 population). Nationally, notifications were most common in October (1,688 reports). Despite the high rates of disease, only six outbreaks were identified during 2001. Three small outbreaks were associated with take-away kebabs and two were associated with the consumption of chicken.[6]

The highest age specific rate of campylobacteriosis was 296 cases per 100,000 population in children aged 0–4 years. Rates according to age group and sex are shown in Figure 16. In the 0–4 year age group the rates were higher in males (341 cases per 100,000 population) than in females (251 cases per 100,000 population). The male to female ratio in this age group was 1.4:1, while overall it was 1.2:1.

Figure 16. Notification rates of campylobacteriosis, Australia, 2001, by age group and sex



Cryptosporidiosis

Cryptosporidiosis is spread by faecal contamination and includes person-to-person, animal-to-person, waterborne and foodborne transmission. The prevalence of infection is between 1 to 4.5 per cent of individuals in developed countries and between 3 to 20 per cent of individuals in developing countries.[16] Children under two years of age, animal handlers, travellers and men who have sex with men are recognised to be at greater risk of infection.

Infections with *Cryptosporidium* are commonly asymptomatic and carriers can shed oocysts in their faeces and be a source of infection to others.[16] The infective dose is very small (approximately a hundred oocysts) and previous exposure in immunocompetent adults is not entirely protective, although it may decrease the severity of the disease caused by subsequent infections. People with markedly impaired immune systems due to HIV infection are susceptible to severe persistent diarrhoea caused by cryptosporidiosis and the infection may spread to the biliary tract. Declines in the prevalence of cryptosporidiosis in HIV and AIDS patients treated with highly active anti-retroviral therapy have been reported.[17]

Notification of cryptosporidiosis to NNDSS was agreed by all Australian states and territories from January 2001. Since addition of new diseases to the notifiable list requires legislative change in each Australian jurisdiction, reports of cryptosporidiosis received by NNDSS in 2001 probably underestimate the national annual total.

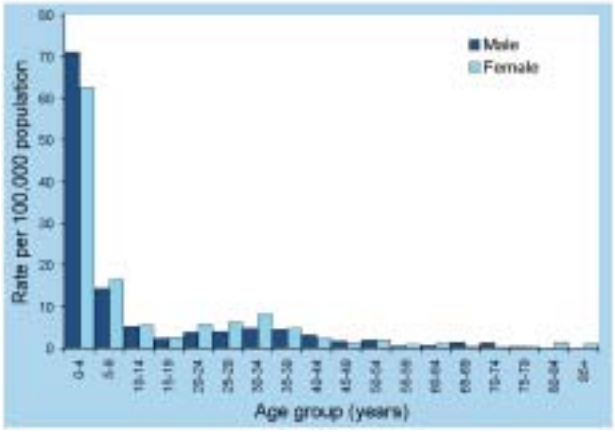
In the autumn quarter of 2001 (April-June), sporadic cryptosporidiosis infections associated with use of swimming pools were reported from several states and territories in Australia. Compared to previous years, Victoria observed increased notifications of cryptosporidiosis, predominantly from the Melbourne

metropolitan area. The majority of cases reported exposure to public swimming pools before becoming ill and small clusters were associated with several pools. Swimming pools are common sources for outbreaks of cryptosporidiosis. In summer 2001 in the United States of America, five protracted outbreaks of cryptosporidiosis associated with swimming pool use were reported.[18] Such outbreaks can be prevented by rigorous control of water pool quality, provision of advice to people that they should not swim if they have gastroenteritis, and enforcement of a faecal accident policy. The Queensland government has published guidelines to prevent outbreaks of cryptosporidiosis in swimming pools (www.health.qld.au/phs/Documents/cdu/5436.pdf).

In Queensland, five linked cases of cryptosporidiosis were reported, which were associated with consumption of unpasteurised milk intended for animal consumption. Of the five cases, three were hospitalised.[19] Cryptosporidiosis infection associated with consumption of unpasteurised products are possibly due to contamination with cow manure.[16] A cluster of 45 *Cryptosporidium* infections occurred in northern Tasmania in November 2001 and an animal nursery at an agricultural show was suspected to be the source. The majority of cases were children who had attended the show, with secondary cases arising in families through person-to-person transmission. (Ashbolt, *Commun Dis Intell* submitted)

The notification rates for cryptosporidiosis by age group and sex are shown in Figure 17. More than half the cases were in children under the age of five years (869 cases, 53% of total). There was no difference in the notification rates between males and females.

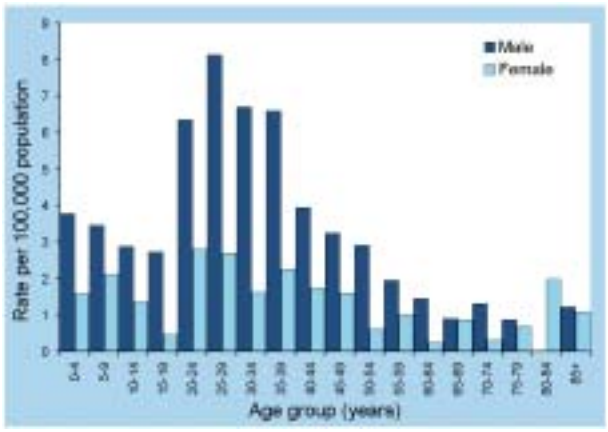
Figure 17. Notification rates of cryptosporidiosis, Australia, 2001, by age group and sex



Hepatitis A

There were 530 notifications of hepatitis A in Australia in 2001, the lowest since NNDSS began in 1991 and a 35 per cent decline from the 812 cases reported in 2000. The majority of cases occurred in the larger states: New South Wales (n=195), Queensland (n=120) and Victoria (n=102). The rates in these states were similar (2.1–3.3 cases per 100,000 population). The Northern Territory had the highest notification rate for hepatitis A (19.0 cases per 100,000 population, n=38). The highest age-specific rates were in males in the 25–29 year age group and females in the 20–24 year age group (8.1 and 2.8 cases per 100,000 population, respectively) and the overall male to female ratio was 2.6:1 (Figure 18).

Figure 18. Notification rates of hepatitis A, Australia, 2001, by age group and sex



Marked declines in the annual number of notifications of hepatitis A have been seen in north Queensland since hepatitis A vaccination was introduced for Indigenous children in the region in early 1999. There were 231 notifications of hepatitis A in Far North Queensland in 1999, 34 cases in 2000, and 11 cases in the first nine months of 2001. The last case in an Indigenous person was in June 2000. The majority of cases in Far North Queensland during 2000 and 2001 were acquired abroad, particularly in Papua New Guinea (PNG) (Jeffrey Hanna, Tropical Public Health Unit Network, personal communication, November 2001).

Apart from rare large outbreaks associated with food, such as the outbreak associated with oysters in 1997,[20] hepatitis A in Australia is most commonly acquired through household or close contact with a case, recreational drug use and overseas travel. Risk exposure information was available for 247 of the 530 cases (47%) in 2001 (Table 9).

Table 9. Risk exposures associated with infection with hepatitis A virus infection, Australia, 2001 by reporting state or territory

	State or territory							
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA
Injecting drug use	3	–	–	12	2	–	23	1
Household /close contact of case	1	–	9	17	3	–	6	2
Overseas travel	2	36	1	20	6	1	26	16
Childcare	–	–	–	32	0	–	0	–
Homosexual contact	–	–	–	6	1	–	6	–
Sex worker	–	–	–	0	–	–	0	–
Other	–	–	–	33*	–	2†	–	–
Total with risk factors identified	6	36	20	120	12	3	61	19
Unknown	8	159	28	0	8	1	41	18
Total	14	195	38	120	20	4	102	37

* Includes exposure to shellfish (n=17) and Indigenous person or contact with Indigenous community (n=13)

† The two cases notified from Tasmania became infected in Queensland.

A national cross-sectional hepatitis A seroprevalence survey of opportunistically obtained serum was performed in 1998 and reported in 2001.[21] This study found 41 per cent of the samples were positive for antibodies to hepatitis A, and the proportion of positive samples increased with age. When combined with declining notifications of hepatitis A, these data support the idea of a declining incidence, with fewer young people being exposed to the virus.

Hepatitis E

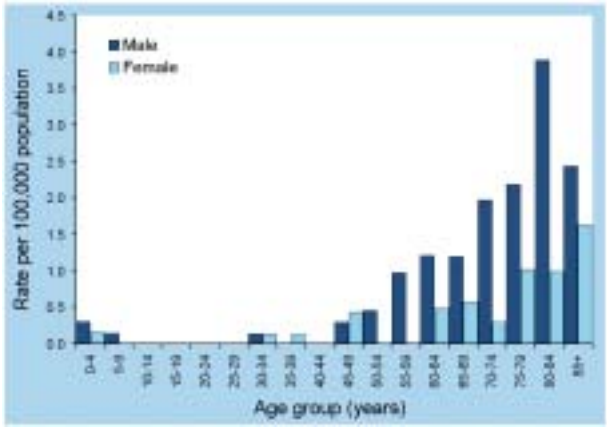
There were 10 cases of hepatitis E reported to NNDSS in 2001, the same number as in 2000. The cases occurred in New South Wales (n=6), Victoria (n=3) and Queensland (n=1). There were six female and four male cases and three of the women were of child-bearing age (15–49 years). All three of the cases reported in Victoria had travelled overseas.

Listeriosis

Listeriosis is a serious bacterial disease caused by the consumption of food contaminated with *Listeria monocytogenes*. Changes in food processing and distribution and a growing population with predisposing risk factors for infection with *Listeria monocytogenes* has raised concerns about this pathogen.[22]

In 2001, 62 cases of listeriosis were notified to NNDSS. This was lower than the 67 cases notified in 2000. The national rate was 0.3 cases per 100,000 population. Rates of 0.6 cases per 100,000 population were reported in Queensland (n=20) and Western Australia (n=11). There were no clusters or outbreaks reported. There was a predominance of male cases, with a male to female ratio of 2.2:1. Rates according to age group and sex are shown in Figure 19. OzFoodNet reported that 6 out of 61 cases were maternal foetal infections, which resulted in three foetal deaths.[6] The majority of listeriosis notifications occurred in the elderly, with 40 cases (64% of total) occurring in people aged more than 60 years. OzFoodNet reported a mortality rate of 13 per cent among non-pregnancy-related cases.[6]

Figure 19. Notification rates of listeriosis, Australia, 2001, by age group and sex



A recent review of the epidemiology of listeriosis in Australia found a stable and low rate of listeriosis, which did not vary from jurisdiction to jurisdiction.[23] There were inconsistencies identified in how a maternal-foetal pair was reported, either as a single case or mother and child reported separately.

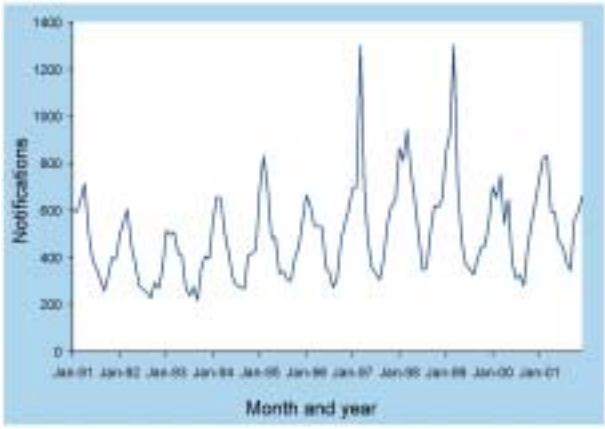
Australia's Imported Food Program undertakes surveillance of imported food, and is a joint activity of Food Standards Australia New Zealand and the Australian Quarantine Inspection Service. All 'ready-to-eat' imported foods, such as soft cheese and smoked fish, must be free of *Listeria*. Data from the Imported Food Program from 1995 to 1998 show an increasing percentage of imported food items (up to 8%) was contaminated with *Listeria*.[24] Surveillance for *Listeria* contamination of imported food is therefore a vital measure for control of listeriosis in Australia.

Salmonellosis (excluding typhoid)

Salmonellosis is the second most commonly notified gastrointestinal disease in Australia and is primarily associated with food.[25] In 2001, there were 7,045 cases reported, an increase of 14.5 per cent on the 6,151 cases reported in 2000. While there has been a variable trend over the last 10 years, improvements in the investigation of foodborne disease by states and territories may have contributed to recent increased notifications.

Cases of salmonellosis were reported from each Australian state and territory in 2001, and the national rate was 36.2 cases per 100,000 population. The highest rate was in the Northern Territory (186.5 cases per 100,000 population). Rates of salmonellosis varied by Statistical Division (Map 2), with the Kimberley district of northern Western Australia having the highest rate (602 cases per 100,000 population). In general, there were higher rates of salmonellosis in more northerly areas of the country. Reports of salmonellosis were highest in summer months (January–March, Figure 20). As in previous years, the highest age-specific rate was in children aged less than five years (196 cases per 100,000 population) and the male to female ratio was 1.1:1. Rates according to age group and sex are shown in Figure 21.

Figure 20. Trends in notifications of salmonellosis, Australia, 1991 to 2001, by month of onset



Map 2. Notification rates of salmonellosis, Australia, 2001, by Statistical Division of residence

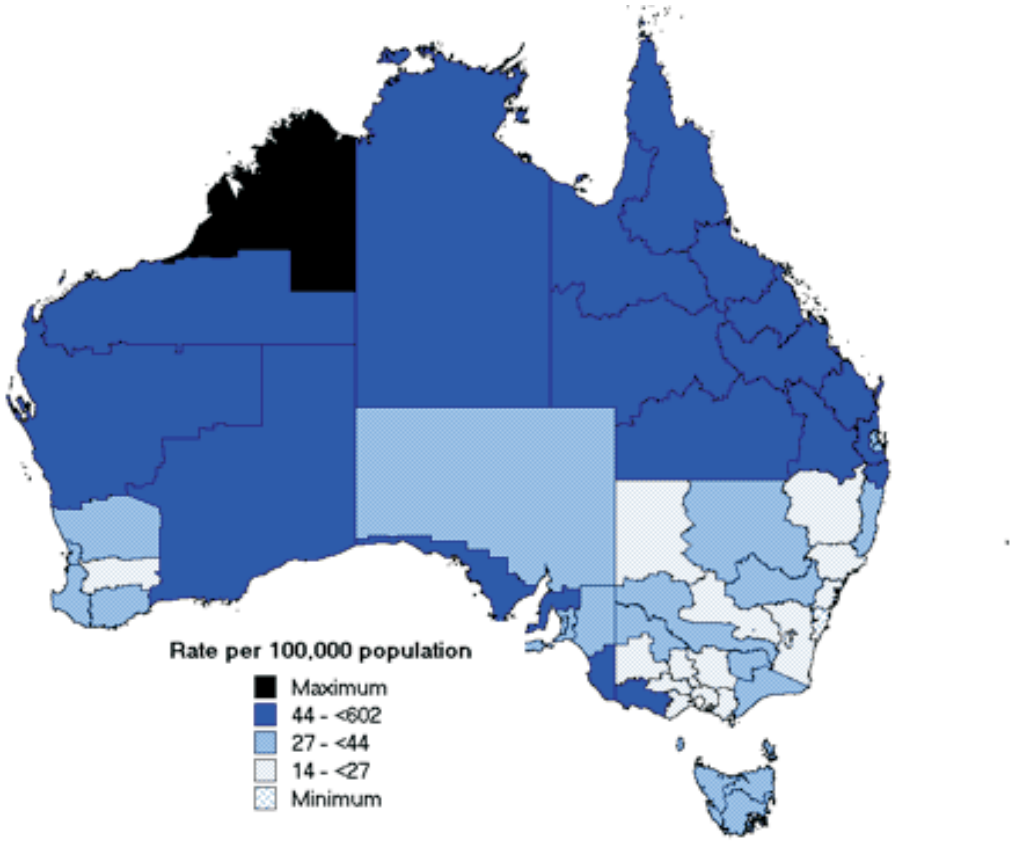
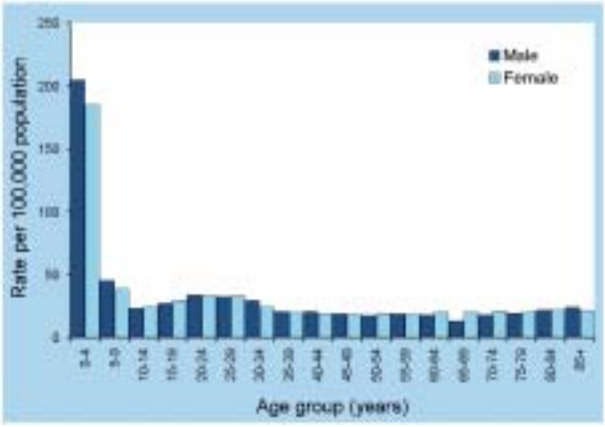


Figure 21. Notification rates of salmonellosis, Australia, 2001, by age group and sex



The National Enteric Pathogens Surveillance Scheme reported 6,932 cases of *Salmonella* infection in 2001.[26] The 10 most frequently isolated serovars and phage types of *Salmonella* which account for 45.2 per cent of all isolates, are shown in Table 10.

Outbreaks of *Salmonella*

Salmonella Typhimurium Definitive Type 104

During 2001 the Victorian Department of Human Services investigated an outbreak of *Salmonella* Typhimurium Definitive Type 104 (STM DT104), which was found to be associated with helva, a sweet made from sesame seeds, sugar and flavourings, imported from Turkey. The investigation in Victoria was in conjunction with Sweden, Norway and other European countries, where salmonellosis cases associated with helva were also identified.[27] Twenty of the 23 (87%) of Australian cases occurred in Victoria, and two cases occurred in New South Wales and one in Queensland.

S. Typhimurium DT104 emerged worldwide during the 1990s and now constitutes 8 to 9 per cent of isolates in the USA. DT104 constituted only 0.4 per cent of isolates in Australia in 2001, almost all of which were cases from the outbreak reported above. The DT 104 strain carries resistance to multiple antibiotics (ampicillin, chloramphenicol, trimethoprim-sulphamethazol, streptomycin and tetracycline). Isolates of DT104 with decreased susceptibility to fluoroquinolones have been isolated in the United Kingdom and the emergence of this additional resistance is linked to veterinary use of these antibiotics.[28]

Salmonella Stanley

An outbreak of 24 cases of *Salmonella* Stanley infection, associated with the consumption of contaminated dried peanuts imported from China, affected several Australian states and territories in 2001. Two people with *Salmonella* Newport infections also reported eating the same brand of peanuts. Three *Salmonella* serovars: Stanley, Newport and Lexington were isolated from the peanuts. These findings triggered an international product recall and assisted health agencies in Canada and the United Kingdom who were investigating similar outbreaks.[29]

Table 10. Top 10 isolates of *Salmonella*, Australia, 2001

Organism	State or territory									
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust	Total %
S. Typhimurium PT135	5	257	9	140	25	4	91	104	635	9.2
S. Virchow	5	65	1	289	21	0	95	2	478	6.9
S. Typhimurium PT9	10	139	0	52	48	11	122	17	399	5.8
S. Typhimurium PT126	4	94	9	73	111	2	18	2	313	4.5
S. Enteritidis	6	79	3	62	20	8	50	66	294	4.2
S. Saintpaul	1	33	20	175	5	2	8	43	287	4.1
S. Birkenhead	2	109	0	132	2	0	6	2	253	3.6
S. Bovismorbificans	5	54	2	54	13	2	30	7	167	2.4
S. Chester	1	30	15	64	13	0	11	31	165	2.4
S. Typhimurium PT64	1	61	3	5	31	1	11	35	148	2.1
Others	44	768	309	1,076	324	131	631	510	3,793	54.8
Total	84	1,689	371	2,122	613	161	1,073	819	6,932	

Source: National Enteric Pathogens Surveillance Scheme, annual report, 2001.

Salmonella Typhimurium phage type 126

A community-wide outbreak of *Salmonella* Typhimurium phage type 126 (STM 126) involving 88 cases occurred in South Australia. The outbreak lasted for several months, with cases emerging in other states and territories later in the epidemic. A case-control study demonstrated that illness was associated with consumption of chicken. Descriptive epidemiology and microbiological evidence of pathogens from samples of raw chicken provided corroborating evidence for this link. The South Australian Department of Human Services observed a decrease in human cases of STM 126 following interventions at breeder farms, hatcheries and processing plants.

Salmonella Bovismorbificans

In June 2001, Queensland investigated a state-wide increase in *Salmonella* Bovismorbificans phage type 32. The outbreak was suspected to be linked to a food product purchased from a fast food restaurant. A case control study implicated a product containing iceberg lettuce, and environmental investigations identified a mechanical slicer at the processing facility that was positive for *Salmonella* Bovismorbificans phage type 32. Thirty-six cases occurred, six of whom were hospitalised.[30]

Salmonella Mgulani

The Northern Territory reported 15 cases of *S.* Mgulani in October and November 2001. Previously, this serovar had rarely been identified in the Territory. Cases were widely dispersed and occurred mostly in non-Indigenous people. Although interviews were conducted, no food source was identified.[31] A cluster of *S. Mgulani* in New South Wales in December 1999 and January 2000 involved 542 cases.[32]

Shigellosis

It is estimated that the majority of shigellosis is due to person-to-person transmission and that only 20 per cent may be foodborne.[25] In Australia, the majority of *Shigella* infections are seen in men who have sex with men, Indigenous communities and travellers returning from overseas. Foodborne outbreaks are rare in Australia, largely as a result of improved standards of sanitation and food-handling.[33] The last outbreak of foodborne shigellosis was in 1998,[34] although outbreaks via person-to-person contact have been reported.[35,36] OzFoodNet reported no outbreaks or confirmed links with food among shigellosis cases notified in 2001.[6]

Shigellosis became a notifiable condition in New South Wales for the first time in 2001. This accounts for the increase in the number of cases (562 cases compared with 487 cases in 2000). Despite this, the national notification rate (2.9 cases per 100,000 population) continued to decline (Figure 22). The highest notification rate was in the Northern Territory, with 51.5 cases per 100,000 population. By age, the highest rates were in children aged less than five years (11 cases per 100,000 population). Overall there was a slight predominance of males (male:female ratio 1.3:1). Rates according to age group and sex are shown in Figure 23.

Figure 22. Trends in notifications of shigellosis, Australia, 1991 to 2001, by month of onset

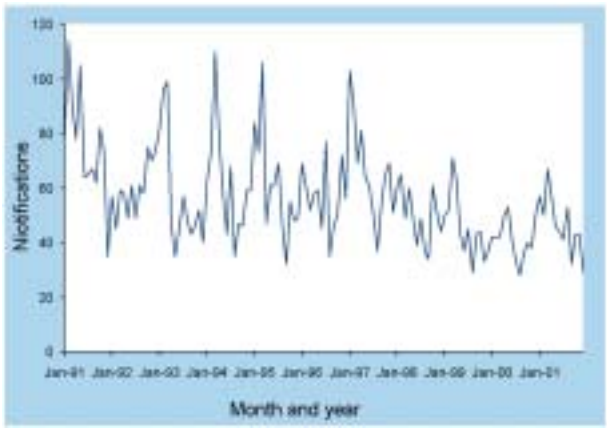
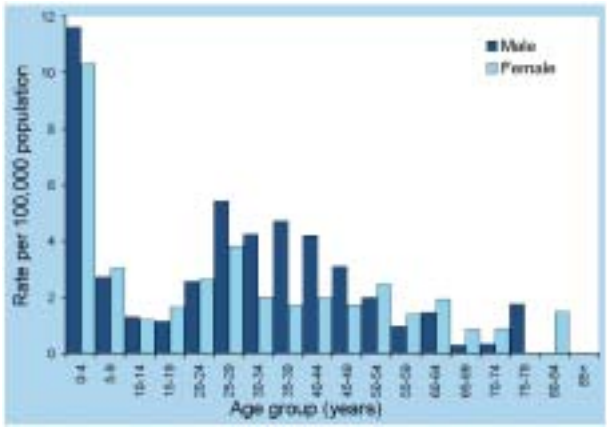


Figure 23. Notification rates for shigellosis, Australia, 2001, by age group and



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

There were 49 cases of SLTEC/VTEC notified to NNDSS in 2001. This was an increase of 48 per cent on the 33 cases reported in 2000. Reports of SLTEC/VTEC infections were received from Queensland and Western Australia for the first time in 2001. The notification rate rose slightly to 0.3 cases per 100,000 population.

As in previous years, more than 50 per cent (27/49) of cases were notified in South Australia, reflecting a policy of screening all bloody stools for toxin genes by polymerase chain reaction. OzFoodNet reported that *E. coli* O157 was identified in 3 of 26 cases in South Australia, 2 of 4 cases in Victoria and 4 of 10 cases in Queensland, although typing methods are difficult to compare between jurisdictions.[6]

Haemolytic uraemic syndrome

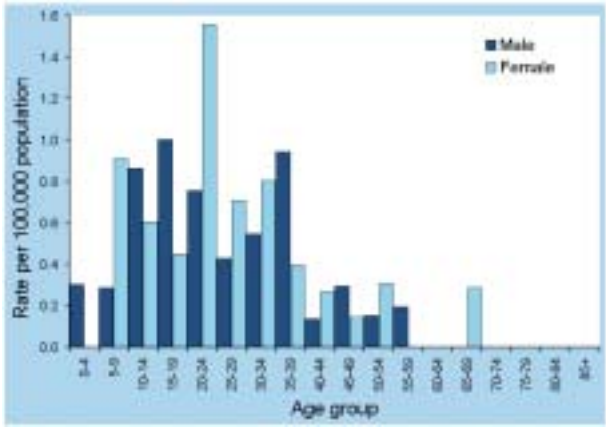
Infections with SLTEC/VTEC have the potential to cause severe and life-threatening illness, including haemolytic uraemic syndrome. Young children are more at risk and HUS in children is typically post-diarrhoeal. *E. coli* O157:H7 infection in children aged less than five years progress to HUS in 10 to 14 per cent of cases.[37]

There were only three cases of HUS notified to the NNDSS in 2001, which was markedly lower than the 15 cases reported in 2000. There is evidence that notified cases of HUS represent only a small proportion of all cases. In California only 44 per cent of cases were reported to public health authorities.[37] The Australia Paediatric Surveillance Unit recorded 325 reports of HUS from paediatricians between July 1994 and December 2000, in children aged less than 15 years. Of these 137 were confirmed and 97 were associated with diarrhoea.[38] In the same period, only 83 cases were notified to the NNDSS. A survey of Australian hospitalisation data conducted by OzFoodNet has shown 90 separations for HUS in the 1998–99 financial year and 47 in the 1999–00 financial year. By contrast, for the same periods there were 21 and 16 notifications of HUS to NNDSS, respectively.[6] Ongoing studies are needed to address the differences seen between the datasets and notification mechanisms in the states and territories.

Typhoid

Typhoid infections in Australia are usually associated with overseas travel. In 2001, there were 84 notifications of typhoid to the NNDSS. This represents a 43 per cent increase on the 60 cases reported in 2000. Of the 84 cases, the highest notification rates were in the 20–24 year age group (1.2 cases per 100,000 population) and there was a male to female ratio of 1:1. Rates according to age group and sex are shown in Figure 24.

Figure 24. Notification rates of typhoid, Australia, 2001, by age group and sex



The National Enteric Pathogen Surveillance Scheme identified 69 isolates of *S.* Typhi in 2001. Fifty-two isolates were from Australian residents, nine from refugees in detention centres and eight from overseas visitors. The percentage known to be acquired overseas was 84 per cent (58/69). There was a single case of typhoid acquired within Australia, which was a laboratory-acquired infection.[26]

Quarantinable diseases

In Australia in 2001, the human diseases which were covered by the *Quarantine Act 1908* were cholera, plague, rabies, yellow fever, and four viral haemorrhagic fevers (Ebola, Marburg, Lassa and Crimean-Congo). These infections are of international public health significance, with mandatory reporting to the WHO. All states and territories notify quarantinable diseases to the NNDSS.

Four cases of cholera were the only reports of quarantinable disease notified in Australia in 2001. All cases were imported and occurred in two males and two females in New South Wales, Queensland, South Australia and Victoria. The organism was identified in one case as *V. cholerae* 01 — El Tor Inaba, imported from Hong Kong; another as *V. cholerae* 01, imported from Bali, Indonesia and a third as *V. cholerae* 01 — Ogawa, which also was acquired in Bali. The occurrence of cholera in returning travellers demonstrates the importance of consuming safe food and drink in areas where cholera is known to occur.

Two human cases of rabies, in which symptoms developed in Australia, were the result of overseas exposure in 1987 and 1990.[39] Although no cases of rabies or yellow fever were reported in Australia in 2001, worldwide these two diseases continue to cause fatalities and travellers should be aware of measures they can take to prevent infection with these viruses. Travellers intending to visit central

Africa or central South America are encouraged to receive the yellow fever vaccine from an approved Australian vaccination centre. Information on quarantinable diseases can be found on the DoHA's website: (www.health.gov.au/pubhlth/strateg/quaranti/index.htm).

Sexually transmitted infections

Sexually transmitted infections (STIs) remain a prevalent public health problem in Australia, despite efforts in prevention and education. In 2001, chlamydial infection, donovanosis, gonococcal infection and syphilis were nationally reportable to NNDSS, while chancroid and lymphogranuloma venereum were removed from NNDSS reporting. During 2001, a total of 27,817 STI notifications were received by NNDSS, which accounted for 27 per cent of all notifications.

A number of systems are involved in STI surveillance in Australia, including the NNDSS, the Laboratory Virology and Serology Reporting Scheme (LabVISE) (for chlamydia and syphilis) and specialist laboratory networks such as the Australian Gonococcal Surveillance Programme.[40] The NCHECR has an interest in STI surveillance, and have further analysed data from the NNDSS and other reporting sources in their annual surveillance report.[41]

The number of chlamydia and gonococcal infections reported in 2001 were the highest since 1991. Increases were also observed for donovanosis, while the number of syphilis notifications were at their lowest level since reporting commenced. Increases in some STIs may be due to higher rates of diagnosis, however, changes in surveillance methods may also account for some of the observed trends.

Chlamydial infection

The rate of chlamydial infections continued to increase in 2001 (Figure 25). During the year, a total of 20,026 notifications of chlamydial infection were received by NNDSS, an 18 per cent increase on the 17,018 cases reported in 2000. The notification rate for chlamydial infections in 2001 was 103 cases per 100,000 population, an increase from 88 cases per 100,000 population in 2000.

The increase in the number of chlamydial notifications occurred in all states and territories (Table 11). South Australia recorded the largest increase in 2001, of 37 per cent.

Notification rates vary widely between states and territories. The rates were above the national average in the Northern Territory (619.4 cases per 100,000 population), Queensland (153.9 cases per 100,000 population) and Western Australia (143.4 cases per 100,000 population, Map 3).

Figure 25. Trends in notification rates of chlamydial infection, Australia, 1994 to 2001, by year of onset

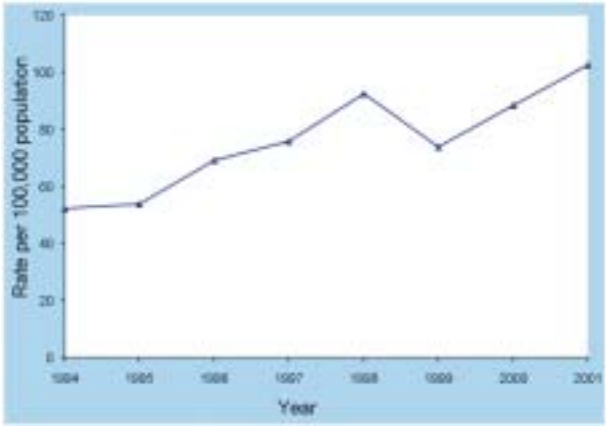
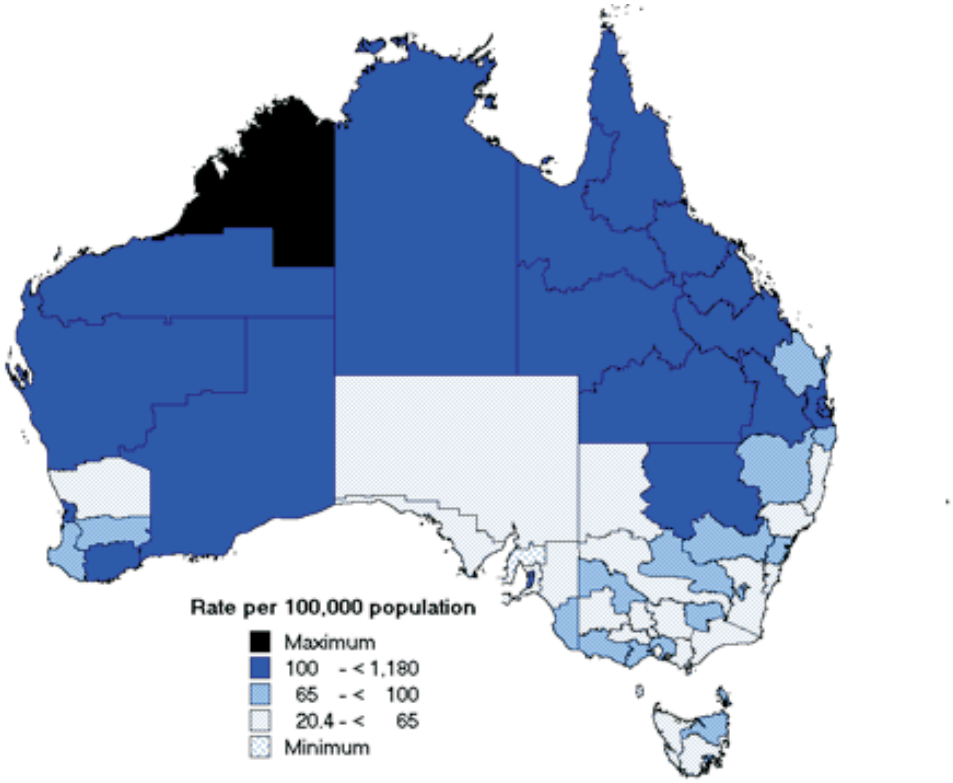


Table 11. Trends in notifications of chlamydial infection, 1994 to 2001, by state or territory

Year	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
1994	88	NN*	737	2,444	727	5	1,318	834	6,179
1995	80	NN	520	2,414	768	283	1,317	1,025	6,439
1996	110	NN	670	3,266	1,024	293	1,559	1,444	8,390
1997	142	NN	629	3,508	1,005	249	2,115	1,591	9,302
1998	194	NN	791	4,076	1,024	202	2,569	2,071	11,490
1999	177	2,461	863	4,476	973	254	2,939	1,903	14,046
2000	244	3,555	1,000	4,932	1,023	332	3,335	2,597	17,018
2001	301	4,389	1,239	5,596	1,402	380	3,924	2,733	19,964
Increase from 2000 (%)	23.4	23.5	23.9	13.5	37.0	14.5	17.7	5.2	17.3

* Not notifiable

Map 3. Notification rates of chlamydial infection, Australia, 2001, by Statistical Division of residence



The overall notification rate was 82.7 cases per 100,000 population for males and 122.2 cases per 100,000 population for females in 2001. The male to female ratio was 1:1.5, which was similar to 2000. Chlamydia is predominantly a disease of young adults. Among the cases in 2001, 77 per cent were in adolescents and young adults between the ages of 15 and 29 years. Notification rates of chlamydia in females exceeded those of males in each age group, with the greatest differences occurring in the 10–14 year age group (male:female ratio 1:7.7) and the 15–19 year age group (male:female ratio 1:3.8). Rates according to age group and sex are shown in Figure 26.

In 2001, the highest notification rate for females occurred in the 20–24 year age group (653.2 cases per 100,000 population), followed by the 15–19 year age group (565.3 cases per 100,000 population). These rates are 5.3 times and 4.6 times the national rate for women, in those aged 20–24 and 15–19 years respectively.

Trends in the sex distribution pattern for the 15–29 year age range since 1997 (Figure 27) show increases for all three age groups (15–19, 20–24, 25–29 years). The largest increases for chlamydia notifications were observed for females in the 15–19 and 20–24 year age groups and for males in the 25–29 years group.

Figure 26. Notification rates of chlamydial infection, Australia, 2001, by age group and sex

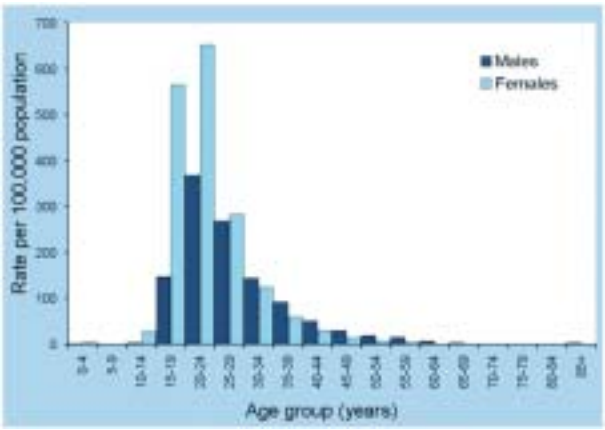
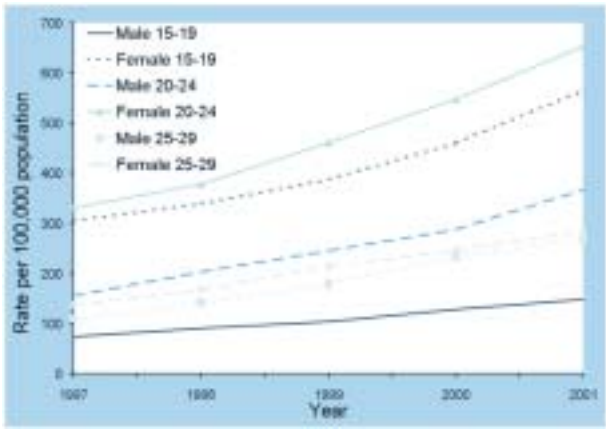


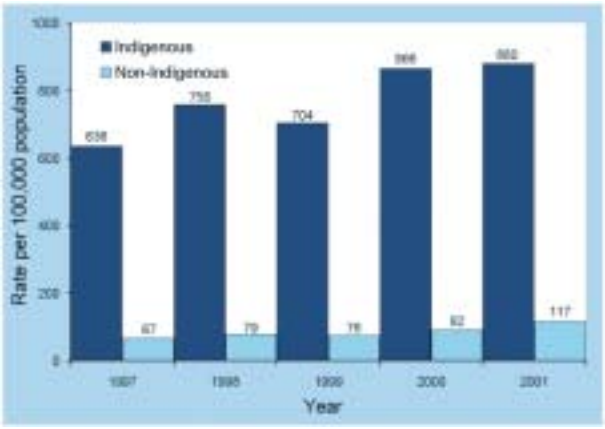
Figure 27. Trends in notification rates of chlamydial infection in persons aged 15–29 years, Australia, 1997 to 2001, by sex



There were 53 cases of chlamydia infection reported in children aged less than 10 years. All of these were cases of chlamydial conjunctivitis. Notifications were from all states and territories except South Australia and Tasmania.

Based on notifications from the Northern Territory, South Australia and Western Australia to NNDSS, the NCHECR have reported further details on chlamydial infection in Indigenous Australians. For these jurisdictions, Indigenous status was identified in 73 per cent of the notifications.[41] In 2001, the estimated age standardised rate of chlamydial infection among Indigenous Australians was 880 cases per 100,000 population, compared with the rate of 117 cases per 100,000 population in non-Indigenous Australians. Trends in notification rates of chlamydia in Indigenous and non-Indigenous Australians between 1997 and 2001 are shown in Figure 28.

Figure 28. Trends in age-standardised notification rates of chlamydial infection, the Northern Territory, South Australia and Western Australia (combined), 1997 to 2001, by Indigenous status*



* Data for 2001 unfinalised

Source: NCHECR HIV/AIDS Annual surveillance report: 2001

Chlamydial infection is caused by *Chlamydia trachomatis.* The infection can be asymptomatic. It has been estimated that in about 80 per cent of females and 40 per cent of males the infections are asymptomatic, and can easily remain undiagnosed.[42] It is difficult, therefore, to estimate the true burden of chlamydial infection in Australia. The disease is usually transmitted by vaginal intercourse or during oral or anal sex. Mother-to-child transmission can also occur during birth and may result in conjunctivitis or pneumonia in the newborn.

There may be a number of reasons for the increase in the number of chlamydial cases over the past decade. The increase may reflect the effect of chlamydia control campaigns that increase the rate of screening. The use of non-invasive tests (e.g., testing of urine) and more sensitive assays for chlamydial infection may increase the number of tests undertaken and the number of positive results. Enhanced surveillance activities by health authorities and greater awareness of the disease by health professionals may improve reporting.

If real, the continued increase in cases of chlamydia infection over the past decade, particularly among adolescents and young adults, is a cause for public health concern. Data on risk factors and evaluations of preventative programs may help understand disease transmission and deliver more comprehensive and effective control programs. In 2001 the Department of Human Services, Victoria, launched the *Chlamydia Strategy for Victoria, 2001 – 2004* to address the continuing increase in chlamydial infections.[43]

Donovanosis

Donovanosis is a sexually transmissible infection caused by *Calymmatobacterium granulomatis,* a gram-negative pleomorphic bacillus. It is characterised by genital ulcerative lesions which may develop into a chronic ulcerative disease if untreated. Lesions may be extensive and extragenital, and may be associated with secondary bacterial infection. The mode of transmission of donovanosis is primarily through sexual contact, although it may also be acquired by a faecal route, or at birth by passage through an infected birth canal.[44]

Internationally, donovanosis is endemic in tropical and sub-tropical areas, particularly PNG, central America, southern Africa and southern India.[16] In Australia, the disease is rare in the general population. It is, however, more common in Indigenous communities in rural and remote areas of northern Australia.[45,46]

Donovanosis was notifiable in all states and territories except South Australia in 2001. Among the notifiable STIs, donovanosis is the least commonly reported. NNDSS received a total of 42 notifications of donovanosis from the Northern Territory, Queensland and Western Australia in 2001, with a rate of 0.2 cases per 100,000 population.

The number of donovanosis cases in 2001 was twice that of 2000 (n=21). The increase of donovanosis notifications may be the result of the donovanosis eradication program, which includes enhanced surveillance in addition to the use of more sensitive and acceptable diagnostic assays, such as polymerase chain reaction.[47,48] Ultimately though, it should lead to eradication. As part of the program, in 2001 a project officer was employed in Western Australia, to raise awareness of donovanosis among health-care workers and communities in rural and remote areas of the State.

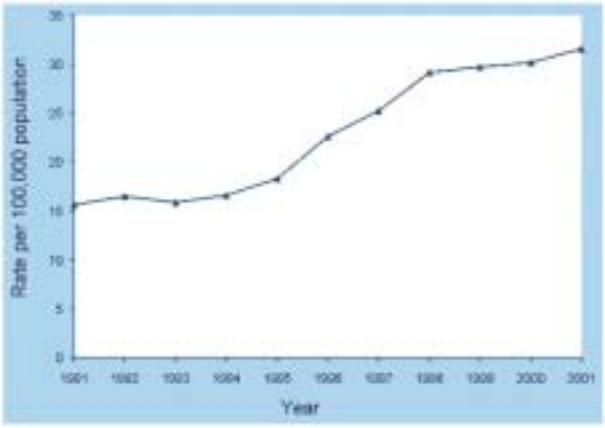
The highest group specific rates in 2001 were reported for females in the 15–19 year age group (0.8 cases per 100,000 population) and males in the 20–24 year age group (0.7 cases per 100,000 population) and with a male to female ratio of 1:1.8. Data on Indigenous status were available for all notifications and all but two cases were in Indigenous Australians.

Gonorrhoea

Infection by *Neisseria gonorrhoeae* is transmitted from person to person through vaginal, anal, or oral sexual contact. The disease may also be transmitted to the new-born from the mother's birth canal to cause gonococcal ophthalmia neonatorum. Humans are the only host for the bacterium.

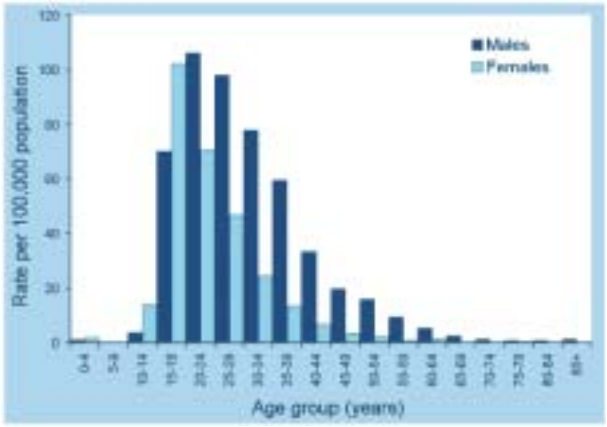
As with chlamydial infection, the number of notifications of gonococcal infection in Australia has increased over the last decade. The annual national notification rate of gonococcal infection has increased steadily from 16 cases per 100,000 population in 1993 to 32 cases per 100,000 population in 2001 (Figure 29). In 2001, a total of 6,158 notifications of gonococcal infection were reported nationally, an increase from the 5,801 reports received in 2000. The increase occurred in the Australian Capital Territory, New South Wales, the Northern Territory and Tasmania. The remaining states and territories showed a decrease in notifications in 2001.

Figure 29. Trends in notification rates of gonococcal infection, Australia, 1991 to 2001



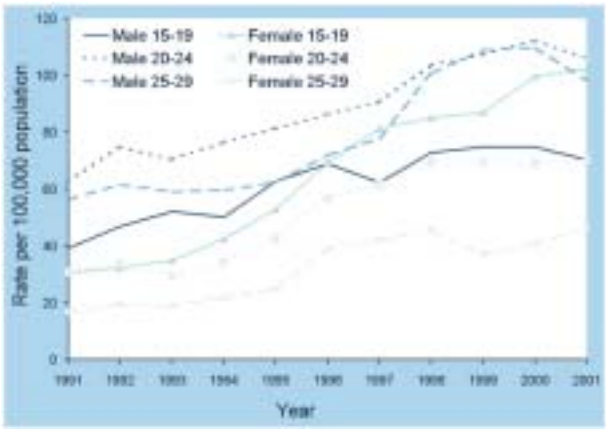
The notification rate was higher than the national average in the Northern Territory and Western Australia, with a rate 22 times the national level in the Northern Territory in 2001. The notification rate of gonococcal infection in 2001 was 42.9 cases per 100,000 population for males and 20.4 cases per 100,000 population for females. As in previous years, the male to female ratio remains 2:1. The age group specific notification rate of gonococcal infection in females was higher than that in males in the 10–14 year age group (male:female ratio; 1:3.8) and the 15–19 year age group (male:female ratio 1:1.5), while higher rates were observed in males compared to females in all other adult age groups (Figure 30).

Figure 30. Notification rates of gonococcal infection, Australia, 2001, by age group and sex



Trends in the sex-specific rates of gonococcal infection in persons aged 15–29 years over recent years all show a general increase. The increase was greatest for females in the 15–19 year group. The highest age group and sex-specific rates over time have been in males aged 20–29 years (Figure 31).

Figure 31. Trends in notification rates of gonococcal infection, in persons aged 15–29 years, Australia, 1991 to 2001, by sex

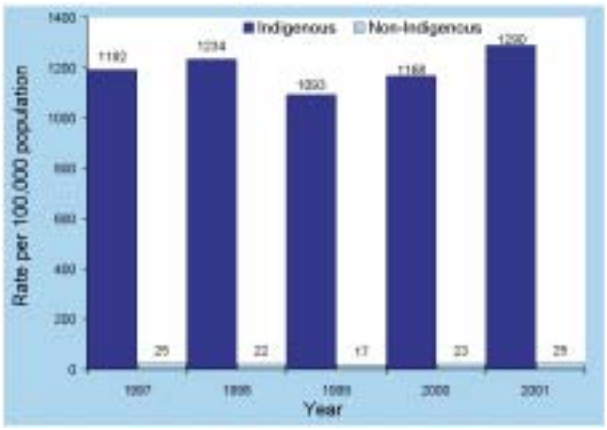


Increased testing, the availability of more sensitive diagnostic tests, and enhanced surveillance may account for some of the increase. True increases in disease may occur in some communities, such as men who have sex with men.[49,50] In the Northern Territory, increased screening for gonococcal infection through four sentinel practices in 2001 may also have increased notification rates. The use of polymerase chain reaction in place of culture for diagnosis in some areas may be reducing the number of gonococcal isolates that can be used to monitor antibiotic resistance.

There was wide geographical variation in the rate of notification of gonococcal infection. The highest rates of notification were reported in the Kimberley (1,580 cases per 100,000 population) and Pilbara (780 cases per 100,000 population) Statistical Divisions in northern Western Australia and in the Northern Territory (765 cases per 100,000 population) (Map 4).

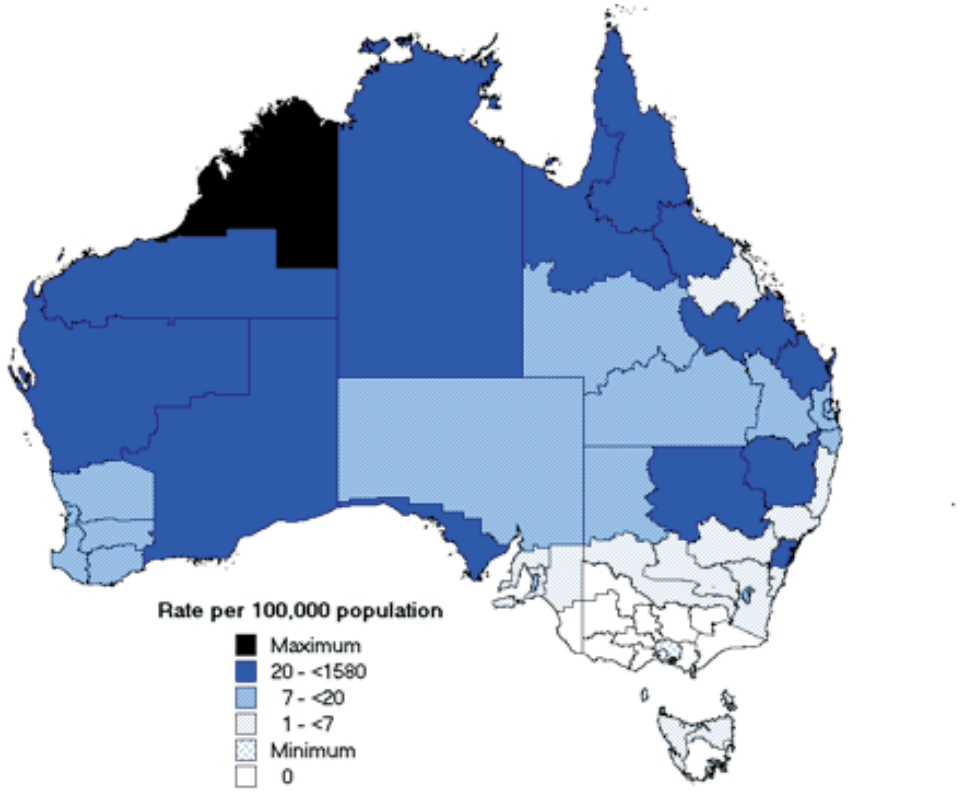
Based on the notifications from the Northern Territory, South Australia and Western Australia to NNDSS, the NCHECR reported further details on gonococcal infection in Indigenous Australians.[41] From these three jurisdictions, data on Indigenous status in 2001 were available for 87 per cent of notifications. The age standardised gonococcal notification rates were estimated to be 1,290 cases per 100,000 population in the Indigenous population, compared with 25 cases per 100,000 population in the non-Indigenous population. This represents an increase in gonococcal notification rates since 1999 for Indigenous Australians. The same trend was also observed in non-Indigenous Australians (Figure 32).

Figure 32. Trends in age-standardised notification rates of gonococcal infection, the Northern Territory, South Australia and Western Australia (combined), 1997 to 2001, by Indigenous status*



Source: NCHECR HIV/AIDS Annual surveillance report: 2001

Map 4. Notification rates of gonococcal infection, Australia, 2001, by Statistical Division of residence



Other surveillance activities for gonococcal infections

The Australia Gonococcal Surveillance Programme is the national laboratory-based surveillance system that monitors the antibiotic susceptibility of the gonococcus. The program is undertaken by a network of reference laboratories in each state and territory, which use an agreed and standardised methodology to quantitatively determine susceptibility of the organism to a core group of antibiotics. The annual results of the Australian Gonococcal Surveillance Programme have recently been published.[51]

In 2001, a total of 3,706 of gonococcal isolates were analysed by the Australian Gonococcal Surveillance Programme, an increase of five per cent on the previous year's total. The most common anatomical sites of isolates obtained for testing were from the urethra for males (80%) and from the cervix for females (92%). Rectal isolates, obtained only from males, comprised ten per cent of the isolates. Of the total number of isolates, 85 per cent were from men, and this ratio was little changed from 2000.

Table 12 presents trends in annual antibiotic resistance rates in Australia between 1998 and 2001. The proportion of isolates resistant to penicillin by chromosomally-controlled mechanisms increased from 10.6 per cent in 2000 to 15.3 per cent in 2001, but this rate is still less than the 22 per cent recorded in 1998. While the level of quinolone resistance in gonococci decreased slightly from the previous year, it became more widespread in Australia in 2001. Antibiotic susceptibility patterns varied considerably between regions and resistance to the penicillins remained high in larger urban centres.[51]

Syphilis

During 2001, 1,406 cases of syphilis were reported to the NNDSS. This represents a decrease of notifications for the second consecutive year and it was the lowest number of cases received by NNDSS since 1991. In Australia, all states and territories report syphilis (including primary, secondary and latent syphilis) and congenital syphilis separately, to NNDSS.

In 2001 the overall notification rate for syphilis was 8.4 cases per 100,000 population for males and 6.1 cases per 100,000 population for females. The male to female ratio was 1:0.7. The peak notification rate was reported in females in the 15–19 year age group (16.2 cases per 100,000 population). The disease is more common in females in their child-bearing years. In 2001, 75 per cent of the cases in females occurred in the 15–44 year age range. The highest age specific notification rate for males was in the 20–24 year age group (14.4 cases per 100,000 population, Figure 33). Since 1991, overall decreases in the syphilis notification rate have been clearly observed in the 15–29 year age range, for both males and females (Figure 34).

The national notification rate for syphilis in 2001 was 7.3 cases per 100,000 population, a decrease from 9.3 cases per 100,000 population in 2000. Decreases were seen in the Australian Capital Territory, New South Wales and Queensland. Increases in the number of syphilis notifications cases in 2001 was reported in the other five states and territories. The increase may reflect a more active follow-up of suspected cases.[52] Significant cleaning of syphilis notification data in some states and territories in 2001 has accounted for decreased numbers of notifications.

Figure 33. Notification rates of syphilis, Australia, 2001, by age group and sex

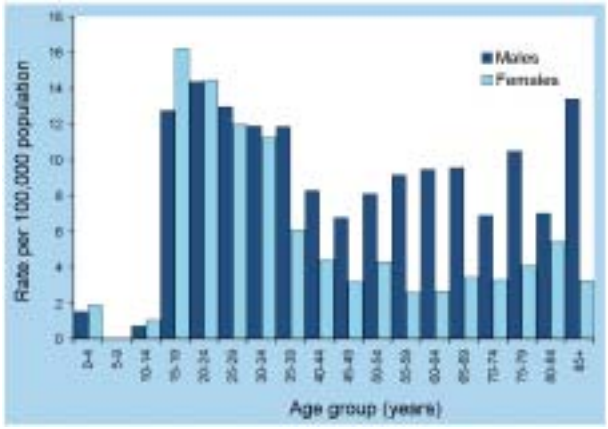
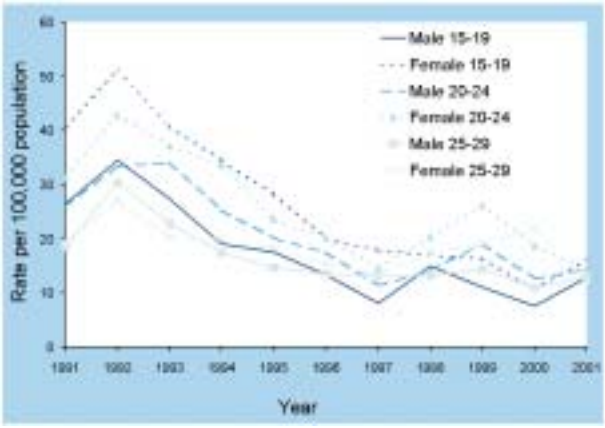


Table 12. Proportion of gonococcal isolates showing antibiotic resistance, Australia, 1998 to 2001

	Penicillin resistance (% resistant)		Quinolone resistance (% resistant)	High level tetracycline resistance (% resistant)
	Plasmid mediated resistance	Chromosomally mediated resistance		
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

NR Not recorded

Figure 34. Trends in notification rates of syphilis, in persons aged 15–29 years, Australia, 1991 to 2001, by sex

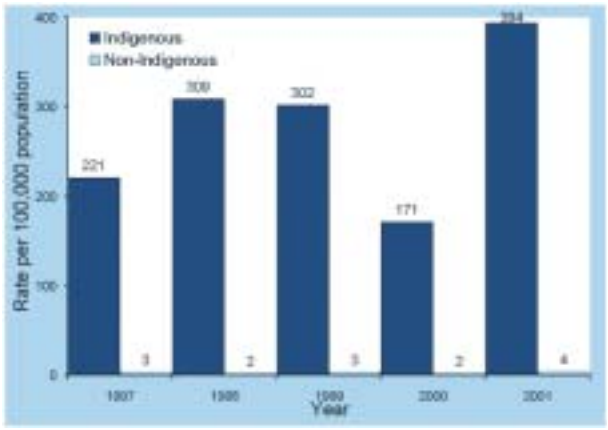


In 2001, there was wide geographical variation in notification rates for syphilis (Map 5). The highest rate occurred in Western Australia in the Kimberley Statistical Division (360.9 cases per 100,000 population).

There were 21 cases of syphilis in children aged less than one year (3 in New South Wales, 17 in the Northern Territory and 1 in Western Australia). All were confirmed as congenital syphilis.

Based on the notifications from the Northern Territory, South Australia and Western Australia to NNDSS, the NCHECR reported further details on syphilis in Indigenous Australians.[41] From these three jurisdictions, data on Indigenous status was available for 93 per cent of notifications in 2001. The age standardised syphilis rates were estimated to be 394 cases per 100,000 population for Indigenous Australians, compared with 4 cases per 100,000 population for non-Indigenous Australians. Trends in notification rates of syphilis in Indigenous and non-Indigenous Australians from these states and territories between 1997 and 2001 are shown in Figure 35.

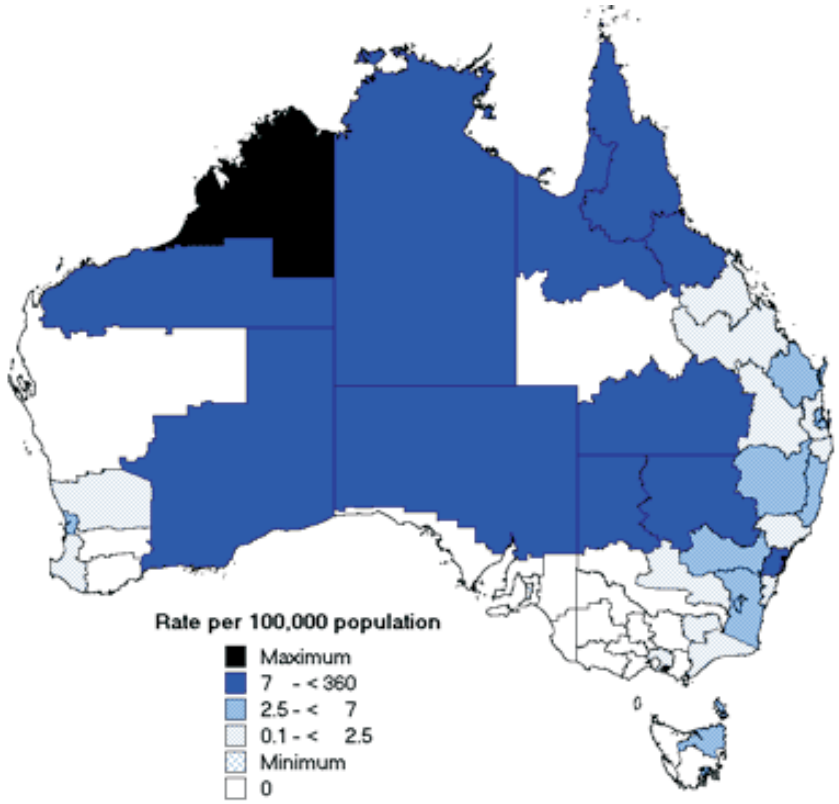
Figure 35. Trends in age-standardised notification rates of syphilis, the Northern Territory, South Australia and Western Australia (combined), 1997 to 2001, by Indigenous status*



* Data for 2001 unfinalised

Source: NCHECR HIV/AIDS Annual surveillance report: 2001

Map 5. Notification rates of syphilis, Australia, 2001, by Statistical Division of residence



Vaccine preventable diseases

This section summarises the national notification data for laboratory-confirmed influenza and invasive pneumococcal disease as well as diseases targeted by the Australian Standard Vaccination Schedule in 2001. This includes diphtheria, *Haemophilus influenzae* type b (Hib) infection, measles, mumps, pertussis, poliomyelitis, rubella and tetanus. The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) have recently published a detailed analysis of vaccine preventable diseases in Australia for 1999 to 2000.[53]

Laboratory-confirmed influenza and invasive pneumococcal disease (IPD), were added to the list of nationally notifiable diseases in 2001. Both these diseases were made notifiable in all Australian states and territories during 2001. Because of the need to change public health legislation to include these diseases, complete data for 2001 were not available from all states and territories.

The rationale for the introduction of surveillance of laboratory-confirmed influenza was to give better national data on the annual incidence of influenza, the circulating viral subtypes and the effectiveness of annual influenza vaccinations. In Australia, annual vaccination against influenza is provided free of charge to non-Indigenous Australians aged 65 years and above and to Indigenous Australians aged 50 years and above. It is also recommended for individuals who are at increased risk of influenza-related complications and those who may transmit influenza to persons at increased risk.[54]

There was only one change to the Childhood Immunisation Schedule in 2001 — a program for at-risk children using the seven-valent conjugate pneumococcal vaccine was recommended and publicly funded (Table 13). The program was introduced with a focus on Indigenous children, who have some of the highest incidences of invasive pneumococcal disease in the world.[55] The conjugate vaccine has been demonstrated to have an efficacy of 94 per cent in preventing invasive pneumococcal disease in young children.[2]

There were 13,030 notifications of vaccine preventable diseases in 2001; one in eight of the total notifications to NNDSS. Pertussis was by far the most common with 9,515 notifications, or 73 per cent of all vaccine preventable disease notifications.

Diphtheria

A single case of cutaneous diphtheria in a 52-year-old man was reported from the Northern Territory in March 2001. A toxigenic strain of *Corynebacterium diphtheriae* var. *mitis* was isolated. The patient acquired the disease in East Timor and had an uncertain vaccination history. This is the first case reported in Australia since 1993.

Haemophilus influenzae type b disease

Notifications of Hib disease have fallen more than 30-fold since 1991, due to the efficacy of Hib conjugate vaccines (Figure 36). There were 26 notifications of Hib disease in 2001, a rate of 0.1 cases per 100,000 population. This is eight per cent less than in 2000, and the lowest number of notifications recorded since national surveillance began in 1991. Most notified cases (14, 53%) were aged less than five years and five were infants aged less than one year. Rates according to age group and sex are shown in Figure 37. There were less notifications of Hib disease for males than for females (male:female ratio 0.7:1) in 2001.

Table 13. Vaccination schedules for seven-valent conjugate pneumococcal vaccine in Australia

Date implemented	July 2001
Serogroups in vaccine	4, 6B, 9V, 14, 18C, 19F, 23F
Target populations	All Aboriginal and Torres Strait Islander infants in a 3-dose series at 2, 4 and 6 months of age, with a booster dose of the 23-valent pneumococcal polysaccharide vaccine (23vPPV) at 18–24 months of age. Catch-up is recommended for Aboriginal children in Central Australia up to the fifth birthday and for Aboriginal and Torres Strait Islander children elsewhere up to the second birthday. All Australian children with underlying predisposing medical conditions at 2, 4 and 6 months of age with a booster dose (of 7vPCV) at 12 months of age and a booster dose of 23vPPV at 4–5 years of age. Catch-up vaccination is recommended for these children up to the fifth birthday. Non-Indigenous children residing in Central Australia up to the second birthday, as catch up vaccination.
Data source	*Australian Immunisation Handbook*, 8th edition

Figure 36. Trends in notifications of *Haemophilus influenzae* type b infections, Australia, 1991 to 2001, by month of onset

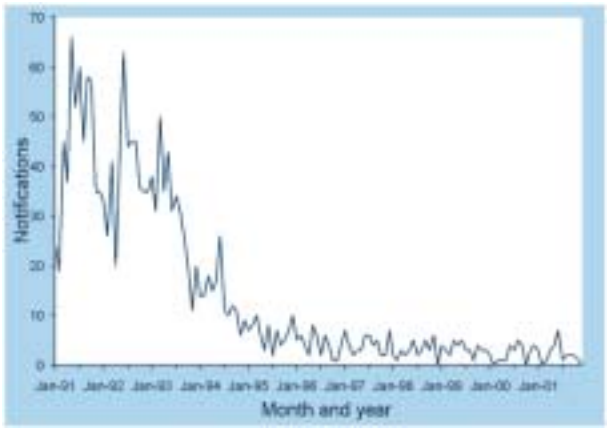
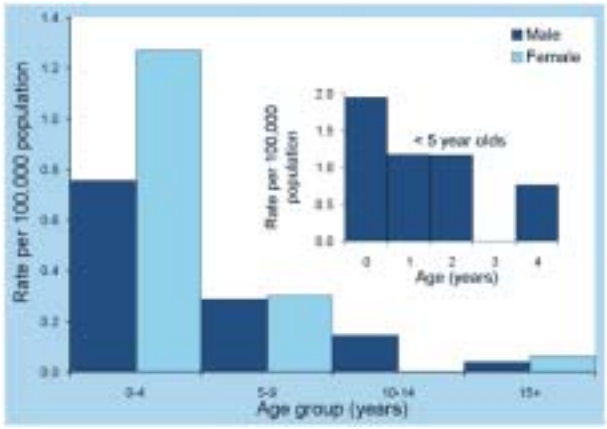


Figure 37. Notification rates of *Haemophilus influenzae* type b infection, Australia, 2001, by age group and



The Northern Territory had the highest notification rate (n=3, 1.5 cases per 100,000 population) although most cases were from New South Wales (9/26). The vaccination status of eleven cases was known; seven were unvaccinated, two partially vaccinated and two cases in Victoria were fully vaccinated. These two children were confirmed cases with documented evidence of receipt of four doses of Hib vaccine and had no identified risk factors.

Laboratory-confirmed influenza

There were 1,286 reports of laboratory-confirmed influenza in 2001 to the NNDSS, a rate of 6.6 cases per 100,000 population. As noted above, data were not available from all states and territories for the full year, consequently these numbers are an underestimate of the true incidence. No notifications were received from Tasmania. Notifications of laboratory-confirmed influenza showed a peak in August and September (late winter). These data, together with the month when reporting began in each state or territory, are shown in Figure 38.

The highest rates of laboratory-confirmed influenza were in children aged less than five years (Figure 39). The male to female ratio was 1.1:1.

In 2001, influenza A was the dominant type, 81 per cent of which were subtype H1N1 and 19 per cent were subtype H3N2. The influenza A (H1N1) isolates analysed were all A/New Caledonia/20/99-like strains. The H3N2 isolates were antigenically similar to the reference strain (A/Moscow/10/99) and the vaccine strain (A/Panama/2007/99). The influenza B isolates, which made up only 10 per cent of all (influenza A and B) isolates, were mainly B/Sichuan/379/99-like strains. Ten per cent of the influenza B isolates though were more closely related to B/Harbin/7/94-like viruses, which have circulated in previous years. The Australian 2001 influenza vaccine therefore represented a good match for the circulating viruses and 77 per cent of the over 65 year age group in Australia was vaccinated in 2001.[56]

Figure 38. Notifications of laboratory-confirmed influenza and month when reporting to the National Notifiable Diseases Surveillance System began in each state or territory, Australia, 2001

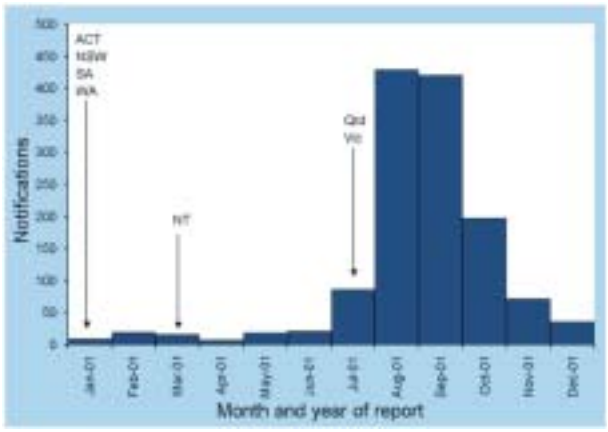
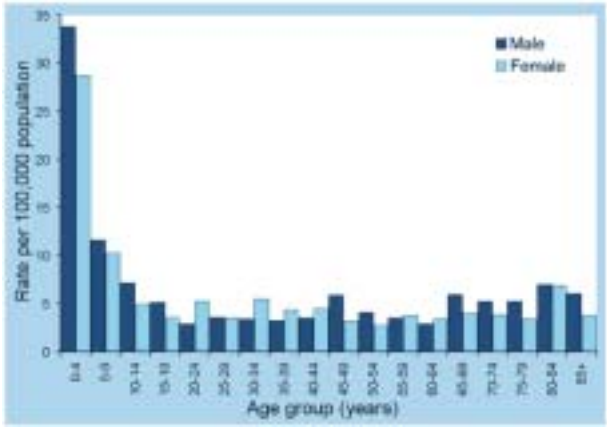


Figure 39. Notification rates of laboratory-confirmed influenza, Australia, 2001, by age group and sex

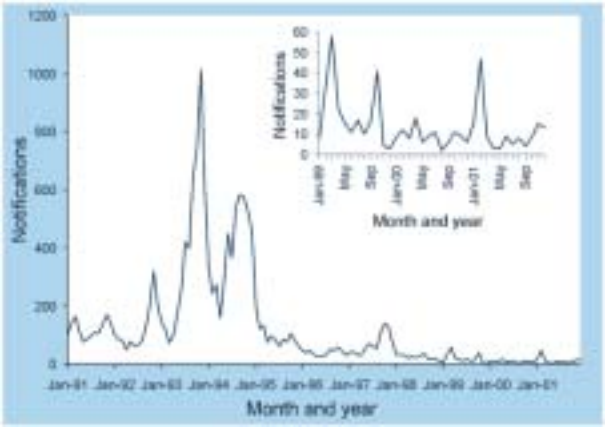


Measles

Measles is the most important cause of vaccine-preventable death in the world. In 1998 an estimated 30 million measles cases and 880,000 measles-associated deaths occurred worldwide, with 85 per cent of deaths occurring in Africa and South-East Asia.[57] In recent years a dramatic reduction in measles incidence and elimination of endemic measles transmission has been achieved in a number of countries with a variety of vaccination strategies.[58]

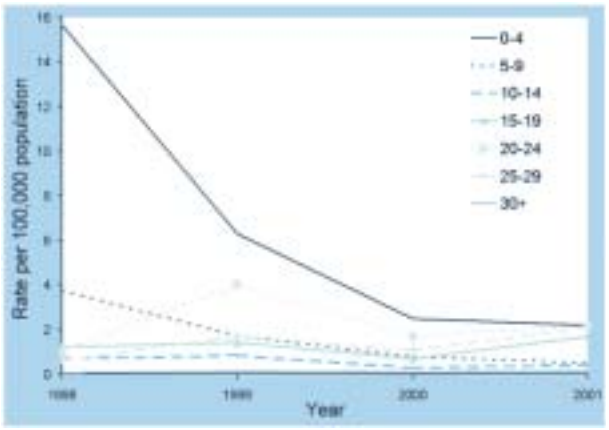
In Australia there were 141 measles notifications in 2001, a national rate of 0.7 cases per 100,000 population. This is a slight increase on the 107 cases reported in 2000, which was the lowest annual rate for Australia since national surveillance began in 1991 (Figure 40). The highest rate was in Victoria with 1.7 cases per 100,000 population (n=83).

Figure 40. Trends in notification rates of measles, Australia, 1991 to 2001, by month of onset



As in recent years, the age-specific notification rate of measles was highest for the 0–4 year age group (2.2 cases per 100,000 population). The rate for this age group was considerably lower than it has been in the past (Figure 41). Within the 0–4 year age group most cases (54%) were aged less than one year old. The rate for the 5–9 year age group (0.5 cases per 100,000 population) was also the lowest on record. Following the 0–4 year age group, the next highest rates were in the 20–24 year age group (2.2 cases per 100,000 population) and the 25–29 year age group (2.1 cases per 100,000 population). The proportion of cases in the 20–29 year age group has been increasing since national surveillance began, from 6 per cent in the early 1990s to above 30 per cent in 1999 to 2000. In 2001, the 20–29 year age range accounted for 41 per cent (58/141) of the reported cases.

Figure 41. Notification rates of measles, Australia, 1998 to 2001, by age group



There were a number of measles outbreaks in Australia in 2001. In January, a young Australian recently returned from India was the index case in an outbreak affecting 50 young adults in Melbourne. All cases were laboratory confirmed.[59] The median age was 25 years (range 10 months to 34 years) with 90 per cent aged 15 to 34 years. Most cases were unvaccinated and four were partially vaccinated against measles. Twenty-two (43%) of the confirmed cases were hospitalised for an average of four days (range 1–10 days) but there were no deaths.

A second outbreak of measles in Victoria was reported in October. The index case had acquired the infection overseas and there were 17 laboratory-confirmed cases linked to the index case. All 18 cases were infected with the D5 measles genotype. The majority of cases were aged between 18 and 34 years and none had a documented history of measles vaccination.

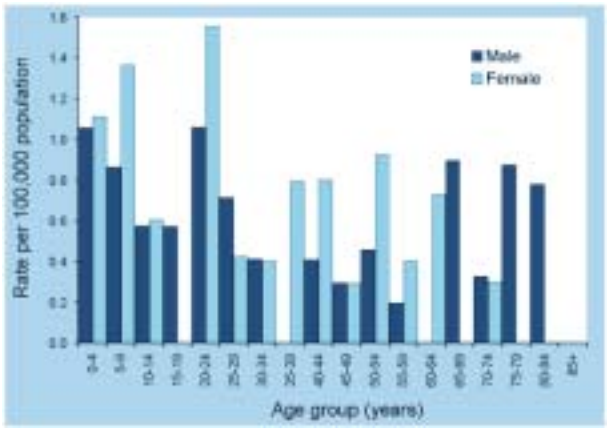
The third cluster of seven cases (five of which were laboratory confirmed) occurred in the second quarter of the year in western Sydney. The index case was probably infected while travelling overseas. Three infants aged 8–12 months and four young adults aged 19–26 years were involved in this outbreak. A measles outbreak in PNG in June 2001 prompted CDNA to warn intending travellers to that country that they should be vaccinated against measles.

In 2001, the Victorian Infectious Diseases Reference Laboratory established the National Measles Laboratory in Melbourne. The laboratory analysed samples from five Australian states and territories during the year, and 18 distinct measles strains belonging to eight different measles genotypes were identified. These observations suggest that indigenous measles transmission has been eliminated in Australia, and that periodic introductions of the virus occurs predominantly from South-East Asia.[60]

Mumps

In 2001, there were 114 notifications of mumps, a rate of 0.6 cases per 100,000 population. This is a decrease of 47 per cent on the 212 cases reported in 2000 and the lowest rate since all states and territories began notifying the disease in 1996 (although mumps was not notifiable in Queensland between July 1999 and December 2000). There were cases in most age groups (Figure 42) but the majority (n=77, 68%) were from people aged 15 years or more. Although rates were also considerably less than in 2000, the 20–24 year age group still had the highest rate of notifications (1.2 cases per 100,000 population). The next highest rates were in the 0–4 and 5–9 year age groups (both 1.1 cases per 100,000 population). Unlike most previous years, there was a slight preponderance of mumps cases in females (male:female ratio 0.8:1).

Figure 42. Notification rates of mumps, Australia, 2001, by age group and sex



Pertussis

Pertussis continues to be the most common vaccine preventable illness in Australia, with periodic epidemics occurring at intervals of 3 to 5 years (Figure 43).[61] There were 9,515 notified cases of pertussis in 2001, 60 per cent more than the 5,942 cases reported in 2000. The annual notification rate was 48.8 cases per 100,000 population.

Since 1999, the 10–14 year age group have had the highest notification rates of pertussis and this pattern continued in 2001 (187 cases per 100,000 population) (Figure 44). Changes in the age distribution of cases is a result of the introduction of a fifth dose of pertussis vaccine in 1994, which is given at four years of age. Children less than one year of age (particularly those under six months who have received fewer than three doses of vaccine) also have high notification rates. This age group has significantly higher morbidity and mortality than any other age group.[53]

For all age groups up to 80 years there were higher notification rates in females than in males (Figure 45), and the overall male to female ratio was 0.8:1.

Notification rates of pertussis varied considerably by geographic location (Map 6). At the state or territory level, rates were highest in South Australia (132.7 cases per 100,000 population) and lowest in Western Australia (11.9 cases per 100,000 population).

Figure 43. Trends in notifications of pertussis, Australia, 1991 to 2001, by month of onset

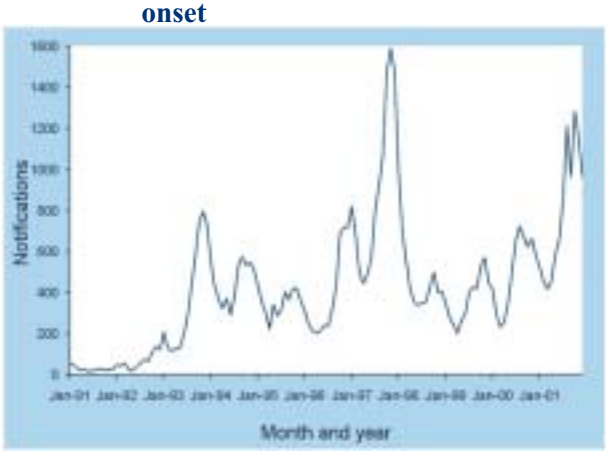


Figure 44. Notification rates of pertussis, Australia, 1996 to 2001, by age group

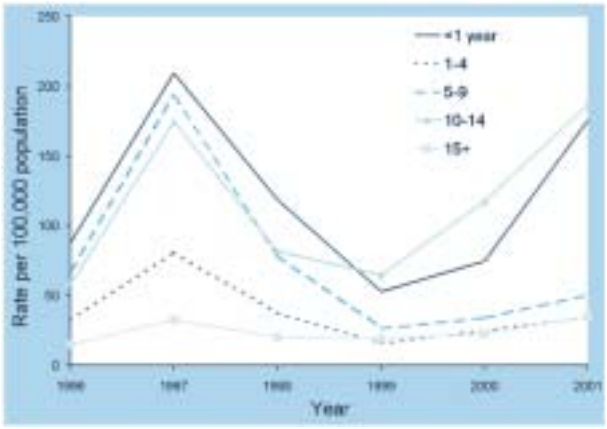
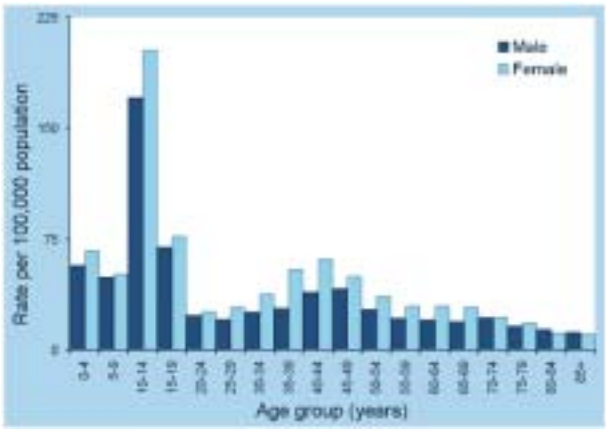
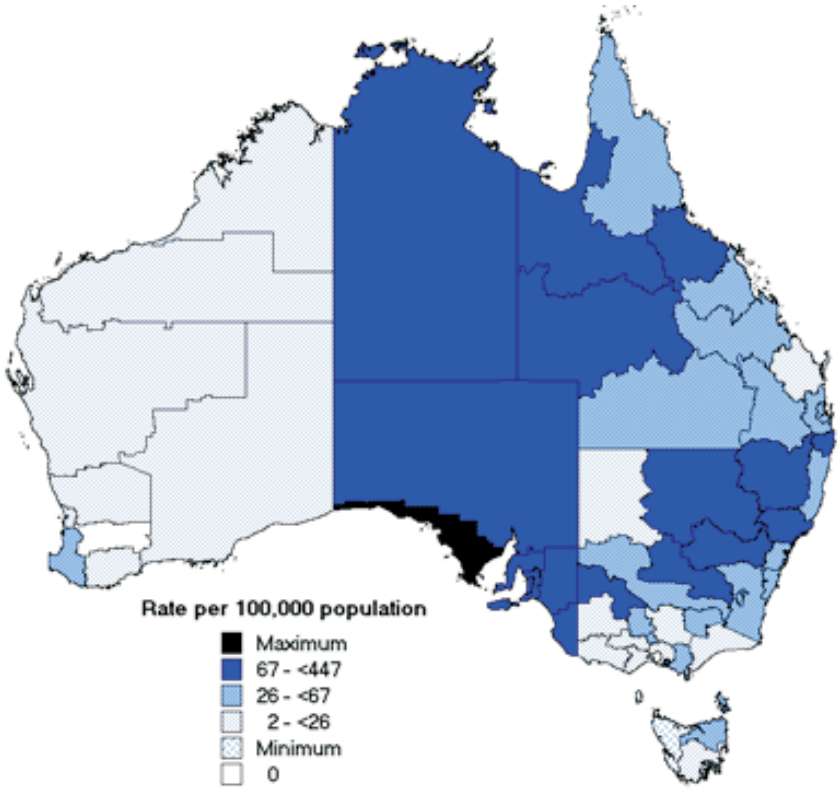


Figure 45. Notification rates of pertussis, Australia, 2001, by age group and sex



Map 6. Notification rates of pertussis, Australia, 2001, by Statistical Division of residence



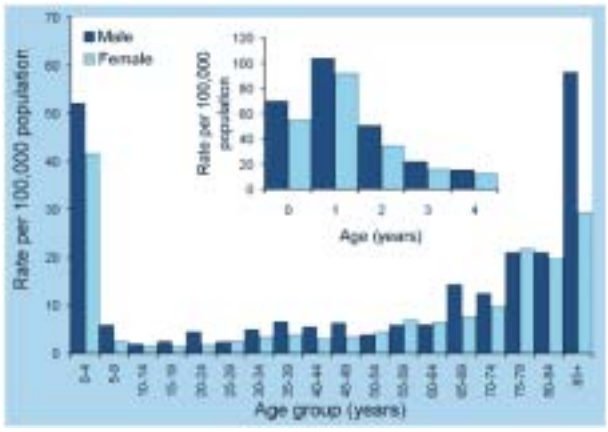
Invasive pneumococcal disease

There were 1,681 notifications of IPD in Australia in 2001, giving a rate of 8.6 cases per 100,000 population. The rates for 2001 are likely to be an under-estimate because for some jurisdictions data on IPD was not available for the whole year. While the largest number of cases were found in New South Wales, Queensland and Victoria, the highest rate occurred in the Northern Territory (48.5 cases per 100,000 population), which was more than five times the national rate. The geographical distribution of IPD varied within states and territories (Map 7), with the highest rates in central and northern Australia.

IPD is largely a disease of the very young and very old. In 2001 the highest rates of disease were in children aged less than five years (47.3 cases per 100,000 population) and adults aged more than 85 years (38.7 cases per 100,000 population). Rates according to age group and sex are shown in Figure 46. There were more cases among males, with a male to female ratio of 1.2:1. A peak of IPD occurred in late winter and early spring, with the largest number, 259 notifications, being reported in August 2001.

Additional data were collected on cases of invasive pneumococcal disease in some Australian states and territories during 2001. Analyses of these data have recently been published.[62,63]

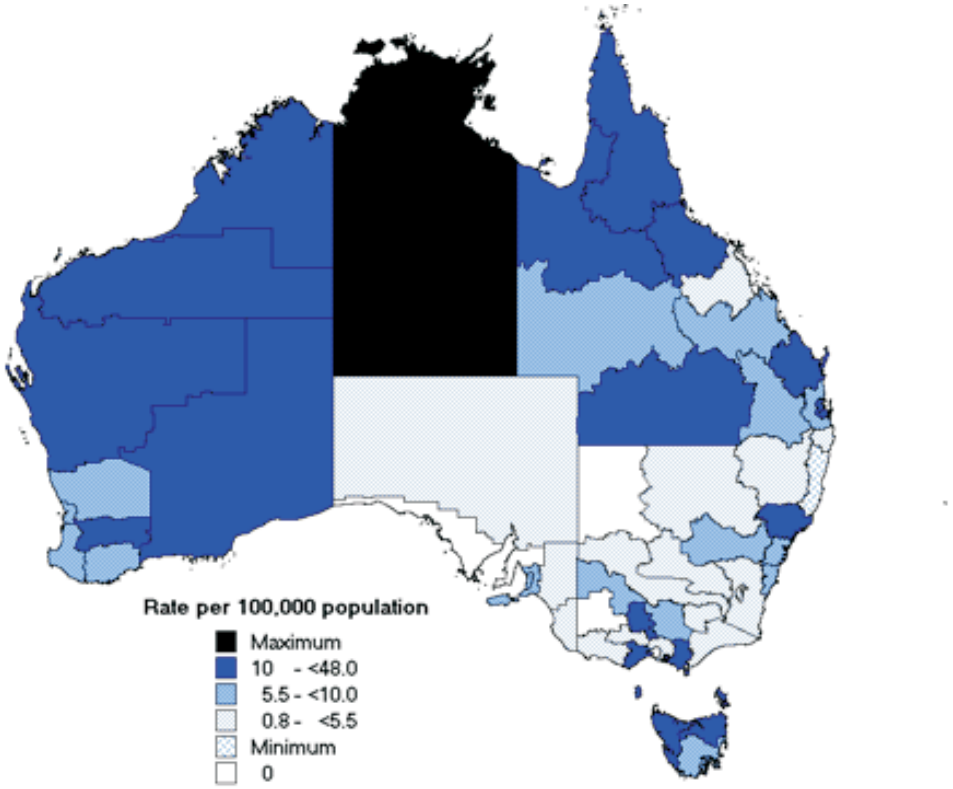
Figure 46. Notification rates of invasive pneumococcal disease, Australia, 2001, by age group and sex



Poliomyelitis

No cases of poliomyelitis were reported in Australia in 2001. The National Poliovirus Reference Laboratory at the Victorian Infectious Diseases Reference Laboratory is responsible for poliovirus testing for Australia. It is also the regional reference laboratory for the Western Pacific Region (WPR) of WHO. Surveillance for acute flaccid paralysis, a clinical manifestation of poliomyelitis, is coordinated at the Victorian Infectious Diseases Reference Laboratory in collaboration with the Australian Paediatric Surveillance Unit.

Map 7. Notification rates of invasive pneumococcal disease, Australia, 2001, by Statistical Division of residence



There were 60 unique notifications of acute flaccid paralysis in 2001, of which 44 were classified by the Polio Expert Committee as eligible non-polio acute flaccid paralysis cases (isolates from patients resident in Australia and aged less than 15 years). Polioviruses were isolated from only one acute flaccid paralysis patient and characterised as Sabin oral poliovirus vaccine-like serotypes 1,2 and 3. In the same patient *Clostridium botulinum* type b organism and toxin were also detected and the case was classified as infant botulism.

As part of the laboratory containment of poliovirus, during 2001 the National Polio Reference Laboratory received viral isolates or samples stored in laboratories across Australia that may contain poliovirus. Forty Sabin-like viruses and five non-Sabin-like polioviruses were identified from 74 referred laboratory isolates and specimens.

The WPR, of which Australia is a member nation, was declared free of circulating wild poliovirus in October 2000. During 2001, however, viruses derived from the Sabin oral polio vaccine caused three cases of poliomyelitis in the Philippines, also a member nation of the WPR. The identification of these three cases has emphasised the necessity of maintaining a high level of vaccination coverage within Australia and an effective surveillance system to detect cases of poliomyelitis.[64]

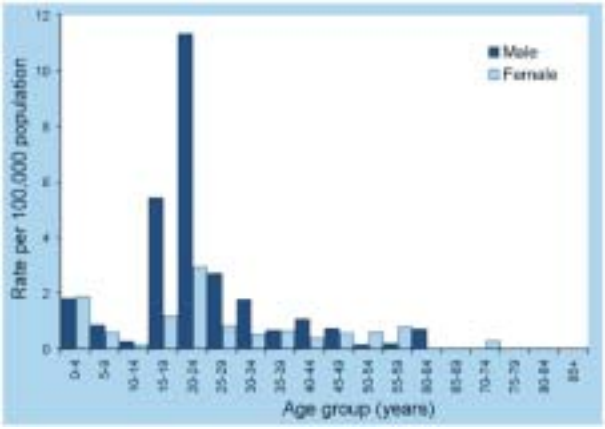
Rubella

Since 1995 the annual number of rubella notifications have been declining. This decrease has occurred at the same time as the measles, mumps and rubella vaccination coverage rates have increased. In 2001, there were 263 notifications, a notification rate of 1.3 cases per 100,000 population, an 18 per cent decrease from the 322 cases of rubella reported in 2000. This is the lowest rate on record since the NNDSS commenced in 1991. As in previous years, the highest number of notified cases occurred in October, reflecting the usual seasonal increase in spring months. The highest notification rate was in Queensland (3.7 cases per 100,000 population).

In 2001, notification rates were highest in males in the 20–24 year age group (11.3 cases per 100,000 population, Figure 47). As in previous years, there were more males than females notified with rubella (male:female ratio 2.5:1) and the ratio is higher than in the past five years.

There were 49 cases of rubella in women of childbearing age (15–49 years) in 2001. Seven cases occurred in young infants, five within two months of birth. There were no notifications of congenital rubella in 2001.

Figure 47. Notification rates of rubella, Australia, 2001, by age group and sex



Tetanus

There have been less than eight cases of tetanus notified each year in Australia since 1995 mainly in adults aged over 70 years. In 2001, there were three cases. Two were aged 70 years or more and the third was also an adult. Two cases were male and one cases was female.

Childhood vaccination coverage reports

Estimates of vaccination coverage both overall and for individual vaccines for children at 12 months of age continued to improve in 2001. This trend was also evident in each state and territory. Vaccination coverage at one year of age is shown in Table 14.

Vaccination coverage at two years of age was first reported in 1998. Coverage estimates for vaccines recommended at 12 and 18 months of age were higher in 2001, compared with the previous year, as were the estimates for being 'fully vaccinated' at two years of age. Vaccination coverage at two years of age is shown in Table 15. The reported 'fully vaccinated' coverage levels are lower than the levels for individual vaccines, because children who have missed vaccination against some diseases are not necessarily those who have missed vaccination against the other diseases. It is important to note that in other countries such as the United Kingdom, three doses of the diphtheria-tetanus-pertussis and Hib vaccines constitute full vaccination for these vaccines at two years of age.

Table 14. Percentage of Australian children born in 2000 vaccinated according to data available on the Australian Childhood Immunisation Register. Estimate at one year of age

Vaccine group	Per cent vaccinated			
	1 Jan–31 Mar 2000	1 Apr–30 Jun 2000	1 Jul–30 Sep 2000	1 Oct–31 Dec 2000
DTP	91.8	91.9	92.2	92.0
OPV	91.7	91.8	92.1	91.9
Hib	94.8	94.5	94.3	94.5
Hepatitis B	NA	NA	94.3	94.4
Fully vaccinated	91.5	91.2	90.4	90.5

DTP Diphtheria-tetanus-pertussis
OPV Oral polio vaccine
NA Not available

Table 15. Percentage of Australian children born in 1999 vaccinated according to data available on the Australian Childhood Immunisation Register. Estimate at two years of age

Vaccine group	Per cent vaccinated			
	1 Jan–31 Mar 1999	1 Apr–30 Jun 1999	1 Jul–30 Sep 1999	1 Oct–31 Dec 1999
DTP	89.5	89.8	90.3	90.2
OPV	93.9	93.9	94.3	94.4
Hib	95.0	95.2	95.3	95.4
MMR	92.8	93.1	93.2	93.4
Fully vaccinated	86.6	87.0	88.0	87.8

DTP Diphtheria-tetanus-pertussis
OPV Oral polio vaccine
MMR Measles-mumps-rubella

Vectorborne diseases

Introduction

Vectorborne diseases reported to the NNDSS include arbovirus infections and malaria. Arboviruses (arthropod-borne viruses) belong to two families, the alphaviruses, which include Barmah Forest virus infection (BF) and Ross River virus infection (RR) and the flaviviruses, which include dengue and the Murray Valley encephalitis virus (MVE), Kunjin virus and Japanese encephalitis virus (JE). Malaria cases recorded by NNDSS include infections caused by four *Plasmodium* species.

Arboviruses and malaria are transmitted to humans through the bite of infected mosquitoes. The human population acts as the host species for dengue and malaria. The remaining arboviruses discussed in this chapter have a complex life cycle involving vertebrate hosts, mosquitoes and humans. The vertebrate reservoirs include marsupials, introduced placental mammals and avian species. During epidemics it is possible that newly infected humans further spread these viruses.

Malaria and dengue cases have been reported to NNDSS since 1991. At this time, all other arboviruses were reported to NNDSS as 'Arbovirus — NEC'. Infection with RR became separately notifiable in 1993 and BF in 1995. Infection with MVE, Kunjin virus and JE were separately reported in 2001.

Alphaviruses

Barmah Forest virus infection and Ross River virus infection

Clinical infections with BF and RR are characterised by arthritis, myalgia, fever, headaches and lethargy. A rash is also usually present. The spectrum of illness ranges from the sub-clinical, through to illness that may last for months or even years.[65] Recent results, however, suggest that persistence for long periods may be overestimated.[66]

Infections with BF and RR are diagnosed by serological tests. As exposure to the viruses may have occurred in the past (resulting in persistent antibodies), it is important to obtain both acute and convalescent sera, approximately two weeks apart, in order to demonstrate seroconversion or the increase in antibody levels that characterises new infections.

In Australia the primary arthropod vectors for both BF and RR are mosquitoes of the *Ochlerotatus* (previously *Aedes*) and *Culex* genera. The primary hosts for RR are believed to be macropods (kangaroos and wallabies), but horses and fruit bats have shown serological evidence of infection. The range of primary hosts for BF is less understood.

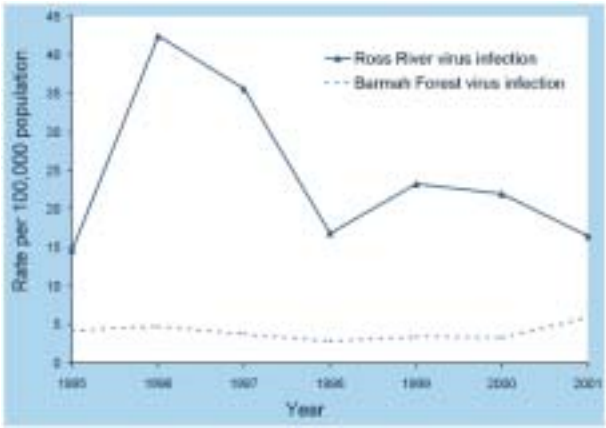
The first human infection with BF was identified in 1986. The first recognised outbreak occurred in Nhulunbuy, on the Gove Peninsula in the Northern Territory in 1992.[67]

RR is the most commonly reported arboviral disease in Australia. During 2001, 3,219 notifications of RR infection and 1,141 notifications of BF infection were received. A comparison of the annual trends in the notification rates of RR infection and BF infection since 1995 is shown in Figure 48. The wide fluctuation in the rates of RR infection probably reflects the occurrence of outbreaks against a background of sporadic cases.

The number of cases of BF notified in 2001 was the largest recorded since it became separately notifiable in 1995. The national notification rate for 2001 was 5.9 cases per 100,000 population. Prior to this rates have ranged between 4.8 cases per 100,000 population (1996) and 2.8 cases per 100,000 population (1998). In 2001 most notifications of BF infection came from Queensland (n=603) and New South Wales (n=398). The highest rate at the state and territory level occurred in the Northern Territory (18.5 cases per 100,000 population).

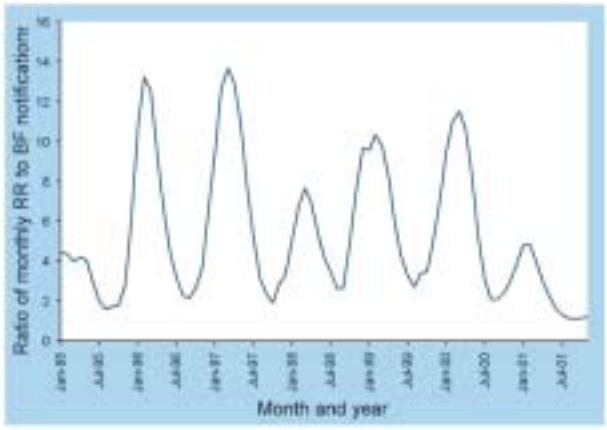
By contrast, the number of notifications for RR infection in 2001 was lower than in 2000 (n=3,219). While this represents a decrease of 25 per cent compared to the previous year, Figure 48 indicates that there is variation in reports over years. The overall rate for 2001 was 16.5 cases per 100,000 population. Most notifications came from Queensland (n=1,569) and the highest rate was observed in the Northern Territory (111.5 cases per 100,000 population). An outbreak of 18 cases of RR was reported in the Morgan Council area, which lies on the lower Murray River in South Australia.

Figure 48. Trends in notification rates of Barmah Forest virus infection and Ross River virus infection, Australia, 1995 to 2001, by year of onset



The co-occurrence of BF and RR infection between 1995 and 2001 by month of onset, measured as the ratio of RR to BF notifications, is shown in Figure 49. The seasonality of the curve possibly represents a more episodic characteristic of BF compared to RR. Further exploration of the epidemiology of the two viruses at smaller geographical areas and over longer periods will allow the interaction between them to be better understood.

Figure 49. Trends in ratio of Ross River virus infection to Barmah Forest virus infection notification, Australia, 1995 to 2001, by month of onset



Both BF and RR infections show similar age group and sex distributions (Figures 50 and 51). For both, there is a relative paucity of notifications in younger children and the early teenage years. The highest rates of notification were observed in 35–60 year old males and females, with the highest rates being about 10 cases per 100,000 population for infection with BF, and between 25 and 30 cases per 100,000 population for RR infection.

Figure 50. Notification rates of Barmah Forest virus infection, Australia, 2001, by age group and sex

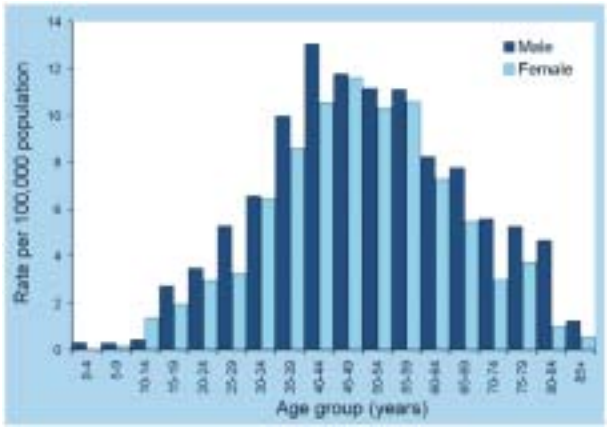
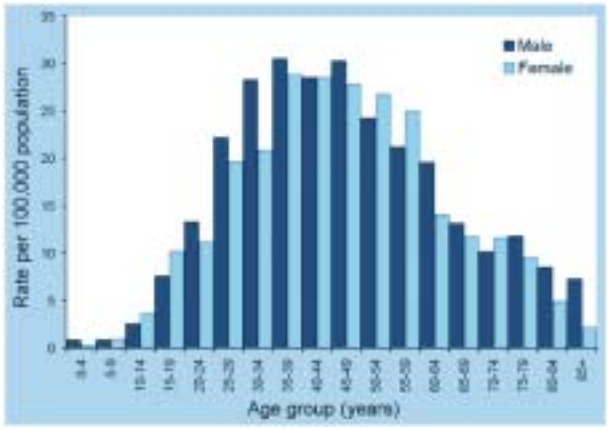


Figure 51. Notification rates of Ross River virus infection, Australia, 2001, by age group and sex



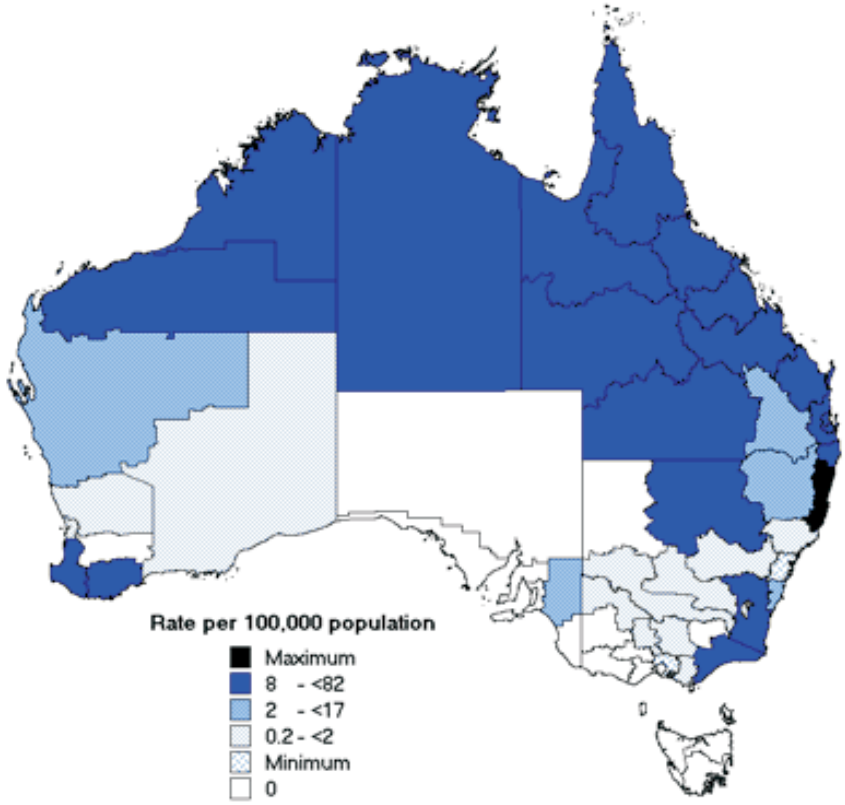
In 2001 infection with RR and BF were notified relatively equally in males and females. For BF infection, the male to female ratio was 1.2:1, and for RR infection the male to female ratio was 1.1:1.

Notification rates by Statistical Division are shown for the two diseases in Maps 8 and 9. The areas of highest rates of occurrence for BF infection were the New South Wales Mid-North Coast Statistical Division (82.2 case per 100,000 population) and the south-west areas of Queensland (44.6 cases per 100,000 population). For notifications of RR infection the highest rates occurred in north-west Queensland (186.4 cases per 100,000 population) and the Kimberley Statistical Division in Western Australia (159.0 cases per 100,000 population).

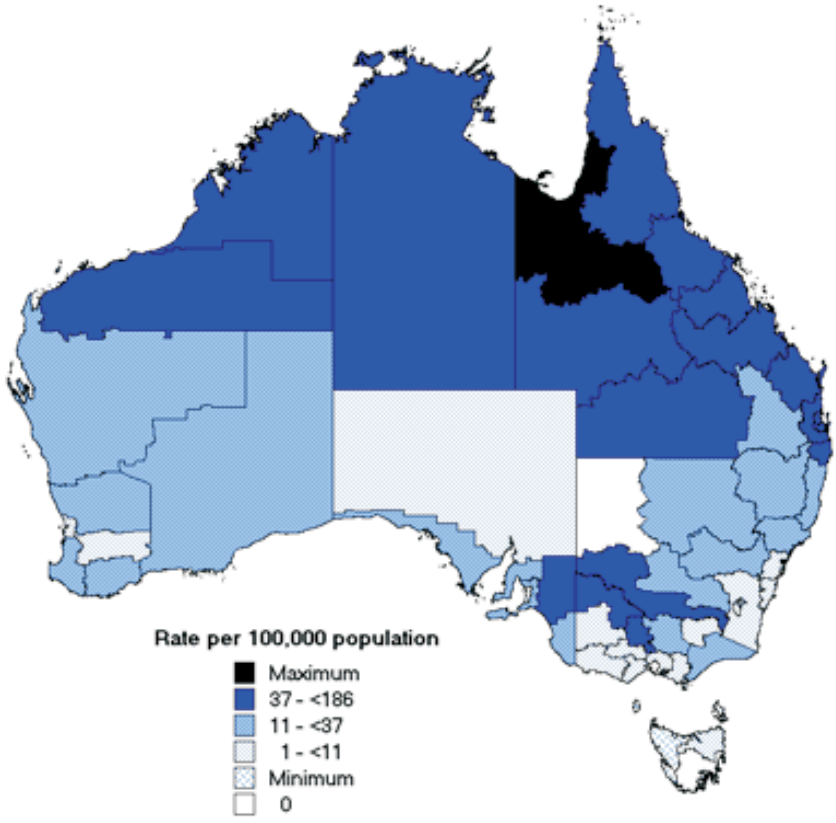
There were geographically isolated outbreaks of BF, in the south-west of Western Australia (the South West and Lower Great Southern Statistical Divisions), and outbreaks of RR along the Murray River region.

There is debate as to whether there is any overall increase in the activity of RR, and whether disease activity is expanding into areas where it has not occurred before.[68,69,70,71] Variations in notifications of RR can reflect the natural ecological variability of virus activity, while the availability of susceptible populations and human geographical and environmental change, also affect rates of RR in humans. At the local level, the numbers of infections have been shown to correlate with the abundance of various mosquito species.[72] Mosquito control programs may affect vector abundance and virus transmission to humans. Possible artefacts should be considered when assessing surveillance data. Increased awareness and testing for RR may account for increases in notifications. The change in reporting methods from clinical diagnosis to laboratory notification together with the adoption of standardised clinical and laboratory definitions may also have contributed to changes in surveillance data.

Map 8. Notification rates of Barmah Forest virus infection, Australia 2001, by Statistical Division of residence



Map 9. Notification rates of Ross River virus infection, Australia 2001, by Statistical Division of residence



Change in the rate of notification of BF may also be affected by surveillance artefacts or may be the result of changes in the epidemiology of this infection. A combination of unusual environmental conditions, together with a highly susceptible (that is, previously unexposed) human population, were the most probable factors contributing to the magnitude of the BF epidemic on the New South Wales south coast in 1995.[73] This outbreak is possibly reflected in the lower than usual ratio of RR to BF notifications for that year in Figure 49.

The extensive outbreak of RR in the Western Pacific in 1979–80 demonstrates the potential of the virus to spread throughout the region.[69] Serological surveys have shown the virus to exist in PNG, areas of Indonesia and in the Solomon Islands,[74,75] and the virus has recently been isolated for the first time from PNG.[76] A competent vector is generally required for transmission to humans. The introduction of *Aedes camptorhyncus* to New Zealand during 1998, highlights the risk to neighbouring countries.[77] During the late 1970s, at the time of the Pacific outbreak, three cases of RR infection were notified in New Zealand but all were acquired outside the country, and none have been notified since.

Murray Valley encephalitis and Kunjin

MVE is enzootic in the Kimberley region of Western Australia and the top two-thirds of the Northern Territory, where it is active principally in the wet season. The virus is epizootic in the Pilbara of Western Australia and the southern part of the Northern Territory, where it is associated with high summer rainfalls and flooding. Human cases occur sporadically in northern Queensland. Since 1974, however, nearly all cases of arboviral encephalitis due to MVE have been reported from Western Australia and the Northern Territory, with MVE activity (as indicated by seroconversion in chicken flocks) and human disease occurring in most years.[75,78] The major vector for the virus is *Cx. annulirostris*, and the main vertebrate hosts are water birds of the ardeid (heron) family.[75,78]

Although most cases are asymptomatic, infection with MVE can lead to serious illness or death. Symptoms include headache, neck stiffness, fever, tremor, weakness, confusion, fitting and sometimes coma.[79] The case fatality rate following symptomatic infection with MVE is 20 per cent and approximately 25 per cent of survivors will be left with significant residual neurological damage.[80] Infection with Kunjin virus generally causes milder disease,[79] but recent cases of Kunjin-associated encephalitis have been reported from Central Australia.[81,82] The number of MVE infections that are asymptomatic have been estimated to be between 500 and 1,500 for each one that is clinically identified.[75,78]

Infections with MVE and Kunjin virus have been separately notifiable since 2001. Prior to this, information on the location of cases and deaths was recorded in individual states and territories. A summary of these cases has previously been reported.[78] In addition, a summary of cases, and of the ecology and epidemiology of Kunjin virus, has been extensively reviewed because of its reclassification as a member of the West Nile virus lineage 1.[83]

In 2001, five cases of MVE and four cases of Kunjin virus infection were notified. Table 16 describes the eight cases that occurred in the first half of the year (corresponding to the 2000–01 arbovirus season). From January to June 2001, four notifications of MVE infection and four cases of infection with Kunjin virus were notified. In the second half of the year (the 2001–02 arbovirus season) only one case of MVE was notified. This was reported in the Northern Territory in July. The case was a two-year-old female. There were no cases of Kunjin notified in the second half of the year.

Table 16. Notifications of infection with Murray Valley encephalitis and Kunjin viruses, Australia, 2001

Notifying jurisdiction	Onset month	Gender	Age (yrs)	Follow-up
	Murray Valley encephalitis virus infection: Jan – June 2001			
Qld	Feb	M	3	Alive, severe neurological sequelae
NT	Feb	F	49	Died
SA	Feb	M	59	Alive
WA*	Mar	M	60	Unknown
	Kunjin virus infection: Jan – June 2001			
NSW	March	Unknown	58	Unknown
NT	March	M	11	Alive
WA	March	F	27	Unknown
NT	May	F	23	Alive

* Possibly acquired in Queensland or the Northern Territory

Although cases of MVE and Kunjin were both notified from Western Australia and the Northern Territory, there was some uncertainty as to where the infections were acquired as they occurred in tourists travelling through northern Australia. An additional case of MVE was diagnosed in Germany in a tourist returning from travel in Northern Australia.[84]

Viral infection of mosquitoes and seroconversion in sentinel animals may be used to monitor the potential for human infections with MVE and Kunjin virus. The locality of sentinel chicken flocks is shown in Map 10, and Figures 52a and 52b chart the number of seroconversions to MVE and Kunjin respectively during 2001. Although seroconversion counts for the different states and the Northern Territory are given

Figure 52a. Numbers of seroconversions to Murray Valley encephalitis virus in sentinel chickens, New South Wales, Northern Territory and Western Australia, 2001

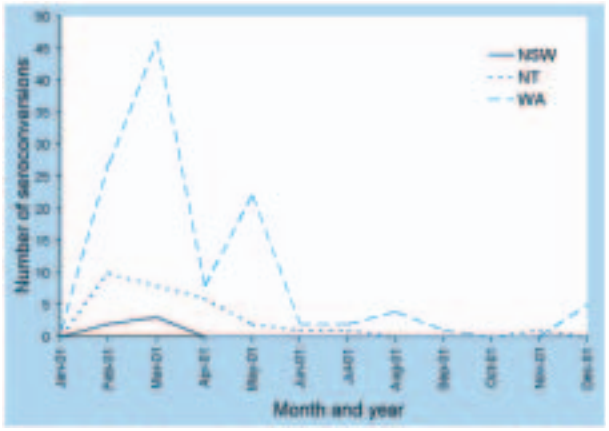
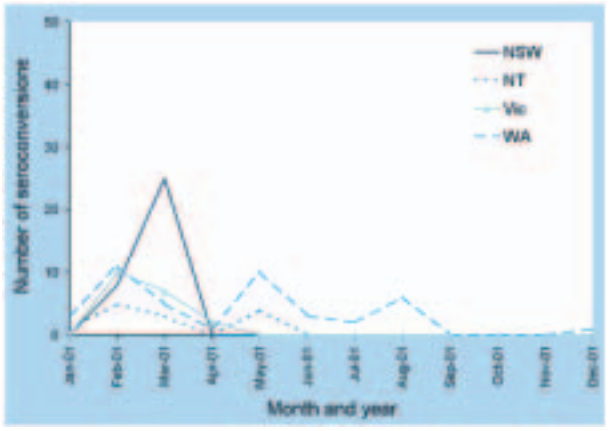


Figure 52b. Numbers of seroconversions to Kunjin virus in sentinel chickens, New South Wales, Northern Territory, Western Australia, and Victoria, 2001



Map 10. Geographical distribution of sentinel chicken flocks for the surveillance of arboviruses, Australia, 2001

they are not directly comparable as the number of flocks and birds within them vary across jurisdictions. Seroconversion in sentinel chicken flocks occurs mostly during the arbovirus season, but also throughout the year.

Surveillance of MVE and its control have been recently reviewed and an integrated system of surveillance based on sentinel chicken and mosquito monitoring was recommended.[78] As viral activity is demonstrated in these systems, public health control measures can then be implemented to prevent human infections.

Japanese encephalitis

Most infections with JE are asymptomatic. Between 1 in 30 and 1 in 300 infections result in clinical disease.[75] The fatality rate in symptomatic cases can be as high as 30 per cent, and neurological sequelae are reported in 50 per cent of survivors. A higher case-fatality rate is reported in the elderly, but serious sequelae (neurological) are more frequent in the very young.

Surveillance efforts in Australia have focussed on the detection of JE virus activity in Torres Strait and are based on the detection of virus carriage in the mosquito vector (*Cx. annulirostris*) and infection of domestic pigs, the amplifying host. In 1995 a localised outbreak of three cases occurred in the Torres Strait region.[85] Two additional sporadic cases were detected in 1998 at Badu Island and at Mitchell River, on the Gulf in Far North Queensland.[86] There were no notifications of JE in Australia in 2001. The absence of human infections in Australia since 1998 can be attributed to a human vaccination program and changes in pig husbandry following the earlier outbreaks.[86]

Feral pigs may be important in the ecology of JE in the Far North Queensland region. Birds, abundant during the wet season in tropical regions, may also play a role in virus transmission cycles. Native Australian macropods though, are thought to be unlikely hosts for JE.[87]

JE is a widespread and emerging disease in South-East Asia, and is probably now endemic in New Guinea.[75,88] The practice of rice-paddy farming throughout much of South-East Asia provides favourable conditions for the mosquito vector, and intensive pig husbandry provides an ideal reservoir species for the virus. These factors, combined with large and growing human populations, provide a fertile environment for the spread of the disease.

Despite an effective vaccine, outbreaks of the disease continue to occur and extend its known range. In Indonesia, the general absence of pig farming is associated with a relative lack of JE. The island of Bali is an exception because of its different religion and attitudes towards pork. The 1995 and 1996 cases of JE in the Torres Strait and West Papua marked a sudden unexplained range extension to the east. The isolation of the virus at three locations since then, and serological detection of more cases suggest that the virus has become endemic on the island of New Guinea.[88]

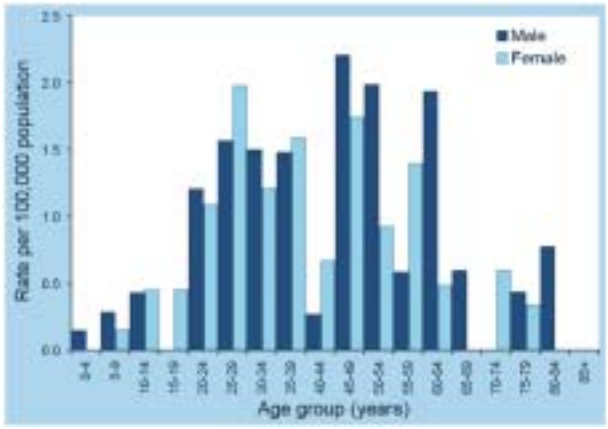
Dengue

Infection with any of four dengue viruses (serotypes 1– 4) is characterised by fever, muscle and joint pain, lymphadenopathy and rash. Secondary infection with a heterologous serotype of dengue virus can result in dengue shock syndrome or dengue haemorrhagic fever.[89,90]

Dengue was reintroduced to Australia in 1981 after an absence of more than 25 years.[91] Dengue virus activity occurs only in Far North Queensland, where the host mosquito *Aedes aegypti* has become established. Because of the absence of the mosquito vector elsewhere in Australia, dengue cases reported from other states and territories are infected either in Queensland or overseas.

During 2001, 176 notifications of dengue were received, corresponding to a national rate of 0.9 cases per 100,000 population. The largest number were from New South Wales (n=50, 0.8 cases per 100,000 population), and 43 notifications came from both the Northern Territory and Queensland (21.5 and 1.2 cases per 100,000 population respectively). Most notifications were for adults between the ages of 20 and 50 years, with a male to female ratio of 1.1:1. Rates according to age group and sex are shown in Figure 53. Relatively low numbers of notifications (n=176) most probably account for gaps in the age distribution.

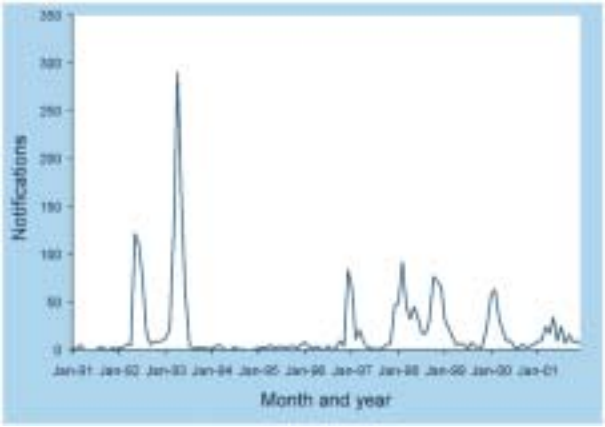
Figure 53. Notification rates of dengue, Australia, 2001, by age group and sex



In Queensland eight of the 43 cases notified were locally acquired, 21 were acquired overseas, while the place of infection was unknown for the remaining 14 cases. All of the remaining cases of dengue notified from other jurisdictions in 2001 were known to have been acquired overseas, except for three cases where the place of acquisition was not known. Information on dengue serotype was available for six cases notified from Victoria. Five were of serotype 1, and one was serotype 3.

The number of dengue notifications by month between 1991 and 2001 is shown in Figure 54. Two extensive outbreaks are represented in these data. The first was in Townsville and Charters Towers, in 1992–93, and the second in Cairns, Mossman and Port Douglas from 1997 to 1999. In these epidemics, 900 and 498 cases, respectively, were notified. The first outbreak was of dengue type 2, and the second outbreak was of dengue type 3. For 2001, the number of notifications are similar to the number notified in the preceding year (n=216), which included an outbreak of 49 cases in Cairns.

Figure 54. Trends in notifications of dengue, Australia, 1991 to 2001, by month of onset



The five-year *Dengue Fever Management Plan for North Queensland 2000–2005* aims to improve disease surveillance in humans, enhance mosquito control and surveillance, and to educate the community and professional groups on mosquito control and the prevention of infection (Queensland Government Health Department: Dengue fever for North Queensland, 2000–2005, http://www.health.qld.gov.au/phs/Documents/tphun/9168dmp.htm). The effectiveness of control measures when undertaken without delay was demonstrated in 2001, when an outbreak in Townsville was limited to only nine cases.[92]

Dengue affects large numbers of people throughout the Pacific region and remains a key public health concern. In 2001 the WHO reported 132,949 cases and 586 deaths from dengue in the WPR (www.wpro.who.int/public/regstatistics/reg_spec.asp). French Polynesia reported 30,000 cases of dengue serotype 1. The epidemic spread to other states in the Pacific, including Western Samoa, New Caledonia, American Samoa, Tokelau and the Cook Islands. New Zealand reported 60 imported dengue cases.

Factors contributing to increases in global dengue incidence include the creation of mosquito breeding sites by poorly planned urbanisation, lapses in mosquito eradication programs in the Americas and large-scale movements of people and cargo around the world. In the 1950s an annual average of 900 cases of dengue haemorrhagic fever were reported worldwide. This increased to over 514,000 cases during the 1990s, and in 1998 1.2 million cases of dengue and dengue haemorrhagic fever infections were reported. Modelling suggests that over 51 million infections in total occur each year.[93]

Arbovirus — not elsewhere classified

Thirty-six notifications were categorised as 'Arbovirus — not elsewhere classified' in 2001. New South Wales and Victoria reported 15 and 16 notifications respectively. Cases may include unspecified flavivirus infections (where serology is inconclusive and Kunjin or MVE cannot be distinguished) or infections caused by other arboviruses.

Malaria

Indigenous transmission of malaria in Australia occurs infrequently. Australia has maintained its WHO malaria-free status since 1983, despite the continued presence of competent *Anopheles* vectors, principally *An. farauti*. This mosquito is found in coastal regions of the Northern Territory and Queensland north of approximately the 17 or 18 degree south of latitude.

All 705 malaria cases reported to NNDSS in 2001 were people who had returned to Australia from malaria endemic regions (Table 17). Most notifications were reported from Queensland (n=300), New South Wales (n=153) and Victoria (n=88). The incidence of malaria has been stable over the last decade, with about 700 notifications occurring each year, giving an annual rate ranging between three and five cases per 100,000 population (Figure 55). Most reported cases were in the 20–24 and 25–29 year age groups. Overall, the male to female ratio was 2.4:1. Rates according to age group and sex are shown in Figure 56.

The species of *Plasmodium* the causing of infection was identified for 274 notifications (38%) (Table 18). Of these most were *P. vivax* (67%) and *P. falciparum* (28%).

Table 17. Notifications of malaria, Australia, 2001, by country of infection

Country of infection	State or territory notifying* (% of total)						
	ACT	NSW	NT	Qld	SA	Tas	Vic
PNG	63.1	42.2	19.3	24.7	19.0	11.1	40.2
Solomon Islands	5.3	3.5	1.8	0.0	3.0	0.0	0.0
Vanuatu	5.3	1.7	0.0	0.0	0.0	0.0	0.0
Indonesia	5.3	8.1	33.3	3.0	6.0	11.1	8.1
Vietnam	0.0	0.6	0.0	0.3	0.0	0.0	0.0
East Timor	0.0	13.9	43.9	3.7	3.0	0.0	14.9
India/Pakistan	10.5	9.8	1.7	0.0	9.0	0.0	0.0
Afghanistan	0.0	0.6	0.0	0.0	0.0	11.1	0.0
Africa	10.5	17.3	0.0	0.7	39.0	55.6	13.8
South America	0.0	0.0	0.0	0.3	0.0	0.0	2.3
Other	0.0	2.3	0.0	0.0	9.0	0.0	20.7
Unknown	0.0	0.0	0.0	67.3	12.0	11.1	0.0

* Data unavailable from Western Australia

Figure 55. Trends in notification rates of malaria, Australia, 1991 to 2001, by year of onset

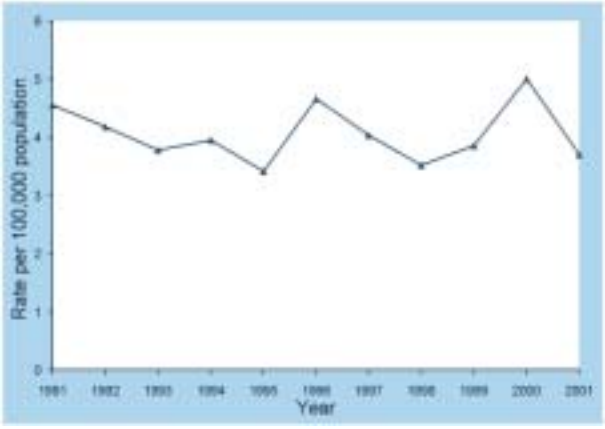


Figure 56. Notification rates of malaria, Australia, 2001, by age group and sex

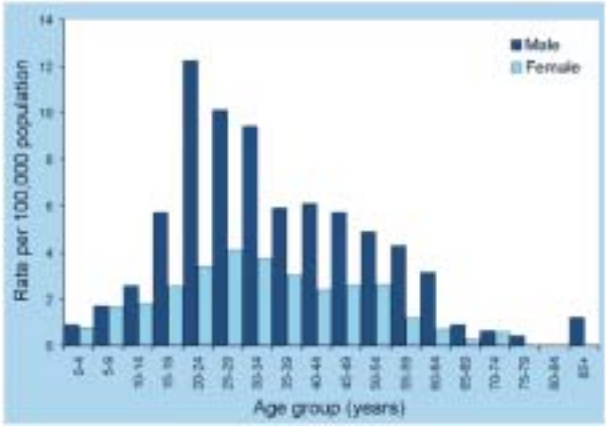


Table 18. Notifications of malaria, Australia, 2001, by *Plasmodium* species

Plasmodium sp.	Number of notifications by species	Per cent species where known
P. vivax	184	67
P. falciparum	76	28
P. vivax AND *falciparum*	2	1
P. malariae	8	3
P. ovale	4	1
Unknown	431	
Total	705	

A large number of malaria infections in Australians have occurred in defence personnel during their peace-keeping operations overseas (LtCdr P. Corrigan, personal communication). The number of malaria cases reported by area of operation (East Timor, Bougainville, Papua New Guinea and 'other') and against *Plasmodium* species by year is shown in Table 19.

These data represent clinical occurrences of malaria, and so include relapses of previously acquired infections. Cases are diagnosed either in the area of operation, or on return to Australia. Infections with *P. falciparum* are commonly diagnosed in East Timor, whereas diagnoses of *P. vivax* are more commonly detected on return to Australia. It should be noted that infections with *P. falciparum* can mask dual infection with *P. vivax*. After treatment for the *P. falciparum* infection, the *P. vivax* infection can then become patent.

Table 19. Number of malaria cases reported to the Army Malaria Institute, 1998 to 2001, by area of operation and *Plasmodium* species*

Year	East Timor					Bougainville/Papua New Guinea					Other				Total
	Pf	Pv	Pf/ Pv	Po	Unk	Pf	Pv	Pf/ Pv	Po	Unk	Pf	Pv	Po	Unk	
1998	0	0	0	0	0	0	9	0	0	0	0	2	0	0	11
1999	23	7	2	0	0	3	22	1	0	0	0	2	1	0	61
2000	26	290	6	1	14	3	27	1	1	1	2	23	0	1	396
2001	1	46	0	0	2	3	5	0	0	2	0	11	0	2	72

* Pf – *Plasmodium falciparum*; Pv – *P. vivax*; Pf/Pv – infected with both; Po – *P. ovale,* Unk — unknown

The endemicity of malaria in South-East Asia and the Pacific and the presence of competent mosquito vectors in Australia underscores the potential for the re-introduction of malaria to Australia.

Zoonoses

The list of zoonoses (diseases transmitted to humans from animals that are the primary host) which are notified to NNDSS was modified in 2001. Hydatid disease was no longer notifiable and three new diseases (anthrax, Australian bat lyssavirus and other lyssaviruses) were made notifiable. Anthrax has been added to the list because of its potential for use as an agent of bioterrorism. The Australian bat lyssavirus came to attention after a human became infected and died after handling a fruit bat in 1996. The other notifiable zoonotic diseases are brucellosis, leptospirosis, ornithosis and Q fever.

Altogether, 1,091 notifications of zoonoses were received. This number accounted for one per cent of the total of all notifications for all diseases during 2001.

Brucellosis

Brucellosis in humans is caused by four species of *Brucella* bacteria, found in four different hosts — *B. melitensis* (sheep/goats), *B. abortus* (cattle), *B. suis* (pigs) and *B. canis* (dogs). Infection occurs principally from exposure through breaks in the skin to the fluid or tissues of infected animals, or from the ingestion of unpasteurised goat or sheep's milk and cheese (most often in visitors from overseas). The disease is characterised by fever, headache, arthralgia, depression and weight-loss.

In Australia during 2001, 19 cases (0.1 cases per 100,000 population) of human brucellosis were notified to the NNDSS. Most of these (n=17) were from Queensland (0.5 cases per 100,000 population), with one each from South Australian and Victoria. The 19 notifications for brucellosis in 2001 are the second lowest since 1991 (Figure 57).

Of the 19 notifications of brucellosis, 17 were in adult males (age range 17–79 years) and the highest notification rates were observed in the 20–25 year age-group(n=4, 0.6 cases per 100,000 population). The two female cases were aged 40 and over 70 years. The male to female ratio was 8.5:1.

Bovine brucellosis was eradicated from Australia in 1989,[94] and the notifications of human disease occurring now are due to infections from the other species. The feral pig population in northern Queensland, estimated to be more than several million (McGaw and Mitchell, referred to in Williams *et. al.*),[95] has been identified as a primary reservoir of brucellosis.[95] Of the 17 notifications from Queensland, only two were typed to species level, both *B. suis*.

Figure 57. Trends in notifications of brucellosis, Australia, 1991 to 2001, by year of onset

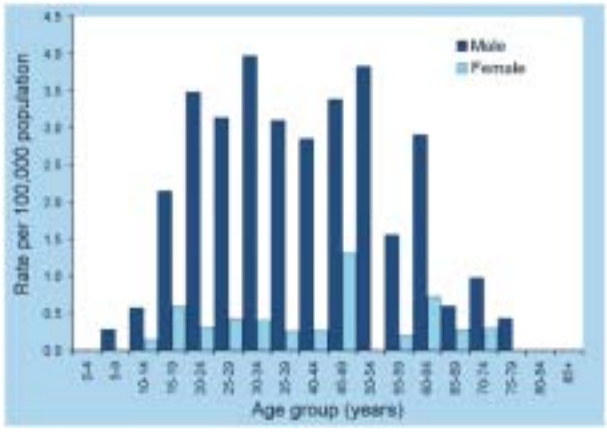
Leptospirosis

Leptospirosis is caused by infection with the *Leptospira* spirochaete. Infection in humans occurs from exposure, through mucosal surfaces or breaks in the skin, to soils or fluids (bodies of water and animal urine) contaminated with the organism. Rats are a common reservoir of infection, and their presence in the cane fields in Australia is a source of exposure to cane growers. Fever, headache, lower limb myalgia and conjunctival suffusion are typical symptoms. There is a wide range of disease severity, from sub-clinical to death from hepatorenal failure. Increased disease severity is associated with increased age and with certain leptospiral serovars. Clinical disease is more likely to be milder in areas of endemic infection.[16]

There were 245 notifications of leptospirosis in Australia during 2001 (1.3 cases per 100,000 population). Notifications were received from all states and territories except the Australian Capital Territory. Most cases occurred in Queensland (n=129) and New South Wales (n=65). The highest rate was observed in Queensland (3.5 cases per 100,000 population). By Statistical Division, the areas with the highest rates were the Central West of Queensland (32.0 cases per 100,000 population) and Far North Queensland (31.4 cases per 100,000 population) (Map 11).

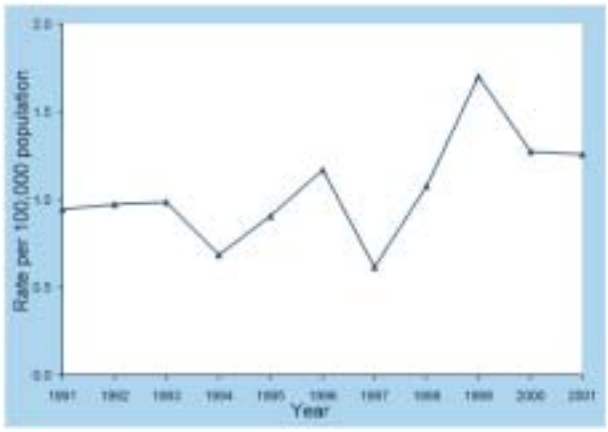
Reflective of a strong occupational association with the stock and horticultural (bananas, sugarcane) industries, males aged 15–64 years accounted for 87 per cent of all notifications. The peak rate of 4.0 notifications per 100,000 population occurred in males in the 30–34 year age group (Figure 58).The male to female ratio was 6.7:1.

Figure 58. Notification rates of leptospirosis, Australia, 2001, by age group and sex



Trends in the notification rate for leptospirosis are shown in Figure 59. A peak in notification rates was observed in 1999, when 184 cases were notified. This has been attributed to prolonged rainfall in northern Queensland, with a concomitant increase in rodent populations.[96] It is possible, however, that reporting artefacts, such as increased awareness, underlie these changes.

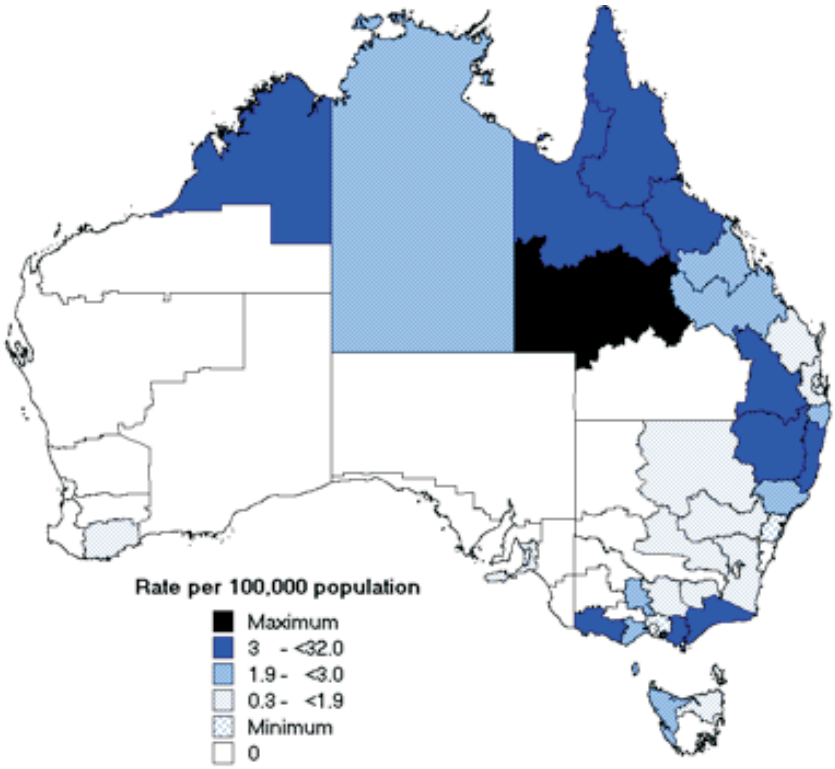
Figure 59. Trends in notification rates of leptospirosis, Australia, 1991 to 2001, by year of onset



The 2001 annual report of the WHO Leptospirosis Reference Laboratory further describes the laboratory characteristics of leptospirosis isolates in Australia (www.health.qld.gov.au/qhpss/qhss/lepto_jandec_2001.pdf). Seventeen serovars were identified in human infections in 2001, and the *Leptospira interogans* serovar *hardjo* was the most commonly identified, in 38 per cent of infections. This serovar is most associated with the cattle and dairy industry, whereas *Leptospira interogans* serovar *australis*, identified in 12 per cent of cases, occurs more in horticultural settings. Since 1992, the number of notifications of *Leptospira interogans* serovar *hardjo* has increased tenfold.

Infection with leptospirosis is a public health concern in the tropical WPR. New Caledonia for example, reported 180 cases per 100,000 population — a rate over 100 times greater than that observed in Australia.[97] Leptospirosis has also been identified as an emerging infectious disease, because of changes in animal husbandry, climate and human behaviour.[98]

Map 11. Notification rates of leptospirosis, Australia, 2001, by Statistical Division of residence



Ornithosis

Ornithosis is an infection caused by the intracellular organism *Chlamydia psittaci*. Symptoms of infection include fever, headache, rash and respiratory tract infections and, especially among older people, atypical pneumonia. *C. psittaci* often infects birds but because of its more particular association with parrots the human disease is also known as psittacosis. As well as directly from birds, the disease can be transmitted to humans through bird detritus (e.g., feathers and dust) and droppings. As infections in birds are commonly asymptomatic, it is prudent to avoid these materials.

During 2001, 131 notifications of ornithosis were reported in Australia. The largest number were from Victoria (n=68) and New South Wales (n=37). The highest rate occurred in Victoria with 1.4 cases per 100,000 population. Notification in Queensland commenced from July 2001. Nationally the rate was 0.7 cases per 100,000 population. The trends in the annual national notification rate between 1991 and 2001 ranged from approximately 0.5 to 1.5 cases per 100,000 population. The peaks in notification rates may reflect particular outbreaks (Figure 60).

In 2001, males and females were equally affected by ornithosis (male: female ratio 1.1:1). The highest notification rates of ornithosis were in males in the 75–79 years age group and in females in the 55–59 years age group (4.4 and 3.0 cases per 100,000 population respectively, Figure 61).

Figure 60. Trends in notification rates of ornithosis, Australia, 1991 to 2001, by year of onset

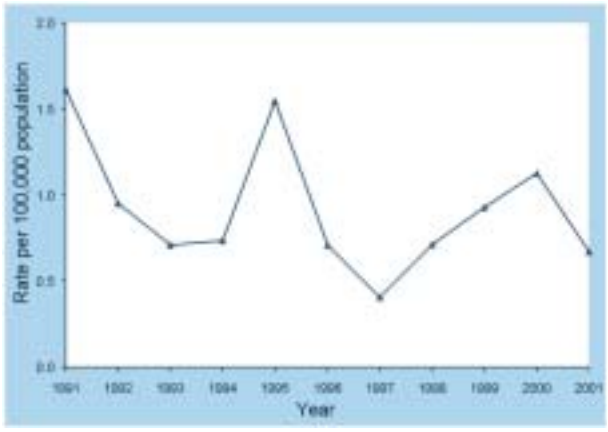
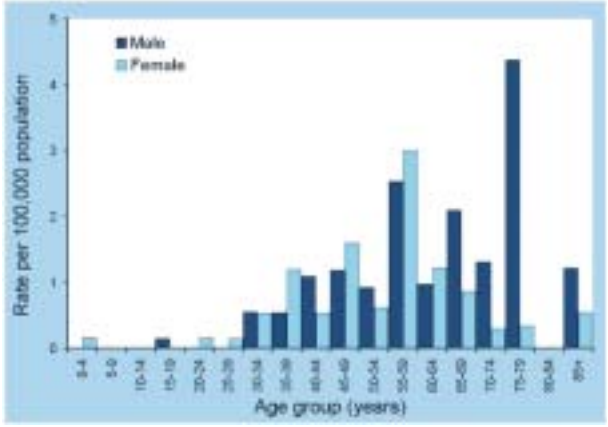


Figure 61. Notification rates of ornithosis, Australia, 2001, by age group and sex



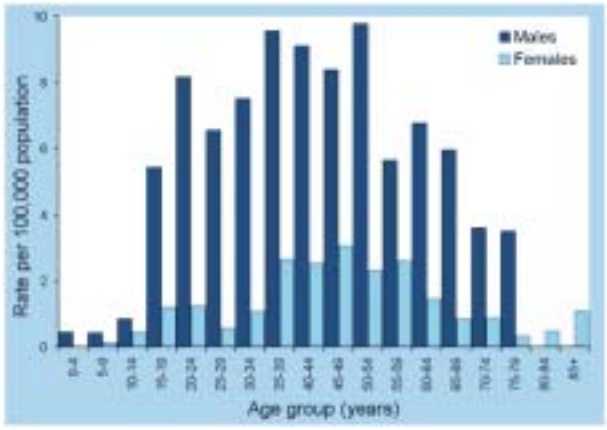
Q fever

Q fever is caused by infection with the rickettsia *Coxiella burnetii*. The disease is particularly associated with workers in the livestock industry. The organism is extremely infectious, and the tissues and fluids of infected animals are sources of infection. The dusts around stock facilities are also sources of infection because the organism is resistant to desiccation.

In 2001, 698 cases of Q fever were notified to NNDSS, a rate of 3.6 cases per 100,000 population. Most cases were from Queensland (n=454, 12.5 cases per 100,000 population). In Victoria, an outbreak of 18 cases occurred in Wodonga between early April and July.

The groups with the highest notification rates were 50–54 year old males (9.6 cases per 100,000 male population) and 45–49 year old females (3.0 cases per 100,000 female population) (Figure 62). The overall male to female ratio was 4.1:1.

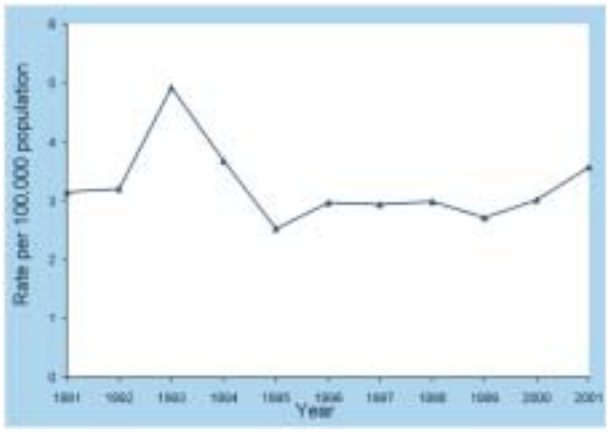
Figure 62. Notification rates of Q fever, Australia, 2001, by age group and sex



Sixteen cases of Q fever in children (aged less than 15 years) were notified from New South Wales (n=4) and Queensland (n=12). Information on exposure was available for 10 cases, all of who lived on or visited farms. Seven cases were linked to other cases of Q fever in family members, neighbours or other contacts.

The rate of Q fever notifications in Australia in recent years has remained relatively consistent since 1995 (Figure 63). Recent increases may be due to increased testing as a result of the Q fever vaccination program.

Figure 63. Trends in notification rates of Q fever, Australia, 1991 to 2000, by year of onset



A Commonwealth program to reduce the occurrence of Q fever commenced in October 2000. In the first phase abattoir workers and shearers were provided with free skin testing and Q fever vaccination. Under this program, Q fever-related medical costs were underwritten by the Commonwealth. The Q fever register has been established to provide a record of the vaccination status of abattoir workers (www.qfever.org). The second phase commenced in October 2001, and is directed at beef, sheep and dairy workers.

Australian bat lyssavirus and lyssavirus (unspecified)

The death of two Queensland women after handling fruit bats in 1996 and 1998 led to the discovery of the Australian bat lyssavirus (ABL). ABL and lyssavirus (unspecified) are closely related to the rabies virus. During 2001 there were no notifications of Australian bat lyssavirus or lyssavirus (unspecified) in Australia. African and European lyssaviruses may infect travellers and thus are included in lyssavirus (unspecified) on the list of notifiable diseases.

The symptoms of lyssavirus infection are similar to those of rabies, and are sometimes indistinguishable. Onset of symptoms occurs weeks after infection (this can be longer again in pre-pubertal individuals). Early symptoms are headache, fever and malaise, and, indicative of neural involvement, a sense of apprehension, and indefinite sensory changes. Following this, excitability, paresis or paralysis, a fear of water, delirium and convulsions then occur. By this stage the disease is inevitably fatal.[16,89]

Between 1996 and 1999, 205 people reported being bitten or scratched by bats in Queensland.[99] The rate of ABL seropositivity in bats involved in human exposures or in sick, injured or orphaned bats was approximately 5.5 per cent .

Anthrax

The bacterium causing anthrax, *Bacillus anthracis*, occurs in many parts of the world, most commonly as spores in the soil. When soil becomes moist and warm, spores enter a reproductive stage and quickly multiply. Animal hosts such as herbivores (sheep, goats, cows) can then become infected, and if not treated, die. Following the animal's death spores are released, which can lie dormant in the soil for decades. Vaccination protects livestock from becoming infected, and also provides protection from infection in humans.

There were no cases of human anthrax infection in Australia in 2001. Human anthrax occurs in three forms (cutaneous, gastrointestinal and pulmonary), depending on the route of infection.[89] Cutaneous anthrax occurs in handlers of animals or animal hides (tannery workers were once a high risk occupational group). The skin lesion is quite characteristic. With treatment the mortality rate is usually less than one per cent. Gastrointestinal anthrax results from the consumption of infected meat. The primary site of infection may be the lower gut or throat. This form has been effectively eliminated in countries where the butchering of livestock is regulated. The non-specific nature of the early symptoms (nausea, vomiting and fever) prevents early diagnosis, and the mortality rates can be substantial.

Pulmonary anthrax results from inhalation of the anthrax spores into the lungs. A two-phase illness results. The first phase produces only mild symptoms, which again are non-specific (e.g., fatigue, myalgia, mild fever), and last for 2–4 days. The second phase of illness is characterised by severe respiratory distress with a sudden onset. The highest rate of mortality from anthrax infection occurs with pulmonary anthrax. In the past, workers in textile mills inhaling fine dusts were often affected by anthrax (wool-sorters disease). Improvements in industrial hygiene and the use of synthetic fabrics have since resulted in fewer cases of pulmonary anthrax being recorded.[89]

In October 2001, 22 people in the United States of America became infected with anthrax. The source of infection was believed to be letters containing anthrax spores. Eleven cases were of the cutaneous form, and the other 11 were inhalational anthrax. Five deaths resulted.[1] Shortly afterwards in Australia and other countries there were many white powder incidents. Over 400 samples were tested for the presence of anthrax in New South Wales. None contained anthrax spores.[100]

Human anthrax infections arising from natural (i.e., soil-borne) sources in Australia have been reported between 1917 and 1990. Infections occurred at very low rates of about 0.8 to 1.0 cases per 100,000 population, and were associated with occupational exposure.[101]

Some areas in Australia are well known for sporadic anthrax infection in livestock. Most cases in livestock in Australia occur in a band running north-south in central New South Wales and into northern Victoria. The occurrence of cases has been correlated with drier periods in summer following wet or humid weather (www.brs.gov.au/usr-bin/aphb/ahsq?Disease=ATX).

Other bacterial infections

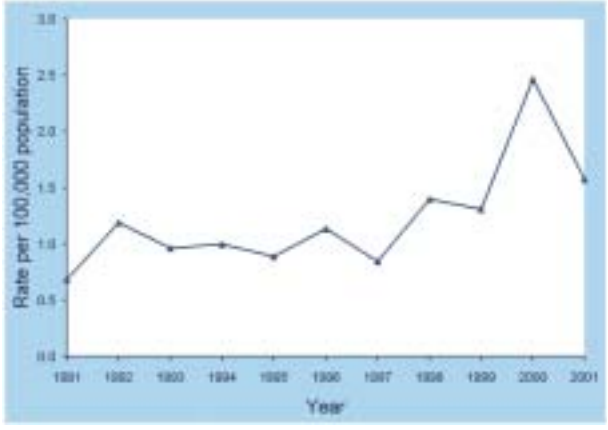
Legionellosis, leprosy, invasive meningococcal infection and tuberculosis (TB) were notifiable in all states and territories in 2001 and classified as 'other bacterial infections' in NNDSS. A total of 1,978 notifications were classified in this group in 2001, which accounted for 1.9 per cent of all the notifications to NNDSS.

Legionellosis

Legionellosis is an acute infection caused by various species of *Legionella* bacteria with two clinical manifestations: Legionnaires' disease and Pontiac fever. Legionnaires' disease, caused commonly by *Legionella pneumophila*, is a severe form of pneumonia, which may be accompanied by involvement of other organs such as the brain, the bowel and the kidneys. Symptoms include a rapid onset of high fever, a non-productive cough, chills, headache and general malaise. Diagnosis is based on isolating and identifying *Legionella pneumophila* from the patient's respiratory secretions or blood. The incubation period is usually 2–10 days. Less than five per cent of exposed persons become ill, but up to 30 per cent of those who become ill may die, depending on the population.[16]

Legionellosis is notifiable in all the states and territories of Australia, and includes notifications of infections caused by all *Legionella* species. The annual rates since 1991 show a marked increase in notifications in 2000 (Figure 64), because of the Melbourne aquarium outbreak.[102] This was followed

Figure 64. Trends in notification rates of legionellosis, Australia, 1991 to 2001, by year of onset



in 2001 by a decrease in notifications, but the number was still greater than those in years prior to 2000. A recent analysis of national legionellosis notification data showed a significant increase between 1991 and 2000, even when outbreak cases were excluded.[103]

There were 307 notifications of legionellosis reported in 2001, giving a national rate of 1.6 cases per 100,000 population. The highest rates of legionellosis were reported in Victoria (2.5 cases per 100,000 population) and Western Australia (2.2 cases per 100,000 population). Legionellosis notifications showed a peak in reports in autumn and spring.

Men accounted for 209 of the 307 (68 %) cases of legionellosis in 2001, to give a male to female ratio of 2.1:1. The highest rates were in the 80–84 year age group for men (10.1 cases per 100,000 population) and the 85 year and over age group for women (4.3 per 100,000 population, Figure 65).

Data on the causative species was available for 286 (93%) of the legionellosis cases. Of these, 152 (52%) were identified as *L. longbeachae,* 131 (46%) as *L. pneumophilia*, and three (1%) as other species (Table 20). In 2001 the proportion of *L. pneumophila* causing legionellosis cases was significantly higher in Victoria (109/121, 90%) than in the rest of the Australia (22/186, 12%, chi-square =180, p<0.0001).

There were 12 deaths identified as due to legionellosis in Australia in 2001, giving a case fatality rate of 3.9 per cent. The breakdown of deaths by state and territory and infecting *Legionella* species is shown in Table 21. The case fatality rate for infections with *L. pneumophila* (11/131, 8.4%) was significantly higher than the case fatality rate for *L. longbeachae* infections (1/152, 0.6%, chi-square=8.5, p<0.005).

Table 20. Notifications of legionellosis, Australia, 2001, by species and state or territory

State or territory	Species of *Legionella*				
	L. longbeachae	*L. pneumophila*	Other species*	Unknown	Total
ACT	2	0	0	0	**2**
NSW	63	0	0	4	**67**
NT	3	0	0	0	**3**
Qld	17	15	0	5	37
SA	29	3	0	0	32
Tas	1	1	0	1	3
Vic	6	109	3	3	121
WA	31	3	0	8	42
Total	152	131	3	21	307

* Other includes *L. micdadei* and *L. bozemannii*.

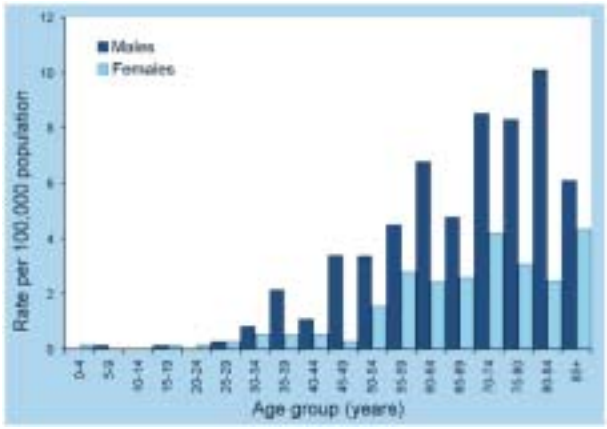
Table 21. Deaths due to legionellosis, Australia, 2001, by species and state or territory

State or territory*	Species of *Legionella*				
	L. longbeachae	*L. pneumophila*	Other species†	Unknown	Total
ACT	0	0	0	0	0
NT	0	0	0	0	0
Qld	0	1	0	0	1
SA	1	1	0	0	2
Tas	0	0	0	0	0
Vic	0	8	0	0	8
WA	0	1	0	0	1
Total	1	11	0	0	12

* No data available for New South Wales.

† Other includes *L. micdadei* and *L. Bozemannii*.

Figure 65. Notification rates of legionellosis, Australia, 2001, by age group and sex



Leprosy

Leprosy is a chronic infection of the skin and peripheral nerves with *Mycobacterium leprae*. Despite elimination from most countries, the disease remains a major endemic public health problem in six countries. One of these, India, accounts for 64 per cent of prevalent infections and 78 per cent of incident cases worldwide.[104] In Australia, leprosy is a rare disease, with the majority of cases occurring in migrants from leprosy-endemic countries or Indigenous communities.

In 2001, five leprosy cases were notified, compared with four in 2000. Three of the five cases occurred in New South Wales and two in Western Australia. Of these, four were male and one female, and the age range was 18–48 years. The country of birth was known for all five cases, and only one was born overseas, in Vietnam. Among the four Australian-born cases, three were identified as Indigenous Australians (two from Western Australia; one from New South Wales), while the Indigenous status of the fourth was not stated.

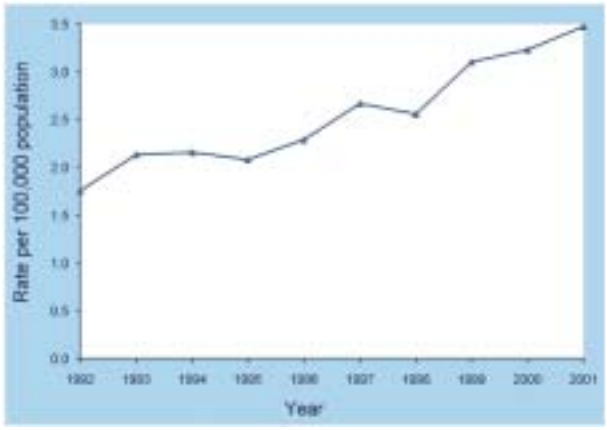
Invasive meningococcal disease

The meningococcus (*Neisseria meningitidis*) is an asymptomatic nasopharyngeal colonising organism found in 25 to 50 per cent of the general population. Invasive infection, however, can cause severe clinical meningitis, which has a high fatality rate.[105] Children, adolescents and the elderly are most at risk of this form of infection.

Meningococcal serogroups A, B, C, Y and W-135 are the main human pathogens. In Australia, serogroups B and C are the major causes of invasive meningococcal disease. WHO estimated that, internationally, there are at least 500,000 cases of invasive meningococcal disease and 50,000 deaths from the disease every year.[106]

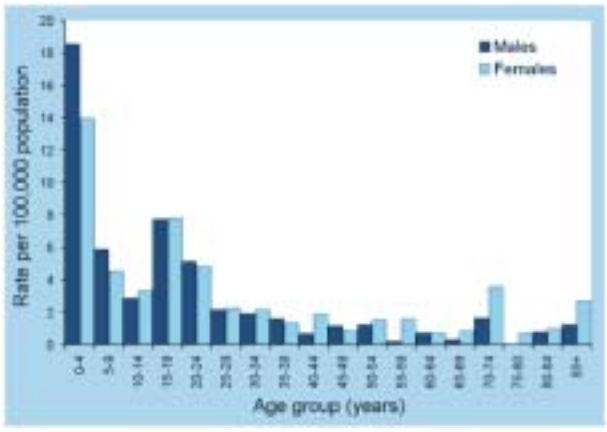
The annual notification rate for meningococcal disease has been increasing in Australia over the past 10 years (Figure 66). In 2001, there were 677 notifications giving a national notification rate of 3.5 cases per 100,000 population, a slight increase from the 622 cases and rate of 3.2 cases per 100,000 population reported in 2000.

Figure 66. Trends in notification rates of invasive meningococcal infection, Australia, 1992 to 2001, by year of onset



The largest number of cases in 2001 occurred in late winter (August, n=93) and early spring (September, n=81). The highest age specific rate was in children in the 0–4 year age group (16.3 cases per 100,000 population) and in the 15–19 year age group (7.8 cases per 100,000 population). Rates according to age group and sex are shown in Figure 67. More cases occurred among male children aged less than five years, and the male to female ratio was 2.6:1.

Figure 67. Notification rates of invasive meningococcal infection, Australia, 2001, by age group and sex



Among 677 meningococcal cases, 452 (67%) had the serogroup identified. Of these, serogroup B occurred in 283 (63%), 155 (34%) were serogroup C and 14 (3%) were serogroup W135 or Y (Table 22).

In 2001 there were 43 deaths due to meningococcal disease giving a crude case fatality rate of 6.3 per cent. The breakdown of deaths by state and territory and serogroup are shown in Table 23. Meningococcal serogroup C disease was associated with a significantly higher case fatality rate (23/155, 14.8%) than serogroup B disease (16/283, 5.6%, chi-square=9.3, $p<0.005$).

There were a number of linked cases of meningococcal disease in 2001 in all jurisdictions except the Northern Territory, where all cases were sporadic. A pair of linked cases in Queensland prompted the vaccination and chemoprophylaxis of more than 2,000 contacts. In Western Australia, four cases of serogroup B invasive meningococcal disease occurred over a period of 10 months in an Aboriginal community in an outer metropolitan area. These cases occurred in November 2000, and February, late July and early August 2001, in children aged between two and nine years. On each occasion, appropriate public health actions, including contact tracing and provision of advice and chemoprophylaxis to identified household and other close contacts, were performed. Mass chemoprophylaxis was provided to all residents of the community and regular visitors to it. There have been no further cases following these interventions.

A large outbreak of 14 cases of serogroup C (phenotype 2a:P1.5,2) occurred in southern Tasmania. In response, antibiotic prophylaxis was offered to contacts of cases and intensified surveillance, through laboratory and clinical services, was carried out. Regular press conferences were also held, in which the importance of early detection and empirical treatment were emphasised. General practitioners, pharmacists and schools were also alerted more directly to relevant disease information. A further two cases of serogroup C occurred in northern Tasmania, but these were shown to be due to a different phenotype.

Table 22. Notifications of invasive meningococcal infection by serogroups, 2001, by state or territory

Jurisdiction	Meningococcal serotype				Total
	Serogroup B	Serogroup C	Serogroups A, Y or W	Unknown serogroup	
ACT	2	0	0	4	6
NSW	85	35	4	106	230
NT	10	2	0	1	13
Qld	67	32	6	22	127
SA	22	7	1	9	39
Tas	5	17	0	1	23
Vic	47	56	2	58	163
WA	45	6	1	24	76
Total	283	155	14	225	677

Table 23. Deaths due to invasive meningococcal infection by serogroups, 2001, by state or territory

Jurisdiction	Meningococcal serotype				Total
	Serogroup B	Serogroup C	Serogroup A, Y or W	Unknown serogroup	
ACT	0	0	0	0	0
NSW	2	5	0	0	7
NT	1	1	0	0	2
Qld	4	5	0	2	11
SA	2	1	0	0	3
Tas	1	4	0	0	5
Vic	2	7	0	2	11
WA	4	0	0	0	4
Total	16	23	0	4	43

The Australian Meningococcal Surveillance Programme was established in 1994 for the purpose of monitoring drug resistance in *Neisseria meningitidis* isolates causing 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 quantitatively determine the susceptibility of *N. meningitidis* to a core group of antibiotics. The results of the surveillance in 2001 have recently been published.[107] In 2001 about two-thirds of all the isolates showed decreased susceptibility to the penicillin group of antibiotics (MIC 0.06-0.5 mg/L), but all isolates tested were susceptible to third generation cephalosporins.

Tuberculosis

TB is an infectious disease caused by *Mycobacterium tuberculosis*. The disease commonly affects the lungs and is usually transmitted person-to-person by airborne droplets.

Australia has one of the lowest rates of TB in the world, with most cases occurring in overseas-born and Indigenous Australians. The Federal Minister for Health and Ageing recently launched *The National Strategic Plan for Tuberculosis Control in Australia Beyond 2000*, prepared by the National Tuberculosis Advisory Committee. The plan consists of three key elements: case finding; treatment; and surveillance. Performance indicators have been developed to allow a regular review of the progress of the Strategic Plan.

In 2001, 989 TB notifications were received by NNDSS, a rate of 5.1 cases per 100,000 population. The notification rates of TB were lower than the national average in the Australian Capital Territory, Queensland, South Australia, Tasmania and Western Australia. The highest rate was reported in the Northern Territory (17.5 cases per 100,000 population).

In 2001, the male to female ratio was equal as it has been in the previous years. TB cases occurred in all age groups, with the highest age-specific rates reported in the 85 years and over age group for both males (29.3 cases per 100,000 population) and females (11.4 cases per 100,000 population) (Figure 68). Detailed analyses of TB in Australia have recently been published.[108]

Figure 68. Notification rates of tuberculosis Australia, 2001, by age group and sex

Other communicable disease surveillance

Laboratory Virology and Serology Reporting Scheme

LabVISE is a passive surveillance scheme based on voluntary reports of infectious agents from virology and serology laboratories around Australia. In 2001, reports from the scheme were analysed and published quarterly in *Communicable Diseases Intelligence.* LabVISE provides information on a number of viruses and other infectious agents (bacteria, parasites and fungi) and the demographic characteristics of those infected. The scheme monitors some infectious agents that are not reported by other surveillance systems. Interpretation of LabVISE data is limited by uncertainties about the representativeness of the data, the lack of denominator data to calculate rates, and variable reporting coverage over time. In addition, there are no consistent case definitions currently in use. Data from LabVISE between 1991 and 2000 were recently analysed.[109]

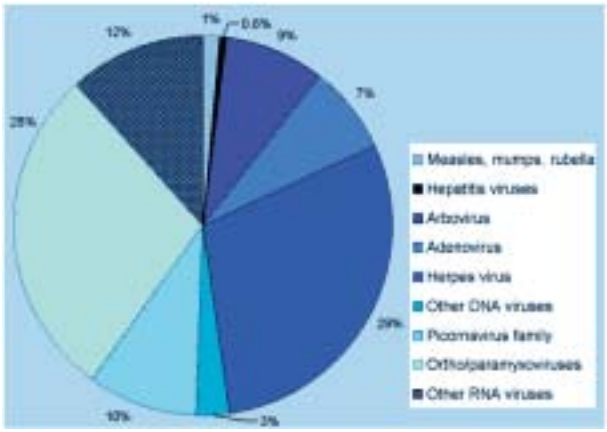
In 2001, 15 laboratories contributed 24,492 reports to LabVISE. This was an increase of 3.5 per cent on the number of reports in 2000 and data were received from one additional laboratory (Canberra Hospital, ACT) in 2001. The total reports received are shown by state and territory in Table 24.

Table 24. Infectious agents reported to the Laboratory Virology and Serology Reporting Scheme, 2001, by jurisdiction

Organism	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Measles virus	0	5	0	5	2	1	99	12	**124**
Mumps virus	0	1	0	1	4	0	8	18	**32**
Rubella virus	1	5	0	53	5	1	12	7	**84**
Hepatitis A virus	0	6	9	41	8	0	4	13	**81**
Hepatitis D virus	0	0	0	2	5	0	3	1	**11**
Hepatitis E virus	0	0	0	0	0	0	3	2	**5**
Ross River virus	0	31	92	463	89	3	33	153	**864**
Barmah Forest virus infection	0	14	7	208	3	0	1	36	**269**
Dengue	0	4	152	0	9	1	1	68	**235**
Murray Valley encephalitis virus	0	0	5	1	0	0	0	1	**7**
Kunjin virus	0	0	3	1	0	0	0	5	**9**
Flavivirus (unspecified)	0	0	2	8	0	0	16	0	**26**
Adenovirus	6	249	32	33	404	14	245	235	**1,218**
Herpes virus	16	508	82	1,117	1,445	26	651	1,018	**4,863**
Other DNA viruses	0	6	5	86	194	0	53	103	**447**
Picornavirus	15	644	50	30	40	18	169	596	**1,562**
Ortho/paramyxoviruses	23	1,383	56	242	1,284	57	443	1,166	**4,654**
Other RNA viruses	21	668	5	1	322	16	333	524	**1,890**
Chlamydia trachomatis	50	529	129	927	773	29	41	930	**3,409**
Chlamydia pneumoniae	0	3	1	1	0	0	0	2	**7**
Chlamydia psittaci	1	4	4	0	0	0	59	9	**77**
Mycoplasma pneumoniae	0	88	28	221	268	15	178	173	**971**
Coxiella burnetii	4	10	0	59	13	0	47	28	**161**
Rickettsia species	0	0	0	0	1	4	2	3	**10**
Streptococcus group A	13	32	36	209	0	0	109	0	**399**
Streptococcus group B	20	0	0	0	0	0	0	0	**20**
Yersinia enterocolitica	0	4	0	0	0	0	1	0	**5**
Brucella species	0	0	0	4	1	0	1	0	**6**
Bordetella pertussis	23	202	35	239	863	7	269	28	**1,666**
Legionella pneumophila	0	2	0	0	0	0	62	3	**67**
Legionella longbeachae	0	1	2	0	3	0	3	28	**37**
Legionella species	0	0	0	0	0	0	15	0	**15**
Cryptococcus species	0	5	0	4	12	0	0	0	**21**
Leptospira species	0	1	1	24	11	1	0	2	**40**
Treponema pallidum	0	91	257	353	377	0	2	41	**1,121**
Protozoa		11		2	5	4	21	3	**46**
Echinococcus granulosus	0	0	0	0	19	0	1	13	**33**
Total	**193**	**4,507**	**993**	**4,335**	**6,160**	**197**	**2,885**	**5,221**	**24,492**

Of reports received in 2001, 19,790 (81%) were of viral infections and 4,702 (19%) were bacterial or other infectious agents. Among the viral infections reported to LabVISE, viruses belonging to the herpes virus family (cytomegalovirus, varicella-zoster virus, Epstein-Barr virus and herpesvirus type 6) were the most commonly reported (4,863 reports, 24.5 per cent of all viral reports) . A similar number of reports of ortho/paramyxovirus infections (laboratory-confirmed influenza, parainfluenza, respiratory syncytial virus) were also received (4,654 reports, 23.5% of viral reports) (Figure 69). Laboratory reports of *Chlamydia trachomatis* made up 72 per cent of all non-viral reports to LabVISE in 2001 (n=3,409).

Figure 69. Reports of viral infections to the Laboratory Virology and Serology Reporting Scheme, 2001, by viral group



There were 1,727 laboratory reports of varicella-zoster virus to LabVISE in 2001. Analysis of these reports over time showed that a peak in January coincided with a peak in hospitalisations for varicella in Australia (Figure 70).[110] Laboratory testing for varicella is an important measure of the disease burden, which will become increasingly important should varicella vaccines be introduced into the Australian Standard Vaccination Schedule.

There were 1,727 laboratory reports of rotavirus to LabVISE in 2001. LabVISE has received reports of rotavirus since 1991. Regular epidemics of rotavirus occur in Australia every winter (Figure 71).

There was a large outbreak of rotavirus gastroenteritis in the Northern Territory in 2001. The outbreak began in Alice Springs and spread throughout the Northern Territory and into western Queensland. Ninety-five per cent of cases were in children under the age of five years and notification rates were five times higher in Indigenous compared with non-Indigenous people. The predominant strain was serotype G9, a strain of rotavirus group A which has only been reported in the Northern Territory since 1999.[111] Surveillance of rotavirus by the National Rotavirus Reference Centre continued in 2001.[112] Between 1 June 2001 and 30 June 2002, 754 samples were examined and this has shown that serotype G9 has become the most prevalent and widely dispersed serotype in Australia.

Figure 70. Laboratory reports of varicella-zoster virus to the Laboratory Virology and Serology Reporting Scheme and hospitalisations with a principal diagnosis of varicella, Australia, 1997 to 1999

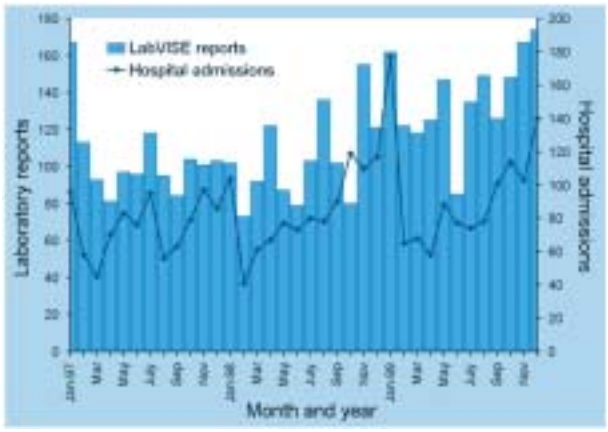
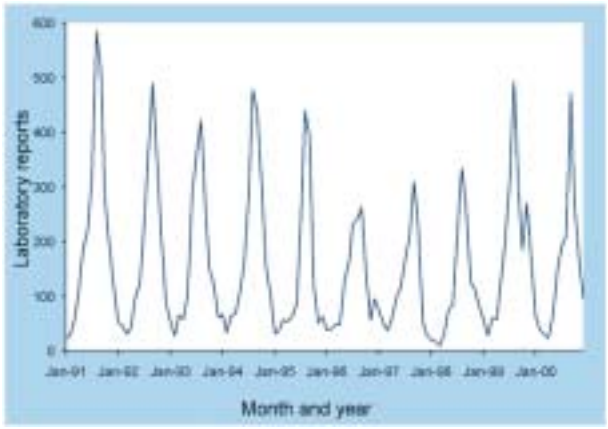


Figure 71. Laboratory reports to the Laboratory Virology and Serology Reporting Scheme of rotavirus infection, Australia, 1991 to 2000, by month of specimen collection



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 allow trends in consultation rates to be detected.

There were approximately 120 general practitioners participating in the scheme from all states and territories in 2001. Approximately 75 per cent of these are located in metropolitan areas and the remainder are in rural areas. Each week, an average of 56 participating practices reported an average 6,476 consultations to the scheme. In 2001, four conditions related to communicable diseases were reported. These were influenza-like illnesses, culture-confirmed influenza, chickenpox and shingles. Case definitions for these conditions were published in *Commun Dis Intell* 2000;24:7–8.

In 2001, a total of 336,745 consultations were reported to ASPREN by participating General Practitioners. Among consultations for communicable diseases, influenza-like illness was the most commonly reported condition (n=1,878), with a further 36 culture positive influenza cases reported to the scheme. Weekly reports of influenza-like illness peaked in winter months.

There were 656 cases of chickenpox and 460 cases of shingles reported to ASPREN in 2001, corresponding to an average weekly rate for chickenpox of 1.9 cases per 10,000 consultations and 1.4 cases per 1,000 consultations for shingles.

Antibiotic resistance in Australia

Since the release of *The Commonwealth Government Response to the Report of the Joint Expert Technical Advisory Group on Antibiotic Resistance* (JETACAR) in October 2000, the government has continued its work toward the development of a national antibiotic resistance management program.[113] Two committees were established to further this aim:

- The Expert Advisory Group for Antimicrobial Resistance (EAGAR), was set up in April 2001 under the auspices of the National Health and Medical Research Council, to provide continuing advice on antibiotic resistance and related matters; and
- The Commonwealth Interdepartmental JETACAR Implementation Group was established in November 2000, to oversee and coordinate the continuing government response to JETACAR, to respond to the policy advice received from EAGAR and to seek funding for implementation purposes.

During 2001, EAGAR developed and commenced the use of a protocol to assess the risk of antibiotic resistance developing in new and existing antibiotics.

Activities undertaken by the Commonwealth Interdepartmental JETACAR Implementation Group and its member agencies in 2001 include:

- an informal consultation meeting in March, *The Monitoring of the Distribution of Antibiotics for Veterinary and Human Use in Australia*; and
- the release in April of the draft report, *National surveillance of healthcare associated infection in Australia,* for consultation.

Other important activities included:

- the workshop on *Antibiotic Resistance Surveillance* (4 May);
- the *National Summit on Antibiotic Resistance* (30 and 31 May);
- a nationwide consultation toward development of a National Antibiotic Resistance Surveillance System for Antibiotic Resistance Management (July–September); and
- the initiation of the EAGAR website — http://www.health.gov.au/pubhlth/strateg/jetacar/eagar.htm.

Progress reports on implementation of the Government Response are available on the implementing JETACAR website — http://www.health.gov.au/pubhlth/strateg/jetacar/index.htm.

Through the *National Summit on Antibiotic Resistance*, representatives from governments, health, agricultural, industry and consumer groups identified priorities for action. In particular, the need for the development of a national system of surveillance for antibiotics was recognised to measure the prevalence of antibiotic resistance. Further needs were also identified including:

- improved education and awareness, leading to more appropriate use of antibiotics;
- clearer research focuses, and better communication and regulation;
- more effective linkages between corporate and peak organisational bodies; and
- reduced incidence of health care-associated infections in Australia.

Creutzfeldt-Jakob disease

This section is based on reports for the period from the Australian National Creutzfeldt-Jakob disease (CJD) Registry, the University of Melbourne.

The Australian National CJD Registry was established in 1993, in response to recognition of four probable cases of iatrogenic CJD resulting from the use of human pituitary hormones. It has subsequently undertaken retrospective case ascertainment in addition to its ongoing monitoring and surveillance activities and the Register now includes cases of CJD dating back to 1 January 1970 (Table 25).

Table 25. Cases reported to the Australian National Creutzfeldt-Jakob Disease Registry: 1970 to 2001

Classification	Number of cases as at December 2001	Change in number of cases during 2001*
Definite	243	+ 7
Probable	170	+10
Possible	0	– 1
Incomplete†	66	– 23
Total	479	– 7

* These changes are due to reclassification of previously notified cases, as more definitive data confirm or exclude provisional diagnoses

The average annual incidence of CJD in Australia between 1988 and 2000 is 1.13 cases per million population. International rates are generally around one case per million population per annum. Of the cases recorded on the Register, 90.3 per cent of cases are sporadic, 7.5 per cent of cases are familial and 2.2 per cent of cases are iatrogenic in origin. No new cases of iatrogenic disease were recorded in Australia in 2001.

The average age of death for sporadic CJD cases by sex was 64 years for males and 67 years for females. For familial CJD the average age for death was 52 years for males and 59 years for females, and for people who acquired the disease iatrogenically, the average age of death was 45 years for both males and females.

As at the end of December 2001, Australia remains free of animal forms of transmissible spongiform encephalopathies and no cases of the variant form of CJD have been detected.

In Australia, autopsy rates have been steadily declining, in line with international trends. Lack of autopsy frequently compromises the ability of the Registry to classify cases as definite, pending the development of additional tests. The Registry has seen an increase in notification of suspected cases since the introduction of diagnostic testing of the 14–3–3 protein in cerobrospinal fluid.

Responses to possible bioterrorism

Public health authorities in Australia and overseas have developed contingency plans to deal with any threats, however unlikely, from chemical, biological, and radiological agents.

Australia has developed its own chemical, biological, and radiological plans through Emergency Management Australia, working in partnership with health agencies and other arms of government.

Australia has had counter-terrorism plans in place for a number of years, and the health system's level of preparedness was increased in preparation for the Sydney Olympics, and again following the events of 11 September 2001 and the cases of anthrax associated with contaminated mail in the USA, and the more recent bombings in Bali. Further details of the anthrax cases in the USA are provided in the zoonoses section of this report.

Preparations by the Department of Health and Ageing include:

- training within health departments and with other agencies;
- adoption of medical treatment protocols suitable for a civilian population;
- stockpiling of appropriate pharmaceutical supplies;
- increasing diagnostic and health surveillance capability;
- improved coordination and advisory arrangements; and
- access to international advice to alert Australian authorities to overseas developments.

The Department of Health and Ageing will continue to be engaged in the development of a coordinated bioterrorism response with state and territory health authorities and with laboratory networks. Australia's laboratories performed extremely well during the anthrax hoaxes and false alarms in October and November 2001, responding rapidly and effectively to the increased workloads and the demand for speedy and accurate results.

Appendices

Appendix 1. Australian Bureau of Statistics population data used in the calculation of rates

Population age structure by state or territory and sex

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Males	156,554	3,251,412	104,326	1,813,215	743,153	231,175	2,392,413	961,442	9,655,422
Females	157,617	3,281,047	93,264	1,814,601	759,244	239,097	2,436,555	948,309	9,731,241
Persons	314,171	6,532,459	197,590	3,627,816	1,502,397	470,272	4,828,968	1,909,751	19,386,663

Population age structure by state or territory and age group

	State or territory								
Age group	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
0–4	20,929	429,371	17,555	241,421	90,576	30,302	301,986	125,117	1,257,471
05–09	21,374	443,504	17,485	259,211	98,110	33,182	322,324	132,796	1,328,290
10–14	21,814	443,803	16,063	259,006	99,695	34,204	323,937	138,589	1,337,457
15–19	24,257	448,117	14,945	265,639	103,119	34,476	330,373	140,871	1,362,106
20–24	28,184	455,001	16,181	258,880	98,520	29,409	353,840	142,244	1,382,408
25–29	25,939	492,684	19,294	268,578	102,042	29,354	369,632	145,634	1,453,387
30–34	24,426	490,605	18,963	268,759	106,708	31,413	375,736	144,348	1,461,251
35–39	23,999	499,952	17,101	274,234	111,753	33,840	369,135	147,882	1,478,222
40–44	24,294	495,174	15,371	275,220	114,779	36,088	364,053	148,827	1,474,085
45–49	23,393	453,004	13,404	254,854	107,217	33,823	335,408	139,489	1,360,860
50–54	22,385	428,163	11,546	243,905	103,730	32,387	315,932	128,061	1,286,335
55–59	15,504	337,496	7,798	190,390	81,069	25,557	245,898	94,574	998,401
60–64	10,917	274,949	4,681	148,752	66,313	21,569	201,683	75,328	804,278
65–69	8,235	233,440	2,880	119,404	57,661	18,155	172,652	60,597	673,063
70–74	6,932	220,337	2,015	109,524	56,630	16,834	161,856	54,266	628,418
75–79	5,707	181,272	1,128	88,449	48,282	13,684	133,025	42,304	513,860
80–84	3,392	114,844	622	56,792	31,003	8,816	82,647	25,988	324,113
85+	2,490	90,743	558	44,798	25,190	7,179	68,851	22,836	262,658

Appendix 2. Completeness of National Notifiable Diseases Surveillance System data received from states and territories, 2001

Completeness of National Notifiable Diseases Surveillance System data received from states and territories, 2001

Total notifications	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Sex									
No. missing	8	93	13	4	0	0	322	69	509
% complete	99.4	99.7	99.7	100.0	100.0	100.0	98.5	99.4	99.5
Age									
No. missing	0	40	38	0	2	10	786	24	900
% complete	100.0	99.9	99.3	100.0	100.0	99.5	96.4	99.8	99.1
Indigenous status									
No. missing	1,037	874	524	20,032	1,659	1,521	18,077	5,380	49,104
% complete	22.2	97.0	89.8	15.7	81.5	25.0	17.2	53.5	52.9

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Composition of Australian influenza vaccine for the 2003 season

In order to select virus strains for the manufacture of the influenza vaccine for the 2003 season, a meeting of the Australian Influenza Vaccine Committee on Influenza Vaccines was convened on 10 October 2002.

Having considered information on international surveillance by the World Health Organization (WHO), and up-to-date epidemiology and strain characterisation presented at the meeting, the Committee considered that the WHO recommendations on the composition of vaccines for the 2003 Southern Hemisphere season should be followed.

A H1N1 strain:	an A/New Caledonia/20/99 (H1N1)-like strain A/New Caledonia/20/99 (IVR-116) is recommended as a suitable vaccine strain.	15 µg HA per dose.
A H3N2 strain:	an A/Moscow/10/99 (H3N2)-like strain A/Panama/2007/99 (RESVIR-17) is recommended as a suitable vaccine strain.	15 µg HA per dose.
B Strain:	a B/Hong Kong/330/2001-like strain B/Shangdong/7/97 and B/Brisbane/32/2002 are recommended as suitable vaccine strains.	15 µg HA per dose.

Pneumococcal disease in Australia: current status and future challenges

A report of the workshop held at the National Centre for Immunisation Research and Surveillance, 8–9 November, 2002

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Introduction

In March 1999, a workshop titled 'Pneumococcal disease in Australia' was held at the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Disease in Sydney. A summary of the workshop[1] and recommendations for surveillance[2] were subsequently published in *Communicable Diseases Intelligence* in April 2000. More detailed papers arising from presentations by a wide range of Australian experts appeared later that year as a supplement of the *Medical Journal of Australia.* The papers in the supplement described the epidemiology of invasive pneumococcal disease (IPD) in areas of Australia with a relatively low proportion (metropolitan New South Wales, Victoria) and a relatively high proportion (Northern Territory and Western Australia) of the population who are Aborigines or Torres Strait Islanders. The most prominent issues identified were the high incidence of IPD among Aboriginal children, particularly in Central Australia, associated with a more diverse range of serotypes than non-Indigenous children; the morbidity and mortality of IPD, particularly meningitis; and increasing levels of antibiotic resistance among strains of *Streptococcus pneumoniae*.

At the time of the first meeting, pneumococcal disease in young children was not vaccine-preventable as the only vaccine then available, the 23–valent polysaccharide vaccine, (23vPPV) is poorly immunogenic under the age of 2 years. Subsequently, the seven-valent conjugate pneumococcal vaccine (7vPCV) was approved for use in Australia by the Therapeutic Goods Administration in December 2000. A Pneumococcal Working Party, jointly chaired by the Australian Technical Advisory Group on Immunisation and the Communicable Diseases Network Australia had been established earlier in 2000. The Pneumococcal Working Party recommended urgent implementation of a publicly funded vaccination program with 7vPCV for children at highest risk of IPD, primarily those with predisposing medical conditions aged under 5 years and all Indigenous children aged under 2 years. The program commenced in July 2001 in Central Australia and was progressively implemented in other states and territories over the next 6 months. At the time of the first workshop, invasive pneumococcal disease was notifiable only in the Northern Territory (since 1995) and Queensland (since 1996).[2] In January 2001, the Communicable Diseases Network Australia agreed to make IPD a notifiable disease in all Australian jurisdictions, with enhanced surveillance systems implemented and funded by the end of 2001. The second national workshop in November 2002 provided an opportunity to evaluate the first year of enhanced surveillance and the 7vPCV vaccination program and to learn from the experience following the introduction of universal 7vPCV vaccination of children under 2 years of age in the United States of America at the end of 2000.

Day 1. Surveillance of invasive pneumococcal disease

Long term population-based surveillance in the United States of America and Australia

United States of America

Cynthia Whitney, a medical epidemiologist from the Centers for Disease Control and Prevention Atlanta, reviewed the current epidemiology of pneumococcal disease in the USA and the impact of the conjugate vaccine. Surveillance of IPD is based on 10 sentinel areas (counties, cities or states) with a total population of 20 to 24 million people. Cases of IPD occurring in these sentinel areas are actively followed up and isolates are collected and analysed for serotype and antimicrobial resistance at the Centers for Disease Control and Prevention or reference laboratories. Surveillance is aimed at assessing vaccine effectiveness. Early assessments indicate a reduction of the order of 70 per cent in the incidence of IPD in children aged less than 2 years. Vaccine failures have been documented in 129 cases of which 47 per cent were cases with non-vaccine serotypes, 37 per cent with vaccine serotypes and 17 per cent with vaccine-related serotypes. Among the 48 cases with vaccine serotypes, only 11 were fully vaccinated and the most common serotype isolated was 6B (n=6). The majority of children who were vaccine failures had other underlying conditions predisposing to pneumococcal infection.

North Queensland

Jeffrey Hanna, Director of the Tropical Public Health Unit based in Cairns, reported on enhanced surveillance of IPD in Far North Queensland since 1992, subsequently expanded to include the whole

of north Queensland.[3] The incidence of IPD in Indigenous children under 2 years of age (297 cases per 100,000 population) is similar to that reported from the Top End of the Northern Territory (326 cases per 100,000 population) but dwarfed by the incidence among Aboriginal children in Central Australia (1,534 cases per 100,000 population). Importantly, the incidence in Far North Queensland of pneumococcal meningitis among Indigenous children (56 cases per 100,000 population) was higher than in non-Indigenous children (17 cases per 100,000 population) and occurred at a significantly younger age (median 0.5 years) than non-Indigenous children (1.0 years). In Indigenous children, only 62 per cent of isolates had serotypes contained in the 7vPCV, compared with 88 per cent among non-Indigenous children.

Metropolitan New South Wales

Peter McIntyre, Deputy Director of the National Centre for Immunisation Research and Surveillance, updated to 2001 the earlier report to 1999 of enhanced surveillance of IPD in metropolitan New South Wales by a laboratory network.[4] The overall incidence of IPD in children aged under 2 years (100 cases per 100,000 population) and pneumococcal meningitis (10 cases per 100,000 population) and the serotype distribution (90 per cent included in 7vPCV) were unchanged. In children less than 5 years of age, additional medical risk factors (extreme prematurity, all premature infants with chronic lung disease and all children with Downs syndrome, diabetes mellitus and cystic fibrosis) were associated with increased numbers of cases. A case-control study in this population has shown that attending child care is most important as a risk factor for IPD in the first 3 months after commencement. Only 15 IPD cases were identified as Indigenous during the study period, giving a very similar incidence to non-Indigenous children. This may suggest that the incidence of IPD in Indigenous children living in an urban setting is lower than in rural and remote settings, but is limited by deficiencies in identifying Indigenous children in urban areas and needs further investigation.

Enhanced surveillance of notifiable invasive pneumococcal disease in states and territories since 2001

Short presentations were made on national data collected from enhanced surveillance for IPD in 2001 and reports of IPD surveillance activities in 2002 in each state and territory. The national report for 2001 has recently been published[5] and the abstracts of each state and territory's presentation are included in the appendix to this report.

Laboratory issues

Lyn Gilbert, Chair of the Laboratory Sub-group of the Pneumococcal Working Party, reviewed the contribution of laboratories to the surveillance of IPD, particularly in measuring the impact of vaccination on serotype distribution and the prevalence of antibiotic resistance. Maintenance of laboratory networks for forwarding IPD isolates and for serotyping and reporting is resource intensive. Current resources will not be sufficient for expanded antimicrobial susceptibility testing. There are a number of more sophisticated methods for serotyping and molecular typing of pneumococcal strains under development. The potential for molecular serotyping of *S. pneumoniae* was discussed in the context of a pilot project which has shown these new techniques correlate well with traditional serological typing, with the potential for micro-array technology to automate typing and antimicrobial susceptibility. In addition, multi-locus sequence-typing based on variations in seven *S. pneumoniae* genes, provides a tool for measuring the molecular epidemiology of antimicrobial resistance and serotype dynamics.

Surveillance of pneumococcal vaccine coverage

While childhood immunisation coverage rates are now monitored in the Australian Childhood Immunisation Register (ACIR), no national register exists for adult vaccines. Hence, there is considerable uncertainty about the proportion of older Australians receiving the pneumococcal polysaccharide vaccine.

Coverage estimates for 23vPPV — Victoria and Australia

Ross Andrews, who is currently evaluating the Victorian funded 23vPPV program, described survey methods aimed at estimating pneumococcal polysaccharide vaccine coverage. In Victoria, vaccine has been offered free of charge to all residents aged 65 years or more since 1997.[6] There are methodological problems in measuring pneumococcal vaccine coverage in older persons by telephone survey. A computer-assisted telephone interview validated against physician records in Victoria has estimated coverage for over 65 year olds receiving the vaccine within the past 5 years, to be between 47 and 51 per cent. Coverage increased over the 5 years 1996 to 2000. By contrast, using Pharmaceutical Benefits Scheme data the coverage in the rest of Australia was estimated to be 25 to 28 per cent, but is also a significant increase over estimates from 1996. In Victoria, preliminary data from a National Health and Medical Research Council funded hospital-based case-cohort study suggest that 23vPPV has an effectiveness of 80 per cent when measured against an outcome of hospitalisation for pneumonia.

Coverage estimates for 7vPCV

Peter McIntyre described the use of the ACIR to measure coverage of eligible children with the conjugate pneumococcal vaccine. ACIR data show that little vaccine was distributed outside the jurisdictions with the highest rural and remote Indigenous populations (the Northern Territory, Western Australia and Queensland) before 2002, with a progressive increase in 2002. Although the ACIR collects information on the Indigenous status of children, these data have not been available or have been poorly utilised until recently. If all doses recorded on the ACIR are assumed to have been given to Indigenous children, then of the estimated Indigenous birth cohort, over 90 per cent of the Northern Territory, 67 per cent of the Western Australian and Queensland and 44 per cent of other jurisdictions received 7vPCV in the first quarter of 2002. The availability of an Indigenous status indicator by state or territory on the ACIR will soon allow more accurate estimates. The currently available data from the ACIR, and other data presented at the meeting from the Northern Territory, Western Australia and Queensland, suggest that coverage of 7vPCV has been satisfactory in the highest risk populations.

Surveillance of invasive pneumococcal disease — where to from here?

The first day concluded with an animated discussion on the needs of surveillance of IPD in the future. The present needs to improve the Commonwealth funded enhanced surveillance for IPD were described by Vicki Krause. These include capturing all incident cases; improving links with laboratories; collecting more complete data on Indigenous status; more broadly defining risk factors for pneumococcal disease; and measuring outcomes, including deaths, more accurately. Beyond these immediate needs, the groups discussed the sustainability of enhanced surveillance nationwide in the context of limited resources. Cynthia Whitney described the USA experience of using sentinel areas in place of nationwide surveillance and some of the limitations of this method. In most Australian states (except the Northern Territory and Western Australia and the far north of Queensland), there will be little measurable effect of the conjugate vaccine on rates of pneumococcal disease, since the majority of children in these regions are not eligible for free vaccine under the current schedule. The meeting concluded that high quality enhanced surveillance should be supported in the Northern Territory, rural Western Australia and Far North Queensland. Surveillance in other areas should be reviewed in light of other priorities. Data from Victoria on the impact of the polysaccharide vaccine should continue to be collected to provide information relevant to the use of this vaccine in controlling pneumococcal disease in the elderly elsewhere in Australia. The costs of adding adult vaccine coverage data to the ACIR are probably prohibitive. Novel methods of increasing adult vaccine coverage, such as linking pneumococcal vaccination to annual influenza vaccinations, should be considered.

Day 2 – Pneumococcal vaccines and their impact

The second day of the meeting focussed on the impact of pneumococcal vaccines on IPD. The coverage and impact of polysaccharide vaccines in the USA, and in non-Indigenous and Indigenous adults in regions of Australia, was reviewed first followed by similar data on 7vPCV.

Pneumococcal polysaccharide vaccines

Issues in the United States of America

Cynthia Whitney reviewed data on the use of the polysaccharide vaccine in the USA, where the 23–valent vaccine has been in use since 1983. As in Australia, the vaccine is recommended for all persons aged 65 years or more and for at-risk groups aged 2 years or more. There have been concerns about an increase in pneumococcal disease following vaccination with 23vPPV in HIV-infected people with advanced disease.[7,8] In the USA, the 23–valent vaccine appears to protect HIV-infected people against pneumococcal disease without adverse effects, but efficacy is greatest when CD4+ cell counts are over 500. Therefore vaccination should be as early as possible after seroconversion or following immune-reconstitution with antiretroviral therapy. The question of re-vaccination of the elderly using 23–valent vaccine is being addressed. The duration of vaccine protection is difficult to measure in elderly populations who have a high prevalence of immunosuppressive disease and a high usage of medications. Currently, re-vaccination is recommended every 5 years; however, the safety of re-vaccination needs to be assessed since local reactions to the vaccine become more significant. Coverage in the USA in targeted populations is estimated to be 65 per cent and there are plans to increase this to 90 per cent in these groups. It is estimated that present levels of vaccination will reduce pneumococcal disease incidence in the elderly by 12 per cent and increasing the vaccine coverage could reduce this by as much as 25 per cent. Use of the 23–valent vaccine in the elderly is cost-effective.

Vaccination programs with 23vPPV in north Queensland and Victoria

Jeffrey Hanna reviewed the impact of the 23–valent polysaccharide vaccine on IPD in Far North Queensland. Over a seven year period, the incidence of IPD in Indigenous adults (eligible for vaccination at 50 years of age) has been reduced from 110 to 28 cases per 100,000 population.[9] This rate is approximately the same as that seen in the non-Indigenous population. A vaccine effectiveness of 50 to 80 per cent has been estimated from these data.

Ross Andrews provided evidence of the impact of the 23–valent vaccine on IPD in Victoria. From the examination of IPD surveillance data and estimated 23vPPV coverage, it is estimated that the funded vaccine program for the elderly in Victoria has prevented 109 cases and 20 deaths since the program was started.

Impact of pneumococcal conjugate vaccine programs

In this session, there were presentations on the impact of 7vPCV from the sentinel surveillance sites in the USA and from three states with significant populations of Indigenous children living in rural and remote areas of northern Australia within the tropics (the Northern Territory, North Queensland and Western Australia). All these regions have demonstrated both significantly higher incidence and serotype diversity among Indigenous children.

Impact of 7vPCV on invasive pneumococcal disease in the United States of America

Cynthia Whitney presented exciting data on the impact of conjugate pneumococcal vaccines in the USA. The 7 valent conjugate vaccine was licensed for use in February 2000 and recommended for use in all children aged less than 2 years and in children aged between 2 and 4 years with risk factors for pneumococcal disease, from June 2000. In some states, vaccine is provided free of charge only to children who fulfil stringent means test requirements, while in other states, vaccine is provided free of charge to all children. Therefore vaccine coverage varies by area.

A comparison of age-specific rates of pneumococcal disease in 2001 in the USA with baseline levels in the pre-vaccine years, 1998-99, shows an overall decline of 69 per cent in the age group 0–12 months and a decline of 44 per cent in one to two-year-olds. Disease caused by vaccine serotypes has declined overall from 156 to 34 cases per 100,000 population and for vaccine serotype-related disease from 20 to 10 cases per 100,000 population. Disease caused by non-vaccine serotypes has increased from 12 to 16 cases per 100,000 population, but this change is not statistically significant. The decline in the incidence of vaccine serotypes range from 62 per cent for serotype 6B to 83 per cent for serotypes 14 and 19F. An interesting correlation between the declining incidence in children and an unexpected decline in adults has been observed — the first evidence of a herd effect of conjugate vaccines. There has been little change in the proportion of isolates resistant to penicillin seen following the introduction of 7vPCV, but to date there is also no evidence of serotype replacement in IPD cases.

Impact of 7vPCV on Invasive Pneumococcal Disease in northern Australia

Northern Territory

Christine Selvey, Director of Immunisation in the Northern Territory Centre of Disease Control, presented data on the impact of 7vPCV in the Northern Territory, adding to data presented the previous day. In the Northern Territory, 50 per cent of the annual birth cohort (all children born in Central Australia and Indigenous children in the rest of the Northern Territory) are eligible for the 7vPCV. Vaccination was implemented from 1 June 2001 (starting with those born after 1 April 2001) and the 'catch-up' program was initiated in September 2001. As at August 2002, 96 per cent of eligible children had received the first dose of vaccine at 2 months of age, 74 per cent of older children had started the 'catch-up' vaccine schedule and 64 per cent of these had completed the vaccine course. Only three mild to moderate adverse events after conjugate vaccination were recorded. A few cases of IPD in children who have received one or more doses of 7vPCV have occurred, mostly among the 'catch-up' group. It is still too early to evaluate the impact of the conjugate vaccine on IPD in the Northern Territory, given the expected small numbers of cases. In Central Australia, evaluation is complicated by the decrease in incidence of IPD among Aboriginal children aged under 2 years, from 1,500 cases per 100,000 population from 1994 to 1998, to 700 cases per 100,000 population from 1999 to 2001, prior to commencement of the 7vPCV program. No change was seen in the incidence of IPD among Aboriginal children in the Top End.

Western Australia

Carolein Giele, epidemiologist in the Communicable Disease Control Branch of the Health Department of Western Australia, presented data on the coverage of 7vPCV and incidence of IPD among Aboriginal children in Western Australia. There was good correlation between data on doses distributed and doses reported to the ACIR, with estimated coverage being high in rural and remote areas and sub-optimal in the urban area of Perth and surrounds. The small numbers of IPD cases in Aboriginal children precluded conclusions about the significance of 3 cases to date in 2002 but 2 of the 3 cases were not serotypes

included in 7vPCV. Presumptive antibiotic treatment before sample collection is likely to reduce the identification of IPD cases in the north of the state.

North Queensland

In north Queensland, the uptake of IPD vaccination in the eligible birth cohort is estimated to be 70 per cent for the first dose, 60 per cent for the second and 50 per cent for the third (within one month of the scheduled age). The number of IPD cases in Indigenous children aged less than 15 years has fallen from 15 cases in 1999 to 6 cases to the end of September 2002. None of the cases in 2002 were caused by vaccine serotypes, while the prevalence of non-vaccine serotypes has shown little change. There is some evidence that the incidence of IPD in non-Indigenous children, who are not eligible for funded vaccination, has also declined in the same period. This, together with data suggestive of a fall in incidence of IPD in Indigenous adults since the introduction of 7vPCV, may be an early indication of herd immunity from the reduction in pneumococcal colonisation among vaccine-eligible children.

Impact of 7vPCV on ear disease

Northern Territory

Amanda Leach, Senior Scientist with the Menzies School of Health Research and the Cooperative Research Centre for Aboriginal and Tropical Health in Darwin, presented background data from her team's previous research into ear disease among Aboriginal children in the Northern Territory. She also discussed research in progress to monitor the future impact of 7vPCV on ear disease in a number of areas of the Northern Territory. Rates of tympanic membrane perforation among Aboriginal people from previous published studies range from 5 to 37 per cent, with the World Health Organization nominating 4 per cent or higher as a 'massive public health problem'.[10] The Prevention of Otitis Media with Prevenar and Training (PROMPT) study, funded by Wyeth-Lederle vaccines, has recruited 20 communities for follow-up, with many having perforation rates among children of more than 25 per cent. The Prevenar Immunisation for Otitis Media Reductions in the Tiwi Islands (PRIORiTI) is a longitudinal study in the Tiwi community, with data on pneumococcal colonisation extending back for almost 10 years. Early results suggest that pneumococcal colonisation has decreased overall, with a decrease in 7vPCV serotypes counterbalanced to some degree by an increase in other serotypes. Clinical implications are unclear, but there appears to be some decrease in tympanic membrane perforations compared with historical data. Close follow-up in both study cohorts will continue. Given the early onset of colonisation previously found in these populations, indirect impacts via decrease in colonisation in older contacts of young infants may turn out to be as important as individual vaccine response in reducing ear disease.

Sydney

Michael Watson, Microbiologist and Infectious Disease Physician at The Children's Hospital Westmead, presented early results of a baseline comparison of serotypes seen in pneumococcal isolates from swabs from ear discharge. The predominant serotypes were very different from those seen in IPD isolates from the same population, with a predominance of 19F, as opposed to serotype 14. This has potential implications for the impact of 7vPCV in non-Indigenous populations when funded vaccination at a population level is introduced.

Summary and conclusions

In relation to surveillance, the predominant issue discussed was universal versus sentinel enhanced surveillance of IPD. In northern Australia, it will be important for enhanced surveillance to continue and to be as complete as possible. There are a number of reasons for this. First, the high incidence and high serotype diversity of IPD in Indigenous children in these areas has prompted the recommendation for boosters with 23vPPV to increase serotype coverage. This makes high quality, comprehensive surveillance essential for national policy. It is also important internationally as such as vaccine program has not been implemented anywhere else but is potentially applicable to other comparable populations. Secondly, the small absolute numbers of cases require data to be accumulated as comprehensively as possible.

In relation to vaccine issues, both 23vPPV and 7vPCV policy are important. There was strong support from the meeting for the recent recommendation from the Australian Technical Advisory Group on Immunisation that both 23vPPV (for those over 65 years) and 7vPCV (for those less than 2 years) be publicly funded as universal programs. With respect to the current programs, there were important issues for Aboriginal and Torres Strait Islander people for both 23vPPV and 7vPCV. For 23vPPV, research is required into both the utility and frequency of boosters in adults as well as any potential role for 7vPCV in adults. Improving the identification of Aboriginal and Torres Strait Islander children is important, especially in urban areas.

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Appendix: Abstracts of presentations from states and territories on invasive pneumococcal disease surveillance in 2002

Invasive pneumococcal disease surveillance in the Australian Capital Territory

Louise Carter, Communicable Disease Control Section, Australian Capital Territory Department of Health and Community Care

Invasive pneumococcal disease (IPD) was listed as a notifiable infection under the Australian Capital Territory *Public Health Act 1997* in September 1999. Public health legislation requires the condition to be notified to the Australian Capital Territory health department by hospitals, medical practitioners and pathology laboratories. The Australian Capital Territory has a passive system which relies on the assistance of hospital infection control practitioners to review medical records and in-patient notes for information on vaccination history and factors which may predispose people to the disease. Serotyping of isolates from the Australian Capital Territory is performed in batches and does provide some feedback on intervention activities pertaining to IPD being conducted locally.

The burden of IPD in the Australian Capital Territory is low (around 5 cases per 100,000 population per annum), however, information other than demographic details provided by the reporting laboratories are often missing and details regarding pneumococcal vaccines previously administered are rarely validated. This presentation supports a uniform national surveillance system for IPD, so that trends regarding the control of this condition and the impact of the National Childhood Pneumococcal Vaccination Program can be accurately monitored.

Invasive pneumococcal disease New South Wales, 2002 — in the beginning

Robin Gilmour, NSW Health Department

Invasive pneumococcal disease became notifiable by all laboratories in New South Wales in 2001, and 2002 saw the commencement of enhanced surveillance for notified cases who were aged less than 5 years or 50 years or older. Surveillance of IPD in New South Wales is derived from three sources: the New South Wales Notifiable Disease Database; the New South Wales enhanced IPD surveillance database (for cases aged less than 5 years or 50 years or more); and typing and antibiotic sensitivity testing from the Children's Hospital at Westmead.

The overall incidence of IPD in New South Wales is 13.4 cases per 100,000 population for the nine-month period. From the enhanced data (n=497) children aged less than 2 years continue to have the highest rate of disease (98.5 cases per 100,000 population). Adults aged more than 65 years accounted for 68 per cent, adults aged more than 85 years have the next highest rate of disease (74.5 cases per 100,000 population). Bacteraemia is the most common infection in children and pneumonia is most common in adults. Meningitis is uncommon in both age groups (3 per cent in adults and 7 per cent in children). Underlying illness was reported in 16 per cent of children and 72 per cent in adults. There have been 3 deaths (1.6%) reported in children aged under 5 years and 73 (29.9%) deaths in adults. Only 9 cases were reported as being Aboriginal or/and Torres Strait Islander. Eighty-two per cent of isolates in the Greater Sydney Region have been serotyped for the period January to June 2002. Thirty-six

possible vaccine failures were identified up to September 2002.

Invasive pneumococcal disease in the Northern Territory 1 January to 30 September 2002. How do the numbers compare to the same period in 2001?

Heather Cook and Vicki Krause, Centre for Disease Control, NT Department of Health and Community Services, Darwin

Invasive pneumococcal disease enhanced data has been reported from the entire Northern Territory since 1994. Very high rates have been documented, specifically in the under two year old Indigenous population with higher rates in this age group in the Centre than in the Top End.

Looking at the first nine months of 2002 there were 49 cases of IPD notified in the Northern Territory compared to 78 cases for the same period in 2001. The greatest reduction was in Top End in Indigenous children under 2 years, dropping from six cases to one case, followed by a decrease of five to zero cases in the same age group for Central Australian non-Indigenous cases in 2001 to 2002, respectively. No change was seen in this same age group in Central Australian Indigenous cases with 4 cases in each year while the Top End non-Indigenous cases decreased from 6 to 3 cases in this age group, in 2001 to 2002, respectively.

A notable decrease was also seen in the overall 15–49 year population where Indigenous cases dropped from 31 to 19 cases and non-Indigenous cases reduced from 6 to 4 cases, in 2001 to 2002, respectively.

Males represented 57 per cent of cases in 2002 and 63 per cent in 2001.

Indigenous status is very well reported in the Northern Territory and 78 per cent of the IPD cases in 2002 (and 67% in 2001) were Indigenous while 27 per cent of the Northern Territory population is Indigenous.

Although slightly decreased, pneumonia continued to be the most common overall primary clinical presentation (67% of cases in 2002 compared to 78% in 2001). In those 15 years and older, 83 per cent of all cases in 2002 presented as pneumonia. The only two cases of meningitis in 2002 occurred in non-Indigenous adults between 15 and 49 years of age.

Three IPD deaths occurred in 2002 with two of these occurring in the Indigenous 15-49 year age range, a case fatality rate of 11 per cent for this age group, and an overall rate of 6 per cent while the overall rate was 3 per cent in 2001.

Serotyping results were available for 92 per cent of the cases from 2002 with 71 per cent of the isolates tested in the under 2 year age group being those found in the 7-valent vaccine and all isolates in this age group were those found in the 23-valent vaccine. For those 2 years and over, 87 per cent of the isolates were covered by the 23-valent vaccine. This figure compares to 76 per cent in 2001.

In 2002, 5 cases of 23-valent vaccine serotype IPD occurred in fully vaccinated persons 5 years of age and older with each having one or more recognised risk factors for IPD as did the 7 same-category cases in 2001. Cases of IPD in children who received the 7-valent vaccine is discussed elsewhere.

Invasive pneumococcal disease trends in Queensland 1997–2002: fact or artefact?

Dr Robyn Pugh, Senior Epidemiologist, Communicable Diseases Unit, Queensland Department of Health

Invasive pneumococcal disease was made a notifiable disease in Queensland in 1996 and enhanced surveillance of cases aged under 5 years began state-wide in July 2001. However, IPD surveillance began in Far North Queensland in 1992 with comprehensive enhanced surveillance data collection on all cases since 1999.

Annual rates

Annual IPD rates increased from 1997 to 2000. There were 11.9 notifications per 100,000 population in 2001 which was slightly lower than in 2000. The highest rates occurred in children aged under 2 years (peaking in 2000 at 133 notifications per 100,000 population) followed by rates for persons aged 75 years or over. Fifty-six per cent of cases were males. In the 9 months to 30 September 2002, there were 351 notifications compared with 343 notifications in same period in 2001. The age distribution of these cases in 2002 was children aged under 5 years (38 per cent, including 26 per cent under 2 years), 5–14 years (5 per cent), 15–49 years (26 per cent) and 50 years or over (32 per cent). The age and gender profile has been similar each year since 1997.

Data collection

Data collection has improved since 1997 especially with the commencement of enhanced surveillance but records are still incomplete, e.g., no Indigenous status was recorded for 56 per cent of cases (29 per cent of cases aged under 5 years) in 2002 to date. There remains an over-representation of cases who are indigenous, with 10 per cent of notified cases aged under 5 years (13 per cent of under 2-year-olds) recorded as Indigenous.

Statistical Divisions in north Queensland

Statistical Divisions in north Queensland had the highest rates of IPD between 1997 and 2001; peak rates occurred in 2000 with 20.8 notifications per 100,000 population in the Far North and 24.0 notifications per 100,000 population in the Northern Statistical Division. The notification rates in divisions are similar this year compared with the same 9 month period in 2001 with some notable exceptions. The rates in the Far North and Northern divisions have fallen and are similar to the rate in Brisbane.

Clinical presentation

Clinical presentation of cases aged under 5 years during this year was similar to the same period last year. The majority presented with a bacteremia (59 per cent) and 32 per cent presented with pneumonia. Only 1.6 per cent of cases presented solely with meningitis.

Death rate

The reported death rate for notified cases was 1.1 per cent for this 5-year period, slightly higher in the earlier years for under 5-year-olds. There has been 4 deaths this year to date, compared with 6 deaths in the same period last year.

Overall, the most common serotype in 2002 to date is serotype 14 but this varied depending on age and Indigenous status of the cases.

Prevenar coverage rate

The Prevenar coverage rate for Indigenous children aged under one year (i.e., for those born after the commencement of the July 2001 schedule) is 58 per cent (fully vaccinated) and 14 per cent (partially vaccinated). The fully vaccinated rates varied from 69 per cent in northern Queensland to 45 per cent in central and 36 per cent in south Queensland. The coverage rates depended on the service provider. Table 1 shows the range of vaccination coverage rates.

Table 1. Prevenar coverage rate for Indigenous children aged under 1 year, Queensland, post July 2001 immunisation schedule, by service provider

Service provider	Fully vaccinated* %	Partially vaccinated* %
Aboriginal Health Service	76	21
Child health/community health services	82	12
Councils	55	23
Hospitals	50	13
Medical practices	32	12

The Pneumovax program for Indigenous adults aged 50 years or over began in north Queensland in 1995 and other areas in Queensland in 1997; the overall coverage up to 30 June 2002 is 53 per cent.

Fact or artefact?

The increase in notifications from 1997 to 2000 may be due to improved surveillance and awareness of this disease. The slight fall in rates in 2001 was chiefly due to the drop in rates for children aged under 2 years. Errors in estimating coverage rates may be due to duplicate records, inaccurate Indigenous identification, inaccurate records and/or incorrect population estimates. It may be too soon to assess the effectiveness of the vaccination strategy and ascertainment of cases may still be improving.

Enhanced surveillance of invasive pneumococcal disease in South Australia, 2002

Sharon Hart, Public Health Nurse, Communicable Disease Control Branch, South Australian Department of Human Services

Enhanced surveillance of invasive pneumococcal disease of cases in all age groups was commenced in South Australia in 2001. The total number of notified cases for 2001 was 118 cases. The dataset for 2001 is not complete, as IPD did not become notifiable in South Australia until January 2002. Data are obtained from a range of sources, including the referring doctor, infection control practitioners and hospital medical records.

The total number of cases from 1 January to 30 September 2002 was 129 cases. Of these, 101 (78.3 per cent) have been serogrouped. Of the remaining cases, 19 (14.7%) are waiting to be serogrouped, 3 (2.4%) isolates were non-viable and 6 (4.7%) were discarded by the primary laboratory. Continuous Quality Improvement activities have been developed to improve the completeness of the dataset.

Half the 2002 cases were female, 53 (41%) were aged under 5 years [36 (28%) aged under 2 years], 11 (8.6%) aged 5–14 years, 21 (16.4%) aged 15–49 years, and 44 (34%) aged over 50 years (31 (24%) aged 65 years or over).

There were 4 (3.1%) indigenous cases, one case aged 2–4 years, one case aged 5–14 years, two cases aged 15–49 years, and there were no indigenous cases aged over 50 years. Both of the cases aged 15–49 years had chronic illness but had not been immunised. Indigenous status is unknown for only one case (<1%) in 2002 compared to 24 cases (20.3%) in 2001.

Of the 36 cases aged under 2 years, 31 (86%) have no other identified risk factors. In the cases aged 2–4

years, 14 of the 17 cases (82.3%) have no other identified risk factors. However, in the cases aged 65 years and over, 25 (80.6%) had identified risk factors.

There have been 7 reported deaths for the year to date. There were no reported deaths in indigenous cases.

In the four cases aged 65 years or over, one case had been vaccinated, one case was not vaccinated and for two cases, vaccination status could not be determined.

Of the 53 cases aged under 5 years, 40 (75.5%) presented with a clinical bacteraemia, 6 (11.3%) presented with pneumonia and 4 (7.5%) presented with meningitis. In the 31 cases aged 65 years or over, 25 (80.6%) presented with pneumonia.

There were 7 cases of invasive pneumococcal disease in vaccinated individuals. Of these, 5 cases were serogroup 14, one was serogroup 9V (with intermediate resistance to penicillin) and one has not been serogrouped to date.

Invasive pneumococcal disease in Tasmania: work in progress

David Coleman, Communicable Disease Surveillance, Health and Human Services, Hobart

History

Invasive pneumococcal disease was made notifiable on 30 September 2000. All initial notifications are made by five laboratories, two of which are in public hospitals and three are private. This gives state-wide coverage of all age groups in the 470,000 population. Pneumococcal surveillance is partly incorporated in general surveillance activities which involves two full time staff.

Case definition

Case definition depends on identification of a culture-positive isolate from a sterile site. There is no local PCR available. Serotyping is done at the Microbiological Diagnostic Unit in Melbourne.

Review of individual hospital records identified 76 paediatric cases between 1994 and 2000. In 1998, 2.6 per cent (2 isolates) showed intermediate penicillin sensitivity; all were fully sensitive to third generation cephalosporins. No serotyping was available. The incidence was 28.5 cases per 100,000 population aged under five years and 54.3 cases per 100,000 population aged under 2 years. Thirty-four per cent of cases had risk factors, and there were 2 deaths.[1]

Surveillance process

The surveillance process involves receipt of laboratory reports, sending a questionnaire to the hospital seeking clinical information and the name of the patient's GP, followed by a questionnaire to the GP. In 2001 (retrospective from the middle of the year) 95 per cent of cases had hospital questionnaires, 44 per cent had GP questionnaires, and 65 per cent of the isolates were typed. By late 2002, 90 per cent of cases had hospital data (our aim is 100%); 48 per cent had GP data (aim is 95%); and typing was done or was pending in 94 per cent of cases.

Summary of cases in 2002 (to end September)

There were 52 cases of invasive pneumococcal disease notified in 2002 up to the end of September, (compared with 42 for same period in 2001) (Table 2). Twelve of the 15 cases aged 0–4 years were under 2 years of age (23% of total). Seventy-seven per cent of all cases were male: 14 of the 15 paediatric cases (93%) were male. Eight of 52 cases died (6 of the 8 deaths had risk factors). One unvaccinated child died of meningitis: this child had no risk factors. One isolate showed reduced sensitivity to penicillin and cephalosporins. Four men aged 58 to 80 years with risk to factors (respiratory/cardiac, renal, or malignant disease) had been vaccinated between 1997 and 2001; serotyping was pending. One boy may have had two episodes of infection: pneumococcal meningitis (serotype 19F) at 11 months; and pneumococcal bacteraemia at 16 months. Another boy who had received three doses of vaccine developed bacteraemia (serotype 6B) at three years of age.

The Tasmanian paediatric pneumococcal vaccination program commenced in 2001, with a target Indigenous population of 745 persons. The Pneumovax Indigenous program targets adults over 50 years, and persons aged 15 years or over, who have risk factors.

Table 2. Summary of invasive pneumococcal disease cases, Tasmania, 1 January to 30 September 2002, by age

Age (years)	Cases n (%)
0–4	15 (28)
<2	12 (23)
5–14	0 (-)
5–49	13 (25)
50+	24 (47)
Total	52

Reference

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Invasive pneumococcal disease surveillance in Victoria

Catherine Ferreira, Megan Counahan and Ross Andrews, Communicable Diseases Section, Department of Human Services, Victoria

Since May 2001, medical practitioners and laboratories have been required to notify the Department of Human Services, Victoria, of all cases of invasive pneumococcal disease. This report covers notification data for the 12 month period from 1 October 2001 to 30 September 2002. There were 449 notifications, 53 per cent were male, less than one per cent were identified as indigenous and there were 21 deaths (case fatality rate: 4.7%). The highest notification rate was among infants (68 cases per 100,000 population of children aged under 2 years).

Serotype information was available for 79 per cent (n=354) of cases. Among infants aged under 2 years, 89 per cent (57 of 64) of the serotypes isolated were either in or related to the 7-valent vaccine while for persons aged 2 years or over years, 98 per cent (284 of 290) were in or related to the 23-valent vaccine.

Pneumococcal vaccine for persons aged 65 years or over has been publicly funded in Victoria since 1998. Data on vaccine failures for cases detected from 1 January 2001 (prior to IPD becoming notifiable) up to 30 September 2002 were reviewed. Twenty-six vaccinated people developed IPD: one was a partially vaccinated infant who had received the 7-valent conjugate vaccine, the remainder had received the 23-valent polysaccharide vaccine. Less than one per cent of cases aged 2–64 years were vaccine failures while 16 per cent of those aged 65 years or over were vaccine failures. The high proportion of vaccine failures in the older age group is consistent with much higher vaccine coverage in this group.

Enhanced surveillance of invasive pneumococcal disease in Western Australia, 1 January to 30 September, 2002

Carolien Giele, Communicable Disease Control Branch, Department of Health, Western Australia.

Enhanced surveillance of invasive pneumococcal disease has been undertaken by the Vaccine Impact Surveillance Network for all age groups in Western Australia since 1996. IPD became notifiable in 2001, and since that time the Department of Health and the Vaccine Impact Surveillance Network have collaborated in data collection. After doctors or laboratories report IPD cases, demographic and clinical information is collected from managing physicians and/or by reviewing case notes.

During the nine-month period from January to September 2002, there were 159 IPD notifications, comprising 91 males and 68 females (notification rate: 11 cases per 100,000 population per year). The majority of cases occurred in three age groups: children under 5 years (n=55, 35%), adults aged 15–49 years (n=37, 23 per cent), and adults over 65 years (n=34, 21%). Notification rates were higher for Indigenous people compared to non-Indigenous people across all age groups. Incidence was highest in children aged under 2 years which was three times higher in Indigenous children (267 cases per 100,000 population per year) compared to non-Indigenous children (90 cases per 100,000 population per year). Of the 121 (76%) cases with ethnicity data, 21 (17 per cent) were Indigenous. There were 12 deaths (case fatality rate: 7 per cent), including 2 deaths in non-Indigenous children aged under 2 years.

Serotype and antibiotic susceptibility data were available for 139 (87 per cent) and 156 (98 per cent) IPD isolates, respectively. Of the 32 isolates from cases aged under 2 years, 78 per cent of serotypes were contained in the 7–valent conjugate vaccine. Of 107 isolates from cases aged 2 years or over, 86 per cent of serotypes were contained in the 23–valent polysaccharide vaccine. Twelve per cent (n=18) of isolates showed reduced susceptibility to penicillin.

These data show that available pneumococcal conjugate and polysaccharide vaccines provide good coverage for strains causing invasive disease in Western Australia. In this State provision of conjugate pneumococcal vaccine to all children as part of the funded childhood immunisation schedule would have considerably greater effect than the meningococcal serogroup C vaccine.

OzFoodNet: enhancing foodborne disease surveillance across Australia: quarterly report, July to September 2002

The OzFoodNet Working Group[1]

Introduction

The Commonwealth Department of Health and Ageing established OzFoodNet in 2000 to improve surveillance and conduct applied research on foodborne disease. The network is a collaborative project with Federal, State and Territory governments, academic institutions, and peak bodies for communicable disease control. During the third quarter, the Department of Health and Ageing commissioned an independent review of OzFoodNet activities. Professor John Kaldor from the National Centre in HIV Epidemiology and Clinical Research lead the review team, which concluded that the network had made substantial improvements to the quality of surveillance activities undertaken in the states and territories and recommended funding for a further five years. A full copy of the review report can be obtained from the Food Safety and Surveillance and Epidemiology Section of Department of Health and Ageing (telephone +61 2 6289 1555 or email: foodsafety@health.gov.au).

In July, the Department of Health and Ageing funded an epidemiologist in both New South Wales Health Department central office and the Northern Territory Department of Health and Community Services to participate in OzFoodNet activities. Previously, New South Wales had enhanced surveillance in the Hunter Region only. This extension of the coverage of OzFoodNet to all states and territories makes a significant improvement to Australia's ability to understand and investigate foodborne disease.

This third quarterly report of OzFoodNet for 2002 summarises the incidence of foodborne disease in the six states of Australia and the Australian Capital Territory between July and September 2002. During the third quarter of 2002, OzFoodNet continued to collect data on the incidence of gastroenteritis and its causes around Australia. All Australian jurisdictions collaborate in the OzFoodNet program of work.

In this report we analyse data by the date that a notification report was received by a health department or its closest equivalent. For historical comparison purposes, total numbers of notifications for the current quarter are compared to a mean of totals for the same quarter of the previous four years. In this report, data are reported for all jurisdictions where available. No data were available for the Northern Territory.

Notifications in the third quarter

In the third quarter of 2002, there were 3,212 notifications of *Campylobacter* infection, which was a decrease of 5.9 per cent from the mean of the same quarter for the previous four years (not including New South Wales). Only two jurisdictions reported increases for the quarter, which were South Australia (4.4%) and Tasmania (1.5%). The median age of cases in different sites ranged from between 27 to 33 years. All States reported that the male to female ratio of cases ranged from 0.9–1.2:1. The Far North Queensland Tropical Public Health Unit investigated one community-wide outbreak of *Campylobacter* infection during the quarter, which was linked to locally produced chicken.

OzFoodNet sites reported 1,010 cases of salmonellosis during the quarter, which was a 7.9 per cent increase from the same quarter in the previous four years. The largest increase was observed in Tasmania (122%). The Australian Capital Territory and Western Australia were the only jurisdictions that recorded decreases (18.5% and 8.3% respectively). The median age of cases ranged from 5 to 27 years in OzFoodNet sites. The male to female ratio ranged from 0.6:1 in South Australia to 2.2:1 in the Hunter.

The OzFoodNet Working Group is (in alphabetical order): Rosie Ashbolt (Tas), Robert Bell (Qld), Scott Crerar (FSANZ), Craig Dalton (Hunter PHU), Rod Givney (SA), Joy Gregory (Vic), Gillian Hall (NCEPH), Brigid Hardy (AFFA), Geoff Hogg (MDU), Rebecca Hundy (SA), Martyn Kirk (FSANZ), Karin Lalor (Vic), Ian McKay (DoHA), Peter Markey (NT), Tony Merritt (Hunter PHU), Geoff Millard (ACT), Jennie Musto (NSW), Leonie Neville (NSW), Jane Raupach (SA), Paul Roche (DoHA), Mohinder Sarna (WA), Craig Shadbolt (DoHA), Russell Stafford (Qld), Nola Tomaska (NCEPH), Leanne Unicomb (Hunter PHU), Craig Williams (FSANZ)

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During the quarter, four phage types of *Salmonella* Typhimurium were the most commonly notified *Salmonella* infections in three or more states: phage types 9, 126, 170 and 135. *S.* Typhimurium 170 continued as a national problem in multiple states, although case numbers decreased from those at the beginning of the year (Table 1).

During the quarter, New South Wales observed the emergence of *Salmonella* Typhimurium 197, and Tasmania reported a significant increase in *Salmonella* Typhimurium 135. Investigations in both jurisdictions did not reveal any obvious common source, although Tasmania reported one family outbreak of *S.* Typhimurium 135 suggesting contaminated egg consumption as the potential source. There were two cases of a novel serovar in humans—*Salmonella* Niarembe—notified in Tasmania from two family members who were refugees from the Sudan. In Western Australia, 12 cases of *Salmonella* Enteritidis 4b infection were reported in travellers who had returned from overseas. Reference laboratories previously categorised phage type 4b, as phage type 4 and have only recently started classifying them separately. Sites reported one *Salmonella* outbreak with a confirmed link to food, and investigated a further 10 clusters where no vehicle or source was identified. The cluster investigations included multiple serovars, including: Ohio (5 cases), Typhimurium 197 (8 cases), Hvittingfoss (11 cases), Lansing (4 cases), Havana (6 cases), Montevideo (6 cases), Typhimurium 141 (7 cases), Okatie (2 cases), Typhimurium 126 (6 cases) and Chester (5 cases).

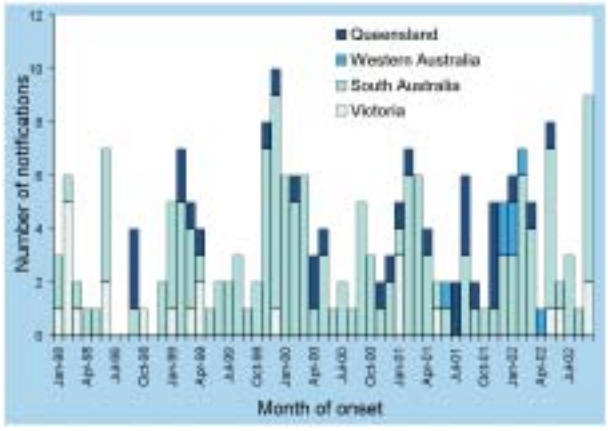
During the third quarter of 2002, the National Enteric Pathogen Surveillance Scheme reported that the five most common *Salmonella* infections nationally were *S*. Typhimurium 135 (79 cases), *S*. Typhimurium 9 (61 cases), *S*. Saintpaul (51 cases), *S*. Typhimurium 170 (50 cases), and *S*. Typhimurium 126 (33 cases). (Personal communication, Mark Veitch, The University of Melbourne, 15 October 2002).

State health departments received 16 notifications of listeriosis during the third quarter of 2002, which was 18.5 per cent higher than the same quarter for the previous four years (14 cases). Fifteen of these cases were reported in older people with severe immunocompromising conditions. The median age of these cases ranged from 42 to 80 years, and the overall male to female ratio was 0.5:1. One of the notifications during the quarter was a materno-foetal infection from Victoria.

OzFoodNet sites reported 15 cases of shiga-toxin producing *E. coli* infections during the quarter, compared to 11 cases for the same quarter in 2001 (Figure 1). South Australia reported 11 cases, Victoria two cases and New South Wales reported two cases of *E. coli*-associated haemolytic uraemic syndrome. No serotype was recorded for 53 per cent (10/19) of notified infections. Four were reported as *E. coli* O157 infections, three of which were from South Australia. The H type was recorded in only one of these cases, which was a case of *E. coli* O157:H7 associated with haemolytic uraemic syndrome in a New South Wales resident. The median age of cases in different sites ranged from 7 to 62 years, with females predominating (male to female ratio, 1:4). South Australia identified a common source outbreak of five cases of *E. coli* O26 associated with a petting zoo. An identical strain of *E. coli* was confirmed in the petting zoo environment as well as from faeces of pigs and alpacas.

OzFoodNet sites reported that during the quarter there were 27 cases of yersiniosis, which represented a 5.9 per cent increase on the mean of the previous four years. Queensland reported 17 cases during the quarter and reports the majority of notifications of yersiniosis in Australia. South Australia received 7 notifications during the quarter, which represents a 115 per cent increase from historical totals. Health agencies have seen continuing declines of yersiniosis in recent years and it is no longer notifiable in Victoria or included in the National Notifiable Diseases Surveillance System. Sites also reported 90 cases of shigellosis and 11 cases of typhoid, which represented decreases of 4.5 and 6.4 per cent from the mean of the previous four years, respectively.

Figure 1. Notifications of shiga-toxin producing *E. coli* in OzFoodNet sites, January 1999 to September 2002



NB. Cases of haemolytic uraemic syndrome where STEC were isolated are not shown on this graph.

Table 1. Number of notifications for the five most common *Salmonella* infections reported to OzFoodNet sites for the third quarter 2002 compared to third quarter 2001.

OzFoodNet site	Top five *Salmonella* infections	Number of cases				
		3rd Qtr 2002	3rd Qtr 2001	YTD 2002	Total 2001	Ratio*
ACT	Typhimurium 135	2	0	9	2	–
	Typhimurium 64	1	0	3	2	–
	Typhimurium 197	1	0	3	0	–
	Typhimurium 170	1	0	2	0	–
	Typhimurium U290	1	0	3	0	–
Hunter	Typhimurium 170	2	2	7	6	1.0
	Typhimurium 197	2	0	3	0	–
	Agona	1	0	8	1	–
	Enteritidis 6a	1	0	1	0	–
	Muenchen	1	0	1	0	–
NSW	Typhimurium 135	23	42	159	201	0.5
	Typhimurium 9	18	21	236	32	0.9
	Typhimurium 170	16	11	107	35	1.5
	Birkenhead	13	8	77	89	1.6
	Typhimurium 197	12	0	23	1	–
Qld	Saintpaul	29	15	199	173	1.9
	Virchow 8	26	22	238	183	1.2
	Birkenhead	17	6	103	134	2.8
	Typhimurium 170	15	2	69	20	7.5
	Aberdeen	14	13	97	81	1.1
SA	Typhimurium 9	6	2	19	49	3.0
	Typhimurium 126	6	49	33	110	0.1
	Typhimurium 108	5	6	25	31	0.8
	Typhimurium 135	5	3	12	24	1.7
	Typhimurium 8	4	1	56	3	4.0
Tas	Typhimurium 135	6	1	18	4	6.0
	Niarembe	2	0	2	0	–
	Mississippi	2	4	62	102	0.5
	Enteritidis 4	1	1	1	3	1.0
	Agona	1	0	2	2	–
Vic	Typhimurium 135	25	12	159	92	2.1
	Typhimurium 9	23	17	119	127	1.3
	Typhimurium 170	19	7	145	72	2.7
	Typhimurium 126	16	3	52	16	5.3
	Hvittingfoss	10	1	13	10	10.0
WA	Enteritidis 4B	12	1	24	3	12.0
	Typhimurium 9	10	1	42	18	0.0
	Chester	7	7	21	31	1.0
	Typhimurium 135A	7	1	25	17	7.0
	Typhimurium 126	6	0		2	–

* Ratio of cases for the third quarter 2002 to the third quarter 2001.

Table 2. Outbreaks reported by OzFoodNet sites, July to September 2002

State	Month of outbreak	Setting	Agent responsible	Number exposed	Number affected*	Evidence	Responsible vehicles
NSW	July	Restaurant	Unknown	130	30	D	Unknown
	August	Restaurant	Unknown	19	5	D	Unknown
Qld	August	Community	*C. jejuni*	Unknown	>24	M	Retail chicken meat
	September	Restaurant	Unknown	23	16	D	Unknown
Vic	August	Nursing Home	*C. perfringens*	69	15	D	Gravy suspected
	August	Restaurant	*S.* Typhimurium 135	Unknown	12	D	Spring rolls or prawn toasts suspected
WA	August	Mine site	Unknown	Unknown	23	D	Unknown
	August	Conference	Suspected viral	1,100	Unknown	A	Oyster shooters

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.
* The number affected is calculated from the proportion of people interviewed who were ill, multiplied by the number of people exposed.

Foodborne disease outbreaks

During the third quarter of 2002, OzFoodNet sites reported 8 outbreaks of gastrointestinal infections with a probable food source, compared to 17 outbreaks for the third quarter of 2001 (Table 2). The outbreaks affected more than 125 people, although the number affected in two outbreaks was unknown. Ten people were hospitalised, however no deaths were reported. Sites conducted two retrospective cohort studies and two case control studies to investigate these outbreaks. The remainder of investigations relied on descriptive information.

Six of the outbreaks occurred in August. One outbreak was due to *Salmonella* Typhimurium 135, one due to *Campylobacter jejuni,* one due to *Clostridium perfringens* and the remainder were of unknown aetiology. Five of the outbreaks occurred in association with meals at restaurants, or conferences or functions. The majority (75%) of investigations relied on descriptive epidemiology, with only one outbreak confirming the vehicle microbiologically and another using a case control study to identify the vehicle.

The outbreak of *C. jejuni* was a community-wide outbreak in Far North Queensland that was associated with locally supplied chicken meat. Investigators conducted a traceback of chicken consumption and found that 80 per cent of all chicken meat sampled for retail sale was contaminated with *Campylobacter*. Isolates from chicken meat supplied by a local abattoir were indistinguishable from human isolates using flagellin A gene typing and pulsed field gel electrophoresis.

Victoria reported an outbreak of *C. perfringens* where 22 per cent (15/69) of residents of a nursing home became ill following a common meal. The suspect food vehicle was gravy. Victoria also reported an outbreak of 12 cases of *Salmonella* Typhimurium 135 that was suspected to be caused by consumption of spring rolls or prawn toasts. The spring rolls contained raw chicken mince and may have been inadequately cooked.

Western Australia reported an outbreak of suspected viral gastroenteritis amongst delegates at an international conference. The vehicle for the outbreak was identified on epidemiological grounds as 'oyster shooters'. Conference organisers served the oysters in small glasses of tomato juice at a cocktail hour. The frozen oysters were imported and the packaging information indicated that the product should be cooked before consumption. No pathogens were isolated from a different batch of the same brand of oysters, although none of the implicated batch was available for testing.

OzFoodNet sites also reported significant numbers of person-to-person spread outbreaks of norovirus, particularly genogroup 2, occurring in hospitals and aged care settings. The numbers of outbreaks appeared to be considerably higher than reports from previous years, although the reasons for this are not known.

Applied research

In the third quarter of 2002, OzFoodNet concluded the national *Campylobacter* case control study with five sites interviewing 1,986 study subjects. The study recruited 881 cases and 883 controls who were aged 5 years or more, and 138 cases and 134 controls under the age of 5 years. South Australia completed data collection for a pilot of a case control study examining risk factors for shiga-toxin producing *E. coli*. OzFoodNet sites interviewed 29 patients as part of the *Listeria* case control study, four of which were infections acquired during pregnancy. Sites continued to recruit patients and controls for the national *Salmonella* Enteritidis case control study and studies into locally endemic *Salmonella* serovars.

In the third quarter of 2002, 900 people were interviewed as part of the national OzFoodNet gastroenteritis survey. Overall 9.8 per cent of people experienced gastroenteritis compared with 9.4 per cent for the previous quarter. Residents of Tasmania reported the highest crude proportion of people experiencing gastroenteritis in the previous month, while Victorian residents reported the lowest.

In the month of August, the national survey concluded after running for a full year. In total 11.2 per cent (683/6,092) of respondents reported any diarrhoea or vomiting in the previous month (Figure 2). There were noticeable differences by season in different jurisdictions (Table 3). This is self-reported gastroenteritis and does not take into account the mode of transmission. The data collected in this survey will contribute to OzFoodNet's estimation of the burden of gastroenteritis due to food in Australia. During the quarter, OzFoodNet held discussions with national and international experts about the proportion of different enteric pathogens that might be ascribed to transmission from food.

Figure 2. Unweighted results of the national OzFoodNet gastroenteritis survey showing the proportion of respondents reporting an episode of gastroenteritis in the previous month (n=6,092), September 2001 to August 2002

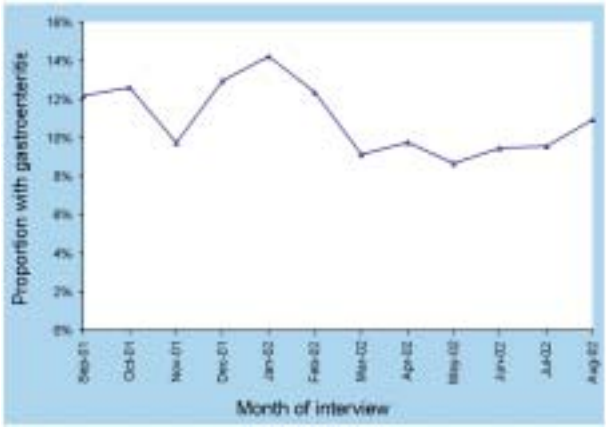


Table 3. Crude proportions of people reporting gastroenteritis in the previous month, Australia, 2001 to 2002, by season (n=6,092)

State	Proportion of respondents reporting gastroenteritis, by season (%)				
	Spring 2001	Summer 2001–02	Autumn 2002	Winter 2002	Total
New South Wales*	9.8	14.8	8.4	8.3	10.3
Northern Territory	19.9	19.3	12.9	12.9	16.3
Queensland	13.1	9.0	7.1	9.1	9.6
South Australia	10.6	12.5	8.4	13.6	11.3
Tasmania	9.5	10.9	8.8	11.6	10.2
Victoria	9.8	12.4	10.1	7.3	9.9
Western Australia	10.5	12.7	6.9	9.2	9.8
Total	11.5	13.2	9.2	10.0	11.0

* Includes the Australian Capital Territory and an over sample for the Hunter region of New South Wales.

Mumps and rubella surveillance in Victoria, 1993 to 2000

Rebecca J Guy,[1,2] Ross M Andrews,[1] Priscilla M Robinson,[1] Stephen B Lambert[2,3]

Abstract

Despite improving childhood coverage of the measles-mumps-rubella vaccine (MMR) in Victoria during the 1990s, mumps and rubella notifications in age groups eligible for vaccination persisted. This study reviewed the mumps and rubella surveillance data from 1993 to 2000 with a specific focus on method of diagnosis. There were 474 notifications of mumps over the seven-year period (annual median 61, range 40 to 77) and 3,544 notifications of rubella (annual median 297, range 66 to 1,165). The highest notifications rates for mumps were consistently among the 1–4 and 5–9 year age groups, whereas there was a marked change in the age distribution of rubella notifications during this interval. A large rubella outbreak occurred in 1995 with 1,165 notifications; the highest notification rates were males aged 15–24 years, infants under one year of age (males and females), and those aged 5–14 years (males and females), respectively. The susceptibility of 5–24 year olds reflects historical changes to the Australian Standard Vaccination Schedule. Rubella notifications returned to baseline levels in 1998 with the highest notification rates in infants aged under one year, and children aged 1–4 years. For both mumps and rubella, the majority of notifications for all age groups were clinically diagnosed, and were most common in children. *Commun Dis Intell* 2003;27:94–99.

Keywords: mumps, rubella, surveillance, immunisation

Introduction

Measles, mumps and rubella were common viral infections in childhood during the pre-vaccine era. Mumps causes aseptic meningitis in up to 10 per cent of cases and was the leading cause of viral encephalitis prior to the implementation of vaccination programs.[1] Whilst acute rubella infection is usually mild, antenatal infection can result in miscarriage, foetal death, and congenital rubella syndrome.[2]

In Australia, monovalent vaccines for measles and rubella were first available 30 years ago, with a monovalent mumps vaccine available for at least 20 years (Table 1).[3] In 1989 a trivalent measles, mumps and rubella vaccine became available and a two-dose schedule has been recommended in the Australian Standard Vaccination Schedule for both males and females since 1993.[3]

Despite improving MMR vaccination coverage in Victorian children aged 2 years (from 78% in 1994–95[4] to 92% in 2000[5]), a review of measles epidemiology between 1992 and 1996 demonstrated continuing high rates of notification in children, with the majority of these based on clinical diagnosis alone.[6] Enhanced measles surveillance in Victoria since 1997 has shown that a clinical diagnosis of measles has a low positive predictive value, overestimating disease incidence, particularly in young children eligible for vaccination.[7]

Surveillance of mumps and rubella during the 1990s showed persisting levels of notifications from age groups who should have been protected by high and improving vaccination coverage rates. To gain a better understanding of the epidemiology of mumps and rubella, surveillance data from 1993 to 2000 were reviewed, with a specific focus on the methods of diagnosis employed.

Methods

Data source

The Victorian 1990 Health (Infectious Disease) Regulations require medical officers and laboratories to notify cases of mumps and rubella to the Department of Human Services within 7 days of diagnosis. The notification form included notifier details, the notified disease, onset date and demographic data of the cases. Notification data were electronically stored in a computer database. Notifications received from laboratories were classified as being laboratory confirmed.

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Case definitions

The National Health and Medical Research Council surveillance case definitions for mumps and rubella during the period under review are shown in the Box.[8] Notifications were not routinely checked to ascertain whether cases met the clinical criteria of the National Health and Medical Research Council definition.

Data analysis

All notifications of mumps and rubella received by the Department of Human Services between 1 January 1993 and 31 December 2000 were collated and analysed using MS Excel and Epi Info version 6.04d.

Age- and sex-specific annual notification rates were calculated using Victorian mid-year population data from the Australian Bureau of Statistics. Rates were calculated for total notifications and laboratory-confirmed notifications for both diseases. Rates were calculated using the date of notification as the date of onset was not available for all notifications. For rubella sex- and age-specific laboratory-confirmed rates in an outbreak year (1995) were compared with a non-outbreak period when the number of notifications was at an all time low (1998 to 2000). Age groups used for analysing each disease were decided on by identifying changes in local epidemiology and vaccination policy (Table 1).

For notifications in the year 2000 for children aged 1–4 years the MMR immunisation status was retrieved from the Australian Childhood Immunisation Register (ACIR).

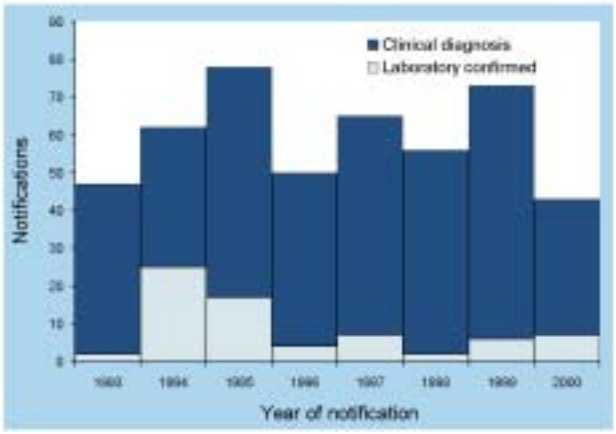
Results

Mumps

There were 474 mumps notifications received by the Victorian Department of Human Services between 1 January 1993 and 31 December 2000. The median number of annual notifications was 61 (range 40 to 77). There was no seasonal variation. Only 15 per cent of these notifications were laboratory confirmed (Figure 1).

The highest notification rates were seen in the 1–4 years age group (average annual rate 5.7 per 100,000 population, range 4.3 to 7.7 per 100,000 population) and those aged 5–9 years (average annual rate 5.6 per 100,000 population, range 2.5 to 8.6 per 100,000 population) (Table 2, Figure 2).

Figure 1. Annual notifications of mumps, Victoria, 1993 to 2000, by notification type



Box. National Health and Medical Research Council case definitions for mumps and rubella, 1994

Mumps

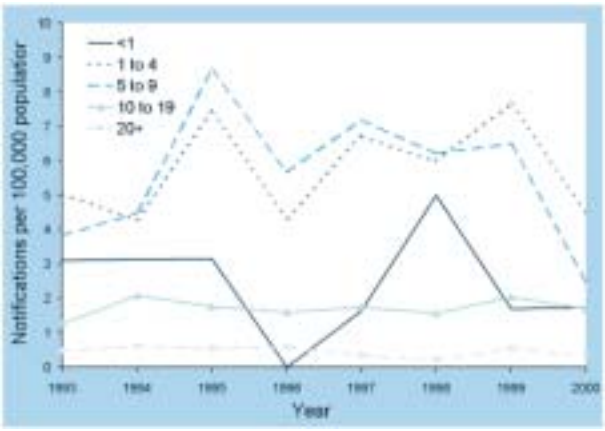
- The mumps virus isolated from a clinical specimen; OR
- a significant rise in mumps antibody level by any standard serological assay, except following immunisation; OR
- a clinically compatible illness (unilateral or bilateral swelling of the parotid or other salivary glands lasting two days or more without other apparent cause).

Rubella

A generalised maculopapular rash and a fever, plus:

- one or more of arthralgia/arthritis, lymphadenopathy and conjunctivitis, plus
- an epidemiological link to a confirmed case; OR
- a demonstrated rubella-specific IgM antibody, except following immunisation; OR
- a fourfold or greater change in rubella antibody titre between acute and convalescent phase sera obtained at least two weeks apart; OR
- rubella virus isolated from a clinical specimen.

Figure 2. Mumps notification rates per 100,000 population, Victoria, 1993 to 2000, by age group



Between 1993 and 2000 there were no laboratory-confirmed notifications in infants aged under one year. The proportion of laboratory-confirmed notifications increased with age, reaching a maximum of 37 per cent for those aged 20 years and above (Table 2).

In 2000, there were 11 notifications in the 1–4 years age group and all were clinically diagnosed. According to the ACIR, 10 cases had received their first MMR vaccination due at 12 months.

Table 1. Significant events in measles, mumps and rubella immunisation practice in Australia

Year	Intervention
1968	Live, attenuated measles vaccine approved
1970	Rubella vaccine approved, measles vaccine widely available
1971	Measles vaccine initially recommended for 15-month-old infants
	School girl rubella program for 10–14 year old girls and non-immune women of child-bearing age
1975	First national immunisation schedule: measles vaccination for infants at 12 months of age
1980	Mumps vaccine approved for infants aged 12–15 months
1982	Combined measles-mumps vaccine (MM) available
1989	Measles-mumps-rubella vaccine (MMR) available
1993	Childhood immunisation schedule included 2nd dose of MMR for children aged 10–16 years (MMR replaced the school girl rubella vaccination program)
1998	Measles Control Campaign with shift in timing of 2nd dose to children aged 4–5 years
2000	Recommended age for 2nd MMR dose changed to 4 years

Adapted from historical table available at http://www.ncirs.usyd.edu.au/publ/publ-79-histtab3.html

Table 2. Laboratory and clinical notifications of mumps, Victoria, 1993 to 2000, by age group

Age group	Clinical only		Laboratory confirmed		Total	Average annual rate (per 100,000 population)
	n	%	n	%	n	
<1	11	(100)	0	(0)	11	2.3
1–4	112	(97)	4	(3)	116	5.7
5–9	136	(94)	8	(6)	144	5.6
10–19	72	(83)	15	(17)	87	1.7
20+	73	(63)	43	(37)	116	0.4
Total	404	(83)	70	(17)	474	1.3

Rubella

There were 3,544 rubella notifications received by the Victorian Department of Human Services between 1 January 1993 and 31 December 2000 (Figure 3), with 2 outbreaks of rubella identified. Figure 3 includes the tail end of an outbreak that commenced in 1992 and concluded in 1993. The second outbreak began in mid-1995, peaked quickly (1,165 notifications received in 1995), with notifications declining, but remaining above pre-1995 baseline levels until 1998. Notifications declined from 189 in 1998, to 123 in 1999 and to 66 in 2000, which was the lowest annual figure on record (Figure 3).

Laboratory-confirmed notifications, as a proportion of all notifications, were highest during the 1995 outbreak reaching 52 per cent in males and 38 per cent in females (Figure 3, Table 3). This proportion decreased to 16 per cent in males and 14 per cent in females for the period between 1998 and 2000. For both 1995 and the period between 1998 and 2000, the proportion of notifications that were laboratory-confirmed was lower in the younger age groups compared with older age groups (Table 3). In 1995 the highest notification rates were in males aged 15–24 years (153 cases per 100,000 population), male and female infants combined aged under one year (98 cases per 100,000 population), and male and females combined aged 5–14 years (38 cases per 100,000 population) (Table 3, Figure 4).

In 1995 the male to female ratio for all notifications in the 15–24 year age range was 14.5:1 but had fallen to 4:1 between 1998 and 2000. In contrast, the male to female ratio for those aged 5–14 years remained relatively unchanged (1.4:1 in 1995 and 1.8:1 between 1998 and 2000). Between 1998 and 2000 (non-outbreak period) the highest notification rate was in infants aged under one year and second highest in the 1–4 year age group (Table 3, Figure 4).

In the year 2000 there were 24 notifications in children aged 1–4 years; 23 were clinically diagnosed (19 had received at least one dose of MMR vaccination according to the ACIR) and one was laboratory diagnosed (no doses of MMR according to the ACIR).

Figure 3. Annual notifications of rubella, Victoria, 1993 to 2000, by notification type

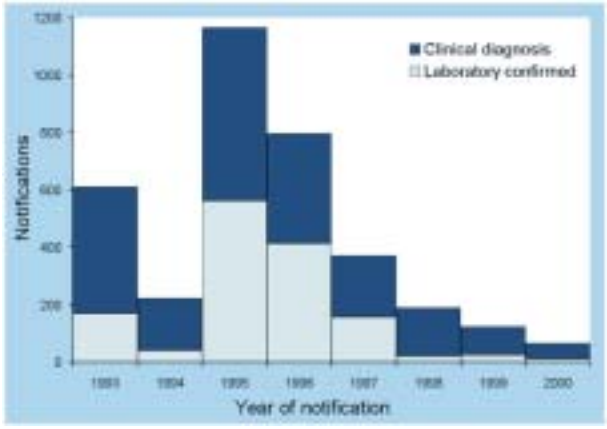


Figure 4. Notification rates for rubella per 100,000 population, Victoria, 1993 to 2000, by age group

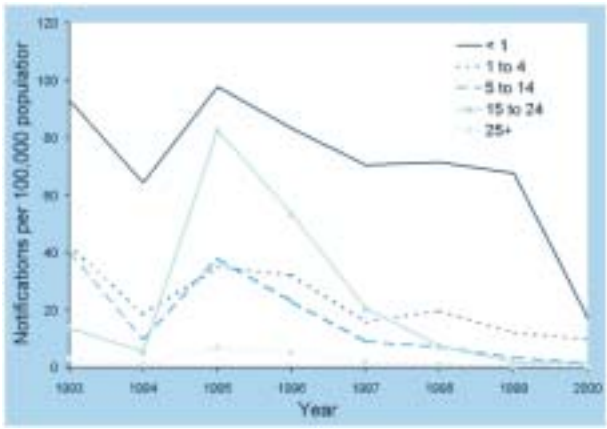


Table 3. Notification rates for rubella per 100,000 population, Victoria, 1995 (annual rate) and 1998 to 2000 (average annual rate), by age group, sex and proportion that were laboratory-confirmed

Age group	1995				1998 to 2000			
	Male		Female		Male		Female	
	Rate	LC %	Rate	LC %	Rate	LC %	Rate	LC %
<1	110.6	19	84.3	8	59.0	0	45.4	0
1–4	38.1	0	32.1	8	13.8	5	12.2	2
5–14	42.7	42	32.3	37	5.3	2	3.0	15
15–24	153.2	56	10.9	50	5.6	42	1.5	25
25+	11.3	69	2.9	73	0.7	47	0.3	55
Total	40.5	52	10.7	38	3.5	16	2.0	14

LC Laboratory-confirmed

Discussion

This review of Victorian surveillance data between 1993 and 2000 demonstrated little change in mumps epidemiology with notification rates, the proportion of laboratory-confirmed cases, and age groups affected remaining stable. The mumps notification rates were highest in the 1–4 and 5–9 year age groups. The rubella surveillance data, by contrast, demonstrated a changing epidemiology. A large rubella outbreak occurred in 1995 with the highest notification rates in males aged 15–24 years, infants under one year of age (males and females), and those aged 5–14 years (males and females), respectively. By 1998 rubella notifications had returned to pre-1995 baseline levels. The highest rubella notification rates between 1998 and 2000 were seen in infants aged under one year, with the second highest rates in the 1–4 year age group. For both mumps and rubella, laboratory confirmation in these younger age groups was infrequent. A similar picture has been seen for measles where it was demonstrated that the low rate of laboratory confirmation for notifications from the younger age groups represented a true absence of disease.[7] On the basis of this, we hypothesise that low rates of laboratory-confirmed mumps and rubella are indicative of low levels of disease.

Enhanced measles surveillance introduced for Victoria in 1997 has shown that passive surveillance based on the clinical diagnosis of measles was inaccurate due to the poor predictive value of a clinical diagnosis. Of 317 notifications, 258 had serology performed and only 19 (6%) of these were laboratory confirmed with measles.[7] Similarly, in populations with high levels of MMR coverage simple rash illnesses (diagnosed as rubella) and parotitis (a classical feature of mumps) may often be due to other aetiologies.[9,10] In this study, a large proportion of notifications for both mumps and rubella were from children in age groups targeted by vaccination and were based on a clinical diagnosis only, which may have resulted in an overestimation of cases. This has been highlighted by other enhanced surveillance systems in countries with widespread vaccination. Enhanced mumps surveillance in the United Kingdom[11] and Texas[12] showed that despite a large proportion of notified cases being laboratory tested only 3 per cent and 7 per cent, respectively, were laboratory confirmed. Rubella enhanced surveillance during the National England and Wales measles and rubella immunisation strategy campaign in 1994 showed only 29 per cent of notified cases were laboratory confirmed as rubella.[13]

The higher rubella notification rates in the 5–14 and 15–24 year age ranges, reflect susceptibility due to lack of exposure to the virus in childhood and lack of vaccination due to changes in the schedule. Infant rubella vaccination (as part of MMR) was only available from 1989 and the second dose strategy for teenagers commenced in Victoria in 1994/95.[14] The national serosurvey performed prior to the 1998 Measles Control Campaign showed the 10–12 year age group had the lowest proportion of immune subjects (60%) but this gap in immunity was removed by the Campaign.[14] Rubella vaccination between 1971 and 1993 targeted adolescent schoolgirls only, leaving contemporary males susceptible.[14,15] Prior to the Measles Control Campaign, females had significantly higher rubella seropositivity rates (97%) than males (85%) in the 16–39 year age range.[14]

High vaccine coverage in children should continue to ensure minimal mumps and rubella virus circulation in the younger age groups. Continuing rubella circulation will result in cases in males currently aged in their 20s[15] and for mumps it is likely that true cases will also be in adults. As has been seen with measles epidemiology, gradual introduction of vaccination programs with slowly increasing coverage rates leads to the development of susceptible cohorts, who neither get wild virus infection nor receive vaccination.[16]

This study highlights the difficulties in interpreting notifications rates derived from passive surveillance data as the incidence of vaccine preventable diseases declines. To gain a better understanding of the true epidemiology of mumps and rubella in Victoria the Department of Human Services is undertaking a period of enhanced surveillance for mumps and rubella. The system will be similar to that introduced for measles in 1997,[7] focussing on laboratory confirmation of all cases. Early results from enhanced mumps surveillance have shown that of 16 notifications made in July 2001, only two were laboratory confirmed.[17] These results suggest mumps, at least, is less common than notifications would lead us to believe.

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Letter to the Editor

Varicella surveillance: simpler than you think?

In the article 'Surveillance of viral pathogens in Australia: varicella-zoster virus' published in the previous issue of *Communicable Diseases Intelligence*,[1] the authors dismiss passive surveillance as unworkable because of 'the large numbers of cases of chickenpox and the small proportion of cases who seek medical attention'. It is not clear if they believe that passive surveillance is impractical because too many or too few notifications would be made.

Effective surveillance rarely requires notification of every single case of a disease. Most often surveillance is a tool to identify change which will prompt investigation and, if necessary, public health action. The conventional notifiable diseases system, for example, depends on clinical or laboratory filtering of surveillance data to achieve an acceptable level of specificity and reduce the volume of data to a manageable quantity.

The objectives of chickenpox surveillance are to measure the impact of immunisation on the age distribution of cases and the form of the disease (primary infection or zoster). There is also some value for policy purposes of directly demonstrating a decline in the number of cases. All of these data are essential in the evaluation of a program.

If varicella immunisation is recommended for the Australian Standard Vaccination Schedule but not funded by the Commonwealth Government, it is possible that the partial immunisation of the population will result in an increase of the average age of acquisition and a parallel increase in the number of cases of severe disease.[2] It is also possible that the absence of regular priming of immunity to varicella-zoster by contact with chickenpox in people who have previously had chickenpox may result in increased numbers of cases of zoster.[3]

In South Australia varicella-zoster infection was made a notifiable disease in January 2002. General practitioners were informed by a single letter posted in April 2002. Since then 1,208 notifications have been received: 662 of chickenpox, 355 of zoster and 191 where the clinical syndrome was not stated. Varicella-zoster notifications made up 15 per cent of all notifications made in South Australia in this period.

The age distribution of the South Australian data is consistent with the expected age distribution for chickenpox (Figure 1) and zoster (Figure 2).

However, the expected temperate climate seasonal peak for chickenpox in late winter and spring[4] is not reflected by the first year's data (Figure 3).

Figure 1. Number of notified cases of chickenpox, South Australia, 1 January 2002 to 31 January 2003, by date of onset, and age group and sex

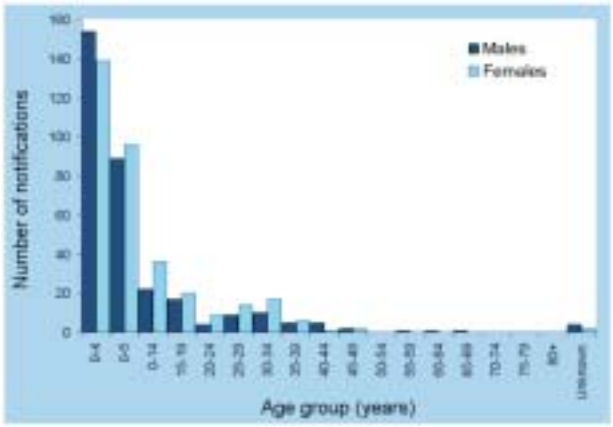


Figure 2. Number of notified cases of zoster, South Australia, 1 January 2002 to 31 January 2003, by date of onset, and age group and sex

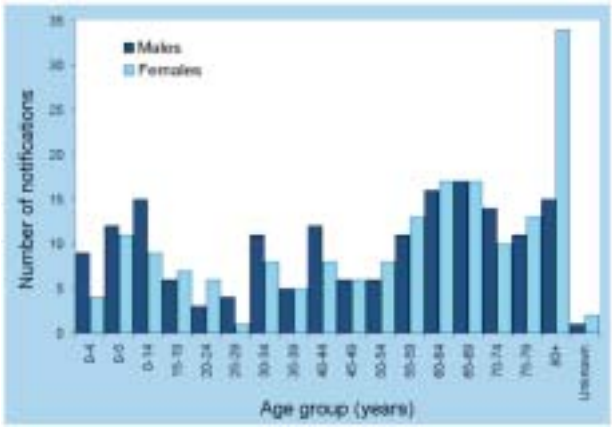
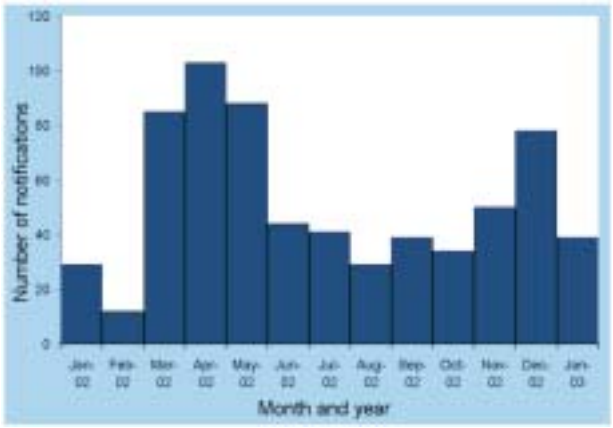


Figure 3. Number of notified cases of chickenpox, South Australia, 1 January 2002 to 31 January 2003, by month of onset



Assuming the real incidence of chickenpox is one birth cohort per year (n=17,000 for South Australia approximately) the sensitivity of this notification system for chickenpox is 4 per cent. The apparent reversal of the expected seasonal chickenpox incidence may reflect the actual situation in 2002 but, more likely, indicates that passive notification is not sensitive enough to detect seasonal trends

Given the very small effort that has been made so far in South Australia to encourage doctors to notify varicella-zoster infection, it is likely that the sensitivity of the system could be markedly improved. Even the current data may be adequate as a baseline to monitor changes in age distribution. The estimated cost to the South Australian Communicable Disease Control Branch of processing these varicella-zoster notifications was just over $1,000 for the year.

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Uptake of influenza vaccine among Aboriginal and Torres Strait Island adults in north Queensland, 2002

Jeffrey N Hanna,[1] Bradley G McCulloch[2]

Abstract

Since 1999, the Commonwealth has provided free annual influenza vaccine for all at-risk Aboriginal and Torres Strait Islander adults. The uptake of the vaccine in this population in north Queensland in 2002 was determined using the state-wide computerised immunisation register. Although 59.4 per cent of Aboriginal and Torres Strait Islander adults aged 50 years or over were vaccinated, the uptake in this age group exceeded 80 per cent in only the Cape York and Torres Strait and North Peninsula Area Health Service Districts (HSDs). Assuming that a third of Aboriginal and Torres Strait Islander adults 15–49 years of age had a medical risk factor, it was estimated that 85 per cent of those at-risk were vaccinated. There was considerable variation between HSDs, ranging from 159 per cent uptake in the Torres Strait and North Peninsula Area Health Service District to 48.7 per cent in the Cairns HSD. *Commun Dis Intell* 2003;27:102–104.

Keywords: influenza, Indigenous population, immunisation

Introduction

Annual influenza vaccination is recommended for those at increased risk of influenza-related complications, including all Aboriginal and Torres Strait Islander adults aged 50 years or over. Since 1999 the Commonwealth has provided funding for free, annual immunisation of all Aboriginal and Torres Strait Islander adults aged 50 years or over and all Aboriginal and Torres Strait Islander adults 15–49 years of age with a medical risk factor.[2]

Since 1999, details of pneumococcal and influenza vaccines provided to Aboriginal and Torres Strait Islander adults in Queensland have been recorded on the state-wide immunisation database, Vaccination Information Vaccination Administration System (VIVAS).[3] This report details the uptake of the influenza vaccine in Aboriginal and Torres Strait Islander adults in north Queensland in 2002, using data extracted from VIVAS.

Methods

A listing of all influenza vaccinations for 2002 was extracted from the VIVAS database. These data were checked for consistency with regards to address, date of birth and vaccination. The data then aggregated into the Health Services Districts,[4] as approximated by an aggregation of 1996 Statistical Local Area boundaries.[5] The counts of Aboriginal and Torres Strait Islander populations in north Queensland by HSDs were obtained from the 2001 national census.[6]

Because the vaccine is recommended for all Aboriginal and Torres Strait Islander adults aged 50 years or over, the uptake in this age group in each HSD could be readily calculated. The prevalence of medical risk factors in Aboriginal and Torres Strait Islander adults aged 15–49 years is not known with any precision in north Queensland. However, the uptake in this age group was calculated assuming that one third of this age group has a risk factor and is therefore eligible for immunisation.

Results

The number of doses given to, and the uptake of influenza vaccine in, Aboriginal and Torres Strait Islander adults aged 50 years or over in north Queensland in 2002 are shown in Table 1. Table 2 compares the number of doses given to Aboriginal and Torres Strait Islander adults aged 50 years or over in 2002 with those given in 2001.

The number of doses given to, and the uptake of influenza vaccine (assuming a risk factor prevalence of 33%) in, Aboriginal and Torres Strait Islander adults 15–49 years of age in north Queensland in 2002 are shown in Table 3.

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Table 1 Influenza vaccine doses given to Aboriginal and Torres Strait Islander adults aged 50 years or over, north Queensland, 2002

Health Service District	Vaccinated n	Uptake* %
Bowen	52	28.4
Cairns	482	39.8
Cape York	405	88.0
Charters Towers	29	26.4
Innisfail	160	61.5
Mackay	131	43.7
Moranbah	4	11.1
Mt Isa and Gulf	556	57.8
Tablelands	235	59.6
Torres Strait and North Peninsula Area	758	87.3
Townsville	531	63.0
Total	3,343	59.4

* Based upon Census 2001 population estimates

Table 2. Influenza vaccine doses given to Aboriginal and Torres Strait Islander adults aged 50 years or over, north Queensland, 2001 and 2002

Health service District	2001	2002	Difference
Bowen	45	52	+7
Cairns	503	482	-21
Cape York	392	405	+13
Charters Towers	49	29	-20
Innisfail	148	160	+12
Mackay	117	131	+14
Moranbah	3	4	+1
Mt Isa and Gulf	561	556	-5
Tablelands	221	235	+14
Torres Strait and North Peninsula Area	722	758	+36
Townsville	514	531	+17
Total	3,275	3,343	+73

Table 3. Influenza vaccine doses given to Aboriginal and Torres Strait Islander adults 15–49 years of age, north Queensland, 2002

Health Service District	Vaccinated n	Uptake* %
Bowen	56	22.7
Cairns	1,009	48.7
Cape York	978	146.6
Charters Towers	49	31.4
Innisfail	335	84.2
Mackay	204	35.4
Moranbah	7	10.8
Mt Isa and Gulf	1,081	77.2
Tablelands	691	105.3
Torres Strait and North Peninsula Area	1,786	159.0
Townsville	1,523	88.4
Total	7,719	85.0

* Based upon the assumption that 33 per cent of the Census 2001 population estimate had a risk factor.

Discussion

There was an overall increase of approximately 480 doses of influenza vaccine used in north Queensland in 2002 compared to 2001 (data not shown). Most (approximately 410 doses) of the increase were in Aboriginal and Torres Strait Islander adults 15–49 years of age, and it is likely that most of the increase was in the Torres Strait and North Peninsula Area Health Services District.

The Commonwealth Department of Health and Ageing has suggested as an uptake target that 80 per cent of Aboriginal and Torres Strait Islander adults aged 50 years or over should receive the influenza vaccine annually.[2] Although both the Torres Strait and North Peninsula Area, and Cape York HSDs have reached the proposed target, the other HSDs have a considerable way to go. This is a concern; not only has this program been in place in north Queensland for at least five years,[7] but it also seems that many HSDs have made little recent progress (Table 2).

Because the prevalence of risk factors in Aboriginal and Torres Strait Islander adults 15–49 years of age is unknown, the uptakes in Table 3 are based on an assumption that 33 per cent of those in this age group have a risk factor. However, the uptakes of more than 100 per cent in three HSDs suggest that an estimated prevalence of risk factors of 33 per cent may be too low, and that an estimate of 45 per cent may be more accurate. Alternatively, the prevalence of risk factors may vary widely by HSD.

Nevertheless, assuming a risk factor prevalence of 33 per cent and an annual uptake target of 80 per cent of this group,[2] the low uptake in two HSDs with large populations in this age group, namely Cairns, and Mt Isa and Gulf, is of concern. The apparent difficultly in providing effective immunisation services to Aboriginal and Torres Strait Islander populations in Cairns, in particular, has been previously documented.[8]

High uptake of influenza vaccine is important for the immediate protection of at-risk Aboriginal and Torres Strait Islander adults. Concerns about pandemic influenza have highlighted the importance of high uptake of the vaccine 'in identified cohorts and high-risk groups' in the inter-pandemic period.[9] Further innovations will be required to improve influenza vaccine uptakes in Aboriginal and Torres Strait Islander adults aged 50 years or over, and in 'at risk' Aboriginal and Torres Strait Islander adults 15–49 years of age, particularly in Cairns.

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Variability of larval identification characters of exotic *Aedes albopictus* (Skuse) intercepted in Darwin, Northern Territory

Gisela D Lamche,[1] Peter I Whelan[2]

Abstract

There is no morphological identification key to satisfactorily distinguish between the larvae of *Aedes (Stegomyia)* species with special consideration to endemic and exotic species in Australia. Difficulty in differentiation between exotic *Aedes (Stegomyia) albopictus* (Skuse) and *Aedes (Stegomyia) scutellaris* Walker has been described previously.[1] Aedes (Stegomyia) larvae were collected during an interception from an overseas vessel in Darwin, Northern Territory, and link bred. The adults were identified as *Ae. albopictus*. The larval skins and larvae were used to describe the variation in hair features of larval segment VII that are used to identify *Ae. albopictus*. The median hair number of hair 1–VII was three, whereas the description in Huang's identification key[2] states four. The median of hair 2–VII was one, confirming Huang.[2] However, the variability was higher than described by Huang[2] and nearly half of the specimens showed different hair numbers on both sides. Individual specimens are therefore not clearly distinguished from other members of the *Aedes (Stegomyia) scutellaris* group. This paper also describes the detection, elimination and surveillance procedures following the interception. These were successful in preventing the establishment of exotic *Ae. albopictus* in the Northern Territory following the interception. *Commun Dis Intell* 2003;27:105–109.

Keywords: Aedes albopictus, mosquitoes, surveillance

Introduction

The larvae of some species of the *Aedes (Stegomyia) scutellaris* group are difficult to identify morphologically.[3] The collection location is often essential in assisting species identification because many species are allopatric.[4] The species *Aedes (Stegomyia) katherinensis* Woodhill, for example, is endemic to the Northern Territory of Australia and the species *Aedes (Stegomyia) scutellaris* Walker is distributed locally in the Torres Strait, Queensland and also in Papua New Guinea.[4,5,6] At present it is impossible to distinguish these two species at the larval stage.[1,7]

Identification keys generally encompass all species of a zoogeographic region. However, importation of exotic species occurs with increased transport and traffic due to globalization and new species can be introduced into a region.[8,9]

Correct identification of larval mosquitoes is essential to prevent the establishment of exotic species of the *Aedes (Stegomyia) scutellaris* group in Australia. *Aedes albopictus* is a vector of dengue fever, and the prevention of its establishment is a public health priority in both tropical and temperate Australia. *Aedes albopictus* has recently spread globally and is now established in many countries.[8,10] The species is present in countries to Australia's immediate north such as East Timor and Papua New Guinea, posing a continual threat to entering Australia.[2,11,12,13] Exotic larvae recently collected in Darwin, Northern Territory, were identified as either *Aedes (Stegomyia) albopictus* (Skuse) or *Ae. scutellaris*, but with the material available no conclusive identification was possible.[1] The differentiation of *Ae. albopictus* and *Ae. scutellaris* is usually carried out using a key for the South-East Asian region by Huang.[2] However, identification can be difficult when one of the key characteristics of intercepted larvae differs from the description. There is also the possibility of the interception of an undescribed species or a species such as *Aedes (Stegomyia) alorensis* Bonne-Wepster of which larvae have not been described.[1,12]

A routine quarantine inspection of unloaded overseas cargo at an international shipping company premise in Darwin Port, Northern Territory, found mosquito larvae in water pooling on mining equipment. The larvae were collected live. Some were identified at this stage while some were link bred to adults to confirm the larval identification. The reared adults

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were identified as *Ae. albopictus*. The larvae of a bulk sample and the larval skins of the link bred specimens provided material for the investigation of morphological features used to identify exotic and endemic *Aedes(Stegomyia)* larvae. A sample of four mosquito larvae sent from a survey in East Timor was also included in this examination.

This paper describes the investigation on the larval identification characters, as well as the interception and the elimination measures taken.

Identification

Twenty-four mosquito larvae were submitted by the Australian Quarantine Inspection Services (AQIS) to the Medical Entomology Branch in Darwin (MEB). Five females and five males were link bred successfully. All were identified as *Ae. albopictus* when using the identification keys by Huang.[2,12] Pupal skins were identified as *Ae. albopictus* according to Huang.[2] Fourteen larvae were not link bred and were preserved in 70 per cent ethanol for further examination.

The larval skins and larvae of the interception sample were analysed to distinguish between *Ae. albopictus* and *Ae. scutellaris* as in Huang.[2] Hair numbers on hairs 1–VII and 2–VII were counted on the left and the right side of each specimen (referred to as sides A and B), as differences on the two sides were observed. Therefore, 48 hair number counts were made for the 24 specimens (i.e., the larval skins of the five females and five males as well as the 14 larvae).

The hair length of hairs 1–VII was assessed in two classes, as 2 times the length or 2.5 times the length of hairs 5–VII. Other classes were not observed. Not all of the specimens were preserved well enough to clearly investigate this hair length parameter in all specimens and there was no difference between sides A and B of the individuals examined. There were 22 measurements of the hair length parameter from 22 specimens.

The variability of the hair characteristics is summarised in Table 1. The median number for hairs 1–VII was three and the median number for hairs 2–VII was one. The standard deviation revealed variability for both of these hair branches (Table 1). The sides A and B of individuals were observed to show different hair numbers in 11 out of 24 specimens examined.

A sample of four larvae sent from East Timor were identified as *Ae. albopictus*, although variability of the key identification characters was observed.[2] The larvae were investigated as described above and the results are also summarised in Table 1.

Detection and eradication measures

AQIS found the mosquito larvae during a routine inspection of a vessel on 2 January 2002 at premises of a shipping company at Darwin Port. The larvae were discovered in pooling of what was probably rainwater on oil exploration machinery. The machinery had been unloaded on 31 December 2001 from a cargo vessel which had arrived on 30 December

Table 1. Larval hair characteristics of *Aedes albopictus* from interception and East Timor sample and data provided by Huang[2]

	Hair 1–VII branches	Hair 2–VII branches	Length of hair 1–VII to hair 5–VII
Interception			
n	48	48	22
Minimum	2	1	2
Maximum	4	3	2.5
Average ± standard deviation	3.25 ± 0.70	1.19 ± 0.45	2.05 ± 0.15
Median	3	1	2
East Timor specimens			
n	8	8	4
Minimum	3	1	2
Maximum	4	2	2
Average ± standard deviation	3.25 ± 0.46	1.25 ± 0.46	2.0 ± 0.0
Median	3	1	2
Huang[2]			
Ae. albopictus	4 (3–4)	1 (1–2)	2
Alternative couplet	2 (2–3)	3–4	2.5

N Number of observations

* Key couplet leads to the *Aedes* (Stegomyia) *scutellaris* group members *Ae. downsi*, *Ae. patriciae*, *Ae. riversi*, *Ae. malayensis*, *Ae. alcasidi*, or *Ae. scutellaris*

2001. The inspecting officer removed the live mosquito larvae with their original water, which were taken to MEB and reared out as described above. Parts of the equipment were sprayed with the residual insecticide deltamethrin. The consignment was fumigated at 1.30pm on 2 January 2002.

The recent history of the exploration equipment was obtained from AQIS. In March 2001 it was used offshore in the Philippines. On 19 December 2001 it was transferred to Singapore by vessel, which then departed with the equipment on 20 December 2001. The equipment arrived in Darwin via Dili, East Timor, on 30 December 2001 and was discharged in the late afternoon on 31 December 2001.

The larval sample taken to MEB also contained 5 pupal skins and one unidentifiable dead adult. This indicated that live adults could have emerged and hence the interception was treated as a risk interception. MEB conducted a fogging operation using a Leco HD aerosol fogger applying bioresmethrin on the evening of 2 January 2002 from 6.00pm to 7.00pm within the premises of the shipping company and neighbouring port areas.

Container breeding survey

A container (receptacle) breeding survey was conducted as a joint operation between MEB and AQIS within the 400 m exclusion zone of the shipping company. Most premises were inspected on 22 January 2002, one week after substantial rain. Two remaining premises were inspected on 11 February 2002, 5 days after substantial rainfall.

No exotic mosquitoes were found (Table 2). All containers were sprayed with the residual insecticide deltamethrin. The container index (the percentage of water holding containers examined that contain mosquito larvae) was calculated according to World Health Organization guidelines (Table 2).[14]

Ovitrap surveillance

MEB set two ovitraps in vegetated areas within the neighbourhood of the shipping company premise for a time period of two months. These were in addition to five routine ovitraps set in the port area. The ovitraps were collected fortnightly, commencing on 7 January 2002 and ending on 4 March 2002 with the trapping results presented in Table 3. Following the interception, exotic *Aedes* species were not recorded from any of the additional traps or from the five routine ovitraps of the MEB, or the six AQIS ovitraps in the area.

Table 2. Summary of container breeding survey conducted around the port area of Darwin

Survey parameter	Number/index
Premises inspected	6
Containers found	258
Containers holding water	103
Containers breeding mosquitoes	17
Container index	16.5
Species found†	
Ochlerotatus notoscriptus	16.5
Culex quinquefasciatus	4.9
Ochlerotatus tremulus	2.9
Culex annulirostris	1.0

* Container index = percentage of water holding containers examined that contain mosquito larvae.[14]

† The index value calculated for each species is derived as: Species container index = percentage of water holding containers that contain mosquito larvae of this species.

Table 3. Ovitrap results of two additional traps set for eight weeks

Site description	Sampled n	Positive n	Positive %	Times species recorded (total number of larvae/pupae)				
				Ochlerotatus notoscriptus pupae		*Ochlerotatus notoscriptus* larvae		Nil mosquitoes
				Count of species*	Sum of pupae	Count of species*	Sum of larvae	Count of occurrence
Near drain along Frances Bay Drive	4	4	100.0	1	4	4	304	0
Mavie Street, under bush left hand side of driveway	4	2	50.0	0	0	2	104	2
Totals	8	6		1	4	6	408	2

* The count of species details the number of times this species was found in a trap.

Discussion

According to the key by Huang[2] the identification of *Ae. albopictus* larvae is carried out using the hair characteristics of segment VII: hair 1–VII is usually 4 branched (3–4 branches), hair 2–VII is single (1–2 hairs) and hairs 1–VII are less than 2 times as long as hairs 5–VII. The alternative features, which lead to identify *Ae. scutellaris*, in the dichotomous key are: hair 1–VII is usually 2 branched (2–3 branches), hair 2–VII is 3–4 branched and hairs 1–VII are at least 2.5 times as long as hairs 5–VII.

The investigation of hair numbers of segment VII of link bred *Ae. albopictus* larvae and larval skins revealed considerable variation. Hair branch 1–VII was found to have 3.25 ± 0.70 hairs, hair branch 2–VII revealed 1.19 ± 0.45 hairs, and hairs 1–VII were 2.05 ± 0.15 times as long as hairs 5–VII. The medians of two out of three parameters confirm the description given in Huang.[2] However, the variability is larger than described in Huang,[2] which is confirmed by the observation that the sides of one specimen differed in hair numbers in nearly 50 per cent of the specimens investigated. Eleven of the 24 larval specimens intercepted would not be accurately identified as *Ae. albopictus* and thus be misidentified as *Ae. scutellaris*.

Larval identification of *Aedes scutellaris* or *Ae. albopictus* from a single specimen using Huang's key[2] therefore poses a substantial potential of misidentification. This problem could be partly overcome by sampling a number of specimens. However, during an exotic interception, the number of larvae available is often small.

Identification keys are generally produced for zoogeographic regions covering the endemic species of a given area. Identification keys for larvae of *Aedes (Stegomyia)* of the Australasian region do not encompass all species of the *Aedes (Stegomyia) scutellaris group* of the nearby South-East Asian region.[15] Keys for the oriental and the South-East Asian regions do not include all of the Australasian species.[2,12] A comprehensive key of *Aedes (Stegomyia*) species is required for quarantine purposes in Australia.

A further difficulty is that larvae of some species already present in Australia such as *Ae. katherinensis* in the Northern Territory and *Ae. scutellaris* in Queensland (and also Indonesia and Papua New Guinea) can not be readily distinguished.[1,2,6,11] In addition, there is no description of larvae of *Ae. alorensis* and there is also the possibility of an undescribed species.[1] In an interception of larvae from overseas vessels, the knowledge of the last port of call or recent ports of call can often provide important information assisting problem identifications.

The world-wide problem of exotic importations of container breeding mosquito species via international transport has been discussed widely.[8,9,10,16,17,18] It is possible that distribution maps of the *Aedes (Stegomyia)* species of the Pacific region are out of date due to undetected species or the recent establishment of species from neighbouring or other countries. This potential increases the need for a comprehensive larval key for the *Aedes (Stegomyia)* species of the Pacific region as a whole.

The requirement to include the exotic or newly established species in identification keys has been successfully solved for *Ae. albopictus* larvae (and adults) in the Nearctic region.[18] However, in this example few *Aedes (Stegomyia)* species needed to be considered.[18] Additional taxonomic investigations with larger samples of specimens from different countries are essential to clarify larval identification of some of the *Aedes (Stegomyia)* species of the Pacific region. The application of molecular biological methods might provide supplementary information towards solving identification issues, but combined morphological and molecular biological studies remain to be done.[3,19]

AQIS routinely carries out inspections on overseas vessels in the Northern Territory and the finding of exotic mosquito larvae is not uncommon.[20] There have been 24 (in 2000/01) and 6 (in 2001/02) exotic mosquito interceptions found during routine inspections in Darwin. Port areas in Darwin are close to the city and therefore it is considered very important to react immediately on interceptions.

Elimination procedures in the above interception included chlorination, spraying and fumigation, and procedures were updated recently to include the rearing of larvae from South-East Asia, Papua New Guinea and the Pacific to adults.[20,21,22] This will assist in both the identification of intercepted specimens and the collection of specimens to establish comprehensive reference material towards the development of a comprehensive key as described above.

The elimination procedures above were successful in preventing the establishment of *Ae. albopictus* in Darwin. Darwin and the Northern Territory remain free of exotic dengue vectors. However, the container breeding survey carried out within the 400 m exclusion zone revealed a container index of 16.5. According to the assessment system of the World Health Organization, this container index is associated with significant risk for dengue transmission in countries with endemic dengue.[14] A container index between 1 and 2 is the objective to ensure that towns and seaports in a dengue endemic area are free from the danger of disease transmission.[14] If exotic *Aedes (Stegomyia)* species are imported into the Darwin port area, there are ample potential breeding places, increasing the potential of establishment of exotic vectors. Darwin is both vulnerable and receptive to

the introduction and establishment of exotic vectors of dengue.

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Rainfall and vector mosquito numbers as risk indicators for mosquito-borne disease in Central Australia

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Abstract

There have been 5 confirmed cases of Murray Valley encephalitis virus (MVE) infection in the Alice Springs region during the high rainfall years of 1999/00 and 2000/01, compared with one case in the preceding 9 years. There also appeared to be an increased prevalence of Ross River virus (RR) infection in the Alice Springs and Tennant Creek regions associated with high rainfall. This paper presents an analysis of summer rainfall from 1990/91 to 2000/01, numbers of seroconversion of sentinel chickens to MVE, and RR cases in both regions. In Alice Springs where summer rainfall (December to February) and average vector numbers in the December to March period are closely correlated, the analysis also included mosquito vector numbers and MVE cases. Summer rainfall over 100 mm was significantly associated with sentinel chicken seroconversions to MVE. From December to March there was also a significant association of average vector numbers (≥ 300) with seroconversions in sentinel chickens following high summer rainfall. MVE appears enzootic in the Tennant Creek region and epizootic in the Alice Springs region. In Alice Springs during December to March, there was a significant association of RR cases with rainfall over 100 mm and with average vector numbers over 300. There was also a significant correlation of summer rainfall with RR cases in Tennant Creek. Summer rainfall is a new and good early indicator of high risk for both MVE and RR disease in the Alice Springs locality and RR in the Tennant Creek locality. Although similar relationships between rainfall and vector abundance, and disease incidence probably exist in other areas of central Australia, rainfall and vector abundance thresholds will probably vary according to local climatic and environmental conditions. *Commun Dis Intell* 2003;27:110-116.

Keywords: Surveillance, Murray Valley encephalitis, Ross River virus infection, flavivirus, rainfall, Culex annulirostris,

Introduction

Murray Valley encephalitis virus (MVE) is a mosquito-borne arbovirus primarily carried by the common banded mosquito *Culex annulirostris*.[1,2] The human disease caused by this flavivirus can result in severe symptoms and has a 25 per cent mortality rate.[3,4] The virus is considered enzootic in the Kimberley region of Western Australia, the Top End of the Northern Territory, and possibly in north Queensland.[5] The virus is responsible for infrequent severe epidemics in eastern Australia, with the latest occurring in 1974.[5] The Alice Springs and Tennant Creek regions in the Northern Territory (Figure)[6] are thought to be epizootic for MVE following widespread and heavy wet season rainfall.[7] The natural host of MVE is thought to involve wild waterbirds, particularly herons and egrets.[2] The mosquito vector *Cx. annulirostris* is found throughout the Northern Territory and reaches very high numbers two to three weeks after heavy rainfall and widespread flooding in arid areas.[7]

There were 18 confirmed cases of MVE disease in the Northern Territory in 6 separate years between 1974 and 1993.[7] Most cases since 1993 have occurred in the Alice Springs region. Recent cases in Central

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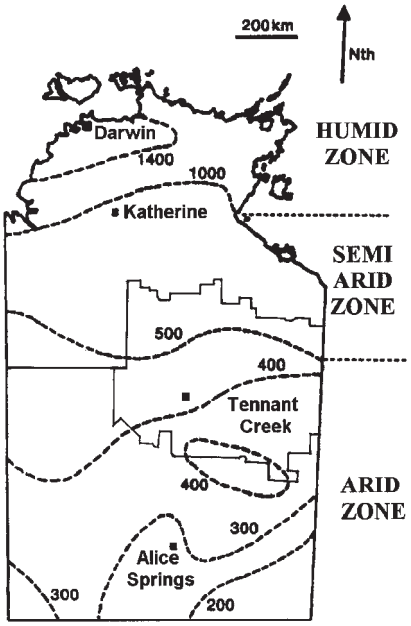
Figure. Northern Territory mean annual rainfall isohyets (mm) and the approximate limits of climatic zones



Adapted from McDonald and Alpine.[6]

Australia were from February to April during 2000 and 2001.[8] It has been suggested that the increase in cases in the Alice Springs region might be associated with high summer rainfall and subsequent high vector numbers.[8]

Regular adult mosquito monitoring has been established in Alice Springs and Tennant Creek to guide vector control and to provide an indication of increased potential for arbovirus disease in these regions.[5] Sentinel chicken flocks were established in 1995 in Tennant Creek and in late 1996 in Alice Springs to monitor MVE activity by testing for general and specific flavivirus seroconversion. Media alerts and media warnings are issued by the Department of Health and Community Services in response to increased vector numbers and to new seroconversions to MVE in sentinel chickens.[5]

Ross River virus (RR) is an alphavirus that causes a debilitating and sometimes chronic polyarthritis disease in humans.[2] The normal vertebrate hosts of RR are native macropods, such as kangaroos and wallabies.[2] *Culex annulirostris* is the major vector in inland areas of Australia, although *Ochlerotatus normanensis* is also a possible vector.[9,10] Epidemic activity of RR in inland areas of temperate Australia is usually associated with summer and autumn rain.[2]

RR disease was not considered common in Central Australia prior to 1991 when improved records were started.[9] Confirmation of local transmission was reported during an outbreak in 1995.[10] It has been suggested that a recent apparent increase of RR disease in Central Australia is related to increased rainfall and the subsequent high number of vector mosquitoes.[11]

A recent outbreak of MVE and RR disease in Central Australia has revived the interest in predicting seasons of increased arbovirus disease potential so that timely warnings can be made to residents in at-risk regions.

This paper describes the association of rainfall, vector numbers, seroconversion to MVE in sentinel chickens, and cases of MVE and RR disease, for use as predictors of outbreaks of mosquito-borne disease in the Alice Springs and Tennant Creek regions of the Northern Territory.

Methods

Rainfall data for Alice Springs and Tennant Creek were obtained from the Bureau of Meteorology records.[12] The daily rainfall totals for each month were combined into a total for December to February combined (summer) and an annual total.

Adult mosquito numbers in Alice Springs were monitored on one night per week by battery operated carbon dioxide baited EVS traps[13] at four regular monitoring sites. The traps were set and retrieved by the Environmental Health Officers of the Alice Springs Town Council. The trap sites included two sites at either end of Ilparpa Swamp, the closest boundary being 2 km south of the southern outskirts of the urban area. One was set in a semi-rural area approximately 500 m east of the eastern swamp boundary, and one within an urban area approximately 3 km north of the swamp. The trap sites were chosen to monitor the dispersal of mosquitoes from the swamp into the semi-rural and urban areas.

The traps were set before sunset and retrieved after sunrise. The mosquitoes were sent to the Medical Entomology Branch in Darwin for identification. All mosquitoes were identified and counted for trap collections that contained up to 200 mosquitoes. For trap collections over 200 mosquitoes, sub-samples of around 100 were identified and counted, and the total estimated by weight. For *Cx. annulirostris* the mean number of mosquitoes per trap night from the four sites was calculated for the periods December to February, December to March, and annually (Table 1). The December to March category was chosen to include the lag in adult vector mosquito numbers resulting from rain in February. Mosquito trap results from Tennant Creek were not available for continuous years.

The sentinel chicken flocks usually contained 10 birds, and were established in semi-urban areas in Alice Springs and Tennant Creek. Veterinary officers of the Department of Business, Industry and Resource Development bled the chickens each month. Blood samples were sent to the Arbovirus Surveillance and Research Laboratory at the University of Western

Australia for both general and specific testing using an epitope-blocking enzyme immunoassay for flavivirus antibody.[5] When the majority of the chickens in a flock seroconverted during the arbovirus season (December to August), the entire flock was replaced. Otherwise it was replaced at the end of each season. The number of new seroconversions to MVE each month were totalled for each financial year.

The MVE cases were the number of annually notified laboratory-confirmed cases for the Alice Springs or Tennant Creek region recorded by the Centre for Disease Control in Darwin.

The RR cases were the number of annually notified laboratory-confirmed cases from Alice Springs or Tennant Creek recorded by the Centre for Disease Control in Darwin.

The rainfall, vector numbers, sentinel chicken seroconversions, MVE cases, and RR cases were tabulated for each year for which they were available (Table 1, Table 4). Correlation analyses were used to determine whether there was a significant linear association between rainfall or vector numbers, and the number of disease cases and seroconversions. In addition, cut-offs for division into a categorical variable were determined to represent differences between years of high and low rainfall and vector numbers. For analysis $p< 0.05$ was considered significant. Analysis was performed using Intercooled STATA version 7.0 (Stata Corporation Texas).

Results

Alice Springs

The mean annual rainfall for Alice Springs was 273.7 mm (Figure).[6,12] The results of rainfall, mosquito vector numbers, sentinel chicken seroconversions, MVE cases, and RR cases for Alice Springs for the last 11 years are displayed in Table 1.

There were 4 years which had an annual rainfall of over 300 mm; 1994/95, 1996/97, 1999/00 and 2000/01 (Table 1). These were the only years where the average numbers of *Cx. annulirostris* per trap per night were over 116 for the whole year, over 198 for December to February and over 327 for December to March. These years, with the exception of 2000/01, also reported a relatively high number of cases of RR (14 cases or over).

During the years when sentinel chickens were in place, there were seroconversions to MVE in all 4 years and, except for 1994/95, cases of MVE were also reported in these years (Table 1). MVE cases were restricted to the Alice Springs locality, except in 1990/91 and 1999/00 (90/91 in Tanami and 99/00 in Willowra, Docker River and Hermannsburg), when 4 cases occurred in small communities in the wider Alice Springs region.

With one exception (2000/01) the years which recorded a rainfall of more than 100 mm during the summer months (December to February), also reported high numbers of RR cases (14 cases or more). Except for 1994/95, seroconversions to MVE in sentinel chickens and cases of MVE were also recorded in these years. The average vector numbers

Table 1. Cases of Murray Valley encephalitis virus and Ross River virus, vector numbers, rainfall, and Murray Valley encephalitis seroconversions in sentinel chickens, Alice Springs, 1990/91 to 2000/01, by financial year

	Financial year										
	1990/91	1991/92	1992/93	1993/94	1994/95	1995/96	1996/97	1997/98	1998/99	1999/00	2000/01
Cx. annulirostris Dec-Feb	23	1	85	7	369	0	199	1	137	496	1,188
Cx. annulirostris Dec-Mar	100	7	64	11	407	2	328	2	109	888	1,046
Cx. annulirostris (annual)	99	29	30	23	130	4	117	6	76	315	431
Summer rainfall (Dec-Feb) (mm)	111.0	25.0	78.4	83.8	170.4	43.0	286.2	48.0	48.8	343.6	285.4
Annual rainfall (mm)	162.8	127.6	264.8	233.6	301.0	115.6	369.4	254.8	216.0	663.2	523.4
New seroconversions to MVE							7	0	0	5	8
Cases of MVE infection	1	0	0	0	0	0	1*	0	0	3	2
Cases of RR infection	14	2	9	1	25	0	26	5	1	14	4

per trap per night for these years were an annual count of over 98 for all years, over 198 for December to February except for 1990/91, and over 327 for December to March except for 1990/91.

Alice Springs statistics

The data on rainfall, vector numbers, sentinel chicken seroconversions, MVE cases and RR cases for Alice Springs were all normally distributed. Mosquito vector numbers from December to February were significantly correlated with summer rainfall for the same months (r=0.72, p=0.012) (Tables 2 and 3). December to March vector mosquito numbers were significantly correlated with summer rainfall (r=0.89, p=0.0003). MVE seroconversions significantly correlated with summer rainfall as a continuous variable (r=0.89, p=0.0003) and were significantly associated with summer rainfall of 100 mm or higher (p=0.017). MVE seroconversions were significantly associated with December to March mosquito numbers of 300 or higher per trap per night (p=0.017), but not with mosquito numbers during December to February.

RR cases were significantly associated with summer rainfall of 100 mm or higher (p=0.026). They were also significantly associated with December to March mosquito numbers of 300 or higher per trap per night (p=0.022). However, there was no significant association of RR cases with December to February mosquito numbers.

Tennant Creek

The mean annual rainfall for Tennant Creek Airport was 451.5 mm (Figure). The results of sentinel chicken seroconversions, rainfall, and RR cases for Tennant Creek are shown in Table 4.

There were 5 years which had an annual rainfall of over 500 mm; namely 1990/91, 1992/93, 1996/97, 1999/00 and 2000/01 (Table 4). Four of these years had relatively high numbers of RR cases (23 cases or over), with the highest number of cases in the year of highest annual rainfall (2000/01). All of the years when sentinel chickens were in place had seroconversions to MVE, except for 1995/96 and 1997/98, which were years of relatively low annual rainfall (Table 4).

All years which reported a high number of RR cases (more than 10), had a summer rainfall of over 400 mm. The exception was 1999/00, when there were 6 cases of RR reported. However, sentinel chicken seroconversion occurred in 2 years when summer rainfall was not over 400 mm (Table 4).

Table 2. Correlation of rainfall, vector and Murray Valley encephalitis virus variables, Alice Springs, December to February 1990/91 to 2000/01

Variable 1	Variable 2	r value	p value
Summer rainfall*	Average vector numbers Dec-Feb*	0.72	0.012
Summer rainfall*	Average vector numbers Dec-Mar*	0.89	0.0003
Summer rainfall*	MVE seroconversions*	0.89	0.0003
Summer rainfall 100 mm*	MVE seroconversions*		0.017
Average vector numbers Dec-Feb	MVE seroconversions		ns
Average vector numbers Dec-Mar 300*	MVE seroconversions*		0.017
Average vector numbers Dec-Mar	MVE cases		ns

* Significant correlation 0.05 level

ns Not significant

Table 3. Correlation of Ross River virus, rainfall and vector numbers, Alice Springs, December to February 1990/91 to 2000/01

Variable 1	Variable 2	r value	p value
Summer rainfall	RR cases	0.58	0.059
Summer rainfall 100 mm*	RR cases*		0.026
Average vector numbers Dec-Feb	RR cases		ns
Average vector numbers Dec-Mar 300*	RR cases*		0.022

* Significant correlation 0.05 level

ns Not significant

Table 4. Cases of Ross River virus, rainfall, and Murray Valley encephalitis virus seroconversions in sentinel chickens, Tennant Creek, 1990/91 to 2000/01, by financial year

	Financial year										
	1990/91	1991/92	1992/93	1993/94	1994/95	1995/96	1996/97	1997/98	1998/99	1999/00	2000/01
Summer rainfall (mm)	448.4	99.0	582.6	338.2	199.8	101.2	615.4	259.2	223.6	450.8	517.8
Annual rainfall (mm)	503.4	189.2	663.0	401.0	304.8	351.4	632.0	320.0	356.6	751.6	947.0
RR cases	27	3	30	0	3	2	23	1	6	6	64
New seroconversions to MVE	-	-	-	-	7	0	7	0	10	8	12

Tennant Creek statistics

There was a significant correlation of summer rainfall with RR cases (p=0.022), with a highly significant association of rainfall over 400 mm with notification of more than 5 RR cases (p=0.003) (Table 5). There was no significant correlation of summer rainfall with MVE seroconversions as either continuous or categorical data.

Discussion

Alice Springs

The highly significant correlation of vector numbers in December to March with summer rainfall indicates that high summer rainfall is a good parameter to predict high numbers of *Cx. annulirostris* in the Alice Springs locality. The weaker correlation between vector numbers in December to February and summer rain is probably due to the delay in a rise in adult vector numbers after rain in January or February.

High levels of summer rainfall are generally widespread in Central Australia and are usually associated with monsoonal influences from the north and north-west.[12] High summer rain (over 100 mm) in Alice Springs is an indicator of rain in other localities in the region, causing localised flooding or pooling that creates breeding habitats for *Cx. annulirostris*. Heavy and widespread summer rain in the arid area would be an indicator of the extent of increased MVE risk.

The highly significant correlation between MVE seroconversions in sentinel chickens and summer rainfall, both as a continuous variable or a categorical variable when rainfall exceeds 100 mm, indicates that summer rainfall is a very good indicator of MVE transmission in the Alice Springs locality. To our knowledge, this is the first published quantitative measure of rainfall and MVE transmission in a locality.[15]

Seroconversions in sentinel chickens in the Alice Springs locality occurred each year when MVE cases in humans occurred in the region. Seroconversions are thus an indicator of risk of MVE in the region and probably in adjacent areas of other states after widespread summer rain.

Two years of below average rainfall and no seroconversion of sentinel chickens in Alice Springs were followed by a year of sentinel seroconversions and MVE cases. The Forbes hypothesis states that an increased risk of MVE disease in south-east Australia follows two years of above average rainfall of catchments in Northern Territory, Queensland, New South Wales and Victoria. It is clear that there is no similar requirement for two consecutive years of above average rainfall in Central Australia for MVE transmission. This suggests either MVE is endemic in Central Australia or there is a rapid spread of either infected birds or mosquitoes from enzootic regions to the north or north-west.

The lack of seroconversion to MVE in sentinel chickens in the Alice Springs locality for 2 consecutive years (1997 to 1999) (Table 1) may indicate a lack of endemicity of MVE in that region. The periodic seroconversion in Alice Springs associated with north-west monsoon weather, and the simultaneous

Table 5. Tennant Creek statistics, December to February 1990/91 to 2000/01

Variable 1	Variable 2	p value
Summer rainfall*	RR cases*	0.022
Summer rainfall ≥ 400 mm*	RR cases 5*	0.003
Summer rainfall	MVE seroconversions	ns
Summer rainfall ≥ 400 mm	MVE seroconversions	ns

* Significant correlation at 0.05 level

Ns Not significant

seroconversion in Alice Springs and Tennant Creek during the 3 years, 1996/97,1999/00 and 2000/01, suggests the periodic introduction of MVE to the Alice Springs region. This introduction is most probably from the north or north-west by infected wind-blown mosquitoes or wind-assisted dispersal of infected birds. The north-south dispersal of MVE through Central Australia may have been an additional mechanism for the introduction of MVE to south and eastern Australia in the widespread outbreak in 1974.[2]

Sentinel chickens have been used as indicators of MVE activity in Western Australia and the Northern Territory in the past and have been useful predictors of disease.[5,16] However, there are delays in receiving data from sentinel chicken programs. The results presented here indicate that high summer rainfall is a good indicator of risk of MVE disease in the Alice Springs locality and probably the region as a whole. The use of accumulated rainfall is independent of sentinel chicken results and therefore offers an earlier indicator of probable MVE activity.

The highly significant correlation with December to March average vector numbers of 300 mosquitoes or higher and MVE seroconversions in sentinel chickens gives a good threshold indicator of vector numbers for expected MVE activity in the Alice Springs area. This is the only published quantitative measure of vector numbers for increased risk of MVE transmission of which we are aware.

High average vector numbers in the above order of magnitude in the December to March period in other localities in the arid zone, in association with widespread heavy summer rain, would be a further indication of increased risk of MVE in that locality.

Summer rainfall over 100 mm is also a good predictor of increased RR risk. However, the relatively low number of cases of RR in 2000/01 did not correlate with high rain and high numbers of cases (Table 1). It is possible that people in Alice Springs protected themselves more from mosquitoes in that year after the considerable publicity following cases of MVE in the town.

An average vector number of over 300 mosquitoes per trap per night for December to March is a vector threshold for RR transmission to humans in the Alice Springs locality. While there have been a number of published qualitative indicators of rainfall and vector numbers with RR transmission, the above indicator is among the few published quantitative indicators for RR transmission.[17,18,19]

The lack of a significant correlation of RR cases with December to February vector numbers could be an indication that the RR virus-mosquito-host cycle takes a longer time to develop before conditions are favourable for transmission to humans. The lack of correlation of RR cases with December to February vector numbers also indicates that the rapid appearance of floodwaters *Ochlerotatus* mosquitoes, which occur very soon after heavy rain, are not the prime vectors of RR to humans in Alice Springs.

Rainfall alone is a significant indicator of probable MVE or RR activity. When the threshold of summer rainfall is exceeded in any part of the region, media warnings urging self-protection against mosquito bites, and enhanced vector control measures should be commenced.

Tennant Creek

RR transmission follows high summer rainfall in the Tennant Creek locality, and probably the whole of the Tennant Creek region. Summer rainfall over 400 mm is a good indicator for enhanced RR transmission in the Tennant Creek locality.

Although there is inconsistent mosquito monitoring data for Tennant Creek, those results available for specific years do indicate high numbers of *Cx. annulirostris* and *Ochlerotatus normanensis* following summer rainfall (Peter Whelan, unpublished data). Either *Cx. annulirostris* or *Oc. normanensis* could be the vectors of RR in the Tennant Creek locality and the region as a whole. However, relatively high numbers of *Cx. annulirostris* tend to remain longer after heavy rain and localised flooding (Peter Whelan, unpublished data), indicating that *Cx. annulirostris* is probably the major vector of RR in this region.

The lack of correlation of summer rainfall with MVE seroconversion of sentinel chickens in Tennant Creek indicates that seroconversions are independent of high rainfall. There were seroconversions in sentinel chickens in most years and with summer rainfall totals as low as 199.8 mm. This suggests the probable endemicity of MVE in the Tennant Creek locality and the Tennant Creek region.

While the frequent annual seroconversion of sentinel chickens suggests that MVE may be enzootic in the Tennant Creek locality, more information is required on both vector numbers and sentinel chickens to make valid conclusions on the endemicity of MVE in the locality and the region. It is possible that the Tennant Creek region is not enzootic for MVE and that the north-west monsoon winds blow MVE infected mosquitoes or assists infected bird dispersal from the north-west of Western Australia or the Top End of the Northern Territory into the region in most years. This speculation regarding the north-south dissemination of infected wind blown mosquitoes is supported by the occasional recovery of the coastal salt marsh mosquito *Ochlerotatus vigilax* in both Tennant Creek and Alice Springs during January and February in some years (Peter Whelan, unpublished data).

It is evident however, that MVE risk is high in the Tennant Creek locality and Tennant Creek region following summer rain. Over 200 mm of accumulated rain in summer appears to be a working indicator for an initial warning for both MVE and RR, with enhanced warnings issued after 400 mm of rain.

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Surveillance of viral pathogens in Australia

For many years, a sentinel laboratory system, the Laboratory Virology and Serology Reporting Scheme (LabVISE) has been collecting data on viral pathogens of public health importance in Australia. This report is one in a series of articles focussing on the epidemiology of viruses and viral groups under surveillance through LabVISE which are of current public health interest.

Respiratory syncytial virus

Paul Roche,[1] Stephen Lambert,[2] Jenean Spencer[1]

Introduction

Since the identification of respiratory syncytial virus (RSV) in the late 1950s, it has been recognised as a major cause of lower respiratory tract infection in young children.[1] RSV is a member of the paramyxovirus family, an RNA virus related to other respiratory viruses such as influenza and parainfluenza. In the United States of America in 1991, disease caused by RSV was estimated to be responsible for the hospitalisation of 100,000 children at a cost of US$300 million.[2] The significance of RSV infections is the high rates of infection in very young infants, and the difficulties in diagnosis, treatment and vaccination of this high-risk group. Given this, the progress towards the development of therapies to prevent infection, or at least prevent the potentially severe consequences of infection, have been watched with great interest.

RSV infections peak annually in winter or spring in temperate climates. The annual peak of RSV infection tends to occur in the absence of other respiratory viral pathogens.[1] Annual RSV epidemics are associated with a relative decline in infections with the parainfluenza viruses and other respiratory viruses. Influenza epidemics tend to occur as the RSV epidemic declines, although RSV and influenza epidemics may coincide. The reasons for this 'interference phenomenon' in the epidemiology of RSV and other respiratory viruses remain unclear.

Viral shedding in respiratory secretions is as high as 10^7/ml in the nasal discharges of infected children. While person-to-person transmission occurs, there is evidence that transmission through contaminated fomites is also important.[3] Fomites are particularly important in hospital settings where nosocomial infection with RSV in paediatric wards is a significant problem.[4] Direct inoculation of contagious secretions from the hands into the eyes and nose of carers, leads to outbreaks of RSV in families and among hospital staff.[2]

RSV infection occurs repetitively in children as infection with RSV does not cause lasting protective immunity. A longitudinal study in a day care centre[5] showed that 98 per cent of children became infected on their first exposure to RSV, a second exposure resulted in 74 per cent of children being infected and 65 per cent of children were infected on a third exposure. There are some well-defined groups of children who are at increased risk of RSV infection. These include children of lower socioeconomic status, children who share a bedroom with more than two other children, children who attend day-care, have siblings in pre-school or school or are in a multiple birth set. Children who are born prematurely or have chronic lung disease or congenital heart disease are at a higher risk of severe RSV disease resulting in admission to hospital.[6] RSV has also been recognised as an important infection in solid organ and haemopoietic stem cell transplant patients of all ages.[7]

Although infants are recognised as the group with the largest burden of RSV disease, infections with the virus resulting in illness continue throughout life. The importance of RSV in the elderly has been recognised. Four per cent of adults with community-acquired pneumonia hospitalised during the RSV season had serological evidence of current RSV infection.[8] More recently it has been recognised that a substantial proportion (between 7 and 41 per cent) of 'influenza-like illness' reported to general practices in the United Kingdom was due to RSV infection.[9] Strains of RSV found in the community and hospital patients in the same year were similar although lineages of RSV varied year by year. These are important findings for the development and use of vaccines and anti-viral agents against respiratory viruses.

Pathogenesis

Primary infection with RSV occurs with the highest incidence in infants aged between two and eight months.[6] The infection is rarely asymptomatic with

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pneumonia, bronchiolitis and tracheobronchitis being the major clinical manifestations. Otitis media may occur in 30 to 40 per cent of infected infants. RSV is responsible for between 50 and 90 per cent of hospital admissions for bronchiolitis, 5 to 40 per cent of those for pneumonia and 10 to 30 per cent of those for tracheobronchitis in children.[2] RSV viruses invade the epithelial cells of the lower respiratory tract and spread from cell to cell by inducing cell fusion and the formation of syncytia. Ciliated epithelium is destroyed and necrosis is observed.[10] The sloughing-off of the epithelia and increased secretion of mucus obstruct small airways leads to the clinical symptoms of bronchiolitis (inflammation of the bronchioles). These symptoms include hyperinflation, atelectasis (absence of gas in part of lung due to failure of alveolar expansion) and wheezing.[2] Treatment of RSV infection in infants is largely supportive and may include administration of supplemental oxygen, mechanical ventilation and fluid replacement. Bronchodilator therapy with alpha- and beta-agonists have been used but their efficacy is uncertain.[6] Treatment with aerosolised ribavirin, a synthetic nucleoside with antiviral properties, was licensed for use in hospitalised children with RSV infection in the United States of America in 1985.[10] A recent review of randomised trials of ribavirin[11] suggests that anti-viral treatment reduces the length of ventilator support and possibly the duration of hospital stay. Larger studies are required to fully assess the efficacy of this treatment.

Various factors have been postulated to influence the clinical severity of RSV disease in infants. Infants with compromised respiratory function, such as bronchopulmonary dysplasia or congenital heart disease with increased pulmonary circulation, are at increased risk of severe RSV disease.[12] There have been suggestions that group A RSV viruses cause more severe disease than group B, but the evidence is inconclusive.[12] More recent studies have suggested the GA3 clade of group A RSV causes more severe disease than clades GA2 or GA4.[13] The viral load in infants with severe RSV disease has been shown to be 10-fold higher than that in infants with mild disease.[14] RSV viral load seems to be associated with lung consolidation and hypoxia.[15] However, other studies have shown no relation between viral titre and severity of illness,[16] possibly because of difficulties in determining when the peak in viral shedding occurs.

Investigations of the pathogenesis of RSV disease have shown a relationship between a single nucleotide polymorphism in the upstream sequence of the interleukin 8 (IL–8) gene. This polymorphism is associated with increased IL–8 production, which is a potent neutrophil chemoattractant. This allele was demonstrated to occur more frequently in infants with RSV bronchiolitis.[17] IL–8 levels in plasma are increased in infants with severe RSV disease[12] and neutrophils are predominant in RSV bronchiolitis.[18]

Further clues to the pathogenesis of RSV come from the trials of the formalin-inactivated RSV vaccine (FI–RSV) in the 1960s, which showed increased morbidity and mortality in vaccine recipients when infected with RSV. Enhanced RSV disease seen in vaccine recipients may have been mediated by the formation of immune complexes and the subsequent activation of complement resulting in haemorrhagic necrosis in the lung.[19]

Approximately 70 per cent of wheezing episodes in the first year of life are associated with respiratory viral infections. RSV, rhinovirus and influenza B are the most frequently identified viruses.[20] Following RSV infection, subsequent wheezing has been noted in 40 to 50 per cent of infants hospitalised with RSV bronchiolitis.[2] These infants have a higher risk of wheezing and abnormal pulmonary function for up to 10 years.[6] Exacerbation of existing asthma by respiratory viruses has been noted, with an asthma attack in 50 to 70 per cent of cases with culture confirmed rhinovirus, RSV or coronavirus infection.[20] A cohort study showed that children with RSV lower respiratory tract infection before age three had a threefold increased risk of infrequent wheeze and a fourfold increased risk of frequent wheeze up to the age of 6 years. The risk of wheeze declined in this group thereafter and by age 13 this group of children were no longer at increased risk.[21] These data are suggestive of an important role for RSV in the inception of childhood asthma via modulation of the local immune response or changes in the neural pathways to the lungs.[22] Control of the current 'epidemic' of asthma in Western countries may be achieved by reducing viral respiratory infections through vaccination.

Prevention of RSV: prophylaxis and vaccination

Two strategies have been employed in the prevention of RSV infection. The passive immunisation of at-risk groups using antibodies to RSV and active immunisation with vaccines. Antibodies are important in protection from RSV disease, especially neutralising antibodies directed to the RSV proteins responsible for viral attachment (G protein) and cell fusion (F protein).[18] Parenteral administration of pooled immunoglobulin with high titres of RSV neutralising antibody (RSVIG) given monthly as prophylaxis during RSV season has been shown to reduce the frequency of infections and consequent hospitalisations. RSVIG has been used in high-risk infants, but requires the administration of a large volume at monthly intervals, which is expensive and exposes infants to the risks associated with blood products. Cost effective use of RSVIG appears to be limited to children with bronchopulmonary dysplasia, where one hospitalisation is prevented by the treatment of 12 children with RSVIG.[10] More recently a RSV monoclonal antibody, ('Palivizumab', Med Immune, Gaithersburg, MD) has been licensed as a prophylactic

agent for RSV in high-risk infants. A randomised, double-blind, placebo controlled trial of ' Palivizumab' in three countries demonstrated that monthly administration during the RSV season resulted in a 55 per cent reduction in hospital admissions for RSV infection as well as a shortening of hospital stay and less need for intensive care unit admission and oxygen support.[23] In the absence of a vaccine, 'Palivizumab' is the most effective prophylaxis against RSV in at-risk children.

The development of vaccines against RSV is complicated by the need to induce an effective immune response in very young infants and in the presence of maternal antibodies. Naturally acquired immunity is neither complete nor durable, although protection from severe disease occurs after primary infection.[2] A RSV vaccine would ideally protect at-risk children who may be deficient in their immune responses. The type of protective immunity to be induced by a vaccine is uncertain, as the balance of different effector mechanisms of the immune system in pathogenesis or protection in RSV disease is unclear. Natural immunity may be limited to groups or even clades of the RSV virus and a vaccine would need to induce broad immunity. RSV vaccine development is haunted by the exacerbation of RSV disease induced by the formalin-inactivated vaccines (FI–RSV) in the 1960s. Current strategies in RSV vaccine development cover a broad range of new vaccine technologies including sub-unit, live attenuated and DNA vaccines and adenovirus and poxvirus vaccine vectors.[12] Live vaccines are more likely to be effective in infants and live attenuated cold-passaged temperature sensitive RSV vaccines have been tested in adults and seropositive and seronegative children.[24] Although there was no exacerbation of RSV disease in vaccinated children or reversion of the vaccine virus to virulence, the protective efficacy of these vaccines has yet to be demonstrated. The introduction of safe and effective vaccines against RSV into childhood vaccination schedules appears to be some years away.

Local epidemiology

In temperate regions of Australia, RSV infections occur in sharply defined annual winter epidemics. RSV infection is not a notifiable disease in Australia so local information about the disease is limited to surveillance systems such as LabVISE, hospitalisation and mortality data and published research literature.

LabVISE reports of RSV numbered between 2,555 and 4,640 annually between 1991 and 2000. The annual peak in cases occurred in July each year, with between 55 and 77 per cent of all cases identified between July and September (Figure 1).

In 2000, 87 per cent of reports were in children aged less than 5 years, 64 per cent in children less than one year, 44 per cent in infants aged less than 6 months and 35 per cent in children aged less than 3 months (Figure 2). Overall, the male to female ratio was 1.3:1.

The peak in LabVISE reports of RSV infection coincided with the peak in hospitalisations for acute bronchiolitis, of children aged less than one year (National Hospital Morbidity database, Australian Institute of Health and Welfare, Figure 3). Bronchiolitis accounted for 56 per cent of all admissions to Australian hospitals of infants aged less than one year in 2000/01. The number of LabVISE reports of RSV was of the same order as total admissions for acute bronchiolitis (Figure 3). It is likely LabVISE captures the majority of hospitalised cases of RSV through its network of tertiary hospital laboratories in major Australian cities.

Figure 1. Laboratory reports to LabVISE of respiratory syncytial virus infection, Australia, 1991 to 2001, by month of specimen collection

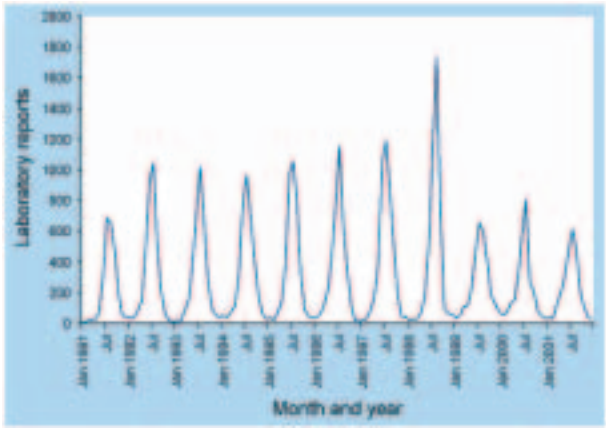


Figure 2. Laboratory reports to LabVISE of respiratory syncytial virus infection, Australia, 2000, by age and sex

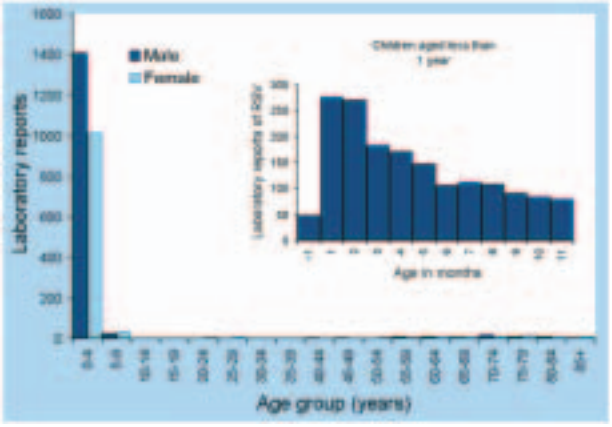
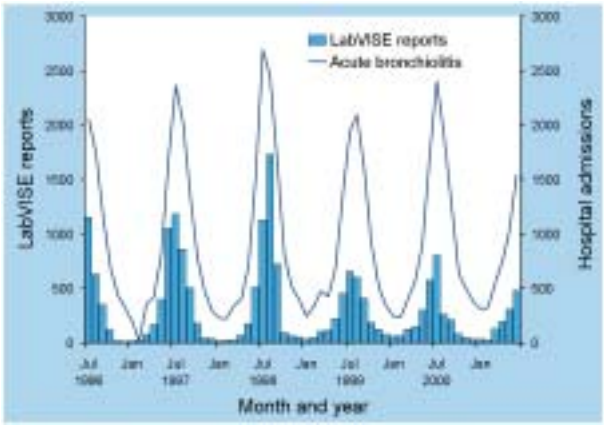
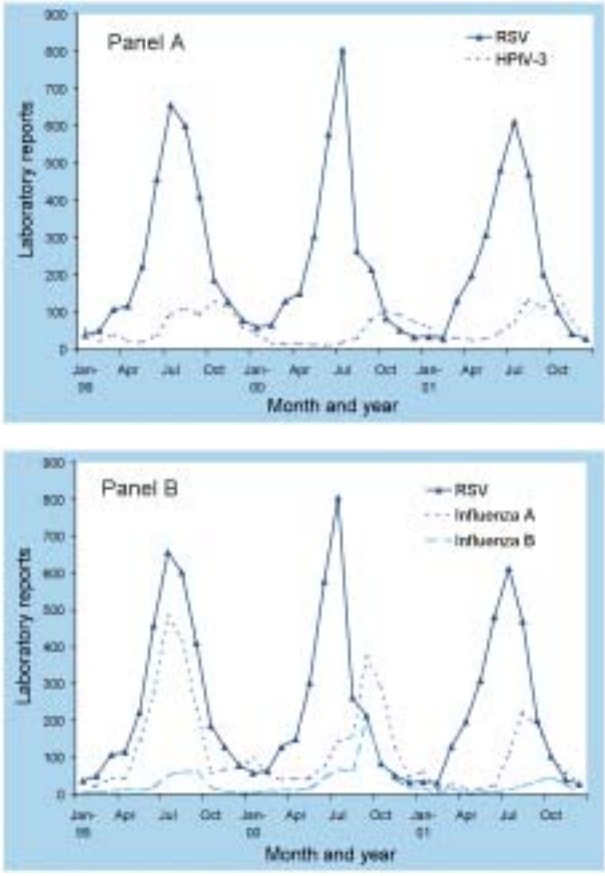


Figure 3. Laboratory reports to LabVISE of respiratory syncytial virus infection and hospitalisations with a principle diagnosis of bronchiolitis* in children aged less than one year, Australia, 1996 to 2001, by month of report or separation



* ICD–10AM code J21

Figure 4. Laboratory reports to LabVISE of respiratory syncytial virus infection and human parainfluenza virus type 3 (HPIV–3), (Panel A) and influenza A and B (Panel B), Australia, 1999 to 2001, by month of report



The relationship between the annual peaks in reports to LabVISE of RSV and human parainfluenza virus type 3 (HPIV–3) and influenza A and B over three seasons is shown in Figure 4. Peaks in RSV activity precede those of HPIV–3 (Panel A) and in some seasons precede, and in other seasons coincide with, peaks in activity of influenza A and B.

Given the ubiquitous nature of RSV infection in infants and young children, there are surprisingly few publications about the incidence or impact of this virus in Australia. Identification of RSV epidemics in Melbourne in 1960 and 1961 confirmed overseas findings of a distinct peak in cases in winter months, a strong link with bronchiolitis and a predilection for infecting infants and young children, causing deaths in the very young.[25,26] The severity of the RSV season in children varied from year-to-year and produced a clinical illness distinguishable from that caused by other viral respiratory pathogens, such as influenza and parainfluenza.[26] The possibility of intra-household transmission was raised with the identification of similar illnesses in the siblings of discharged children.[26] Australian papers since this time have largely consisted of case series reporting hospitalisation episodes with RSV identification, or bronchiolitis and other consistent clinical syndromes.[27,28,29,30] These papers confirm RSV as the major respiratory pathogen causing lower respiratory tract infections in infants and young children, and the seasonal nature of the annual RSV epidemics in Australia. A three year prospective study demonstrated the dominance of RSV group A infection, but suggested RSV group A and group B were similar in relation to relative clinical severity of infection.[30] Nosocomial transmission of RSV in a paediatric teaching hospital in New South Wales using at-risk days for hospital-acquired RSV as the denominator, showed the rate of transmission was 2.9 cases per 1,000 at-risk days.[31]

The epidemiology of RSV in New South Wales was further examined in a review of laboratory isolations, hospital admissions for acute bronchiolitis, and deaths due to acute bronchiolitis.[32] From January 1993 to December 1997 there were between 770 and 1,131 isolations of RSV annually, with the majority of these occurring in children less than six months of age. Between 1990 and 1995 there were 22,969 admissions where the principal diagnosis was acute bronchiolitis, with approximately three-quarters occurring in infants less than 6 months of age. There were 7 deaths from acute bronchiolitis in New South Wales children between 1992 and 1996 — five in infants aged less than 3 months, and the other 2 children aged between one and 2 years of age.[32]

Other papers have examined therapeutic options in the management of patients with RSV infections. A study on the use of RSV immunoglobulin or

monoclonal antibody to prevent hospitalisation and reduce the length of stay in hospital for Australian children with RSV, came to the conclusion that its routine use was inappropriate, even in children at high risk of serious consequences of infection.[33]

The importance of bronchiolitis in Australian Indigenous children has been assessed to determine whether this group is at higher risk. Rates of hospital admission for lower respiratory tract infections were compared in Indigenous and non-Indigenous children born in Western Australia during 1986, using linked hospitalisation and birth and death datasets.[34] The cumulative incidence of hospitalisation for bronchiolitis (not RSV specific), in the first 2 years of life was 116 admissions per 1,000 live births in Indigenous children, compared with 15 admissions per 1,000 live births in non-Indigenous children. While there may be other causes of bronchiolitis in Indigenous children, a review of hospitalisation for RSV proven bronchiolitis in Townsville from January 1997 to October 1999, showed that the annual admission rate was 46 admission per 1,000 live births in Indigenous children compared with 14 admissions per 1,000 live births in non-Indigenous children.[35] There was a seasonal variation in hospitalisations in Indigenous children with March being the peak month for RSV admissions, but there was apparently no seasonality in admissions for non-Indigenous children. This peak in RSV activity is distinct from the peak seen in national hospitalisation data (Figure 3) because Townsville is in a tropical region of Australia.

Surveillance of RSV

Given the ubiquity of RSV infection in childhood, what kind of disease surveillance is appropriate in Australia? Laboratory surveillance for RSV through LabVISE appears to routinely capture data on a large proportion of hospitalised cases of RSV annually, in a timely fashion and provides important information on the interaction between the epidemiology of RSV and other respiratory viruses. The surveillance of influenza-like illness through sentinel general practice schemes such as the Australian Sentinel Practice Research Network, indirectly measures the annual impact of RSV epidemics on community respiratory disease. Since RSV infection in older children and adults may not be readily distinguished from infection with influenza and other respiratory viruses, there is a need for simpler diagnostic tools for RSV to assess the true dimensions of the RSV disease burden. For the past several seasons, Victorian influenza surveillance has provided laboratory support to sentinel sites. In 2001, samples from patients presenting with influenza-like illness were tested by multiplex polymerase chain reaction for influenza A and B, RSV, adenovirus, enterovirus and rhinovirus.[36] This project will be important in defining the relative contribution of RSV to influenza-like illness in Australia.

Conclusions

Acute respiratory infections in children accounted for an estimated 1.9 million deaths in 2000 worldwide; 70 per cent of these deaths occurred in Africa and South-East Asia.[37] Preventing RSV-associated morbidity and mortality through vaccination is a high priority. RSV is the principal cause of bronchiolitis and a major contributor to other lower respiratory tract infections, particularly in the very young. In the coming years, there may be new vaccines to prevent infection and/or the serious consequences of infection due to viral respiratory pathogens, such as RSV, parainfluenza viruses, and influenza viruses. Monitoring the relative importance of these infections and collecting more comprehensive information about their incidence and impact in our community are important prerequisites for the implementation of appropriate vaccine strategies. Use of surveillance data (from systems such as LabVISE and the Australian Institute of Health and Welfare hospitalisation and death data), expansion of sentinel influenza surveillance systems to include other respiratory viral pathogens, and targeted research studies appear to be the most efficient means of gathering these data in Australia.

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Australian Sentinel Practice Research Network

Ian Wilson

Background

During the 1970s and 1980s the state-based Research Committees of the Royal Australian College of General Practitioners (RACGP) in South Australia and New South Wales developed independent networks of sentinel general practices. The networks monitored a number of conditions, but did not work cooperatively.

In the late 1980s Dr Ian Steven, as the head of the RACGP research secretariat, brought the networks together and expanded the project into other states, leading to the establishment in 1990 of the Australian Sentinel Practice Network (ASPREN), a national group recording infectious and other diseases.

Each year since then a small, dedicated group of general practitioners record basic epidemiological data on up to 14 conditions.

Method

Late in each year a meeting of interested people consisting of a mix of RACGP members, academic general practitioners and epidemiologists, is held to determine the conditions to be recorded in the following year. The choices are made from the interests of the group and expressions of interest from groups or people external to the RACGP. A strong emphasis is given to recording information that will be used for research. As well as determining the conditions, this group also approves the definitions that the general practitioners will use.

Forms that can be optically scanned are then printed and distributed to the network. Each time a general practitioner sees a patient who meets the criteria, the general practitioner records gender and age range. At the end of each week the form, which also includes details to identify the doctor and the total number of patients seen by the general practitioner in that week, is returned to the RACGP in South Australia. The records are scanned on a weekly basis and the data analysed on a rolling basis. At this stage, crude data are used to provide weekly estimates of prevalence.

Reporting

Each week, interim reports are sent by email to interested parties. Every three months recorders receive a consolidated report allowing them to compare their practice to the rest of the recorders. In future, recorders will also receive educational literature relating to the topics being recorded. Recorders earn 20 points toward the continuing education requirements of the RACGP.

The management group and researchers who have nominated a topic receive the crude analysis on a weekly basis and the final total collection of data at intervals during the year. *Communicable Diseases Intelligence* reports the ASPREN infectious disease data quarterly. An annual report is prepared which overviews each condition.

Strengths of the ASPREN network

ASPREN is a national network that monitors conditions seen in the community by general practitioners. The results are collated and disseminated electronically on a weekly basis. ASPREN data are timely and sensitive to temporal and seasonal changes in disease prevalence.

Weaknesses of the ASPREN network

There are two significant weaknesses of the network.

1. The distribution of the recorders does not cover all of Australia. Almost all recorders are in urban practice. Some jurisdictions (Western Australia, Victoria, Tasmania, the Northern Territory and the Australian Capital Territory) have few or no recorders.
2. We have not ascertained whether the doctors undertaking the recording are typical of other general practitioners in Australia.

These limitations severely restrict the ability to generalise the results to the rest of Australian general practice. Attempts have been made to overcome these limitations, but financial restrictions have prevented such projects.

Another problem resides in the denominator used for analysis: the number of consultations. In Australia it is not possible to use community size as the denominator as this is unknown. In the United Kingdom, where a capitation system is used it is possible to extrapolate to the community level.

What would improve the network?

Currently the South Australian Faculty of the Royal Australian College of General Practitioners funds the network from other resources of revenue. ASPREN needs to funded effectively and needs the support of the medical community to be able to recruit more

recorders and to undertake the research that is relevant and accurate.

Examples of findings

The prevalence of influenza-like illness has been monitored since the inception of the network. We have been able to show in 2001 and 2002 that New South Wales had a far higher prevalence of the disease than the other jurisdictions. A decreasing prevalence of influenza in the 65 and over age group has also been evident.

We have also been able to demonstrate variations in prevalence of gastroenteritis by state and territory, with higher levels of presentation to general practitioners in Queensland and Western Australia.

It also appears from our work that general practitioners in Victoria offer immunisations less frequently than other states. Possibly patients attend other services for immunisations.

Special investigations

At times, we have been asked to undertake special investigations or analyses. It is now possible to change the conditions being recorded during the year and short-term special investigations can be carried out.

At one time, in South Australia, ASPREN showed a high prevalence of influenza-like illness but surveillance at the viral laboratory did not support this. Recorders in Adelaide were asked to take blood specimens from some of these patients which showed the virus causing the outbreak of 'flu' was respiratory syncytial virus.

A few years ago an outbreak of acute flaccid paralysis associated with hand, foot and mouth disease occurred in Perth. The ASPREN recorders in South Australia were asked to record cases of hand, foot and mouth disease. Thankfully there was not an increase.

We have also provided information to researchers. Investigations into water quality have tried to match water quality with the prevalence of gastroenteritis.

Restrictions

Any researcher can access raw data, but data from individual general practices remain confidential. Where more than one recorder works in a given postcode region we are able to provide data by postcode. The small number of recorders limits this facility.

Contacts

We are always on the lookout for recorders and any general practitioners wishing to become a recorder should contact:

Ms Michelle Chalk
RACGP (SA Faculty)
15 Gover St
NORTH ADELAIDE SA 5006
Telephone: (08) 8267 8312
Facsimile: (08) 8267 3577
Email: michelle.chalk@racgp.org.au

Researchers interested in projects or further information should contact:

Dr Ian Wilson
Manager ASPREN
Senior Lecturer
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Telephone (08) 8303 3460
Facsimile: (08) 8303 3511
Email: ian.wilson@adelaide.edu.au

Surveillance systems reported in *CDI,* 2003

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 for the timely and effective detection and management of outbreaks. The national surveillance system combines a subset of 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 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 may be 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. The major features of the surveillance schemes for which *CDI* publishes regular reports are described below. Other surveillance schemes for which *Communicable Diseases Intelligence* publishes occasional reports include the National Mycobacterial Surveillance System (*Commun Dis Intell* 2002;26:525-536), the Australian Mycobacterium Reference Laboratory Network (*Commun Dis Intell* 2002;26:226-233).

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) and is currently being re-developed.

The system coordinates the national surveillance of more than 50 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. Computerised, de-identified unit records of notifications are supplied to the Department of Health and Ageing for collation, analysis and reporting in *CDI*.

Data provided for each notification include a unique record reference number, state or territory, disease code, date of onset, date of notification to the relevant health authority, sex, age, Indigenous status and postcode of residence. Additional data collected in the re-developed system includes infecting organism and subtype, the diagnosis method, full details of vaccination where appropriate, resident location as defined in the National Localities Index, dates of specimen collection, notification and date when notification was received by health authorities, Indigenous status defined as per the Australian Bureau of Statistics format, 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 (www.cda.gov.au) each fortnight. Data are published in *CDI* every quarter and in an annual report. Cases reported to state and territory health authorities for the current reporting period are listed by state or territory, and totals for Australia are presented for the current period, the year to date, and for the corresponding periods of the previous year. A commentary on the notification data is included with the tables in the 'highlights' section of each issue of *CDI*.

HIV infection and AIDS notifications are not included in NNDSS. Surveillance for these conditions is conducted separately by the National Centre for HIV Epidemiology and Clinical Research and is reported in the HIV and AIDS surveillance reports (see below).

Australian Sentinel Practice Research Network

The 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 who report presentations of defined medical conditions each week. Data collected provide an indicator of the burden of disease in the primary health care setting and are used to detect trends in consultation rates.

There are currently about 50 general practitioners participating in the network from all states. 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 2003, 13 conditions are being monitored, five are related to communicable disease issues. These include influenza, gastroenteritis, antibiotic prescription for acute cough, varicella and shingles. The other recordable conditions are aspirin prescription; micro-albumin testing in diabetes mellitus; steps 2 and 3 of the +++ asthma plan; corticosteroid prescription for asthma; and new diagnoses and recurrences or relapses of depression.

Data for communicable diseases are published in *Communicable Diseases Intelligence* 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

(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:

- sudden onset (within 12 hours);
- cough;
- rigour or chills;
- fever;
- prostration and weakness;
- myalgia, widespread aches and pains;
- no significant respiratory physical signs other than redness of nasal mucous membrane and throat;
- influenza in close contacts.

Gastroenteritis

Intestinal disease presumed or proven to be infective in origin. A stool sample is not carried out and one episode only is recorded per patient.

Antibiotics for acute cough

Record any patient, two years or older, who is prescribed antibiotics for an acute cough of less than 14 days duration and at least one other symptom of a respiratory infection, such as symptoms of upper respiratory tract infection; sore throat; sputum production; dyspnoea; wheeze; or chest pain; for which there is no other explanation. This illness is usually labelled acute bronchitis, chest infection or lower respiratory tract infection.

Excludes

1. Patients that have a history of chronic respiratory illness that requires ongoing treatment, such as chronic obstructive pulmonary disease, bronchiectasis or asthma.
2. Patients with suspected or confirmed pneumonia.

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.

HIV and AIDS surveillance

National surveillance for HIV and AIDS is coordinated by the National Centre in HIV Epidemiology and Clinical Research within the University of New South Wales, 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, either by the diagnosing laboratory (the Australian Capital Territory, New South Wales, Tasmania and Victoria) or by a combination of laboratory and doctor sources (the Northern Territory, Queensland, South Australia and Western Australia). Cases of AIDS are notified through the state and territory health authorities to the National AIDS Registry. To minimise duplicate notifications while maintaining confidentiality, diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code.

Currently, quarterly data presenting HIV infection diagnoses, AIDS diagnoses and AIDS deaths are published in each *CDI*. Tabulations of diagnoses of HIV infection and AIDS 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. Annual data are published on the *Communicable Diseases Australia* website.

Each year from 1997, the National Centre in HIV Epidemiology and Clinical Research has published *HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia annual surveillance report*. The annual surveillance report, available through http://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.

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 (MVE) and Kunjin. MVE virus infection can cause 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. MVE virus is also responsible for occasional severe epidemics of encephalitis in eastern Australia. The most recent was in 1974 when there were 13 fatalities and cases were reported from all mainland states. Since then, 70 cases of MVE have been reported, 63 from the north of Australia and seven from central Australia. In addition, one case of encephalitis caused by MVE and/or Kunjin virus(es) was reported from the north of South Australia in 2000.

Since 1974, a number of sentinel chicken flocks have been established in Australia to provide an early warning of increased MVE virus activity. These programs are supported by individual state and territory health departments. Each jurisdiction has a contingency plan that will be implemented if one or more chickens in a flock seroconverts to MVE virus.

Currently, 32 flocks are maintained in the north of Western Australia, eight in the Northern Territory, six in New South Wales, 10 in Victoria and two in northern Queensland (Map). The flocks in Western Australia and the Northern Territory are tested all year round but those in New South Wales, Victoria and Queensland are tested only in the summer months, during the main MVE risk season. Results are posted on the *Communicable Diseases Australia* Website. A yearly summary is presented in *Communicable Diseases Intelligence*.

Map. Geographical distribution of sentinel chicken flocks for the surveillance of arboviruses, Australia, 2003

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 NNDSS (see above).
- In 2002, five sentinel general practitioner schemes contributed reports of influenza-like illness: the Australian Sentinel Practice Research Network, the Northern Territory Tropical Influenza Surveillance Scheme, the New South Wales Sentinel General Practice Scheme, the Victorian Sentinel General Practice Scheme and the Western Australia sentinel general practices.
- The Virology and Serology Laboratory Reporting Scheme (LabVISE) contributes 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 Disease Australia Website* as the National Influenza Surveillance Scheme.

Annual reports on influenza in Australia are published in *CDI* each year (*Commun Dis Intell* 2002;26:204-213). 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 World Health Organization Collaborating Centre for Influenza Reference and Research.

Australian Gonococcal Surveillance Programme

The Australian Gonococcal Surveillance Programme includes 10 reference laboratories in all states and territories and in New Zealand. These laboratories report data on sensitivity to an agreed core group of antimicrobial agents on a quarterly basis. The antibiotics which are currently routinely surveyed are the penicillins, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens. When *in vitro* resistance to a recommended agent is demonstrated in 5 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 intermittently test isolates for the presence of high level resistance to the tetracyclines and azithromycin. Comparability of data is achieved by means of a standardised system of testing and a program-specific quality assurance process. Expanded annual reports are published in *CDI* (*Commun Dis Intell* 2002;26:242).

Virology and Serology Laboratory Reporting Scheme

The Virology and Serology Laboratory Reporting Scheme began operating in 1977. The scheme comprises 15 laboratories from all states and the Australian Capital Territory. Contributors submit data fortnightly on the laboratory identifications 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. Each report includes summary tables of total numbers of organisms identified by state or territory and numbers of reports by month and participating laboratory. The delay between date of specimen collection and date of publication ranges from two weeks to several months.

Data derived from this scheme must be interpreted with caution. The number and type of reports received are subject to a number of biases. These include the number of participating laboratories, which varies over time. The locations of participating laboratories also create bias, as some jurisdictions are better represented than others. 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 these data, general trends can be observed, for example with respect to seasonality and the age-sex distribution of patients. (*Commun Dis Intell* 2002;26:323)

Australian Paediatric Surveillance Unit

The Australian Paediatric Surveillance Unit conducts nationally based active surveillance of rare diseases of childhood, including specified communicable diseases and complications of rare communicable diseases in children. 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 Australian Paediatric Surveillance Unit are clinicians known to be working in paediatrics and child health in Australia. In 2001, over 1,000 clinicians participated in the surveillance of 15 conditions through the Australian Paediatric Surveillance Unit, with an overall response rate of 98 per cent.

The Australian Paediatric Surveillance Unit communicable diseases studies include: acute flaccid

paralysis, congenital cytomegalovirus infection, congenital rubella, HIV infection, AIDS and perinatal exposure to HIV, neonatal herpes simplex virus infection and hospitalised pertussis in infancy.

National Enteric Pathogens Surveillance System

The National Enteric Pathogens Surveillance System collects, analyses and disseminates data on human enteric bacterial infections diagnosed in Australia. These pathogens include *Salmonella, E. coli, Vibrio, Yersinia, Plesiomonas, Aeromonas* and *Campylobacter*. *Communicable Diseases Intelligence* quarterly reports include only *Salmonella*.

Data are based on reports to National Enteric Pathogens Surveillance System from Australian laboratories of laboratory-confirmed human infection with *Salmonella*. *Salmonella* are identified to the level of serovar and, if applicable, phage-type. Infections apparently acquired overseas are included. Multiple isolations of a single *Salmonella* serovar/phage-type from one or more body sites during the same episode of illness are counted once only. The date of the case is the date the primary diagnostic laboratory isolated a *Salmonella* from the clinical sample.

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 the Health Insurance Commission (HIC). 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 98 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 and territory 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* each quarter. Coverage estimates are provided for each state and territory and Australia as a whole and for each individual vaccine assessed at each milestone. Changes in 'fully immunised' coverage from the previous quarter are also included in the tables.

A commentary on ACIR immunisation coverage estimates provided by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases is included with the tables in each issue.

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;17:226-236.

CDI instructions for authors

Communicable Diseases Intelligence (*CDI*) is a quarterly publication of the Surveillance and Epidemiology Section, Communicable Diseases Branch, Commonwealth Department of Health and Ageing. Its aim is to provide timely information about communicable diseases in Australia to those with responsibility for their control.

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.

The approximate publication schedule for *CDI* is March, June, September and December.

Submission procedure

Manuscripts submitted to *CDI* for peer review must be offered exclusively to the Journal.

Manuscripts must be submitted electronically by email or on 3¼" disk, including text, tables and illustrations (see below). No hard copies are required.

Submission addresses and contact details

Contributions and requests for further information should be sent to:

The Editor
Communicable Diseases Intelligence
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Department of Health and Ageing
GPO Box 9848 (MDP 6)
CANBERRA ACT 2601
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Facsimile: (02) 6289 7791
E-mail: cdi.editor@health.gov.au

Manuscript

All

Authors are asked to provide an electronic copy of the manuscript by email or on a computer disk (on 3.5 inch diskette). Microsoft Word for Windows 97 (or earlier version) is preferred, or alternatively Rich Text Format (RTF) files should be used. Arial font is preferred, and if not available; Times New Roman. Do not use headers or footers, or automatic numbering for references or footnotes. Do not use numbered paragraphs.

Label disks with the title of the article, authors' name, and the word-processing format.

All manuscripts should have a title page that should include:

- title (e.g. Professor, Doctor, Ms, Miss, Mrs, Mr) and full name, including middle initial of each author;
- position held, and address of the institution where the article was produced;
- name of corresponding author; and
- current postal address, telephone number, facsimile number and email address of the corresponding author.

All manuscripts should be accompanied by a covering letter that should include:

- signatures of all authors if sent by mail, or list of all authors if sent by email;
- confirmation that the manuscript contents (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.

All articles, short report and letters to the Editor may be edited for style.

Abstract and keywords

Include up to 10 keywords. Do not cite references in the abstract. Abstracts should not exceed 250 words.

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 2,000 words or less and are peer-reviewed. A separate word count of the main text and of the abstract must be included on the title page.

Short reports

Short reports of less than 1,000 words are not peer-reviewed and 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. Surveillance summaries should be structured like articles including abstracts.

Outbreak reports

Short (500 to 1,000 words) unstructured reports of communicable disease outbreaks. Outbreak reports 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. Case reports will be considered based on their public health significance. Authors must note the instructions on protection of patient's right to privacy (see 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 *Communicable Diseases Intelligence* in the form of letters to the Editor. Letters should normally be less than 500 words, include no more than a single figure and less than six references.

Copyright

All authors are asked to transfer copyright to the Commonwealth before publication.

Authors

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.

Style

Avoid too many abbreviations. Use standard abbreviations only; do not make up abbreviations. Spell out on first mention and use only if acronym is used again.

Tables

Submit all tables on separate pages; simplify the information as much as possible, keeping the number of columns to a minimum and the headings short. Information in tables should not be duplicated in the text.

Tables are to be submitted without borders, blank rows or blank columns for spacing. Do not use paragraph returns. 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.

Figures and illustrations

Supply a copy of all figures on a separate page, labelled with the figure number and title. Histograms and graphs should be produced in Microsoft Excel and created on a separate worksheet. The numerical data on which these are based must be provided to enable editing for in-house style. Worksheets should be appropriately titled to distinguish each figure. Do not include the graph heading on the Excel worksheet.

All other figures should be provided in an appropriate graphic format. Do not embed figures or graphs in the manuscript text document. Use Arial font for figure lettering. Figures, symbols, lettering and numbering should be clear and large enough to be legible when reduced.

All table and figure headings should be provided in the manuscript at the end of the text. All tables and figures should be referred to within the results section and should not duplicate information in the text.

Black and white illustrations or photographs can be included if required.

Electronic copies of computer-generated illustrations should be saved in Adobe Photoshop, JPEG, EPS, GIF, or TIFF formats. Electronic versions of photos need to be at least 300 dpi.

References

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

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) and abbreviate journal names as in Medline (e.g. *Commun Dis Intell*). Give 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, a 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 give their titles, positions and affiliations.

Protection of patients' rights to privacy

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. 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 also appear in the manuscript. When informed consent has been obtained it should be included in the article.

Ethical approval and patient consent may also be required for case reports.

Review process

Short reports, surveillance summaries, reviews and correspondence are not subject to peer review.

On receipt of a manuscript, authors will be sent a brief acknowledgment. The articles then undergo a review process that may include peer review by two experts in the topic area. Articles may be rejected without peer review. 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. All authors are required to sign a copyright release form transferring all copyright to the Commonwealth. The Commonwealth copyright will be rescinded if the article is not accepted for publication. Accepted manuscripts are edited and final proofs returned for checking prior to printing.

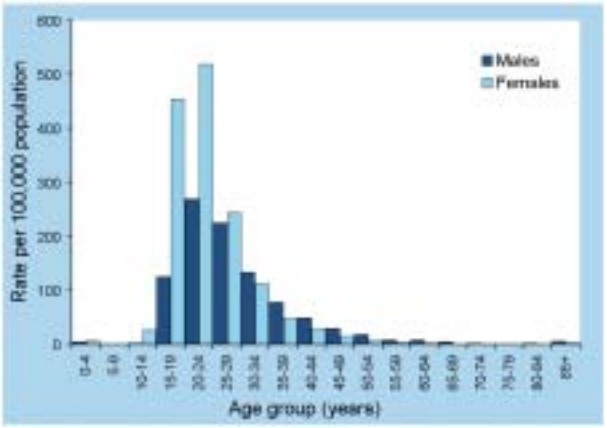
Errata

Australia's notifiable diseases status, 2000: annual report

In the Australia's notifiable diseases status, 2000: annual report of the National Notifiable Diseases Surveillance System (*Commun Dis Intell* 2002;26:155), the rate of chlamydial infections by age and sex as shown in Figure 20 was incorrect. The correct age sex distribution is shown below.

In the Additional reports section, under the Australian Paediatric Surveillance Unit, HIV infection, AIDS and perinatal exposure to HIV (*Commun Dis Intell* 2002;26:624), the text should read 'Between 1997 and 2001, 97 children were reported through either the Australian Paediatric Surveillance Unit or the National Centre in HIV Epidemiology and Clinical Research national HIV/AIDS surveillance, to have been exposed to HIV perinatally. Of these 9 children were diagnosed with perinatally acquired HIV infection'.

Figure 20. Notification rates of chlamydia, Australia, 2000, by age group and sex



Acute flaccid paralysis surveillance in Tasmania and the Northern Territory

In the editorial 'Polio eradication in Australia and the world' which appeared in *Commun Dis Intell* 2002;26:113-117, it was stated: 'While this [acute flaccid paralysis, (AFP)] surveillance target [1 per 100,000 population] is being met nationally, low AFP detection rates in Tasmania and the Northern Territory as reported in this issue suggest that AFP surveillance is sub-optimal in these jurisdictions.'

It should be noted that the reason for low AFP detection rates in these jurisdiction is the difficulty of any surveillance system to detect a small number of AFP cases in a sparsely populated state or territory.

The editors did not intend any criticism of AFP surveillance activities in these jurisdictions but want to point out the difficulties of achieving the surveillance targets in these settings.

National Enteric Pathogens Surveillance System

The figures for the National Pathogens Surveillance System in Table 12 of the Additional reports section, published in the previous issue of *Communicable Diseases Intelligence* (*Commun Dis Intell* 2002;26: 630) were for the third quarter 2002, not the second quarter as published.

Communicable diseases surveillance

Highlights for 4th quarter, 2002

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 disease notifications with an onset in the fourth quarter of 2002, compared with the 5-year fourth quarter mean. Disease notifications above or below the 5-year mean, plus- or minus- two standard deviations are marked with an asterisk. There were no diseases where the number of cases reported was two standard deviations above the mean of the same reporting period in the last 5 years in the current quarter. The reports of hepatitis C (unspecified), leptospirosis and tuberculosis were two standard deviations below the 5-year mean in this quarter. These and other disease trends are discussed below with additional commentary provided by State and Territory health authorities.

Due to difficulties in data transmission this quarter, Victorian data for sexually transmissible infections were not updated (Table 2).

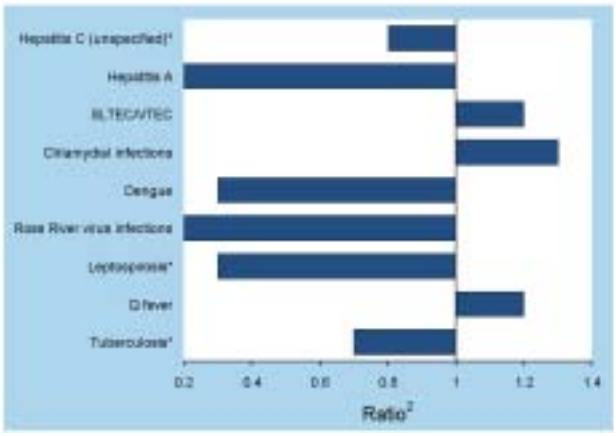
Gastrointestinal disease

Salmonellosis

There were 3 per cent more cases of salmonellosis in the fourth quarter 2002 than there were in the fourth quarter of 2001 (Table 2). Notifications from October to December 2002 were the second highest fourth quarter on record since 1998. Salmonellosis notifications are generally lowest in the winter months and show a peak in March (Figure 2). There have been several major *Salmonella* cluster investigations around Australia this quarter. The Hunter Public Health Unit investigated an outbreak of S. Montevideo in Newcastle that was traced back to Egyptian tahini imported by a company based in Sydney. Tahini is a paste made from sesame seeds and used as an ingredient for humus. To date there have been 43 notified cases, 32 of these cases had eaten kebabs. The investigation lead to a consumer-level recall of products containing the tahini.

The New South Wales Department of Health identified an increase in the number of *S.* Potsdam cases notified in early December. Other jurisdictions reported similar increases and an investigation was undertaken by OzFoodNet to determine the source of the outbreak. The cases were spread from the mid-north coast of New South Wales to Tasmania in the south and South Australia in the west. There are approximately 60 cases to date. All jurisdictions have conducted hypothesis-generating questionnaires. The source of the outbreak remains unclear.

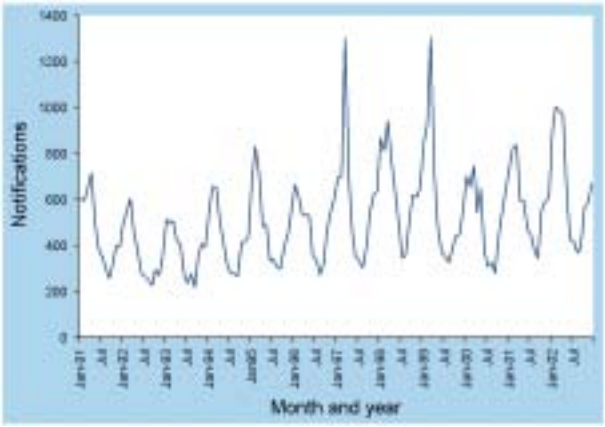
Figure 1. Selected[1] diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 October to 31 December 2002, with historical data[2]



1. Selected diseases are chosen each quarter according to current activity.
2. Ratio of current quarter total to mean of corresponding quarter for the previous five years.

* Notifications above or below the 5-year mean for the same period plus- or minus- two standard deviations.

Figure 2. Trends in notifications of salmonellosis, Australia, 1991 to 2002, by month of onset



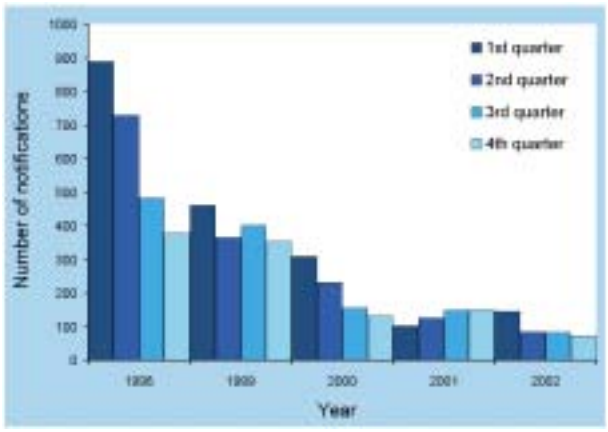
In October 2002, the South Australian Communicable Disease Control Branch investigated an outbreak of *Salmonella* Typhimurium phage type 99 associated with the consumption of cream filled cakes from a metropolitan bakery. In total, 111 environmental swabs and food samples were collected from the bakery. Of these, a composite sample of six piping bags yielded *Salmonella* Typhimurium phage type 99. An environmental investigation revealed that reusable piping bags were being used to pipe raw meat and cream products.

In October, four laboratory-confirmed cases (three males and one female aged between one and five years) of *Salmonella* Typhimurium PT170 infection were reported among attendees of a child care centre located on the Sunshine Coast, Queensland. The centre conducted a chicken hatching program over a two week period in September. Children were allowed to handle the chickens. The supplier of the chicken hatching equipment obtained their eggs from a local hatchery. *Salmonella* Typhimurium PT170 was detected in two poultry breeder sheds operated by the hatchery.

Hepatitis A

Hepatitis A notifications have been decreasing over the past five years. Notifications are typically highest in the first quarter and lowest in the final quarter (Figure 3). The lowest number of notifications for any quarter over the past five years were reported in the final quarter of 2002. Even though notifications have been low, the Northern Sydney Public Health Unit investigated a cluster of 8 cases of hepatitis A linked to a yum cha restaurant. The cases had all eaten in the restaurant in late September. An inspection of the premises did not identify any high-risk food preparation practices. The staff of the restaurant were interviewed and agreed to blood tests for hepatitis A serology. No evidence of recent acute infection was found in any of the food handlers. Detailed interviews were conducted with the cases and other patrons, but no obvious source of infection was identified. A similar outbreak occurred at a restaurant in south-eastern Sydney in 1997. In that outbreak, a case control study found that the likely source was undercooked prawns imported from Burma. While the exact cause of the current hepatitis A outbreak in northern Sydney remains unclear it is likely that the cause was from the ingestion of food contaminated with hepatitis A, although the route of contamination is undefined. Given the negative serology from food handlers, it would seem most likely that a food product was contaminated through exposure to human effluent.

Figure 3. Notifications of hepatitis A infection, Australia, 1998 to 2002, by quarter of notification



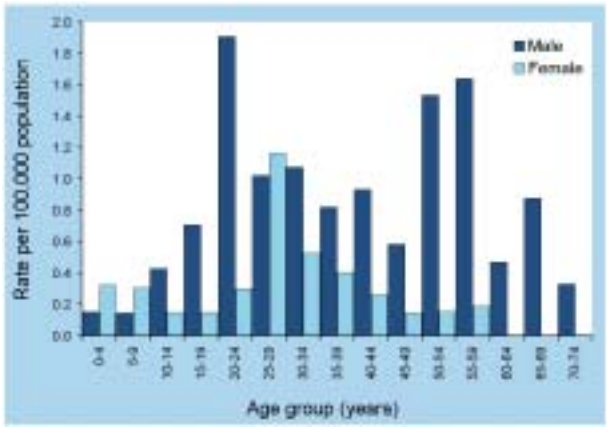
Vectorborne diseases

Malaria

Of the 108 cases of malaria reported to NNDSS this quarter, 10 were part of an outbreak in Far North Queensland. The outbreak occurred during the first two weeks of October among tourists who camped at Noah Beach in the Daintree National Park.[1] The individual believed to be the source of the outbreak stayed at the campsite for 4 days in late September and was diagnosed with *Plasmodium vivax* malaria the day after he left Noah Beach. He had a history of travel to Africa in 2002 and Indonesia in 2001. The Tropical Public Health Unit conducted mosquito trapping in the area and found a large number of *Anopheles farauti*, which can transmit malaria in northern Queensland. Fogging was undertaken at Kuranda as three of the cases lived there and *Anopheles* mosquitoes were found there.

Of all notifications of malaria, from October to December 2002, the majority of people with malaria were males (74%). The notification rates of malaria were highest in males aged 20–24 years, 50–54 and 55–59 years (Figure 4). The highest notification rate of malaria in females was in the 25-29 year age group.

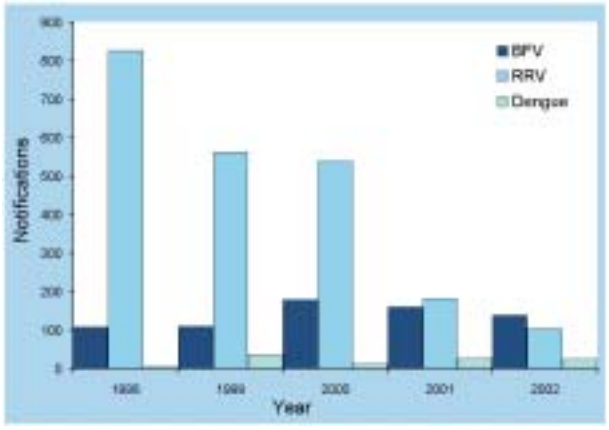
Figure 4. Notification rates of malaria, Australia, 1 October to 31 December 2002, by age group and sex



Other vectorborne diseases

Ross River virus infections have decreased steadily over the past five years, from 826 notifications in 1998 to 104 notifications in 2002 (Figure 5). Barmah Forest virus infections have remained fairly stable in the fourth quarter, while the number of notifications of dengue virus infections have fluctuated from four in 1998 to 36 in 1999. The fluctuations in dengue notifications are attributable to the outbreak in Cairns, Mossman and Port Douglas in 1997 to 1999.[2]

Figure 5. Notifications of Ross River virus infections, Barmah Forest virus infections and dengue virus infections, Australia, 1 October to 31 December, by year of notification



Other bacterial infections

Tuberculosis

Precautionary screening for tuberculosis was carried out across Australia from October 2002, after a worker from a resort in Queensland was diagnosed with tuberculosis. The Queensland Department of Health was given the responsibility of collating the national figures from the screening. Approximately 1,500 people have been offered screening, 26 in the Australian Capital Territory, 575 in New South Wales, 8 in the Northern Territory, 347 in Queensland, 21 in Tasmania, 102 in South Australia, 407 in Victoria, 33 in Western Australia and 42 people from overseas. Queensland Health are currently collating figures, but to date there is no evidence of local transmission of tuberculosis from the worker to people attending the resort.

Other non-notifiable diseases

Viral gastroenteritis

Viral gastroenteritis outbreaks were reported throughout Australia this quarter. Western Australia received several reports of probable or confirmed norovirus (Norwalk-like virus) gastroenteritis outbreaks in institutions in the fourth quarter of 2002, as well as evidence of a high incidence in the community.

In October 2002, a child-care centre reported 34 cases of a vomiting illness in children over the previous 12 days. Secondary spread in households and other features suggested a norovirus outbreak, and PCR tests on faecal specimens collected from some children confirmed human calicivirus infection.

Later in the month, an investigation was commenced when 10 people presented overnight at a public hospital emergency department with diarrhoea and vomiting. Another nine were treated at another public hospital within the same 24-hour period. Cases ranged in age from 12 to 94 years, with 13 females and 6 males. All cases reported profuse vomiting and diarrhoea, accompanied by abdominal pain, with median duration of illness being 3 days. Secondary cases occurred in staff members in both hospitals, and two cases had previous known contact with persons with a similar illness. No commonalities in food consumption, or places visited or eaten at, could be identified. Faecal specimens from two of the cases were positive for calicivirus by PCR testing. The investigation concluded that most, if not all, of the cases were community-acquired norovirus infection.

In November, an outbreak of gastroenteritis in a nursing home was confirmed to be due to norovirus infection. Twenty-one of 40 residents reported illness (attack rate: 52%). Six staff members also experienced gastroenteritis, and there was a secondary spread in the families of two staff members. Three of four specimens were positive for norovirus by PCR testing. No food sources were identified. Similar outbreaks in several other nursing homes were reported around the same time, as well as an outbreak in a group of teachers who had participated in a professional development day.

LabVISE

During the period October to December 2002, 17 participating laboratories (5 in New South Wales, 3 each in Western Australia and Victoria, 2 each in Queensland and Tasmania and one each in South Australia and the Australian Capital Territory), contributed 5,136 reports to LabVISE. Although there were no contributing laboratories in the Northern Territory, samples from this jurisdiction were included in reports from participating reference laboratories.

Of the 5,136 reports received, 3,289 (64%) were of viral infections and the remainder (1,847) were bacterial, spirochaete, fungal, protozoan or helminthic infections. Of the viral infections, reports of rotavirus (590 reports) represented 18 per cent of all viral identifications. The number of norovirus reports to LabVISE has doubled from 138 reports in 2001 to 268 reports in 2002. Among reports of non-viral pathogens, *Chlamydia trachomatis* (916 reports) represented 50 per cent of the total.

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References

1. Lawrence J. European travellers affected by the outbreak of *Plasmodium vivax* malaria in Northern Queensland, Australia. *Eurosurveillance* 2002 (www.eurosurveillance.org/ew/2002/021121.asp#2)

2. Blumer C, Roche P, Spencer J, Lin M, Milton A, Bunn C, *et al*. Australia's notifiable diseases status, 2001: annual report of the National Notifiable Diseases Surveillance System. *Commun Dis Intell* 2003;27:1–78.

Tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 21,494 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 October and 31 December 2002 (Table 2). The notification rate of diseases per 100,000 population for each State or Territory is presented in Table 3.

There were 5,136 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 October to 31 December 2002 (Tables 4 and 5).

Table 1. Reporting of notifiable diseases by jurisdiction (4th quarter 2002)

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
Hepatitis (NEC)	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
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible diseases	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions except SA
Gonococcal infection	All jurisdictions
Syphilis	All jurisdictions

Disease	Data received from:*
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Laboratory-confirmed influenza	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease - invasive	All jurisdictions
Poliomyelitis	All jurisdictions
Rubella	All jurisdictions
Tetanus	All jurisdictions
Vectorborne diseases	
Arbovirus infection NEC	All jurisdictions
Barmah Forest virus infection	All jurisdictions
Dengue	All jurisdictions
Japanese encephalitis	All jurisdictions
Kunjin	All jurisdictions except ACT†
Malaria	All jurisdictions
Murray Valley encephalitis	All jurisdictions except ACT†
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions except SA
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
Invasive meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

* Jurisdictions not yet reporting on diseases either because legislation has not yet made some diseases notifiable in that jurisdiction or data are not yet being reported to the Commonwealth

† In the Australian Capital Territory, infections with Murray Valley encephalitis virus and Kunjin are combined under Murray Valley encephalitis

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 October to31 December 2002, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 4th quarter 2002[1]	Total 3rd quarter 2002[1]	Total 4th quarter 2001[1]	Last 5 years mean 4th quarter	Year to date 2002	Last 5 years YTD mean	Ratio†
Bloodborne diseases															
Hepatitis B (incident)	0	13	8	9	1	6	24	8	69	60	88	77	230	331	0.9
Hepatitis B (unspecified)	18	782	NN	202	48	12	404	102	1,568	1,903	1,902	1,766	7,288	7,283	0.9
Hepatitis C (incident)	1	14	NN	NN	8	2	23	33	81	71	144	106	328	388	0.8
Hepatitis C (unspecified)	53	1,493	62	658	107	88	896	315	3,672	4,297	4,344	4,456	16,737	18,533	0.8
Hepatitis D	0	0	0	0	0	0	2	0	2	7	4	7	18	19	0.3
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0.0
Campylobacterosis[2]	98	NN	44	1,155	748	200	1,488	650	4,383	3,386	4,745	3,971	14,089	13,514	1.1
Cryptosporidiosis	4	43	52	56	10	16	57	55	293	241	442	N/A	3,235	N/A	N/A
Haemolytic uraemic syndrome	0	1	1	0	0	0	0	0	2	2	1	5	10	12	0.4
Hepatitis A	0	32	9	11	4	2	8	7	73	82	151	330	375	1,688	0.2
Hepatitis E	0	0	0	0	0	1	0	0	1	4	1	1	12	8	1.0
Listeriosis	0	5	0	4	1	1	1	4	16	11	16	15	57	64	1.1
Salmonellosis	27	529	67	597	121	49	256	176	1,822	1,171	1,761	1,706	7,744	7,006	1.1
Shigellosis	0	17	28	27	0	1	7	29	109	108	113	140	490	599	0.8
SLTEC, VTEC[3]	0	0	0	2	8	0	2	0	12	14	13	10	51	36	1.2
Typhoid	0	8	0	1	2	0	1	1	13	15	17	16	70	69	0.8
Quarantinable diseases															
Cholera	0	0	0	1	1	0	0	0	2	3	0	0	6	3	0.0
Plague	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
Viral haemorrhagic fever	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
Sexually transmissible diseases															
Chlamydial infection	93	1,274	436	1,618	389	121	§	771	4,702	5,094	4,895	3,666	21,469	14,374	1.3
Donovanosis	0	0	1	2	NN	0	§	0	3	4	10	8	20	34	0.4
Gonococcal infection[4]	7	300	346	238	39	6	§	330	1,266	1,402	1,421	1,305	5,879	5,539	1.0
Syphilis[5]	0	195	102	22	1	10	§	18	348	459	344	394	1,624	1,623	0.9

Table 2 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 October to 31 December 2002, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 4th quarter 2002[1]	Total 3rd quarter 2002[1]	Total 4th quarter 2001[1]	Last 5 years mean 4th quarter	Year to date 2002	Last 5 years YTD mean	Ratio†
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Haemophilus influenzae type b	0	2	1	0	0	0	0	2	5	3	3	8	30	36	0.6
Laboratory- confirmed influenza	0	79	10	58	13	0	11	29	200	2,681	191	N/A	3,774	N/A	N/A
Measles	0	0	0	0	1	0	0	0	1	13	36	99	17	322	0.0
Mumps	0	6	0	2	1	0	1	2	12	20	14	33	66	177	0.4
Pertussis	13	401	1	520	51	14	218	37	1,255	1,155	3,320	2,515	5,358	7,258	0.5
Pneumococcal disease - invasive	11	187	15	94	44	11	104	44	510	890	419	N/A	2,257	N/A	N/A
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Rubella[6]	0	12	0	49	3	0	0	1	65	80	77	154	236	620	0.4
Tetanus	0	0	0	0	0	0	0	0	0	0	2	2	2	5	0.0
Vectorborne diseases															
Arbovirus infection NEC	0	4	0	1	0	0	7	0	12	3	2	9	21	51	1.3
Barmah Forest virus infection	0	45	1	85	0	0	0	7	138	106	160	136	901	728	1.0
Dengue	0	10	2	10	0	0	0	2	24	38	27	70	215	256	0.3
Japanese encephalitis	0	0	0	0	0	0	0	0	0	0	0	N/A	0	1	N/A
Kunjin virus infection	-	0	0	0	0	0	0	0	0	0	0	N/A	0	3	N/A
Malaria	4	12	6	59	1	4	18	4	108	83	139	147	463	762	0.7
Murray Valley encephalitis	0	0	0	0	0	0	0	0	0	0	0	N/A	2	4	N/A
Ross River virus infection	0	16	9	54	0	0	0	25	104	98	181	492	1,438	4,319	0.2
Zoonoses															
Anthrax	0	0	0	0	NN	0	0	0	0	0	0	N/A	0	N/A	N/A
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	N/A	0	N/A	N/A
Brucellosis	0	0	0	11	0	0	0	0	11	8	4	12	40	36	0.9
Leptospirosis	0	10	1	2	0	1	3	1	18	16	44	55	146	226	0.3
Lyssavirus (unspecified)	0	0	0	0	0	0	0	0	0	0	0	N/A	0	N/A	N/A
Ornithosis	0	10	0	0	0	0	5	2	17	89	41	29	193	85	0.6
Q fever	0	92	0	63	6	0	2	4	167	179	165	145	738	578	1.2

Table 2 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 October to 31 December 2002, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 4th quarter 2002[1]	Total 3rd quarter 2002[1]	Total 4th quarter 2001[1]	Last 5 years mean 4th quarter	Year to date 2002	Last 5 years YTD mean	Ratio†
Other bacterial infections															
Legionellosis	1	5	0	0	16	0	23	20	65	69	85	73	268	290	0.9
Leprosy	0	0	0	0	0	0	0	0	0	0	3	2	3	6	0.0
Invasive meningococcal infection	1	45	2	22	3	5	61	15	154	238	151	141	666	573	1.1
Tuberculosis	3	73	10	17	8	3	63	14	191	235	249	267	961	1,031	0.7
Total	334	5,715	1,214	5,650	1,635	553	3,685	2,708	21,494	24,338	25,725	22,369	97,527	88,493	1.0

1. 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.
2. Not reported for New South Wales because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
3. Infections with Shiga-like toxin (verotoxin) producing *E. coli* (SLTEC/VTEC).
4. Northern Territory, Queensland, South Australia, Victoria and Western Australia: includes gonococcal neonatal ophthalmia.
5. Includes congenital syphilis.
6. Includes congenital rubella.

* Date of notification = a composite of three dates: (i) the true onset date from a clinician, if available, (ii) the date the laboratory test was ordered, or (iii) the date reported to the public health authority.

† The number of notifications received from Victoria this quarter were fewer than expected due to technical difficulties in data transmission.

‡ Ratio = ratio of current quarter total to mean of the same reporting period over the last 5 years calculated as described above.

§ The number of notifications received from Victoria were not reported due to difficulties in data transmission.

NA Not calculated as only notifiable for under 5 years.

NN Not notifiable

NEC Not elsewhere classified.

\- Elsewhere classified.

Table 3. Notification rates of diseases by State or Territory, 1 October to 31 December 2002. (Rate per 100,000 population)

Disease[1]	State or Territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis B (incident)	0.0	0.2	4.0	0.2	0.1	1.3	0.5	0.4	0.4
Hepatitis B (unspecified)	5.6	11.7	NN	5.4	3.2	2.5	8.3	5.3	8.0
Hepatitis C (incident)	0.3	0.2	NN	NN	0.5	0.4	0.5	1.7	0.4
Hepatitis C (unspecified)	16.4	22.4	31.0	17.7	7.0	18.6	18.3	16.3	18.6
Hepatitis D	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacterosis[2]	30.3	NN	22.0	31.1	49.1	42.2	30.5	33.7	22.2
Cryptosporidiosis	1.2	0.6	26.0	1.5	0.7	3.4	1.2	2.9	1.5
Haemolytic uraemic syndrome	0.0	0.0	0.5	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis A	0.0	0.5	4.5	0.3	0.3	0.4	0.2	0.4	0.4
Hepatitis E	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0
Listeriosis	0.0	0.1	0.0	0.1	0.1	0.2	0.0	0.2	0.1
Salmonellosis	8.3	7.9	33.5	16.1	7.9	10.3	5.2	9.1	9.2
Shigellosis	0.0	0.3	14.0	0.7	0.0	0.2	0.1	1.5	0.6
SLTEC,VTEC[3]	0.0	0.0	0.0	0.1	0.5	0.0	0.0	0.0	0.1
Typhoid	0.0	0.1	0.0	0.0	0.1	0.0	0.0	0.1	0.1
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.1	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
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 diseases									
Chlamydial infection	28.7	19.1	217.9	43.6	25.6	25.5	§	40.0	23.9
Donovanosis	0.0	0.0	0.5	0.1	NN	0.0	§	0.0	0.0
Gonococcal infection[4]	2.2	4.5	172.9	6.4	2.6	1.3	§	17.1	6.4
Syphilis[5]	0.0	2.9	51.0	0.6	0.1	2.1	§	0.9	1.8
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.0	0.5	0.0	0.0	0.0	0.0	0.1	0.0
Laboratory-confirmed influenza	0.0	1.2	5.0	1.6	0.9	0.0	0.2	1.5	1.0
Measles	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0
Mumps	0.0	0.1	0.0	0.1	0.1	0.0	0.0	0.1	0.1
Pertussis	4.0	6.0	0.5	14.0	3.4	3.0	4.5	1.9	6.4
Pneumococcal disease - invasive	3.4	2.8	7.5	2.5	2.9	2.3	2.1	2.3	2.6
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella[6]	0.0	0.2	0.0	1.3	0.2	0.0	0.0	0.1	0.3
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Vectorborne diseases									
Arbovirus infection NEC	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Barmah Forest virus infection	0.0	0.7	0.5	2.3	0.0	0.0	0.0	0.4	0.7
Dengue	0.0	0.2	1.0	0.3	0.0	0.0	0.0	0.1	0.1
Japanese encephalitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection	-	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	1.2	0.2	3.0	1.6	0.1	0.8	0.4	0.2	0.5
Murray Valley encephalitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	0.0	0.2	4.5	1.5	0.0	0.0	0.0	1.3	0.5

Table 3 (continued). Notification rates of diseases by State or Territory, 1 October to 31 December 2002. (Rate per 100,000 population)

Disease[1]	State or Territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	NN	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.0	0.1
Leptospirosis	0.0	0.2	0.5	0.1	0.0	0.2	0.1	0.1	0.1
Lyssavirus (unspecified)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.2	0.0	0.0	0.0	0.0	0.1	0.1	0.1
Q fever	0.0	1.4	0.0	1.7	0.4	0.0	0.0	0.2	0.8
Other bacterial infections									
Legionellosis	0.3	0.1	0.0	0.0	1.1	0.0	0.5	1.0	0.3
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Invasive meningococcal infection	0.3	0.7	1.0	0.6	0.2	1.1	1.2	0.8	0.8
Tuberculosis	0.9	1.1	5.0	0.5	0.5	0.6	1.3	0.7	1.0

1. Rates are subject to retrospective revision.
2. Not reported for New South Wales because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
3. Infections with Shiga-like toxin (verotoxin) producing *E. coli* (SLTEC/VTEC).
4. Northern Territory, Queensland, South Australia , Victoria and Western Australia: includes gonococcal neonatal ophthalmia.
5. Includes congenital syphilis.
6. Includes congenital rubella.

§ The notifications rates for Victoria this quarter were not reported due to technical difficulties in data transmission.

NN Not notifiable

NEC Not elsewhere classified.

– Elsewhere classified.

Table 4. Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 October to 31 December 2002, and total reports for the year[2]

	State or Territory[1]								This period 2002	This period 2001	Year to date 2002[3]	Year to date 2001
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	0	0	0	0	0	0	1	0	1	20	16	124
Mumps virus	0	0	0	0	0	0	2	1	3	1	16	32
Rubella virus	0	0	0	8	2	0	1	1	12	33	92	84
Hepatitis viruses												
Hepatitis A virus	0	2	2	1	7	0	0	5	17	17	71	81
Hepatitis D virus	0	0	0	0	0	0	1	0	1	1	7	11
Hepatitis E virus	0	0	0	0	0	2	2	0	4	1	9	5
Arboviruses												
Ross River virus	0	2	4	20	1	0	0	19	46	56	423	864
Barmah Forest virus	0	1	0	23	0	0	2	6	32	29	203	269
Dengue type 2	0	0	0	0	0	0	0	2	2	-	3	1
Dengue type 3	0	0	0	0	0	0	0	1	1	-	1	2
Dengue not typed	0	0	2	0	0	0	0	6	8	44	163	229
Murray Valley encephalitis virus	0	0	0	0	0	0	0	1	1	-	7	7
Flavivirus (unspecified)	0	0	0	3	0	0	3	0	6	5	43	26
Adenoviruses												
Adenovirus type 1	0	1	0	0	0	0	0	0	1	1	1	4
Adenovirus type 40	0	0	2	0	0	0	0	15	17	6	47	49
Adenovirus not typed/pending	0	33	5	11	103	0	7	146	305	331	995	1,097
Herpesviruses												
Herpesvirus type 6	0	2	0	0	0	0	0	0	2	-	2	2
Cytomegalovirus	7	52	2	20	164	1	8	7	261	374	1,110	1,277
Varicella-zoster virus	1	37	11	128	32	2	9	160	380	447	1,714	1,727
Epstein-Barr virus	10	18	24	101	184	1	16	120	474	546	1,789	1,857
Other DNA viruses												
Vaccinia virus	0	1	0	0	0	0	0	0	1	-	1	-
Molluscum contagiosum	0	0	0	0	0	0	0	8	8	7	26	17
Parvovirus	0	0	3	17	17	0	19	16	72	170	323	421
Picornavirus family												
Coxsackievirus A16	0	1	0	0	0	0	0	0	1	2	4	6
Echovirus type 6	0	1	0	0	0	0	0	0	1	18	61	20
Echovirus type 9	1	1	0	0	0	0	0	0	2	6	18	81
Echovirus type 11	0	1	0	0	0	0	0	0	1	2	6	8
Enterovirus - not typed	0	1	3	2	8	0	2	137	153	183	560	784
Poliovirus type 1 (uncharacterised)	0	12	0	0	0	0	0	0	12	3	34	20
Poliovirus type 2 (uncharacterised)	0	4	0	0	0	0	0	0	4	3	16	18
Poliovirus type 3 (uncharacterised)	0	2	0	0	0	0	0	0	2	-	6	7
Rhinovirus (all types)	1	103	0	0	6	0	2	55	167	143	516	462
Picornavirus not typed	0	0	0	0	0	2	0	0	2	7	14	16
Ortho/paramyxoviruses												
Influenza A virus	0	4	1	8	96	0	2	28	139	191	1,793	873
Influenza B virus	0	0	3	0	32	0	1	14	50	83	546	219
Parainfluenza virus type 1	0	0	0	0	25	0	0	6	31	25	290	58
Parainfluenza virus type 2	0	0	0	0	8	0	0	1	9	6	78	51
Parainfluenza virus type 3	0	54	0	7	93	0	2	79	235	248	590	776
Respiratory syncytial virus	0	16	2	3	70	4	9	73	177	172	2,945	2,645

Table 4 (continued). Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 October to 31 December 2002, and total reports for the year[1]

	State or Territory[1]								This period 2002	This period 2001	Year to date 2002[3]	Year to date 2001
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other RNA viruses												
HTLV-1	0	0	2	0	1	0	0	1	4	1	7	18
Rotavirus	2	161	0	1	323	2	21	80	590	428	1,952	1,730
Calicivirus	0	0	0	0	0	0	0	14	14	4	22	5
Norovirus	0	0	0	0	0	0	40	0	40	8	268	138
Other												
Borrelia burgdorferi	0	0	0	0	1	0	0	0	1	-	2	0
Brucella species	0	0	0	1	0	0	0	0	1	2	5	5
Chlamydia trachomatis not typed	4	113	49	258	235	8	0	249	916	912	3,838	3,408
Chlamydia pneumoniae	6	0	0	0	0	0	0	11	17	-	32	7
Chlamydia psittaci	0	0	0	0	2	0	10	3	15	19	61	77
Mycoplasma pneumoniae	1	22	0	41	63	11	56	32	226	350	1,222	970
Coxiella burnetii (Q fever)	1	6	0	6	20	0	12	14	59	37	251	162
Rickettsia (spotted fever)	0	0	0	0	0	0	0	1	1	1	1	100
Streptococcus group A	0	8	6	64	0	0	8	0	86	137	508	400
Yersinia enterocolitica	0	0	1	0	0	0	0	0	1	1	9	5
Bordetella pertussis	1	5	0	80	32	1	23	7	149	741	936	1,666
Legionella pneumophila	0	1	1	0	1	0	34	3	40	19	118	67
Legionella longbeachae	0	1	0	0	9	0	8	12	30	18	78	37
Legionella species	0	0	0	0	0	0	2	0	2	3	15	15
Cryptococcus species	0	0	0	2	2	0	0	0	4	3	29	21
Treponema pallidum	0	39	61	92	88	0	0	5	285	235	1,382	1,121
Entamoeba histolytica	0	0	0	0	0	0	4	2	6	2	28	11
Toxoplasma gondii	0	1	0	0	1	0	1	0	3	8	26	35
Echinococcus granulosus	0	0	0	0	3	0	0	2	5	16	30	33
Total	35	706	184	897	1,629	34	308	1,343	5,136	6,126	25,359	24,261

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.
2. From January 2000 data presented are for reports with report dates in the current period. Previously reports included all data received in that period.
3. Totals comprise data from all laboratories. 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.

– No data received this period.

Table 5. Virology and serology laboratory reports by laboratories for the reporting period 1 October to 31 December 2002[1]

	Laboratory	October 2002	November 2002	December 2002	Total this period
Australian Capital Territory	The Canberra Hospital	10	3	7	20
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	103	101	34	238
	New Children's Hospital, Westmead	89	45	36	170
	Repatriation General Hospital, Concord	-	-	-	-
	Royal Prince Alfred Hospital, Camperdown	-	3	6	9
	South West Area Pathology Service, Liverpool	133	90	21	244
Queensland	Queensland Medical Laboratory, West End	280	416	392	1,088
	Townsville General Hospital	-	-	-	-
South Australia	Institute of Medical and Veterinary Science, Adelaide	655	555	416	1,626
Tasmania	Northern Tasmanian Pathology Service, Launceston	10	8	14	32
	Royal Hobart Hospital, Hobart	-	-	-	-
Victoria	Monash Medical Centre, Melbourne	20	-	-	20
	Royal Children's Hospital, Melbourne	44	-	21	65
	Victorian Infectious Diseases Reference Laboratory, Fairfield	100	61	59	220
Western Australia	PathCentre Virology, Perth	446	380	335	1,161
	Princess Margaret Hospital, Perth	146	-	-	146
	Western Diagnostic Pathology	43	25	29	97
Total		2,079	1,687	1,370	5,136

1. The complete list of laboratories reporting for the 12 months, January to December 2003, will appear in every report regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.

–- Nil reports

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia. The system provides the national surveillance of more than 50 communicable diseases or disease groups endorsed by the Communicable Diseases Network Australia and the National Public Health Partnership. Notifications of these diseases are made to State and Territory health authorities under the provisions of their respective public health legislation. De-identified core unit data are supplied fortnightly for collation, analysis and dissemination. For further information, see Commun Dis Intell 2002;26:57.

LabVISE is a sentinel reporting scheme. Currently 16 laboratories contribute data on the laboratory identification of viruses and other organisms. This number may change throughout the year. Data are collated and published in Communicable Diseases Intelligence *quarterly. These data should be interpreted with caution as the number and type of reports received is subject to a number of biases. For further information, see* Commun Dis Intell *2002;26:57.*

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 66 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 2002, 10 conditions are being monitored, six of which are related to communicable diseases. These include influenza, gastroenteritis and acute cough. Definitions of these conditions were published in Commun Dis Intell 2002;26:57.

Data from 1 January to 31 December 2002 are shown as the rate per 1,000 consultations by week in Figures 6, 7 and 8.

Figure 6. Consultation rates for influenza-like illness, ASPREN, 1 January to 31 December 2002, by week of report

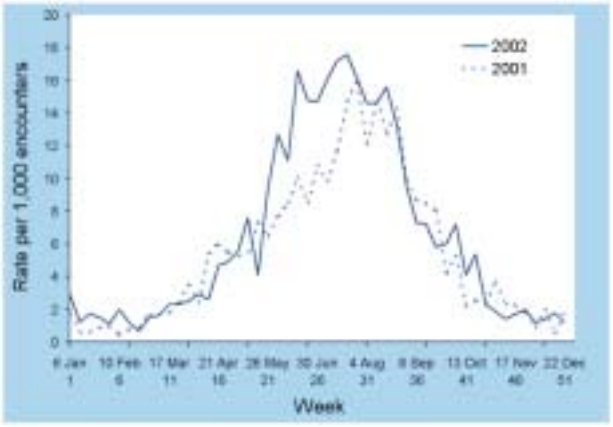


Figure 7. Consultation rates for gastroenteritis, ASPREN, 1 January to 31 December 2002, by week of report

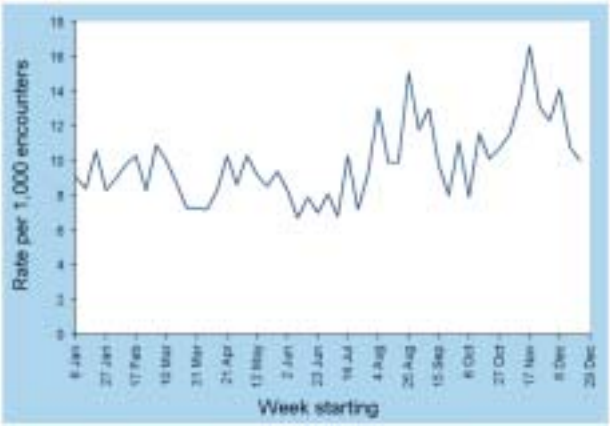
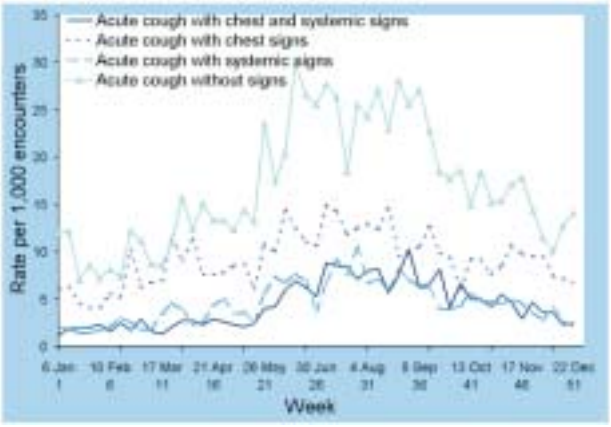


Figure 8. Consultation rates for acute cough, ASPREN, 1 January to 31 December 2002, by week of report



Gonococcal surveillance

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

The Australian Gonococcal Surveillance Programme 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 *2002;26:57.*

Reporting period 1 July to 30 September 2002

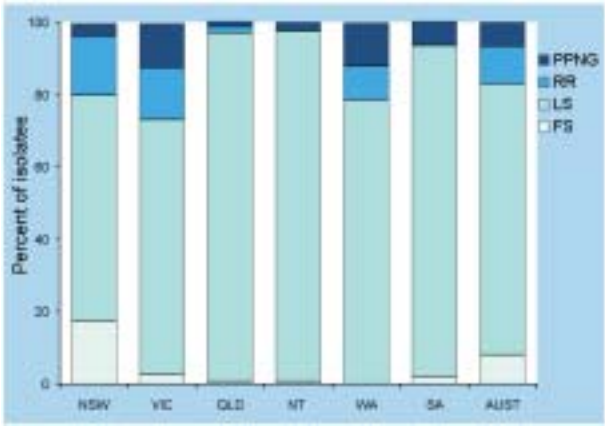
The Australian Gonococcal Surveillance Programme (AGSP) laboratories examined a total of 893 isolates in this quarter and another 20 strains were non-viable, little different from the 913 viable strains in 2001, but substantially more than the 794 examined in the same period in 2000. About 41 per cent of this total was from New South Wales, 17 per cent from Victoria, 13 per cent from Queensland, 15 per cent from the Northern Territory, 9 per cent from Western Australia and 5 per cent from South Australia. Isolates from other centres were few.

Penicillins

Figure 9 shows the proportions of gonococci fully sensitive (MIC ≤0.03 mg/L), less sensitive (MIC 0.06 – 0.5 mg/L), relatively resistant (MIC ≥1 mg/L) or else penicillinase producing (PPNG) aggregated for Australia and by state or territory. A high proportion of those strains classified as PPNG or else resistant by chromosomal mechanisms fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

In this quarter about 17 per cent of all isolates were penicillin resistant by one or more mechanisms — 7 per cent PPNG and 10 per cent by chromosomal mechanisms (CMRNG). The proportion of penicillin resistant strains ranged from 2.4 per cent in the Northern Territory to 26 per cent in Victoria.

Figure 9. Categorisation of gonococci isolated in Australia, 1 July to 30 September 2002, 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.*

This proportion is a decrease from the 26 per cent penicillin resistance seen in gonococci in the third quarter of 2001. The number of PPNG isolated across Australia (n=59) continued to decline slowly. Sixty-six PPNG were detected in the equivalent quarter of 2001 and 70 in 2000. The highest proportion of PPNG was found in isolates from Victoria and Western Australia (12%). PPNG were present in all jurisdictions including 3 (2.4%) in the Northern Territory.

More isolates were resistant to the penicillins by separate chromosomal mechanisms (n=93). This is however, a substantial decrease in CMRNG compared to the same period in 2001 when 173 CMRNG were detected. CMRNG were concentrated in New South Wales (16%), Victoria (14%) and Western Australia (9%). CMRNG were not detected in the Northern Territory or South Australia.

Ceftriaxone

Low numbers of isolates with decreased susceptibility to ceftriaxone were present in Victoria, New South Wales and Queensland. The persistence of these isolates in Australia and their presence in nearby countries[2,3,4] suggests that continued monitoring is warranted. A Japanese report recorded treatment failure with cefixime (an oral third generation cephalosporin not available in Australia), but not ceftriaxone, with infections caused by gonococci with slightly raised ceftriaxone MICs.[5]

Spectinomycin

All isolates were susceptible to this injectable agent.

Quinolone antibiotics

Quinolone resistant *N. gonorrhoeae* (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.

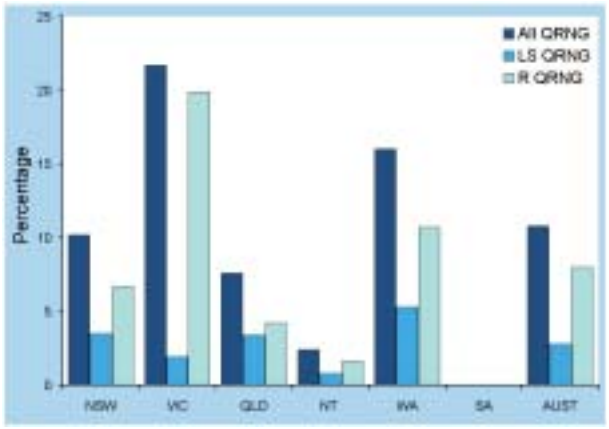
The total number (n=96) and proportion (11%) of QRNG were substantially less than those for the September quarter in 2001 (n=151, 17%). QRNG were again widely distributed, although none were detected in South Australia. High rates were maintained in Victoria (22%) and Western Australia (16%). Rates in New South Wales (10%) and Queensland (8%) declined. Three QRNG were detected in the Northern Territory.

In this quarter most of the QRNG (71 out of 96) exhibited higher levels of resistance as measured by MICs (Figure 10). This continues a trend of increasing MIC in QRNG.

High level tetracycline resistance

The number (n=95) and proportion (11%) of tetracycline resistance (TRNG) detected rose slightly in this quarter from the corresponding data in the September quarter of 2001. TRNG represented 20 per cent of isolates from Queensland, 14 per cent from Western Australia, 11 per cent from Victoria, 10 per cent from New South Wales, 4 per cent from South Australia and 2 per cent from the Northern Territory.

Figure 10. Distribution of *N. gonorrhoeae* displaying quinolone resistance, Australia, 1 July to 30 September 2002



LS QRNG Ciprofloxacin MICs 0.06 — 0.5 mg/L

R QRNG Ciprofloxacin MICs ≥1 mg/L

References

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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 *2002;26:57.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 July to 30 September 2002, as reported to 31 December 2002, are included in this issue of Communicable Diseases Intelligence (Tables 7 and 8).

Table 7. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 July to 30 September 2002, by sex and state or territory of diagnosis

		State or territory								Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2002	This period 2001	Year to date 2002	Year to date 2001
HIV diagnoses	Female	0	5	2	0	0	1	6	2	16	24	63	71
	Male	2	88	0	33	9	2	35	7	176	188	502	514
	Sex not reported	0	0	0	0	0	0	0	0	0	0	0	1
	Total[1]	2	93	2	33	9	3	43	9	194	212	570	587
AIDS diagnoses	Female	0	0	0	0	0	1	1	0	2	7	9	14
	Male	2	12	0	4	1	0	9	0	28	49	108	119
	Total[1]	2	12	0	4	1	1	10	0	30	56	118	134
AIDS deaths	Female	0	0	0	0	0	0	0	1	1	5	3	9
	Male	0	6	0	3	2	0	3	0	14	27	41	56
	Total[1]	0	6	0	3	2	0	3	1	15	32	44	65

1. Persons whose sex was reported as transgender are included in the totals.

Table 8. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 30 September 2002 and reported by 31 December 2002, by sex and state or territory

		State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	28	688	14	185	77	7	270	144	1,413
	Male	235	11,823	115	2,227	749	86	4,336	1,006	20,577
	Sex not reported	0	235	0	0	0	0	24	0	259
	Total[1]	263	12,771	129	2,419	826	93	4,648	1,156	22,305
AIDS diagnoses	Female	9	208	0	53	29	4	82	31	416
	Male	90	4,872	38	909	373	46	1,761	387	8,476
	Total[1]	99	5,093	38	964	402	50	1,852	420	8,918
AIDS deaths	Female	4	122	0	36	18	2	57	20	259
	Male	71	3,363	25	600	246	30	1,322	267	5,924
	Total[1]	75	3,494	25	638	264	32	1,386	288	6,202

1. Persons whose sex was reported as transgender are included in the totals.

Childhood immunisation coverage

Tables 9, 10 and 11 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 2001, at 24 months of age for the cohort born between 1 July and 30 September 2000, and at 6 years of age for the cohort born between 1 July and 30 September 1996 according to the Australian Standard Vaccination Schedule.

A full description of the methodology used can be found in 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 1256, Email: brynleyh@chw.edu.au.

Immunisation coverage for children 'fully immunised' by 12 months of age for Australia has increased from the last quarter by 0.5 percentage points to 91.7 per cent (Table 9). The change in 'fully immunised' coverage varied by state and territory but all jurisdictions experienced an increase in coverage, except for Western Australia (-0.5%) and the Northern Territory (-0.9%). South Australia (+1.4%) and Victoria (+1.1%) experienced the greatest increases in coverage. The remaining states experienced smaller increases in coverage over the quarter. Coverage is hovering around the 91 to 93 per cent level in almost all jurisdictions with the highest level in South Australia (93.2%) and the lowest in Western Australia (89.9%). There were no significant changes in coverage across any jurisdiction for any individual vaccine.

The second consecutive quarterly increase in coverage at 12 months of age for almost all jurisdictions and for most vaccines is encouraging and further indicates that coverage has not reached a plateau as first thought. Every jurisdiction has coverage greater than 90 per cent for all individual vaccines and three jurisdictions have greater than 92 per cent for 'fully immunised' coverage. The highest coverage for an individual vaccine at 12 months of age is for hepatitis B vaccine. National coverage is greater than 95 per cent and six jurisdictions have reached over 95 per cent coverage — New South Wales (95.2%), the Northern Territory (95.9%), Queensland (95.3%), South Australia (96.2%), Victoria (95.2%) and Tasmania (95.3%).

Coverage measured by 'fully immunised' at 24 months for Australia increased from the last quarter by 1.6 percentage points to 89.4 per cent (Table 10). Coverage increased from the previous quarter in five jurisdictions but the increases were all quite small, except in Tasmania (+2.8%) and Western Australia (+2.4%). Despite the increases, only three jurisdictions achieved greater than 90 per cent coverage for 'fully immunised' at 24 months of age, (Tasmania, Queensland and Victoria). Coverage for individual vaccines by 24 months for Australia, however, is much greater. Coverage for OPV is 94.8 per cent and 94.0 per cent for Hib suggesting that at least part of the lower figure for fully immunised may relate to data issues. As with the last quarterly coverage report, the most important changes in coverage at 24 months occurred for Hib vaccine. There were decreases in Hib coverage at 24 months of age in all jurisdictions except for the Australian Capital Territory. The decreases were not dramatic but have occurred for a second consecutive quarter.

Table 9. 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 2001; assessment date 31 December 2002

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,018	21,687	845	12,751	4,507	1,412	15,556	6,196	63,972
Diphtheria, tetanus, pertussis (%)	93.3	92.6	91.5	92.8	93.9	93.6	93.1	90.7	92.7
Poliomyelitis (%)	93.0	92.5	91.1	92.8	93.9	93.6	93.0	90.6	92.6
Haemophilus influenzae type b (%)	93.9	94.7	95.7	95.0	95.9	95.5	95.3	94.1	94.9
Hepatitis B	94.9	95.2	95.9	95.3	96.2	95.3	95.2	93.9	95.1
Fully immunised (%)	91.0	91.4	90.4	91.8	93.2	93.0	92.4	89.9	91.7
Change in fully immunised since last quarter (%)	+0.2	+0.4	-0.9	+0.4	+1.4	+0.1	+1.1	-0.5	+0.5

Table 11 shows immunisation coverage estimates for 'fully immunised' and for individual vaccines by 6 years of age for Australia and by state or territory. These are the third set of officially published ACIR figures of immunisation coverage estimates for this age group. 'Fully immunised' coverage at 6 years of age for Australia increased from the last quarter by 1.5 percentage points to 82.2 per cent. The greatest increase in coverage occurred in the Northern Territory (+10.8%) and New South Wales (+2.4%). All jurisdictions experienced increases in 'fully immunised' coverage for this age group. National coverage by individual vaccine also increased from the last quarter for all vaccines for this age group but there was some small variation in the changes in coverage by jurisdiction. The recent report published by NCIRS shows that true levels of coverage at 6 years of age are probably higher than reported here as late immunisation is still common.[1]

Figure 11 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. The recent increase in coverage over the past three quarters for all age groups is encouraging and indicates that in part the various initiatives and efforts that are taking place at present are probably having some impact on parents' decisions to immunise and immunisation providers' decisions to notify to the ACIR. However, the increase may also be a consequence of children in these recent cohorts being on the new schedule where receipt of only 2 doses of Hib vaccine is considered full immunisation for Hib at 12 months, according to the ACIR coverage algorithm. The greatest increase in coverage for individual vaccines at 12 months was for the Hib vaccine, an overall increase of 1.2 per cent compared with 0.7 per cent for both diphtheria-tetanus-pertusis and oral polio vaccine.

Figure 11. Trends in vaccination coverage, Australia, 1997 to 2002, by age cohorts

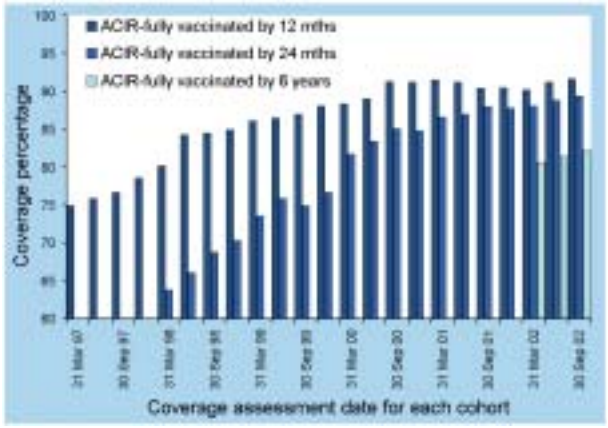

Table 10. Proportion of children immunised at 2 years of age, preliminary results by disease and State or Territory for the birth cohort 1 July to 30 September 2000; assessment date 31 December 2002[1]

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,043	21,942	788	12,593	4,437	1,494	15,376	6,293	63,966
Diphtheria, tetanus, pertussis (%)	89.3	90.8	86.4	92.0	91.4	93.4	92.0	91.2	91.4
Poliomyelitis (%)	94.4	94.4	96.1	94.6	95.6	96.3	95.5	94.1	94.8
Haemophilus influenzae type b (%)	94.3	93.6	93.0	94.0	94.1	95.2	94.6	93.2	94.0
Measles, mumps, rubella (%)	94.5	93.6	94.7	94.2	94.5	95.5	94.7	93.8	94.2
Hepatitis B (%)	-	-	-	-	-	-	-	-	-
Fully immunised (%)[2]	87.4	88.5	85.0	90.3	89.5	92.4	90.2	88.7	89.4
Change in fully immunised since last quarter (%)	-1.1	+1.6	-0.9	+1.5	+2.0	+2.8	+1.4	+2.4	+1.6

1. The 12 months age data for this cohort was published in *Commun Dis Intell* 2002;26:88.
2. These data relating to 2 year-old children should be considered as preliminary. The proportions shown as 'fully immunised' appear low when compared with the proportions for individual vaccines. This is at least partly due to poor identification of children on immunisation encounter forms.

Table 11. Proportion 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 1996; assessment date 31 December 2002

Vaccine	State or Territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,126	22,832	16,214	13,382	4,842	6,770	1,702	789	67,657
Diphtheria, tetanus, pertussis (%)	84.6	84.2	86.0	84.7	84.0	81.5	84.0	84.5	84.5
Poliomyelitis (%)	84.7	84.2	86.3	85.0	84.1	81.9	84.5	85.7	84.7
Haemophilus influenzae type b (%)	-	-	-	-	-	-	-	-	-
Measles, mumps, rubella (%)	83.3	82.4	85.9	84.5	83.3	81.3	83.0	85.9	83.7
Hepatitis B (%)	-	-	-	-	-	-	-	-	-
Fully immunised (%)[1]	81.9	80.8	84.8	82.9	81.8	79.6	81.6	82.8	82.2
Change in fully immunised since last quarter (%)	+0.6	+2.4	+10.8	+0.3	-0.0	+1.8	+1.5	+1.3	+1.5

1. These data relating to 6 year-old children should be considered as preliminary. The proportions shown as 'fully immunised' appear low when compared with the proportions for individual vaccines. This is at least partly due to poor identification of children on immunisation encounter forms.

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1. National Centre for Immunisation Research and Surveillance. Immunisation coverage: Australia 2001. Report. Canberra: Department of Health and Aged Care, 2001. Available from: http://www.health.gov.au/pubhlth/immunise/report.pdf

Acknowledgment: These figures were provided by the Health Insurance Commission, to specifications provided by the Commonwealth Department of Health and Ageing. For further information on these figures or data on the Australian Childhood Immunisation Register please contact the Immunisation Section of the Health Insurance Commission: Telephone: +61 2 6124 6607.

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. These pathogens include Salmonella, E. coli, Vibrio, Yersinia, Plesiomonas, Aeromonas and Campylobacter. Communicable Diseases Intelligence quarterly reports include only Salmonella.

Data are based on reports to NEPSS from Australian laboratories of laboratory-confirmed human infection with Salmonella. Salmonella are identified to the level of serovar and, if applicable, phage-type. Infections apparently acquired overseas are included. Multiple isolations of a single Salmonella serovar/phage-type from one or more body sites during the same episode of illness are counted once only. The date of the case is the date the primary diagnostic laboratory isolated a Salmonella from the clinical sample.

Note that the historical quarterly mean counts should be interpreted with caution, and are affected by surveillance artefacts such as newly recognised (such as S. Typhimurium 197 and S. Typhimurium U290) and incompletely typed Salmonella.

Reported by Joan Powling (NEPSS Co-ordinator) and Mark Veitch (Public Health Physician), Microbiological Diagnostic Unit — Public Health Laboratory, Department of Microbiology and Immunology, University of Melbourne. NEPSS can be contacted at the above address or by telephone: +61 3 8344 5701, facsimile: +61 3 9625 2689. For more information see Commun Dis Intell 2002;26:57.

Reports to the National Enteric Pathogens Surveillance System of Salmonella infection for the period 1 October to 31 December 2002 are included in Tables 12 and 13. Data include cases reported and entered by 30 January 2003. Counts are preliminary, and subject to adjustment after completion of typing and reporting of further cases to NEPSS.

Acknowledgement: Thanks to contributing laboratories and scientists.

Table 12. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 October to 31 December 2002, as reported to 30 January 2003

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total all *Salmonella* for quarter	22	527	45	411	105	47	223	170	1,550
Total contributing *Salmonella* types	12	92	27	101	39	10	68	70	207

Table 13. Top 25 *Salmonella* types identified in Australian States and Territories, 1 October to 31 December 2002

National rank	*Salmonella* type	State or territory								Total 4th quarter 2002	Last 10 years mean 4th quarter	Year to date 2002	Year to date 2001
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
1	*S.* Typhimurium 135	3	68	1	15	0	1	18	14	120	119	646	636
2	*S.* Typhimurium 170	2	54	0	33	0	0	17	1	107	20	419	148
3	*S.* Typhimurium 9	2	28	0	4	6	8	31	2	81	119	585	399
4	*S.* Potsdam	4	25	0	10	4	13	17	1	74	13	130	60
5	*S.* Saintpaul	0	16	8	28	1	0	8	11	72	65	385	289
6	*S.* Typhimurium 197	4	42	0	7	0	1	6	0	60	<1	108	8
7	*S.* Birkenhead	0	17	0	32	2	0	1	0	52	55	246	253
8	*S.* Montevideo	0	40	0	1	0	0	2	3	46	5	105	27
9	*S.* Chester	0	6	0	17	5	1	0	14	43	33	174	166
10	*S.* Typhimurium 126	0	5	0	4	7	2	12	1	31	23	203	313
11	*S.* Infantis	0	11	1	1	2	0	13	2	30	27	115	123
12	*S.* Muenchen	0	6	2	10	3	1	0	8	30	27	131	125
13	*S.* Virchow 8	0	6	0	20	0	0	1	1	28	24	273	245
14	*S.* Waycross	0	9	0	15	0	0	0	0	24	14	106	54
15	*S.* Agona	0	8	1	8	0	0	3	3	23	14	88	56
16	*S.* Hvittingfoss	0	7	1	14	0	0	1	0	23	12	154	89
17	*S.* Typhimurium U290	0	10	0	0	0	0	7	5	22	2	99	26
18	*S.* Typhimurium 99	0	0	0	0	22	0	0	0	22	1	33	40
19	*S.* Mississippi	0	1	0	1	0	17	1	0	20	13	93	124
20	*S.* Kottbus	0	9	0	3	1	0	5	1	19	8	51	26
21	*S.* subsp I ser 16;l,v:-	0	6	1	8	2	0	0	1	18	6	48	17
22	*S.* Enteritidis 4b	0	6	0	0	0	0	3	9	18	1	67	13
23	*S.* Enteritidis 4	0	5	0	5	2	0	2	3	17	48	40	90
24	*S.* Anatum	0	1	1	8	1	0	3	3	17	16	84	58
25	*S.* Stanley	1	4	0	5	1	0	1	5	17	10	59	107

Overseas briefs

World Health Organization

This material has been summarised from information on the World Health Organization Internet site. A link to this site can be found under 'Other Australian and international communicable diseases sites' on the Communicable Diseases Australia homepage.

Yellow fever in Senegal

As of 28 November 2002, the Ministry of Health has confirmed 60 cases of yellow fever and 11 deaths. The outbreak has affected 14 districts in 7 regions. Immunisation campaigns in 5 districts will begin within the next week. The World Health Organization is supporting these immunisation campaigns with both technical assistance and vaccine supplies.

West Nile virus

United States of America

As of 26 November 2002, the World Health Organization Collaborating Centre for Arthropod-Borne Viruses — Western Hemisphere, at the Centers for Disease Control and Prevention, has reported 3,737 human cases of the West Nile virus, with 214 deaths occurring in 39 states and the District of Columbia. During 2002, West Nile virus activity (evidence of infections in birds, humans, mosquitoes, and other animals, primarily horses) has been documented in 43 states and the District of Columbia. For more information about this outbreak see the Centers for Disease Control and Prevention's website at: http://www.cdc.gov/ncidod/dvbid/westnile/surv&control.htm.

Canada

As of 12 November 2002 Health Canada has reported a total number of 141 human cases of West Nile virus infection: 84 suspected cases and 57 confirmed cases and 2 deaths. One confirmed case was resident in the province of Alberta, one suspected case and 7 confirmed cases were resident in the province of Quebec, and the remaining 83 suspected cases and 49 confirmed cases were resident in the province of Ontario. One additional death in a confirmed case and 5 deaths in suspected cases are under investigation in Ontario to determine whether these can be attributed to West Nile virus infection. The one confirmed case in Alberta and one confirmed case in Ontario most likely acquired the infection while travelling in the United States of America whereas all other cases acquired their infection within their home province.

For more information about this outbreak see the Health Canada website at: http://www.hc-sc.gc.ca/pphb-dgspsp/wnv-vwn/ index.html

Meningococcal disease in the Great Lakes area, Burundi

As of 22 September 2002 the Ministry of Health in Burundi had reported a total of 934 cases of meningococcal infection and 68 deaths. Between 23 and 27 September, two cases were laboratory confirmed as *Neisseria meningitidis* serogroup W135. To date, only serogroup A had been identified in 53 samples from all affected provinces. Systematic sampling of suspected cases is ongoing in order to detect whether there has been a shift in the dominant serogroup from group A to group W135. In the meantime, the vaccination campaign with the current polysaccharide bivalent vaccine for serogroups A and C will continue in the affected provinces.

ProMED-mail

This material has been summarised from information provided by ProMED-mail (http://ww.promedmail.org). A link to this site can be found under 'Other Australian and international communicable diseases sites' on the Communicable Diseases Australia homepage.

Dengue update

Taiwan

Source: Taipei Times, 28 December 2002 (edited)

There have been a record number of dengue fever cases (5,227) reported in Taiwan in 2002. Outbreaks of dengue fever may occur throughout the year in Taiwan even without imported cases. The epidemic may not be wiped out in southern Taiwan this winter. Outbreaks of dengue fever in Taiwan in the past few years have been caused by dengue virus type 2.

Kelantan, Malaysia

Source: New Straits Times (Malaysia) online, 17 December 2002 (edited)

A total of 1,166 cases of dengue had been detected in the Kelantan State capital since early 2002, with one confirmed death. The figures show an increase on the 626 cases reported for the whole of 2001.

Malaysia

Source: Borneo Bulletin (Brunei), Agence France Presse, 17 December 2002 (edited)

In the first 10 months of 2002, the dengue fever outbreak in Malaysia had claimed the lives of 40 people. In September 2002, Malaysia placed its capital city, Kuala Lumpur, and four states, on dengue fever alert following an alarming surge in cases.

Health-care facilities — Washington State, United States of America

Source: Karen Steingart (edited)

In December 2002, two retirement centres in Clark County, Washington reported outbreaks of gastrointestinal illness. Over the course of the following 3 weeks, 8 similar outbreaks were reported from nursing homes, health care facilities, and the county jail. A case was defined as acute onset of gastrointestinal illness, including vomiting or diarrhoea, with onset of symptoms after 30 November 2002 in a person working or residing in one of the outbreak settings. Three hundred and fifty-four cases were identified, of which 326 (92%) were associated with four facilities.

The following preliminary analysis is based on cases from these four facilities. Eight cases were hospitalised. One of the 8 cases subsequently died of complications of pneumonia. Stool cultures from ill persons at one of the facilities were negative for enteric pathogens, however, stool samples from 2 patients at two different sites were positive for noroviruses by reverse transcriptase-polymerase chain reaction. Inspections of food preparation and handling practices at all of the facilities have not suggested a foodborne etiology. Control measures have included excluding ill staff from work; emphasising hand hygiene; and requiring meticulous cleaning of environmental surfaces with a 10 per cent solution of household bleach or other virucidal agent. While the origin of the outbreaks has not been identified, the distribution of onset dates suggests person-to-person transmission.

Cruise ships — United States of America

Source: The State/NY Daily News, 24 December 2002 (edited)

In recent months, there have been major outbreaks of gastrointestinal illness involving several thousand passengers in more than 20 outbreaks on cruise lines in the United States of America. This is triple the number from last year and more than the 4 previous years combined, according to the Centers for Disease Control and Prevention's vessel sanitation program. The health agency declares an outbreak when 3 per cent or more of cruise ship's passengers or crew members get the stomach illness. More passengers than ever are taking cruises. The industry expects to tally 7.5 million passengers by the end of this year, a 50 per cent increase since 1997.

Hospitals — British Colombia, Canada

Source: Canada.com, 24 December 2002 (edited)

At Nanaimo Regional General Hospital, British Columbia, two units are now closed after being hit by norovirus infection. The transitional care unit reports that 9 patients and 4 staff have symptoms, while 8 patients and one staff member have come down with the virus in the psychiatric input unit.

Lady Minto Hospital on Saltspring Island reports 33 staff and 16 patients in extended care have had norovirus infection symptoms since 10 December 2002. A further 38 patients and an unknown number of staff at Mount Tolmie extended-care facility are showing norovirus infection symptoms.

Tuberculosis — Russia

Source: Dr Alina Martynov (edited)

There is increasing tuberculosis (TB) morbidity in the territory of the Russian Primorsky region in the far eastern area of Russia. Tuberculosis has increased from 45 cases per 1,000 population in 1997 to 167 cases per 1,000 population in 2002. In addition, there has been a 54 per cent to 140 per cent increase in the rate of TB-related deaths among adults aged 25–44 years.

A vital contributing factor is HIV co-infection. A recent review of autopsy reports from the Vladivostok AIDS Centre showed that 67 (42.4 per cent) of 158 adult patients with AIDS were diagnosed with TB, indicating that this disease is the most common opportunistic infection for persons with AIDS. Commensurate with the increase in TB cases is a surge in the prevalence of multi-drug resistant tuberculosis in adult patients. Tuberculosis laboratories have reported an increase in the prevalence of multi-drug resistant strains of *Mycobacterium tuberculosis* from12.5 per cent in 2000 to 17.8 per cent in 2002.

Glycopeptide intermediate resistant Staphylococcus aureus death in Scotland

Source: Eurosurveillance Weekly Issue 51, 19 December 2002 (edited)

Laboratory tests have confirmed that a patient who died in Scotland last week was infected with a glycopeptide intermediate resistant *Staphylococcus aureus* (GISA).[1] GISA has increased resistance to first line antibiotics used for treating infections caused by methicillin resistant *Staphylococcus aureus* (MRSA), namely, vancomycin and teicoplanin.[2] This is the second report of an infection with this organism in Scotland and possibly the first report in the United Kingdom of an associated death.[3]

The patient, a woman in her early 50s, was admitted for routine bowel surgery but contracted MRSA while in an intensive therapy unit. Although the patient was treated for over 70 non-consecutive days with either vancomycin or teicoplanin in combination with meropenem or gentamicin or rifampin, the MRSA persisted and eventually developed resistance to glycopeptide antibiotics (vancomycin minimum inhibitory concentration 8 mg/L). Postmortem findings confirmed tricuspid endocarditis with metastatic abscesses throughout the lungs. The patient had been previously fit and well, apart from mild obesity and hypertension.

All patients, staff, and family members in contact with the patient during the period in which the MRSA acquired this additional resistance, have been offered screening as a precautionary measure. There is little evidence to show transmission of this organism from person to person and it is unlikely that there is any danger to the general public.[4] The unit was closed, cleaned, and screened before reopening.

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Human European bat lyssavirus death — Scotland

Source: Eurosurveillance Weekly, vol. 6, issue 50, 12 December 2002 (edited)

A human case of European bat lyssavirus (EBL) 2 infection in Scotland in November 2002 was confirmed by the Veterinary Laboratories Agency. This is the first confirmed human infection of EBL 2 in the United Kingdom (UK), and the first human rabies-like infection acquired in the United Kingdom since 1902. The man who became infected was admitted to hospital with an acutely progressing neurological illness and died on 24 November 2002. The clinical presentation was compatible with rabies and the man was a licensed bat handler who had been bitten by bats on several occasions in Scotland, including once during the period of incubation before he developed this illness. He had not recently travelled abroad to countries where rabies is endemic. The patient had not received rabies immunisation before or after exposure to bat bites.

This is the fourth confirmed human EBL infection in the world and only the second one with EBL 2. Prior to the recent diagnosis of rabies-like infection in Scotland, two EBL 2 infections have been confirmed in bats in the UK, one in Newhaven on the southern coast of England in 1996 and another in Lancashire in 2002. Both of these bat rabies cases were in Daubenton's bats. It was thought in 1996 that the bat might have migrated from continental Europe. However, the finding in 2002 of an infected bat well away from mainland Europe and the infection of a bat handler is consistent with EBL being now endemic in UK bats, although probably at very low levels. The Veterinary Laboratories Agency in the UK tests about 200 bats every year for this virus and has not so far identified EBL in any bats other than the two cases in 1996 and 2002.

EBL 1 and 2 are rabies-like viruses that are carried by insectivorous bats in Europe. They are from the same family of viruses (the Rhabdoviridae) as cause terrestrial rabies and bat rabies in the Americas and Australia, and are classified in the genus Lyssavirus, but differ in genotype and serotype. In a global context, classical rabies remains a greater public health burden than EBL, with 30,000 to 50,000 deaths per year. EBL 1 and 2 continue to present a small risk to human health in countries in Western Europe that are otherwise rabies-free.

vCJD update — United Kingdom

Source: UK Department of Health, press release 2002/0507, 2 December 2002 (edited)

On 6 December 2002, the United Kingdom Department of Health issued the latest information about the numbers of known cases of Creutzfeldt-Jakob disease. This includes cases of variant Creutzfeldt-Jakob disease (vCJD); the form of the disease thought to be linked to bovine spongiform encephalopathy. The following is a summary of vCJD cases:

- Deaths from definite vCJD (confirmed): 93
- Deaths from probable vCJD (without neuropathological confirmation): 25
- Deaths from probable vCJD (neuropathological confirmation pending): 1
- Number of deaths from definite or probable vCJD (as above): 119
- Total number of definite or probable vCJD (dead and alive): 129

Hepatitis A — New Zealand

Source: Lester Calder

In the first 5 months of 2002, 81 cases of hepatitis A virus infection were notified in New Zealand. This sharp increase was investigated with descriptive and analytical epidemiology, virology, product trace back, and an orchard investigation.

Consumption of raw blueberries was the only significant risk factor identified (adjusted odds ratio 7.6; 95 per cent confidence intervals 2.6–22.4). Trace-back of product through retailers and wholesalers implicated a single commercial orchard. hepatitis A virus was detected in cases' faeces and in blueberries from the orchard. A sanitary audit of the orchard revealed multiple opportunities for faecal contamination of product by pickers and the possibility of contamination by sewage-contaminated ground water. A child with confirmed hepatitis A was in the orchard during harvest. Extensive food safety improvements in the berry fruit industry are under way.

Campylobacter in chickens — United Kingdom

Source: Telegraph (UK), 20 November 2002 (edited)

Free-range and organic chickens are twice as likely to carry the food poisoning bacteria *Campylobacter* than battery hens, research said yesterday. The study investigated 60 organic and 130 conventional flocks and found *Campylobacter* in 58 per cent of indoor-reared flocks, but in 100 per cent of organic flocks. All chickens studied were destined for human consumption.

Campylobacter jejuni has become a major public health hazard and the main etiologic contributor — at least, quantitatively — to food poisoning in many countries, with contaminated raw poultry meat playing the main role. A survey conducted in the United Kingdom indicated that 50 per cent of United Kingdom-produced retail chickens are contaminated with *Campylobacter*. The emergence of quinolone and macrolide resistance in *Campylobacter jejuni* is a reason for additional concerns. Reducing *Campylobacter* levels in chickens is an essential part of cutting food poisoning cases in the UK.

Viral gastroenteritis

These reports have been edited to replace 'flu-like', 'Norwalk-like', 'bug', etc, with the ICTV approved designation 'Norovirus' for the agent responsible for sudden onset viral gastroenteritis.

Clinic-acquired cluster of hepatitis C — United States of America

Source: AP Online, 19 November 2002 (edited) and Nebraska Health and Human Services System, 19 November 2002 (edited)

At least 81 people treated at a Nebraska cancer clinic have tested positive for hepatitis C virus in an outbreak that may have been caused by a contaminated vial of medicine. Poor medical practices at the clinic may be to blame. The patients, who were suffering from cancer or blood disorders, were treated at the clinic in 2000 and 2001. It is possible that a clinic worker used a syringe to administer medicine to a patient who had hepatitis C, then drew more medicine from the same vial for the patient with the same syringe.

Of the 612 patients of the oncology clinic who received letters advising them to be tested, 485 chose to be tested for the virus, and of these, 81 tested positive. The apparent attack rate of 17 per cent (81 cases out of 485 tested) is high and suggests that unsafe injection practices were occurring over a period of time.

Malaria

Virginia, United States of America

Source: Donald Roberts. http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5141a1.htm

Local transmission of *Vivax malaria* in Virginia and Maryland have been recently reported.

The evidence consists of 2 locally-acquired cases of *Vivax malaria* and a remarkable abundance of competent malaria vector mosquitoes. Cases occurred in Virginia, close to the Potomac River, which is popular with people who fish along the river at all hours of the day and night. A proportion of people using the river comes from other countries. In 2001 there were 34 documented cases of imported malaria in Montgomery County, Maryland alone.

The residences of the locally-acquired cases were about 7 miles from Dulles International airport. The probability that a single mosquito was carried by plane then travelled 7 miles to transmission sites and successfully transmitted malaria to 2 individuals is thought to be remote.

During this outbreak investigation, mosquitoes, *Anopheles quadrimaculatus* in particular, have been collected from many localities around the Potomac River. *Anopheles quadrimaculatus*, which is historically the primary malaria vector in this region, has been the overwhelmingly dominant mosquito in Maryland collections since early August 2002. Mosquitoes collected in the vicinity of the 2 malaria cases, and in other locations, were also predominantly *Anopheles quadrimaculatus*.

The epidemiological factors listed above and cited in the Centers for Disease Control and Prevention's Morbidity and Mortality Weekly Report of 5 October 2002 suggest that local transmission of *Vivax malaria* occurred in late summer on at least two occasions on the Virginian side of the Potomac River.

Malaria and mosquito genomes decoded

Source: Press Release WHO/75 (edited)

The decoding of the genomes of the most dangerous malaria parasite, *Plasmodium falciparum*, and of the most important mosquito which transmits it, *Anopheles gambiae*, signals a turning point for global public health.

Malaria infects more than 300 million people every year, killing at least one million of them. About 90 per cent of the deaths are in children aged under 5 years. Both the mosquito and the parasite have evolved mechanisms to escape the limited, affordable technologies available in the developing world. Drugs targeting the parasite are losing their effectiveness. Today, resistance to choloroquine, the cheapest and most widely used antimalarial, is common throughout Africa.

Anopheles gambiae is an extremely efficient transmitter of the disease because of its strong preference for humans, and humans within its range can be bitten hundreds of times a day.

The breakthroughs announced in the journals *Nature* and *Science* open an entirely new field to public health researchers. With this new knowledge, malaria scientists will be able to design new insecticides, repellents, and drugs. TDR has for the last 2 years been training over 100 scientists from Latin America, Africa, and Asia in how to search the genomes, identify vulnerabilities, and build new genetically based drugs and insecticides.