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Front cover: Following the first case of inhalation anthrax in the United States on 4 October 2001 and subsequent anthrax scares and hoaxes in Australia, the nation's media has focused heavily on the planning and preparedness of health authorities to deal with a biological incident. The newspaper articles featured on the front cover are all from Australian newspapers and all relate to Australian issues. Cover art designed by the Public Health Media Unit, Commonwealth Department of Health and Ageing.

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Contents, *continued*

Editorial: looking back, looking forward

Jenean Spencer

This issue of *CDI* contains the annual report of the National Notifiable Diseases Surveillance System (NNDSS) for 1999, as well as the usual array of articles and latest surveillance reports. It will be our final issue in 2001, and is our third with the 'new look', which we hope everyone is enjoying.

The articles in this volume reflect some of the current issues in communicable diseases surveillance and control in Australia — the threat of intentional use of biological agents as weapons, transmissible spongiform encephalopathies (TSEs), tuberculosis (TB), meningococcal disease, arboviral disease, Q fever, foodborne illness, and antibiotic resistance. We have published the first in a series of reviews highlighting the networks that are vital to communicable disease surveillance in Australia. In this issue we have included an overview of the Communicable Diseases Network Australia (p266). While many people working in public health in this country are more than familiar with these networks, we recognise that not all of our readers may be aware of all functions and we hope that these articles will provide an interesting look at their history and development.

The events of 11 September 2001 and the use of anthrax as a biological weapon in the USA has impacted on communicable disease surveillance across Australia, with public health workers responding to numerous incidents. It has been a poignant reminder of our need for preparedness. The editorial on page 188 addresses the risk of anthrax and smallpox in Australia.

The article by Boyd and colleagues (p248) describes the Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR), one part of Australia's response to TSEs (for a review of Australia's response see *Commun Dis Intell* 2001; 25:99-100). At the time of writing the registry contained 405 probable or definite cases, reflecting a 30 year period of surveillance. While initially established in response to CJD associated with cadveric human pituitory hormone treatment for infertility or short stature, the ANCJDR has broadened its focus and now has the capacity to survey other forms of CJD, including variant CJD, should it occur in Australia. Familial, sporadic and healthcare acquired cases are captured in the surveillance system. The long incubation period highlights the need for continued and sustained vigilance. New certification requirements for imported beef (p253) are another part of Australia's response to TSEs.

Surveillance of TB notifications and TB drug resistance are covered by two articles in the current issue. The report from the National TB Advisory Committee summarises TB notifications for 1999 (p254). This report shows that in Australia, while our overall incidence rate is low, TB remains a concern for the health of indigenous Australians, as well the health of people born in countries where TB rates were high. In 1999 rates of new TB cases in overseas-born Australians was 20.6 cases per 100,000 and 8.3 per 100,000 in indigenous Australians, while the overall rate in Australia was 6.1 per 100,000 population.

Surveillance of TB drug resistance in Australia is co-ordinated by the Australian Mycobacterium Reference Laboratory Network. The report from this network by Dawson and colleagues (p261) covers the 1998 to 1999. The report indicates that single- and multi-drug resistance has remained relatively stable between 0.3–2.0, since 1994, with 0.5 per cent of all isolates showing multi-drug resistance in 1999. The results from this Network currently cannot be stratified according to whether the case was newly acquired or relapsed, nor can the laboratory isolates be linked back to notifications at the national level.

Enhancing the surveillance in NNDSS in 2002 will improve the quality of TB reporting. Linkage of notification, laboratory and clinical data will give a national perspective on issues such as drug resistance, effectiveness of treatment regimes and the identification of groups at higher risk. Drug resistance will be able to be correlated with country of birth and treatment history. This information will inform policy development in the area of TB treatment and management, and will contribute to efforts towards TB control in the WHO Western Pacific Region.

This year has been a time of change in the Surveillance Section of the Commonwealth Department of Health and Aged Care. In addition to changing the look of *CDI* , we have strived to improve the quality of reporting of national surveillance data. We continue, with the participation of State and Territory Health Departments, to implement a new data acquisition system for NNDSS. This system, which captures a much broader range of data, should be fully operational in 2002. The revision of the NNDSS system will have many advantages, including improved data quality, improved timeliness and decreased administrative load, allowing more time for us to work on the 'Intelligence' in*CDI* . Some of our goals for 2002 include improving timeliness (we hope the 2000 annual report will appear hot on the heels of the 1999 report contained in this issue), further improving the quality of our reporting, and implementing enhanced surveillance systems for a number of key diseases.

We welcome our new staff member, Patricia Hurtado , who has recently joined the *CDI* team as administrative assistant. I would also like to take this opportunity to thank the rest of the team (Paul Roche, Alison Milton and Ming Lin) who have all worked extremely hard to produce *CDI* this year. I would also like to acknowledge the support and contribution from our Director, Angela Merianos, who continues to be a guiding light and meticulous editor.

Finally, we would like to thank all of the people who have contributed to*CDI* this year, including authors of articles, the reviewers who took time out of their busy lives to review articles, and the vast number of people who contribute to all communicable diseases surveillance systems across the country. We wish everyone a Merry Christmas and a safe and prosperous New Year, and we look forward to your contributions to *CDI* in 2002.

Editorial: The risk of anthrax and smallpox in Australia

The world has changed since the September 11 terrorist attack on New York. Bioterrorism has raised its spectre and globally, anthrax false alarms and worse, deliberate hoaxes, have placed an enormous burden on emergency services and public health systems. The threat of anthrax has highlighted the importance of a multi-disciplinary and high level inter- agency approach to all biological emergencies so that security intelligence is wedded to health intelligence and that the lessons learnt from disaster management can be used when dealing with such events.

The threat of biological terrorism in Australia is low. Evidence supporting a domestic source of the anthrax letters in the United States (US) further reduces Australia's risk.[1] In spite of this intelligence, community anxiety since the first case of inhalational anthrax was confirmed in the US on 4 October has led to a need for information and reassurance. Health departments have been inundated with calls from people seeking information about the disease, what they should do to protect themselves and the availability of vaccination against anthrax and smallpox. In Australia, public health professionals and emergency services personnel have worked hand in hand to manage 'white powder' incidents. Health authorities have also acted quickly to build on existing plans to deal with anthrax and other threats, however unlikely.

Despite the high number of white powder incidents reported in the Australian media no anthrax spores have been detected in any of the samples tested and there have been no cases of anthrax. However, such incidents cause significant anxiety to the people involved and to the public in general. A large component of the work for health professionals is therefore risk communication — reassuring the worried well, providing information to the public should a real incident ever occur, and preventing the mental health consequences of an intangible but implied global threat. General practitioners, State and Territory health authorities and the Commonwealth Department of Health and Ageing Care (formerly Department of Health and Aged Care) have already played a key role in reassuring people and providing reasoned responses to inquiries.

Australia's federal system of governance means that a response to any biological emergency requires a collaborative process between the Commonwealth, States and Territories. Preparedness planning to meet the challenge of new and emerging infectious diseases, including those released deliberately, should build on existing disease and disaster surveillance systems and infrastructures. The Communicable Diseases Network Australia and the Public Health Laboratory Network have been working towards strengthening surveillance systems, outbreak response capacity and laboratory technologies for the early detection and confirmation of all infectious diseases of public health importance as recommended by the National Communicable Diseases Surveillance Strategy, 1995.[2] More recently, both networks have been working towards finalising a revised set of disease case definitions, including those for biological agents that have the potential to be used as weapons. In addition, strong communication linkages with other government agencies and the general public are required and are being actively pursued.

Anthrax

Public health professionals have drawn on the international literature on anthrax and other biological agents, especially advice from the US Centers for Disease Control and Prevention (CDC), the World Health Organization (WHO) and the UK Public Health Laboratory Service, as guides to policy development, adapted to meet Australia's needs. Anthrax treatment and post-exposure prophylaxis guidelines have been developed as a consensus document between public health physicians, microbiologists and infectious diseases specialists and endorsed by Australia's Chief Health Officers and Directors of Public Health services. Work is progressing on other public health and clinical protocols.

Agreement has also been reached that primary care providers should not prescribe or supply chemoprophylaxis in the event of suspected anthrax. Instead they should notify their local, State or Territory public health unit immediately by phone for advice about referral for diagnosis and further management (see contact details for State and Territory health authorities on the Anthrax Fact Sheet at: http://www.health.gov.au). General practitioners should not supply individuals requesting a contingency supply of antibiotics for treatment or prophylaxis, in order to prevent inappropriate use of these antibiotics.

Health authorities in all jurisdictions have taken steps to ensure that Australia has adequate supplies of essential antibiotics in the case of an emergency. The Commonwealth is working with pharmaceutical companies to ensure supply continuity.

Anthrax vaccine is not registered for use in Australia.

Smallpox

Initial indications that the US Government intended to procure 250-300 million doses of smallpox vaccine for mass vaccination appears to have been modified following expert advice, both international and from within the US. Existing vaccines have proven efficacy but also have a high incidence of adverse effects. The risk of adverse events is sufficiently high that mass vaccination is not warranted if there is no or little real risk of exposure.

World Health Organization guidance[3] is that vaccination of entire populations is not recommended.

Nowhere in the world has there been a smallpox release and, despite the USA's announcement to develop a new vaccine supply against a possible bioterrorism incident, no country in the world is routinely giving smallpox vaccine to its citizens. DA Henderson,[4] interviewed on the SBS program *News Hour*,[5] agreed with the WHO guidance that case detection, post-exposure vaccination and 'ring fencing' an outbreak were the appropriate responses to the reintroduction of smallpox. This approach is the basis of the CDC interim smallpox response plan announced on 26 November.[6,7] Unlike many communicable diseases,

smallpox is not transmissible during the incubation period so cases only become infectious when they develop symptoms, hence the effectiveness of search and containment of cases during the global eradication program. In addition, post-exposure vaccination can prevent smallpox even after exposure to the virus.[3]

The calf lymph-derived live smallpox vaccine that was used in the WHO smallpox eradication program, is associated with a post vaccinal encephalitis rate of 3–4 per million primary vaccine doses. Forty per cent of cases are fatal and some patients are left with permanent neurological deficits.[8] In addition, progressive vaccinia occurs among those who are immunocompromised and there is no smallpox vaccine available today with proven safety for use in this group of people. HIV/AIDS was unknown when the last doses of smallpox vaccine were administered in the 1970s. Unlike today when the risks far outweigh the benefits, mass vaccination was used to eradicate smallpox globally and the last case of naturally acquired smallpox occurred in Somalia in 1979.

Due to the eradication of smallpox, Australia has no smallpox vaccine available currently and there is no indication for vaccinating the Australian public against smallpox. However, as a precautionary measure, the Commonwealth Department of Health and Ageing has had discussions with international agencies to secure access to vaccine in the most unlikely event of a smallpox incident. Finally, in the unlikely event that smallpox was re-introduced, Australia needs to be well prepared to implement a strategy of surveillance, quarantine and vaccination. WHO has pledged to support any country in controlling smallpox should it occur as the incident would be considered an international emergency. WHO will help countries to pool available resources so as to contain the disease as rapidly and effectively as possible.

Fact sheets on the Internet

For the assistance of doctors there is now a comprehensive guide to dealing with patients' inquiries posted on the Commonwealth Department of Health's Website at: http://www.health.gov.au.

This guide covers enquires by patients, patients presenting with clinical symptoms, requests for diagnostic testing, and links to Fact Sheets on anthrax and smallpox. There is also a list of contacts for public health authorities around Australia.

Professor Richard Smallwood
Chief Medical Officer
Commonwealth Department of Health and Ageing

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1. Hoaxers spread panic across the globe. New Scientist, 31 October 2001, http://www.newscientist.com/hottopics/bioterrorism/hoaxers.jsp
2. The National Communicable Diseases Surveillance Strategy. Report prepared by the Steering Committee, National Communicable Diseases Surveillance Strategy and the Commonwealth Department of Health and Family Services on behalf of the Chief Health Officers of Australia. www.health.gov.au/pubhlth/publicat/document/ncdss.pdf
3. Statement WHO/16 2 October 2001 Statement to the press by the Director-General of the World Health Organization, Dr Gro Harlem Brundtland — World Health Organization announces updated guidance on smallpox vaccination. http:/www.who.int/inf-pr-2001/en/state2001-16.htm
4. Professor Henderson is a distinguished academic at the Johns Hopkins University, holding an appointment in the Department of Epidemiology. Prof Henderson directed the World Health Organization's global smallpox eradication campaign (1966-1977) and helped initiate WHO's global program of immunisation in 1974. He is currently Director of the Office of Public Health Preparedness at the Department of Health and Human Services in Washington, DC.
5. SBS News Hour Wednesday 21 November 2001.
6. Laura Meckler, Associated Press, 27 November 2001. CDC releases Smallpox Response Plan. http://news.excite.com/news/ap/011127/08/attacks-smallpox
7. Reuters Health eLine, 26 November 2001. CDC releases. Plan for a smallpox emergency. Http://www.reutershealth.com/frame2/eline.html
8. Henderson DA. Smallpox: Clinical and epidemiological features. *Emerging Infectious Diseases* 1999;5:537-539.

Australia's Notifiable Diseases Status, 1999

Annual report of the National Notifiable Diseases Surveillance System

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With Contributions From:

National organisations

Communicable Diseases Network Australia
Australian Childhood Immunisation Register
Australian Gonococcal Surveillance Programme
Australian meningococcal Surveillance Programme
Australian Sentinel Practice Research Network
Australian Quarantine Inspection Service
National Centre in HIV Epidemiology and Clinical Research
National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases
National Enteric Pathogens Surveillance Scheme
National Rotavirus Research Centre
Sentinel Chicken Surveillance Programme
The National CJD Registry
WHO Collaborating Centre for Reference and Research on Influenza

State and Territory health departments

Communicable Diseases Control Unit, ACT Department of Health and Community Care, ACT
Communicable Diseases Surveillance and Control Unit, NSW Health Department, New South Wales
Centre for Disease Control, Territory Health Services, Northern Territory
Communicable Diseases Unit, Queensland Health, Queensland
Communicable Diseases Control Branch, South Australian Health Commission, South Australia
Communicable Diseases Surveillance, Department of Health and Human Services, Tasmania
Communicable Diseases Section, Department of Human Services, Victoria
Communicable Diseases Control Branch, Health Department of Western Australia, Western Australia

Abstract

In 1999 there were 88,239 notifications of communicable diseases in Australia reported to the National Notifiable Diseases Surveillance System (NNDSS). The number of notifications in 1999 was an increase of 3 per cent on notifications in 1998 (85,227) and the second largest reporting year since the NNDSS commenced in 1991. Notifications in 1999 consisted of 29,977 bloodborne infections (34% of total), 22,255 gastrointestinal infections (25%), 21,704 sexually transmitted infections (25%), 5,986 vector borne infections (7%), 5,228 vaccine preventable infections (6%), 1,967 (2%) other bacterial infections (legionella, meningococcal, leprosy and tuberculosis), 1,012 zoonotic infections (1%) and 3 quarantinable infections (0.003%). Notifications of bloodborne viral diseases particularly hepatitis B and hepatitis C and some sexually transmitted infections such as gonorrhoea and chlamydia continue to increase in Australia. Steep declines in vaccine preventable diseases such as *Haemophilus influenzae* type b, measles, mumps and rubella continued in 1999. This report also summarises data on communicable diseases from other surveillance systems including the Laboratory Virology and Serology Surveillance Scheme (LabVISE) and sentinel general practitioner schemes. In addition this report comments on other important developments in communicable disease control in Australia in 1999. *Commun Dis Intell* 2001;25:190-245.

Keywords: Surveillance, communicable diseases, epidemiology

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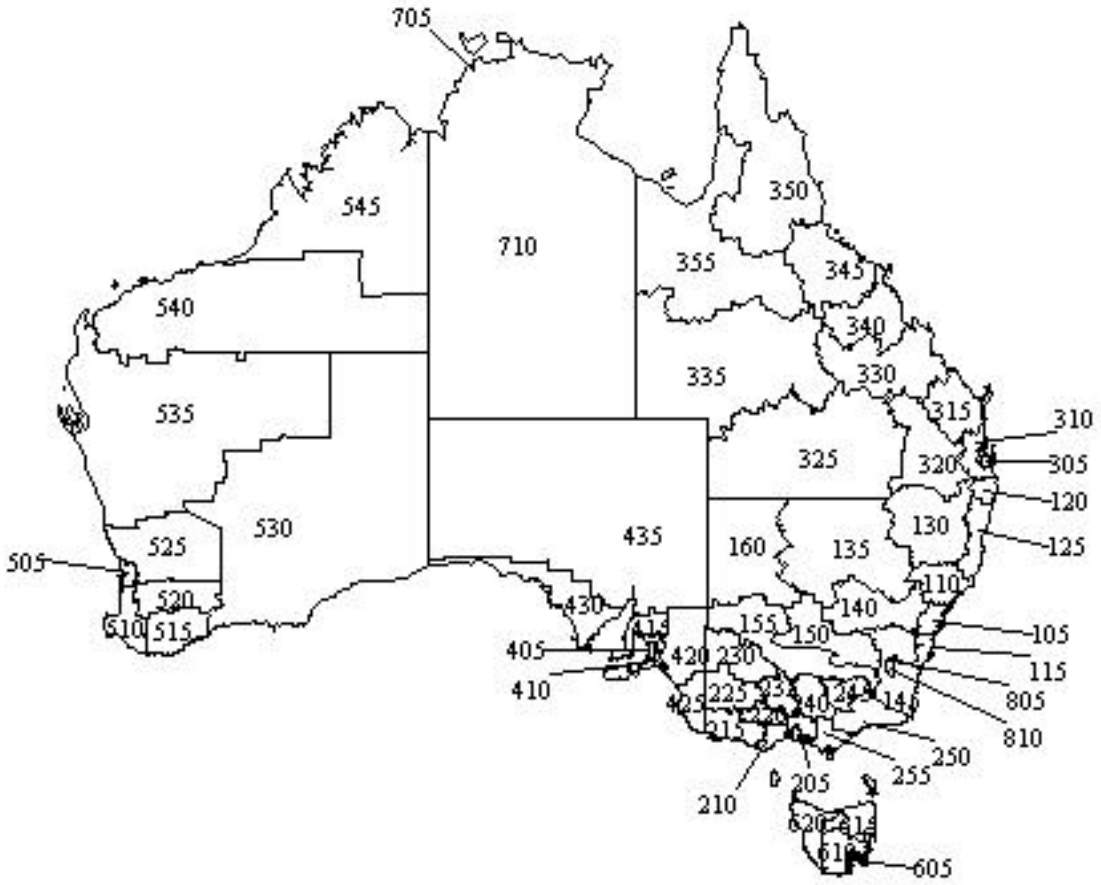
Abbreviations

ABS	Australian Bureau of Statistics
ACT	Australian Capital Territory
ACIR	Australian Childhood Immunisation Register
AGSP	Australian Gonococcal Surveillance Programme
AIHW	Australian Institute of Health and Welfare
Ag	Antigen
APSU	Australian Paediatric Surveillance Unit
ASPREN	Australian Sentinel Practice Research Network
ATAGI	Australian Technical Advisory Group on Immunisation
BF	Barmah Forest virus
BSE	Bovine spongiform encephalopathy
CDC	Centers for Disease Control and Prevention
CDI	*Communicable Diseases Intelligence*
CDNA	Communicable Diseases Network Australia
CJD	Creutzfeldt-Jakob disease
CSF	Cerebrospinal fluid
DHAC	Department of Health and Aged Care
DTP	Diphtheria, tetanus, pertussis (vaccine)
ELISA	Enzyme-linked immunosorbent assay
GP	General practitioner
GPII	General Practitioner Immunisation Incentives
HBcAg	Hepatitis B core antigen
HBsAg	Hepatitis B surface antigen
HBV	Hepatitis B virus
HCV	Hepatitis C virus
HDV	Hepatitis D virus
HEV	Hepatitis E virus
Hib	*Haemophilus influenzae* type b
HIV	Human immunodeficiency virus
HUS	Haemolytic uraemic syndrome
ICD10	International Classification of Diseases Version 10
IFA	Immunofluorescence assay
IgG	Immunoglobulin G
IgM	Immunoglobulin M
JE	Japanese encephalitis
JETACAR	Joint Expert Technical Advisory Committee on Antibiotic Resistance
LabVISE	Laboratory Virology and Serology Surveillance Scheme
LGV	Lymphogranuloma venereum
MMR	Measles-mumps-rubella (vaccine)
MMWR	Morbidity and Mortality Weekly Report
MRLN	Australian Mycobacterial Reference Laboratory Network
MVE	Murray Valley encephalitis

NNDSS	National Notifiable Diseases Surveillance System
NCHECR	National Centre in HIV Epidemiology and Clinical Research
NCIRS	National Centre for Immunisation Research and Surveillance (of Vaccine Preventable Diseases)
NEC	Not elsewhere classified
NEPSS	National Enteric Pathogen Surveillance Scheme
NHMRC	National Health and Medical Research Council
NLV	Norwalk-like virus
NMSS	National Mycobacterial Surveillance System
NN	Not notifiable
NSW	New South Wales
NT	Northern Territory
OPV	Oral polio vaccine
PCR	Polymerase chain reaction
PHLN	Public Health Laboratory Network
Qld	Queensland
RR	Ross River virus
SA	South Australia
SD	Statistical Division
SLTEC, VTEC	Shiga-like toxin producing *Escherichia coli*, verotoxigenic *E. coli*
STI	Sexually transmitted infection
Tas	Tasmania
TB	Tuberculosis
UK	United Kingdom
VAPP	Vaccine associated paralytic poliomyelitis
Vic	Victoria
WA	Western Australia
WHO	World Health Organization

Map 1. Australian Bureau of Statistics Statistical Divisions



Statistical Division		Population
Australian Capital Territory		
805	Canberra	309,850
810	ACT - balance	323
New South Wales		
105	Sydney	4,041381
110	Hunter	572,802
115	Illawarra	385,489
120	Richmond-Tweed	209,281
125	Mid-North Coast	271,330
130	Northern	174,955
135	North Western	117,588
140	Central West	173,306
145	South Eastern	181,608
150	Murrumbidgee	148,968
155	Murray	110,727
160	Far West	24,245
Northern Territory		
705	Darwin	88,124
710	NT - balance	104,758
Queensland		
305	Brisbane	1,601,417
310	Moreton	657,927

Statistical Division		Population
Queensland continued		
320	Darling Downs	201,446
325	South West	25,711
330	Fitzroy	181,202
335	Central West	12,255
340	Mackay	125,977
345	Northern	197,302
350	Far North	222,451
355	North West	35,683
South Australia		
405	Adelaide	1,092,857
410	Outer Adelaide	109,065
415	Yorke & Lower North	44,058
420	Murray Lands	68,435
425	South East	62,905
430	Eyre	33,251
435	Northern	82,503
Tasmania		
605	Greater Hobart	194,166
610	Southern	34,689
615	Northern	133,016
620	Mersey-Lyell	108,390

Statistical Division		Population
Victoria		
205	Melbourne	3,417,218
210	Barwon	245,582
215	Western District	99,050
220	Central Highlands	137,353
225	Wimmera	51,503
230	Mallee	88,204
235	Loddon-Campaspe	161,419
240	Goulburn	186,683
245	Ovens-Murray	90,541
250	East Gippsland	80,730
255	Gippsland	153,890
Western Australia		
505	Perth	1,364,188
510	South West	182,837
515	Lower Great Southern	51,840
520	Upper Great Southern	19,734
525	Midlands	52,697
530	South Eastern	58,778
535	Central	60,262
540	Pilbara	41,153
545	Kimberley	29,527

Table 1. Notifications of communicable diseases by State or Territory health authorities in 1999, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Bloodborne									
Hepatitis B (incident)	3	68	19	55	19	5	93	45	307
Hepatitis B (unspecified)[1]	67	4,327	NN	839	257	34	2,174	393	8,091
Hepatitis C (incident)	20	77	0	-	87	18	70	113	385
Hepatitis C (unspecified)[1]	280	9,215	232	3,104	924	306	6,162	1,021	21,244
Hepatitis D	0	16	0	5	0	0	0	NN	21
Hepatitis (NEC)	0	0	0	0	0	0	0	NN	0
Gastrointestinal									
Botulism	0	0	0	0	0	0	0	NN	0
Campylobacterosis[2]	290	-	237	3,200	2,405	411	4,686	1,414	12,643
Haemolytic uraemic syndrome	0	11	1	1	3	0	8	0	24
Hepatitis A	8	413	89	360	121	5	269	292	1,557
Hepatitis E	1	0	0	0	0	0	1	NN	2
Listeriosis	0	22	0	11	3	2	13	12	63
Salmonellosis	65	1,467	356	2,231	963	145	1,214	713	7,154
Shigellosis[2]	5	-	111	129	72	1	117	112	547
SLTEC,VTEC[3]	0	0	0	NN	39	0	4	NN	43
Typhoid	0	38	0	3	5	0	17	9	72
Yersiniosis[2]	1	-	0	101	18	0	17	6	143
Quarantinable									
Cholera	0	2	0	0	0	0	1	0	3
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									
Chancroid	0	0	0	0	0	0	0	0	0
Chlamydial infection	177	2,477	856	4,472	1,012	251	2,940	1,897	14,082
Donovanosis	0	0	6	3	NN	0	0	7	16
Gonococcal infection[4]	20	1,306	1,138	1,186	235	19	785	987	5,676
Lymphogranuloma venereum	0	0	0	0	0	0	0	NN	0
Syphilis[5]	10	668	328	829	19	9	6	110	1,979

Table 1. (continued) Notifications of communicable diseases by State or Territory health authorities in 1999, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Vaccine preventable									
Diphtheria	0	0	0	0	0	0	0	0	0
Haemophilus influenzae type b	1	13	3	12	4	0	3	4	40
Measles	5	32	10	33	5	11	111	23	230
Mumps	8	33	3	12	12	4	73	39	184
Pertussis	83	1,426	2	963	218	611	997	96	4,396
Poliomyelitis	0	0	0	0	0	0	0	0	0
Rubella[6]	17	46	3	157	4	7	121	21	376
Tetanus	0	1	0	1	0	0	0	0	2
Vectorborne									
Arbovirus infection NEC	0	0	1	12	0	0	46	3	62
Barmah Forest virus infection	0	249	18	310	0	2	13	47	639
Dengue	1	13	31	62	6	0	0	18	131
Malaria	22	180	67	304	29	9	81	32	724
Ross River virus infection	8	963	157	2,308	41	67	247	625	4,416
Zoonoses									
Brucellosis	0	0	0	49	0	0	3	0	52
Hydatid infection	0	NN	0	5	3	1	17	3	29
Leptospirosis	0	57	1	218	3	1	29	9	318
Ornithosis	0	NN	0	NN	9	2	66	7	84
Q fever	0	165	0	297	10	0	26	20	518
Other bacterial infections									
Legionellosis	2	40	4	34	63	2	61	43	249
Leprosy	0	1	0	1	0	0	1	3	6
Meningococcal infection	5	220	8	85	27	13	138	72	568
Tuberculosis	12	469	100	105	69	9	298	91	1,153
Total	1,111	24,015	3,781	21,497	6,685	1,945	20,908	8,287	88,229

1. Unspecified hepatitis includes cases with hepatitis in whom the duration of illness can not be determined.
2. Notified as 'foodborne disease' or 'gastroenteritis in an institution' in New South Wales
3. Infections with Shiga-like toxin (Verotoxigenic 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 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 the NNDSS

NN Not notifiable

NEC Not Elsewhere Classified

\- Elsewhere classified

Table 2. Notification rates of diseases by State or Territory, 1999 (rate per 100,000 population)

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Bloodborne diseases									
Hepatitis B (incident)	1.0	1.1	9.9	1.6	1.3	1.1	2.0	2.4	1.6
Hepatitis B (unspecified)[1]	21.4	67.5	NN	23.9	17.2	7.2	46.1	21.1	42.7
Hepatitis C (incident)	6.4	1.2	0	-	5.8	3.8	1.5	6.1	2.5
Hepatitis C (unspecified)[1]	89.4	143.7	120.3	88.4	61.9	65.1	130.8	54.9	112.0
Hepatitis D	0.0	0.2	0.0	0.1	0.0	0.0	0.0	NN	0.1
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	NN	0.0
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	NN	0.0
Campylobacterosis[2]	92.5	-	122.9	91.1	161.1	87.4	99.4	76.0	100.7
Haemolytic uraemic syndrome	0.0	0.2	0.5	0.0	0.2	0.0	0.2	0.0	0.1
Hepatitis A	2.6	6.4	46.1	10.2	8.1	1.1	5.7	15.7	8.2
Hepatitis E	0.3	0.0	0.0	0.0	0.0	0.0	0.0	NN	0.0
Listeriosis	0.0	0.3	0.0	0.3	0.2	0.4	0.3	0.6	0.3
Salmonellosis	20.7	22.9	184.6	63.5	64.5	30.8	25.8	38.3	37.7
Shigellosis[2]	1.6	-	57.5	3.7	4.8	0.2	2.5	6.0	4.4
SLTEC,VTEC[3]	0.0	0.0	0.0	NN	2.6	0.0	0.1	NN	0.3
Typhoid	0.0	0.6	0.0	0.1	0.3	0.0	0.4	0.5	0.4
Yersiniosis[2]	0.3	-	0.0	2.9	1.2	0.0	0.4	0.3	1.1
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sexually transmitted									
Chancroid	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Chlamydial infection	56.5	38.6	443.8	127.3	67.8	53.4	62.4	101.9	74.2
Donovanosis	0.0	0.0	3.1	0.1	NN	0.0	0.0	0.4	0.1
Gonococcal infection[4]	6.4	20.4	590.0	33.8	15.7	4.0	16.7	53.0	29.9
Lymphogranuloma venereum	0.0	0.0	0.0	0.0	0.0	0.0	0.0	NN	0.0
Syphilis[5]	3.2	10.4	170.1	23.6	1.3	1.9	0.1	5.9	10.4

Table 2. (continued) Notification rates of diseases by State or Territory, 1999 (rate per 100,000 population)

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Vaccine preventable									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.3	0.2	1.6	0.3	0.3	0.0	0.1	0.2	0.2
Measles	1.6	0.5	5.2	0.9	0.3	2.3	2.4	1.2	1.2
Mumps	2.6	0.5	1.6	0.3	0.8	0.9	1.5	2.1	1.0
Pertussis	26.5	22.2	1.0	27.4	14.6	129.9	21.2	5.2	23.2
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella[6]	5.4	0.7	1.6	4.5	0.3	1.5	2.6	1.1	2.0
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	0.0	0.5	0.3	0.0	0.0	1.0	0.2	0.3
Barmah Forest virus infection	0.0	3.9	9.3	8.8	0.0	0.4	0.3	2.5	3.4
Dengue	0.3	0.2	16.1	1.8	0.4	0.0	0.0	1.0	0.7
Malaria	7.0	2.8	34.7	8.7	1.9	1.9	1.7	1.7	3.8
Ross River virus infection	2.6	15.0	81.4	65.7	2.7	14.2	5.2	33.6	23.3
Zoonoses									
Brucellosis	0.0	0.0	0.0	1.4	0.0	0.0	0.1	0.0	0.3
Hydatid infection	0.0	NN	0.0	0.1	0.2	0.2	0.4	0.2	0.2
Leptospirosis	0.0	0.9	0.5	6.2	0.2	0.2	0.6	0.5	1.7
Ornithosis	0.0	NN	0.0	NN	0.6	0.4	1.4	0.4	0.9
Q fever	0.0	2.6	0.0	8.5	0.7	0.0	0.6	1.1	2.7
Other bacterial infections									
Legionellosis	0.6	0.6	2.1	1.0	4.2	0.4	1.3	2.3	1.3
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0
Meningococcal infection	1.6	3.4	4.1	2.4	1.8	2.8	2.9	3.9	3.0
Tuberculosis	3.8	7.3	51.8	3.0	4.6	1.9	6.3	4.9	6.1

1. Unspecified hepatitis includes cases with hepatitis in whom the duration of illness can not be determined.
2. Notified as 'foodborne disease' or 'gastroenteritis in an institution' in New South Wales
3. Infections with Shiga-like toxin (verotoxigenic 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

NN Not notifiable
NEC Not Elsewhere Classified
- Elsewhere classified.

Table 3. National Notifiable Diseases Surveillance System notifications and rates, 1995 to 1999, by year and disease

Disease	Notifications					Rate per 100,000 population				
	1995	1996	1997	1998	1999	1995	1996	1997	1998	1999
Bloodborne										
Hepatitis B (incident)	331	213	268	262	307	1.8	1.2	1.4	1.4	1.6
Hepatitis B (unspecified)[1]	6,477	5,911	7,044	6,620	8,091	35.8	32.3	38.0	35.3	42.7
Hepatitis C (incident)	70	75	154	345	385	0.5	0.5	1.0	2.3	2.5
Hepatitis C (unspecified)[1]	9,959	9,712	19,331	19,006	21,244	55.1	53.0	104.4	101.4	112.0
Hepatitis D	37	14	17	10	21	0.2	0.1	0.1	0.1	0.1
Hepatitis (NEC)	13	16	5	4	0	0.1	0.1	0.0	0.0	0.0
Gastrointestinal										
Botulism	0	0	0	1	0	0.0	0.0	0.0	0.0	0.0
Campylobacterosis[2]	11,298	12,117	11,742	13,282	12,643	94.6	100.1	95.8	107.0	100.7
Haemolytic uraemic syndrome	-	-	4	13	24	-	-	0.0	0.1	0.1
Hepatitis A	1,645	2,112	3,069	2,443	1,557	9.1	11.5	16.6	13.0	8.2
Hepatitis E	5	4	7	1	2	0.0	0.0	0.0	0.0	0.0
Listeriosis	60	68	73	55	63	0.3	0.4	0.4	0.3	0.3
Salmonellosis	6,041	5,791	7,089	7,489	7,154	33.4	31.6	38.3	39.9	37.7
Shigellosis[2]	734	677	796	594	547	6.1	5.6	6.5	4.8	4.4
SLTEC,VTEC[3]	-	-	20	14	43	-	-	0.2	0.1	0.3
Typhoid	75	79	81	63	72	0.4	0.4	0.4	0.3	0.4
Yersiniosis[2]	309	272	246	190	143	2.6	2.2	2.0	1.5	1.1
Quarantinable										
Cholera	5	5	2	4	3	0.0	0.0	0.0	0.0	0.0
Plague	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Rabies	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0
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										
Chancroid	2	1	1	1	0	0.0	0.0	0.0	0.0	0.0
Chlamydial infection	6,398	8,445	9,242	11,339	14,082	35.4	46.1	49.9	60.5	74.2
Donovanosis	84	49	48	27	16	0.5	0.3	0.3	0.2	0.1
Gonococcal infection[4]	3,315	4,175	4,692	5,398	5,676	18.3	22.8	25.3	28.8	29.9
Lymphogranuloma venereum	1	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Syphilis[5]	1,810	1,510	1,355	1,677	1,979	10.0	8.2	7.3	8.9	10.4

Table 3. (continued) National Notifiable Diseases Surveillance System notifications and rates, 1995 to 1999, by year and disease

Disease	Notifications					Rate per 100,000 population				
	1995	1996	1997	1998	1999	1995	1996	1997	1998	1999
Vaccine preventable										
Diphtheria	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	78	49	52	34	40	0.4	0.3	0.3	0.2	0.2
Measles	1,194	481	858	290	230	6.6	2.6	4.6	1.5	1.2
Mumps	157	126	191	182	184	0.9	0.7	1.0	1.0	1.0
Pertussis	4,247	4,384	10,941	5,739	4,396	23.5	23.9	59.1	30.6	23.2
Poliomyelitis	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0
Rubella[7]	4,590	2,556	1,389	745	376	25.4	14.0	7.5	4.0	2.0
Tetanus	7	3	7	8	2	0.0	0.0	0.0	0.0	0.0
Vectorborne										
Arbovirus infection NEC	69	47	21	83	62	0.4	0.3	0.1	0.4	0.3
Barmah Forest virus infection	759	859	696	531	639	4.2	4.7	3.8	2.8	3.4
Dengue	39	123	175	509	131	0.2	0.7	0.9	2.7	0.7
Malaria	623	866	751	647	724	3.4	4.7	4.1	3.5	3.8
Ross River virus infection	2,684	7,853	6,643	3,128	4,416	14.9	42.9	35.9	16.7	23.3
Zoonoses										
Brucellosis	27	39	40	43	52	0.1	0.2	0.2	0.2	0.3
Hydatid infection	51	44	61	40	29	0.3	0.2	0.3	0.2	0.2
Leptospirosis	165	215	128	189	318	0.9	1.2	0.7	1.0	1.7
Ornithosis	186	86	35	64	84	1.3	0.6	0.2	0.4	0.9
Q fever	466	554	580	560	518	2.6	3.0	3.1	3.0	2.7
Other bacterial infections										
Legionellosis	164	201	157	262	249	0.9	1.1	0.8	1.4	1.3
Leprosy	10	7	12	4	6	0.1	0.0	0.1	0.0	0.0
Meningococcal infection	381	421	484	453	568	2.1	2.3	2.6	2.4	3.0
Tuberculosis	1,154	971	1,043	972	1,153	6.4	5.3	5.6	5.2	6.1
Total	65,720	71,131	89,550	83,321	88,229					

1. Unspecified hepatitis includes cases with hepatitis in whom the duration of illness can not be determined.
2. Notified as 'foodborne disease' or 'gastroenteritis in an institution' in New South Wales.
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

NN Not notifiable

NEC Not Elsewhere Classified

\- Elsewhere classified.

Table 4. Diseases notified to the National Notifiable Diseases Surveillance System in 1999

Disease Group	Disease	Comments
Bloodborne disease	Hepatitis B (incident)	All jurisdictions
	Hepatitis B (unspecified)	All jurisdictions except NT
	Hepatitis C (incident)	All jurisdictions except Qld
	Hepatitis C (unspecified)	All jurisdictions
	Hepatitis D	All jurisdictions except WA
	Hepatitis (NEC)	All jurisdictions except WA
Gastrointestinal disease	Botulism	All jurisdictions except WA
	Camplyobacteriosis	All jurisdictions except NSW
	Haemolytic uraemic syndrome	All jurisdictions
	Hepatitis A	All jurisdictions
	Hepatitis E	All jurisdictions except WA
	Listeriosis	All jurisdictions
	Salmonellosis	All jurisdictions
	Shigellosis	All jurisdictions except NSW
	SLTEC, VTEC	All jurisdictions except Qld, WA
	Typhoid	All jurisdictions
	Yersiniosis	All jurisdictions except NSW
Quarantinable diseases	Cholera	All jurisdictions
	Plague	All jurisdictions
	Rabies	All jurisdictions
	Viral haemorrhagic fever	All jurisdictions
	Yellow fever	All jurisdictions
Sexually transmitted infections	Chancroid	All jurisdictions
	Chlamydial infections	All jurisdictions
	Donovanosis	All jurisdictions except SA
	Gonococcal Infections	All jurisdictions
	Lymphogranuloma venereum	All jurisdictions except WA
	Syphilis	All jurisdictions
Vaccine preventable diseases	Diphtheria	All jurisdictions
	Haemophilus influenzae type B	All jurisdictions
	Measles	All jurisdictions
	Mumps	All jurisdictions
	Pertussis	All jurisdictions
	Poliomyelitis	All jurisdictions
	Rubella	All jurisdictions
	Tetanus	All jurisdictions

Table 4. (continued) Diseases notified to the National Notifiable Diseases Surveillance System in 1999

Disease Group	Disease	Comments
Vectorborne diseases	Arbovirus infection (NEC)	All jurisdictions
	Barmah Forest virus	All jurisdictions
	Dengue	All jurisdictions
	Malaria	All jurisdictions
	Ross River virus	All jurisdictions
Zoonoses	Brucellosis	All jurisdictions
	Hydatid disease	All jurisdictions except NSW
	Leptospirosis	All jurisdictions
	Ornithosis	All except NSW and Qld
	Q fever	All jurisdictions
Other bacterial infections	Legionellosis	All jurisdictions
	Leprosy	All jurisdictions
	Meningococcal infection	All jurisdictions
	Tuberculosis	All jurisdictions

1999: The year in review

In 1999, control of communicable diseases in Australia enjoyed some notable successes, weathered some major challenges and prepared for some major threats.

In Australia in 1999 measles and mumps were reported at record low rates in children and rubella was reported at a record low rate in women of childbearing age. This was in part the result of the Measles Control Campaign in late 1998, in which 1.7m children were immunised with a second dose of the measles-mumps-rubella vaccine. It was estimated that immunity to measles increased to 94 per cent among Australian children as a consequence of improved vaccination coverage.[1]

A major challenge in 1999 was the influx into Australia of refugees from Kosovo and East Timor under the 'Safe Havens' initiative. In May and June 1999, 3,920 ethnic Albanians from Kosovo arrived in Australia. After initial processing in Sydney, refugees were accommodated in 8 centres in 5 States. There were significant presentations to medical authorities of refugees with upper respiratory tract infections, gastrointestinal illness and ear problems.[2] In September 1999, 1,863 people were evacuated from East Timor to Darwin. All evacuees had a mandatory health screen on arrival, 100 were admitted to hospital, 324 were reviewed in a 'fever/chest' clinic, 1,218 were reviewed in a transit camp and there were 7 births. Communicable diseases detected included 14 cases of malaria, 61 cases of tuberculosis (TB), 17 laboratory-confirmed cases of infectious diarrhoea and 3 laboratory-confirmed cases of measles and 14 suspected cases.[3] Up to this time there had been no health surveillance guidelines in Australia for such a rapid response setting. Protocols for future health screening of refugees arriving in emergency situations have since been developed and published.[4]

The future of the treatment of microbial infections is increasingly uncertain given the rise of antibiotic resistant bacteria in 1999. Beta-lactamase producing vancomycin resistant *Enterococcus faecilis* was for the first time reported in Australia.[5] A national response to the threat of antimicrobial resistance was the establishment of a Joint Technical Advisory Committee on Antibiotic Resistance (JETACAR). The report of this committee was released in September 1999. Details of the JETACAR report are included in this report. In response to the report, unprecedented co-operation between human and animal health practitioners has started to develop new ways to control and combat the development of antibiotic resistant microbes in Australia.

In common with other countries, Australia faces a threat of bloodborne viruses, particularly hepatitis C. Two major documents were produced in 1999, one describing the epidemiology of hepatitis C in Australia[6] and another on an Australian plan to control hepatitis C.[7] These 2 documents along with the Hepatitis C surveillance strategy are guiding Australia's response to the hepatitis C epidemic.

In 1999 there were important moves toward uniform baseline and enhanced disease-specific surveillance. The Communicable Diseases Network Australia New Zealand (now Communicable Diseases Network Australia), agreed in 1999 to revise the list of diseases which are designated as notifiable in Australia and to collect a more comprehensive set of data on each case. In addition, 'enhanced' surveillance systems for tuberculosis measles and hepatitis C were discussed and designed. These 'enhanced' systems aim to collect important additional information at a national level, that is critical for the surveillance and control of these diseases.

Internationally, there was considerable concern over bovine spongiform encephalopathy (BSE) transmission to humans causing variant Creutzfeldt-Jakob disease (vCJD). There is

now convincing evidence that the human disease has been caused by the consumption of foods contaminated with the BSE prion. It is still too early to predict the total number of cases of vCJD that may appear in the United Kingdom (UK) in the next two decades. In January 1999, the offspring of BSE affected cattle born after June 1996 were slaughtered in the UK, to avoid the possibility of transmission of the disease into the food chain. Deferral of blood donations from Australian citizens who were resident in the UK between 1980 and 1996 was instituted in 2000 to protect recipients from the theoretical risk that vCJD can be transmitted by blood transfusion. No cases of BSE have been found in Australian cattle herds nor have there been any cases of vCJD. The classical form of CJD does occur in Australia at a rate of 1.5 cases per million annually, consistent with international rates.[8]

In 1999, an outbreak in Malaysia and Singapore of a viral encephalitis among workers with exposure to pigs was reported.[9] Investigations led to the identification of a previously unrecognised virus, similar to the Hendra virus which caused deaths in horses and one human handler in Queensland in 1994. The virus has since been named the Nipah virus after the area in which most infections occurred. In 1999, 265 people were infected, of whom 105 died. Consequently, 1.1 million pigs were destroyed.[10] A recently published serosurvey of piggeries in Queensland confirmed that the herds were free of infection with either the Hendra or Nipah virus.[11]

In late August 1999, an unusual clustering of cases of meningoencephalitis was reported in New York City. The cause of the outbreak was confirmed as the West Nile virus.[12] This was the first time this virus had been detected in the Western Hemisphere. Associated with the human cases were an unusually large number of deaths among birds, particularly crows. Necropsies on these birds revealed West Nile virus infection. This outbreak appears to be associated with the appearance of a new variant West Nile virus, which first appeared in Romania in 1996 and Israel in 1998.[13] The disease has since spread along the eastern seaboard of the United States (US), and is carried by at least 14 species of mosquito. Mosquito larval control measures have limited subsequent human disease to small geographic foci. West Nile viruses are flaviviruses and are part of the 'Japanese encephalitis complex' of viruses, which include Kunjin and Murray Valley encephalitis viruses. Kunjin has been described as a subtype of lineage 1 West Nile virus. Recent studies on the relationship between Kunjin virus and West Nile virus,[14] demonstrate that Kunjin virus is one of several subgroups of West Nile virus and that Australian Kunjin virus shows genetic and antigenic differences to both the West Nile virus isolated in New York in 1999 and the Kunjin virus from Malaysia. Kunjin virus is not associated with the same morbidity and mortality caused by infection with West Nile virus.

In summary, communicable disease surveillance and control in Australia was advanced in 1999 by important new initiatives and strategic control measures. The sudden influx of refugees stretched the resources of the public health system; nonetheless refugees received adequate quality medical care. Communicable diseases were diagnosed and treated and there was no spread of disease to the broader Australian community. Important new diseases have been recognised while research and surveillance suggests they will have a limited impact in Australia.

Introduction

Surveillance of communicable diseases is an essential public health activity. Surveillance allows the detection of disease outbreaks prompting the appropriate investigation and control measures to be instigated. Surveillance also allows for the monitoring of trends in disease prevalence and considers the impact and effectiveness of interventions to control the spread of diseases. Surveillance systems exist at national, state and local levels. State and local surveillance systems are crucial to the timely and effective detection and management of outbreaks and in assisting in the effective implementation of national policies. The national surveillance system combines some of the data collected from State and Territory-based systems to provide an overview at a national level. Specific functions of the national surveillance system include: detection and management of outbreaks affecting more than one jurisdiction; monitoring the need for and impact of national control programs; guidance of national policy development; 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 agreed international collaborations such as reporting to the World Health Organization.

The National Notifiable Diseases Surveillance System (NNDSS) was established in its current form in 1991, under the auspices of the Communicable Diseases Network Australia (CDNA, formally the Communicable Diseases Network Australia New Zealand, CDNANZ). The CDNA monitors trends of an agreed list of communicable diseases in Australia. Data are regularly published in the *Communicable Diseases Intelligence (CDI)* and on the Internet site Communicable Diseases Australia. This is achieved through the national collation of notifications of these diseases received by health authorities in the States and Territories. In 1999, 49 diseases or disease categories were included (Table 4), largely as recommended by the National Health and Medical Research Council (NHMRC).[15] At present the list of notifiable diseases and categories is undergoing review and revision. Information collected on notifiable diseases has been published in the Annual Report of the NNDSS since 1991.[16,17,18,19,20,21,22]

Methods

Australia is a federation of 6 States (New South Wales, Queensland, South Australia, Tasmania, Victoria and Western Australia) and 2 Territories (the Australian Capital Territory and the Northern Territory). The States and Territories collect notifications of communicable diseases under their public health legislation. The Commonwealth (or Federal) Government does not have any legislated responsibility for public health apart from human quarantine. States and Territories have agreed to forward data on communicable diseases to the Commonwealth Department of Health and Aged Care (DHAC) for the purposes of national communicable disease surveillance.

In 1999, the States and Territories transmitted data to the Commonwealth, fortnightly. Summaries of the data were

published fortnightly on the *Communicable Diseases Australia* Website and in the *Communicable Diseases Intelligence (CDI)* every 4 weeks. The Commonwealth received final data sets from the States and Territories of cases reported in 1999, by August 2000. Where possible, missing data and apparent errors were corrected, in consultation with the States and Territories. For the purposes of the NNDSS, where a patient being treated in one jurisdiction was diagnosed in another, notifications were from the State or Territory where the case was diagnosed.

Case definitions for each disease can be found in Appendices 1a-1h. For each case, the national data set includes fields for a unique record reference number; a code for the disease; age, sex, indigenous status; postcode of residence; the date of onset of the disease and date of report to the State or Territory health authority; and the confirmation status of the report. Analysis of the data by indigenous status was not possible because of the incomplete reporting of this information. Additional information was available on the species and serogroups isolated in cases of legionellosis, brucellosis, meningococcal disease, malaria and enterotoxigenic (verotoxigenic) *Escherichia coli*.

Analyses in this report are based on date of disease onset. For analysis of seasonal trends, notifications were reported by month of onset. Population notification rates were calculated using 1999 mid-year estimates of the resident population supplied by the Australian Bureau of Statistics. An adjusted rate was calculated where a disease was not notifiable in a State or Territory using a denominator which excluded that population. The data were analysed in Excel.

Maps were generated using MapInfo based on the postcode of residence and allocated to Australian Bureau of Statistics Statistical Divisions (Map 1). The 2 Statistical Divisions that make up the Australian Capital Territory were combined, as the population for one Division is very small. Notifications for Darwin and the remainder of the Northern Territory were also combined to calculate rates for the Northern Territory as a whole. In general, notification rates for Statistical Divisions were depicted in maps or discussed in the text only where the number of notifications was sufficiently large for these to be meaningful.

Notes on interpretation

The notifications compiled by the NNDSS may be influenced by a number of factors that should be considered when interpreting the data. Due to under-reporting, notified cases are likely to only represent a proportion of the total number of cases that occurred. This proportion may vary between diseases, between States and Territories and with time (Appendix 2). Methods of surveillance vary between jurisdictions, 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 jurisdictions.

Postcode information usually reflects the postcode of residence. However, the postcode of residence may not necessarily represent the place of acquisition of the disease, or the area in which public health actions were taken in response to the notification.

As no personal identifiers are collected in records, duplication in reporting may occur if patients moved from one jurisdiction to another and were notified in both. Data from those Statistical Divisions with small populations (Map 1) may result in high notification rates even with small numbers of cases. Notifications of diseases with longer incubation periods are more likely to be affected in this way than short incubation diseases.

The completeness of data in this report is summarised in Appendix 5. Missing data were patients' sex in 0.9 per cent notifications (n = 780) and patients' age in 0.3 per cent notifications (n = 256). The proportion of reports with missing data in these fields varied by State or Territory, and also by disease.

This is the first annual report where data are analysed by date of disease onset. 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. Analysis by date of onset should be interpreted with caution, particularly for chronic diseases such as hepatitis B and C. NNDSS data from previous years (1994–1998, Table 3) show totals and rates for those years as analysed in August 2000. States and Territories continue to revise totals from previous years as duplicates are removed and other data are corrected. For this reason the totals and rates shown in Table 3 differ from totals and rates published in the annual reports from these years. All comparisons in this report are to the most recent totals, which are more accurate than those previously published.

Rates per 100,000 population were calculated using State, Territory and national population estimates for mid-year 1999, suppled by the Australian Bureau of Statistics (ABS). Mortality statistics for 1999 were available from ABS in 2001. The Australian Institute of Health and Welfare (AIHW) supplied hospital admission data for the financial year 1998/1999.

Data were analysed every 4 weeks and a short report published in *CDI*. This report is based on 'finalised' annual data from each jurisdiction, from which duplicate or erroneous records have been removed. For this reason, totals in this report may vary from the cumulative totals of the numbers reported in the four-weekly *CDI* reports. This report is informed by the discussions and comments of members of the CDNA, who met fortnightly by teleconference to discuss developments in communicable disease in their jurisdiction. The contribution of State and Territory data managers, to ensure that the data in this report are accurate, is gratefully acknowledged.

Results — surveillance notifications and reports

There was a total of 88,239 communicable disease notifications in 1999 (Table 1). Notification rates per 100,000 population for each disease by State or Territory are described in Table 2. Comparative data for 1998 and the preceding 4 years are shown in Table 3.

In 1999, cases of haemolytic uraemic syndrome became notifiable in all States and Territories and shiga-toxin producing *E. Coli* (SLTEC, also called verotoxigenic *E. Coli* (VTEC)) infections were reported in all jurisdictions except Queensland and Western Australia.

The number of notifications in 1999 was an increase of 3 per cent on notifications in 1998 (85,227) and the second largest

number of reports since the NNDSS commenced in 1991 (Figure 1). In 1999 there were 29,977 bloodborne infections (34% of total), 22,255 gastrointestinal infections (25%), 21,704 sexually transmitted disease (25%), 5,986 vector-borne diseases (7%), 5,228 vaccine preventable diseases (6%), 1,967 other bacterial infections (2%), 1,012 zoonotic infections (1%) and 3 quarantinable diseases (<1%), (Figure 2).

The major changes in notifications in 1999 are shown in Figure 3 as a ratio of 1999 notifications compared with a 5-year mean. Only diseases with major changes in numbers of notifications in 1999 are shown. There was more than 50 per cent increase in notifications of hepatitis C (incident notifications) and leptospirosis. Smaller increases were noted in the reporting of chlamydial, gonococcal and meningococcal infections, legionellosis, mumps and syphilis. Measles notifications fell by more than 50 per cent compared with 1998. Declines in *Haemophilus influenzae* type b (Hib) infections and mumps were also noted.

In 1999, infectious and parasitic diseases (ICD-10 codes A00-B99) accounted for 1.25 per cent of all deaths in Australia (1,603 deaths). Pneumonia and influenza (ICD-10 codes J10-J18) accounted for a further 1.5 per cent of deaths (1,898 deaths). Death rates increased with age and were greater for males than females aged 45 years and over (Causes of death Australia 1999, Ausstats 3303.0 ABS, 2000).

Bloodborne viruses

The bloodborne viruses notified to NNDSS include hepatitis B, C and D. New HIV diagnoses are notified directly to the National Centre in HIV Epidemiology and Clinical Research (NCHECR), which reports separately in its Annual Surveillance Report. Information on the HIV data collection can be obtained through the NCHECR Website at: www.med.unsw.edu.au/nchecr.

Incident hepatitis C virus (HCV) infections are diagnosed by seroconversion (positive for anti-hepatitis C antibodies, with a negative test in the previous year), or by a clinical illness compatible with acute viral hepatitis where other causes have been excluded. Incident hepatitis B virus (HBV) infections are diagnosed by serology (presence of anti-HBc IgM antibodies) or by a clinical illness compatible with acute viral hepatitis where other causes have been excluded. Some jurisdictions may include cases with a previous negative HBsAg test in the last 12 months. Notifications of hepatitis B and hepatitis C that do not meet the incident case definition are recorded as 'unspecified'. Collectively, cases of hepatitis B and C represented 34 per cent of all notifications to the NNDSS in 1999, similar to the proportion in 1998.

Hepatitis B

Incident cases of hepatitis B have been notified to the NNDSS by all jurisdictions since 1994. In 1999, 307 incident cases were reported to the NNDSS at a national notification rate of 1.6 per 100,000 population, consistent with the rate reported in 1998 (1.4 per 100,000 population). The highest rates were recorded in the Northern Territory (9.9 per 100,000 population), Western Australia (2.4 per 100,000 population) and Victoria (2.0 per 100,000 population).

The majority of incident hepatitis B notifications were in the 15–34 year age range (Figure 4). Infections in males exceeded those in females (male to female ratio of 1.8:1). Risk factor information on incident hepatitis B cases were only available from Victoria, where 77 per cent of the cases occurred in injecting drug users (IDU) and their sexual partners. Three incident cases in Victoria were household contacts of a patient with chronic hepatitis B.

Figure 1. Notification rate (per 100,000 population) to NNDSS 1991 to 1999)

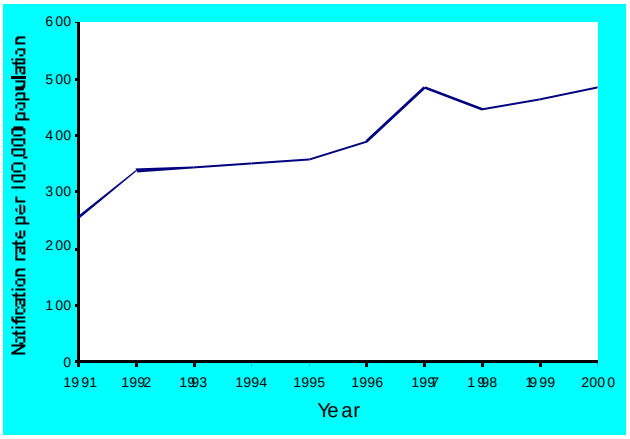


Figure 2. Breakdown of communicable disease notifications by disease category

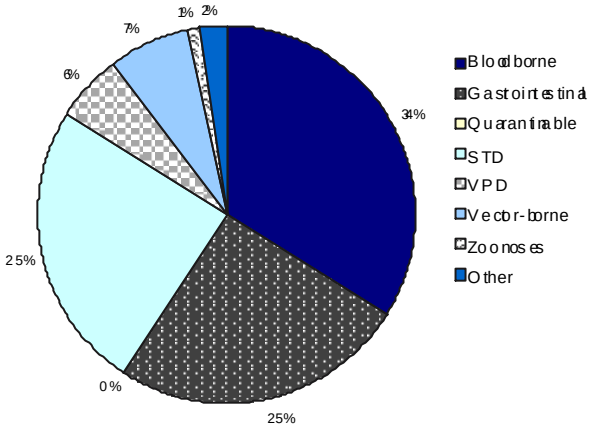
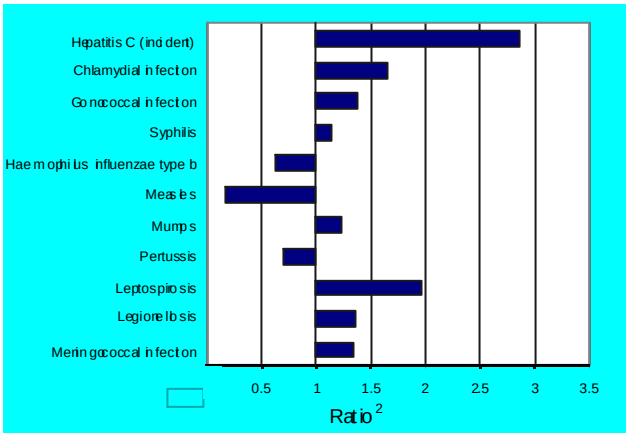


Figure 3. Comparison of selected disease totals in 1999 with historical data (5 year mean)



Unspecified hepatitis B has been notified to the NNDSS by all jurisdictions except the Northern Territory since 1997. In 1999, 8,091 unspecified cases were notified at a rate of 42.7 per 100,000 population (Tables 1 and 2), a rate higher than that recorded in 1998 (35.6 per 100,000 population). The male to female ratio for unspecified cases was 1.2:1. The highest rates of notification were in New South Wales (67.5 per 100,000 population), Victoria (46.1 per 100,000 population) and the Australian Capital Territory (21.4 per 100,000 population). The highest rates were in the 35–39 year age group for men (87.3 per 100,000 population) and the 25-29 year age group for women (77.3 per 100,000 population, Figure 5).

Vaccination against HBV commenced nationally for 'at-risk' infants in Australia in 1987 (except in the Northern Territory which started in 1988 and South Australia which started in 1996) and in adolescents in 1998 (except New South Wales and South Australia which started in 1999). National universal infant vaccination against HBV started in May 2000. In the Northern Territory, vaccination for Aboriginal infants started in 1988 and universal vaccination in 1990).[24] Rates of HBV infection in Australian children are low and it will be some years before the impact of HBV vaccination is seen in older age groups. In the Northern Territory, where infant immunisation with HBV vaccine started 10 years before the rest of the country, there were no child cases of hepatitis B reported in 1999.

Figure 4. Notification rate for incident HBV, Australia, 1999, by age and sex

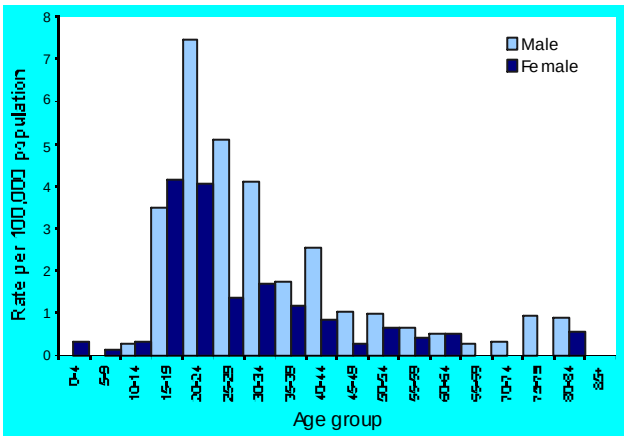
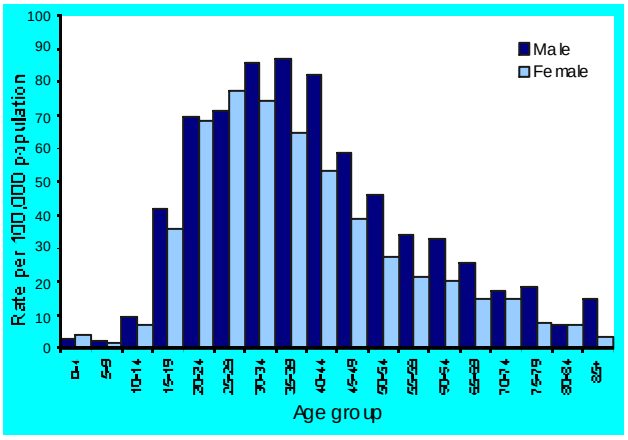


Figure 5. Notification rate for unspecified HBV, Australia, 1999, by age and sex

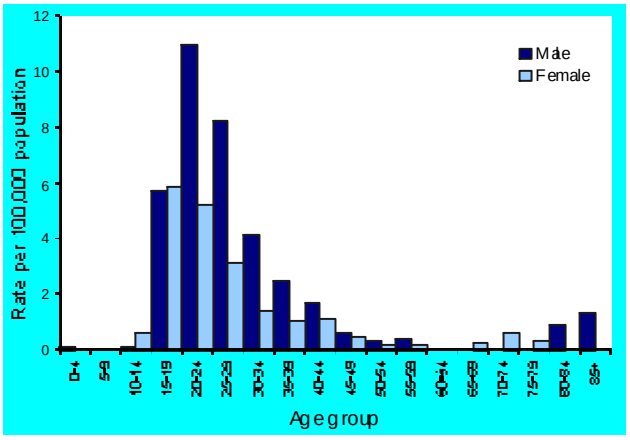


Hepatitis C

It has been estimated that more than 170 million people in the world have been infected with Hepatitis C, five times the number of people infected with HIV worldwide.[25] The virus was only identified in 1988, and was one of the first viruses to be identified solely by molecular biology. Serological screening tests became commercially available only in 1990 and while the first generation of assays had problems with both sensitivity and specificity, these have improved in recent years. Nucleic acid-based diagnostic methods are now commercially available. Hepatitis C infection has been notifiable in most Australian jurisdictions since 1991, and the number of 'unspecified' hepatitis C notifications remains stable at around 20,000 notifications per year. Incident cases of hepatitis C have been separately notifiable since 1993. Most cases of hepatitis C are asymptomatic during the acute phase, and incident cases are rarely identified. Most cases are diagnosed when the patient presents with symptoms of chronic disease, or by screening. As the timing of infection is often unknown, most cases are notified as 'unspecified'. The number of notifications to the NNDSS of incident hepatitis C has increased over recent years, although it is recognised that the number of notifications vastly underestimates 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 these notifications are largely a product of improved surveillance, increased awareness, and more widespread testing which vary across the jurisdictions.

In 1999, all States and Territories reported unspecified cases of hepatitis C. Incident cases were reported from all jurisdictions except Queensland. The total number of hepatitis C notifications (incident and unspecified) was similar in 1998 and 1999. There were 385 incident cases of hepatitis C reported in 1999, at a rate of 2.5 per 100,000 population. The proportion of all notifications that were known incident cases was 1.8 per cent in 1999, similar to the proportion in 1998 (1.7%). The highest rates of incident hepatitis C infection were reported from the Australian Capital Territory (6.4 per 100,000 population), Western Australia (6.1 per 100,000 population) and South Australia (5.8 per 100,000 population). The majority of incident hepatitis C notifications were in the 15–29 year age range (Figure 6).

Figure 6. Notification rate for incident hepatitis C, Australia, 1999, by age and sex



In 1999 only limited data were collected nationally on incident hepatitis C infections. The Australian Capital Territory, South Australia, Tasmania, Victoria and Western Australia collected additional data on risk factors for infection and the reason for testing or reporting source. The following analyses refer only to incident hepatitis C cases identified in these jurisdictions.

Demographic profile of incident hepatitis C cases

The age and sex of incident hepatitis C cases notified in 1999 are summarised in Table 5, according to the State or Territory of diagnosis. The age of incident cases ranged from 13 to 85 years. The majority of cases were, however, between 20 and 40 years of age. Overall, the male to female ratio of cases was 1.8:1, although the proportion of male cases did vary across jurisdictions, with South Australia recording the highest proportion of male cases.

Method of diagnosis of incident hepatitis C

Diagnosis was based either on seroconversion or on the clinical diagnosis of acute hepatitis. In some patients, seroconversion and acute hepatitis were recorded. Data were not available for Victoria, however, data for the other 4 jurisdictions are shown in Table 6. In some jurisdictions the decision of whether a cases in incident or unspecified is made by the clinician. In some of these cases information regarding the method of diagnosis may not be made available to the health department (recorded as unknown in Table 6).

Reporting sources for incident hepatitis C infections

In South Australia the reporting source is recorded, while in the other jurisdictions (the Australian Capital Territory, Tasmania, Victoria and Western Australia), the reason for testing is documented. Recording of multiple responses was possible in Tasmania, while in the remaining jurisdictions recording of one reporting source/reason for test was possible.

A general practitioner was the reporting source for 14 (70%) of the 20 notifications from the Australian Capital Territory, while the remaining 6 cases were identified by screening at a detoxification unit. Prisons were a major reporting source in South Australia, while an investigation of symptomatic hepatitis was the major reason for testing in Tasmania. The reason for testing in Western Australia was often not reported, or reported as 'other'. In Victoria the most commonly identified reason for testing was having another medical problem. The variation across jurisdictions in the recording of the reason for testing or reporting source limits the utility of this information.

Exposure assessment for incident hepatitis C infections

The Australian Capital Territory, Tasmania, Western Australia and Victoria provided exposure assessment data for incident hepatitis C infections. In Tasmania, Western Australia, and Victoria more than one exposure factor was recorded.

Injecting drug use is the major exposure factor for incident infections in all jurisdictions (range 65% in the Australian Capital Territory to 92% in Victoria). Multiple exposures were often recorded, but there were few cases that were not IDU associated. Other risk factors included surgery, tattooing and sexual contact with a hepatitis C-infected person. A proportion of cases had no exposure factor identified, ranging from 1 per cent in Victoria to 24 per cent in Western Australia.

Unspecified hepatitis C accounted for 21,244 notifications; a notification rate of 112 per 100,000 population, similar to the 102.7 per 100,000 population reported in 1998. 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 New South Wales (143.7 per 100,000 population), Victoria (130.8 per 100,000 population) and the Northern Territory (120.3 per 100,000 population). The male to female ratio was 1.8:1. The highest notification rates were in the 20 to 49 year age range for both males (318 per 100,000 population) and females (177 per 100,000 population, Figure 7).

Hepatitis D

Hepatitis D is a co-infection occurring in HBV-infected people and particularly prevalent among injecting drug

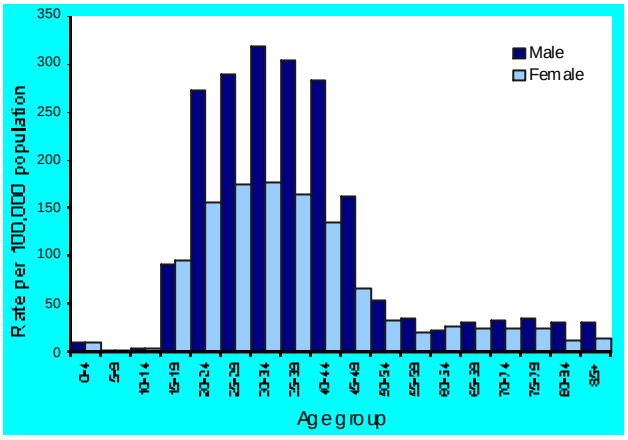
Table 5. Demographics of incident hepatitis C cases in the Australian Capital Territory, South Australia, Tasmania, Victoria and Western Australia, 1999

	ACT	SA	Tasmania	Victoria	WA
% Males	45	70	56	66	63
Median age (range)					
Males	21 (18-30)	25 (17-44)	23 (17-42)	21 (14-40)	27 (18-85)
Females	29 (19-37)	24 (17-74)	31 (17-42)	20 (13-59)	24 (15-50)

Table 6. Method of diagnosis, incident hepatitis C cases in the Australian Capital Territory, South Australia, Tasmania, and Western Australia, 1999

Method of diagnosis	ACT	SA	Tas	WA
Seroconversion %	75	95	49	58
Clinically defined acute hepatitis %	25	5	-	9
Illness and seroconversion %	-	-	-	2
Unknown %	-	-	51	31

Figure 7. Notification rate for unspecified hepatitis C, Australia, 1999, by age and sex



users.[26] There were 21 notifications of hepatitis D to the NNDSS in 1999 at a notification rate of 0.1 per 100,000 population. Of the 21 notifications, 16 (76%) of the notifications came from New South Wales (0.2 per 100,000 population). The majority (85%) of notifications was in males aged between 15 and 49 years.

Gastrointestinal diseases

Introduction

Gastrointestinal and foodborne diseases are a major cause of illness in Australia, despite the often mild nature of symptoms. The exact burden of disease due to food is not easy to quantify, as there is a significant under-estimate in surveillance data and there are multiple modes of transmission for gastrointestinal disease. Surveillance data may be biased by different levels of reporting of gastrointestinal disease in different age groups, with children and the elderly more likely to be seen by a medical practitioner.

It is important to recognise that differences in laboratory testing practices and surveillance methods in States and Territories may account for the difference in observed notification rates. This is particularly true for diseases such as *Shiga*-toxin producing *E. coli* (SLTEC/VTEC), where laboratory diagnosis is difficult, and screening practices vary between laboratories and jurisdictions. States and Territories also have different reporting requirements for doctors and laboratories, which can make national comparison difficult. To overcome some of these difficulties, the CDNA agreed to standardise reportable conditions in each jurisdiction from 1 January 2001.

In January 1999, the NSW Health Department established sentinel surveillance for foodborne disease in the Hunter Public Health Unit. This program of work was modelled on the Centers for Disease Control and Prevention (CDC) FoodNet Active Surveillance Network. The Hunter Public Health Unit heightened surveillance within the region for diarrhoeal disease and syndromic illnesses associated with food. It also established case control studies for *Salmonella* and *Campylobacter*. This pilot program of enhanced surveillance has run for over 2 years, and was used as a model for an Australia-wide program of heightened surveillance — coined OzFoodNet, in 2000.

The major outbreaks of foodborne illness in 1999 were the nationwide outbreak of typhoid following a cruise in the Pacific, and an outbreak of *Salmonella* Typhimurium PT135a associated with orange juice in South Australia. The outbreak of typhoid required multi-state cooperation, as cases were reported to various State and Territory health departments. The CDNA collaborative investigation was lead by the Victorian Department of Human Services.

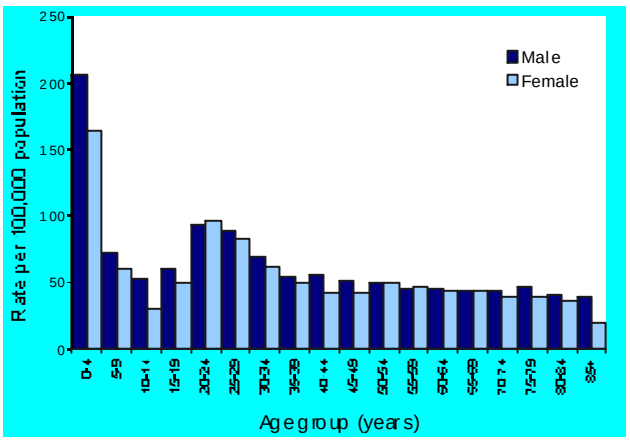
Botulism

There have been no cases of foodborne botulism reported to the NNDSS since the inception of the system in 1991. A single case of infant botulism was reported in 1998. There were no cases of botulism reported in 1999.

Campylobacteriosis

There were 12,643 cases of campylobacteriosis notified to the NNDSS with symptom onset in 1999 (Table 1), which was a decrease of 6 per cent from 13,282 cases notified in 1998. *Campylobacter* species are the most common cause of gastrointestinal disease notified to the NNDSS. Despite this there are very few outbreaks detected due to the lack of a robust typing method. The median age of cases in 1999 was 26 years (range 0-94 years) and 53.4 per cent of notified cases were male. The highest age-specific rate was 188.2 cases per 100,000 population in 0–4 year-old children (Figure 8). The highest notification rates were in South Australia (161.1 per 100,000 population) and the lowest rates were in Western Australia (76.0 per 100,000 population, Table 2). Analysis by Statistical Division showed the highest rates of *Campylobacter* occurred in Outer Adelaide (186 per 100,000 population), the Western District of Victoria (163 per 100,000 population) and Yorke and Lower North in South Australia (163 per 100,000 population) (Map 2). Reports of campylobacteriosis were greatest in

Figure 8. Notification rate for campylobacteriosis, Australia, 1999, by age and sex



Spring and Summer (Figure 9). *Campylobacter* infections were not specifically notifiable in New South Wales.

Hepatitis A

There were 1,557 cases of hepatitis A notified to NNDSS with symptom onset in 1999 (Table 1), which was a decrease of 38 per cent from 2,443 cases notified in 1998 (Table 3). Although a faecal-oral route through

Figure 9. Notifications of campylobacteriosis, Australia, 1991 to 1999, by month of onset

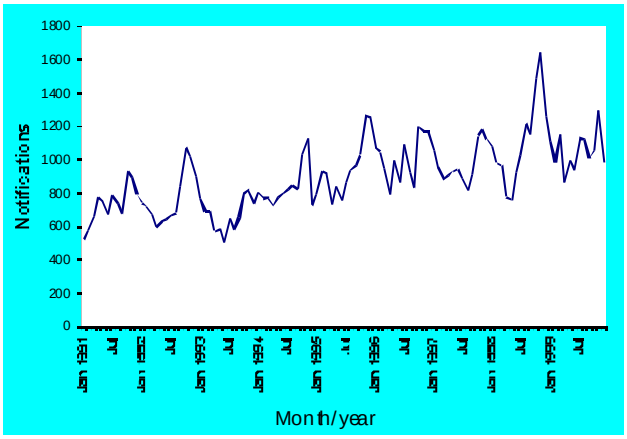
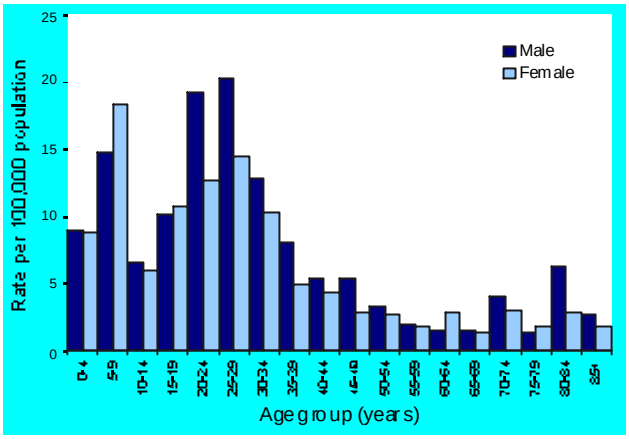
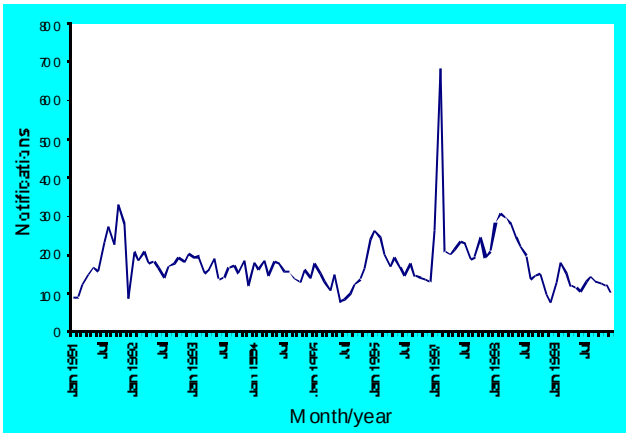


Figure 10. Notification rate for hepatitis A, Australia, 1999, by age and sex

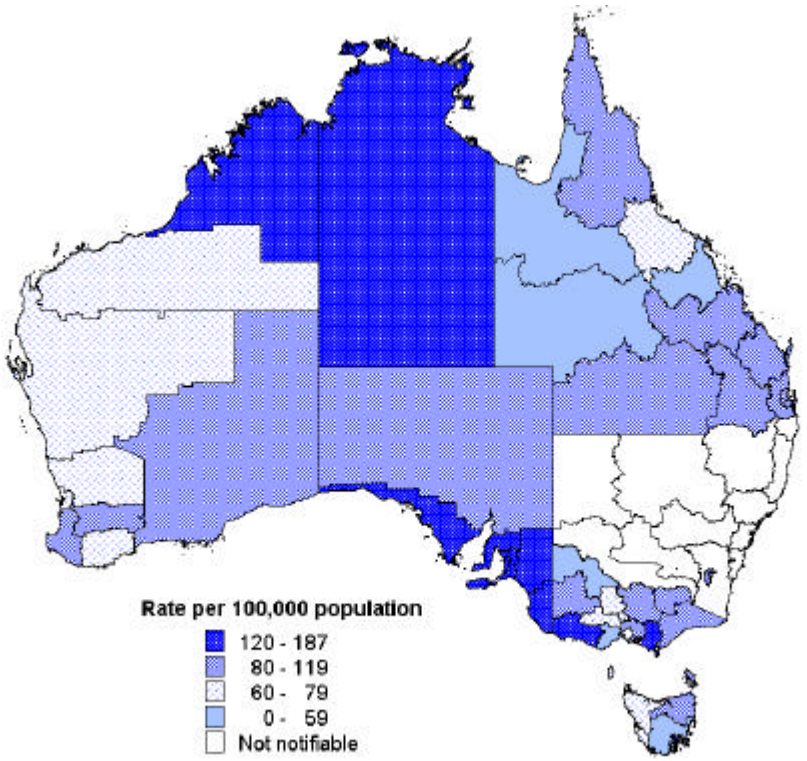


contaminated food or shellfish from contaminated waters commonly spreads hepatitis A, recent outbreaks in the USA and Europe have been associated with injecting drug users. Similarly, in Australia in 1999, there were reports of hepatitis A outbreaks among injecting drug users.[27,28] The median age of cases reported to NNDSS in 1999 was 24 years (0–98 years) and 54.7 per cent of notified cases were male. The highest age-specific rates were in 5–9 year-old children (16.5 cases per 100,000 population), and among adults aged between 20–29 years (16.9 cases per 100,000 population, Figure 10). The highest notification rates were in the Northern Territory (46.1 per 100,000 population) and the lowest rates were in Tasmania (1.1 per 100,000 population, Map 3). Reports of hepatitis A were received throughout the year, but were greatest in the month February (Figure 11).

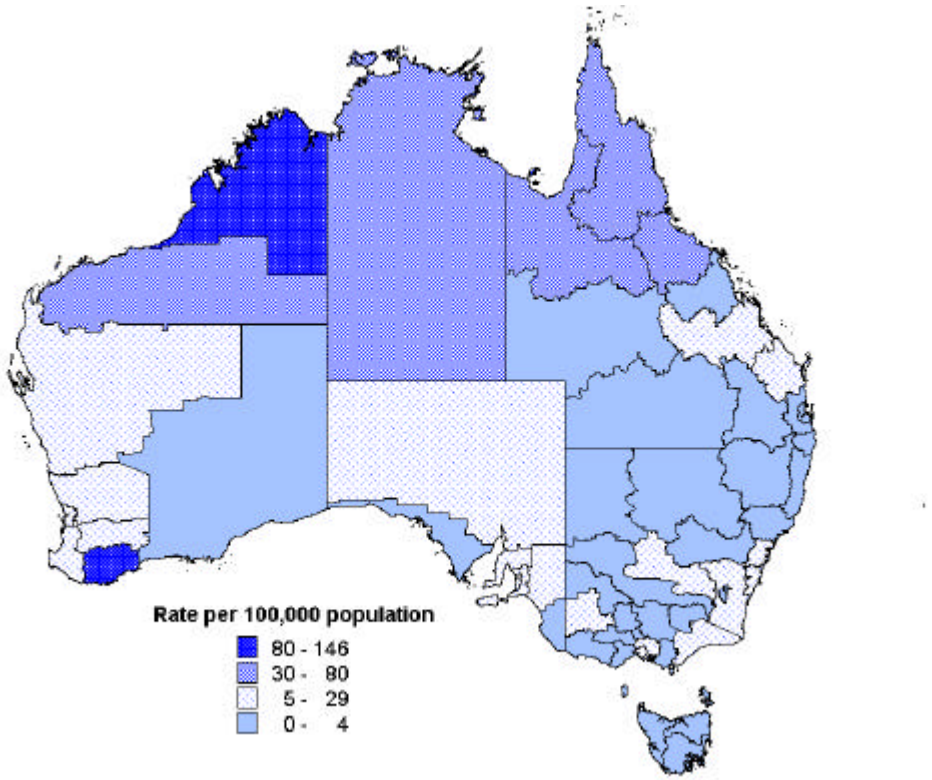
Figure 11. Hepatitis A notifications, Australia, 1991 to 1999, by month of onset



Map 2. Campylobacteriosis notification rate by Statistical Division of residence



Map 3. Hepatitis A notification rate by Statistical Division of residence



Hepatitis E

Hepatitis E virus is now recognized taxonomically as the type species of the genus 'Hepatitis E-like viruses'. Hepatitis E virus (HEV) exhibits similarity in structure and genome organisation with the caliciviruses, and low amino acid similarity with rubella virus and the alphaviruses of the family Togaviridae. It is unrelated to the other hepatitis viruses (A, B, C, D, and G). HEV is associated with sporadic cases of enterically transmitted acute hepatitis. HEV is considered to be endemic in tropical and subtropical regions of Asia, Africa, and Central America. Antibody prevalence suggests global distribution of strains of low pathogenicity. Antibodies to HEV or closely related viruses have been detected in primates and swine. Women in the third trimester of pregnancy are susceptible to fulminant hepatitis E disease that has a case fatality rate as high as 20 per cent.[29] Outbreaks in South Asia among young adults in recent years pose a risk to Australian travellers to these regions. There were 2 cases of hepatitis E notified to NNDSS in 1999, one of which was a 19-year-old male from Victoria and the other a 38-year-old male from the Australian Capital Territory. Both cases had a history of overseas travel and in one case it appeared the infection was acquired in India.

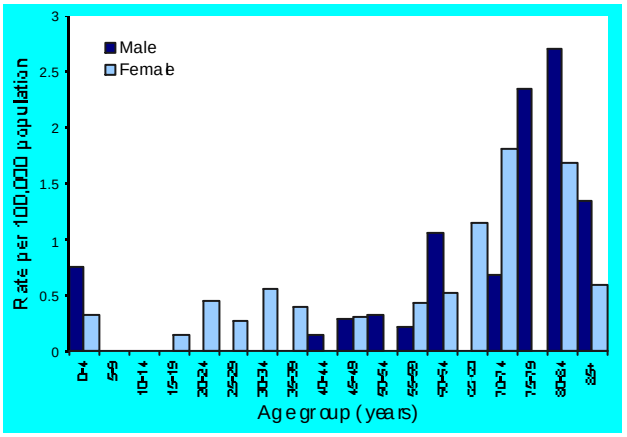
Listeriosis

Listeriosis is a serious but relatively rare foodborne disease to which neonates, pregnant women, the immunocompromised and the elderly are particularly susceptible. Infection during pregnancy can be transmitted to the foetus. Infants may be stillborn, born with septicaemia or develop meningitis in the neonatal period. Clusters of cases of listeriosis have been noted in hospitals, nurseries and aged care facilities.[29]

The interpretation of State and Territory comparisons of listeriosis data is complicated by reporting practices. Some jurisdictions report both cases of maternal-foetal pairs while others report the pair as a single case.

There were 63 cases of listeriosis reported to NNDSS with onset of symptoms in 1999, which was similar to previous years (Table 1). The median age of cases in 1999 was 60 years (0–86 years) and 55.6 per cent of notified cases were female. The highest age-specific rate was 2.1 cases per 100,000 population in 80–84 year-old people (Figure 12). The highest notification rates were in Western Australia (0.6 cases per 100,000 population) and there were no cases reported from the Northern Territory or the Australian Capital Territory. There was no evidence of clustering of cases of listeriosis. Victoria reported that 5 cases occurred as maternal-foetal cases, from which there were only 2 live births.

Figure 12. Notification rate for listeriosis, Australia, 1999, by age and sex



Salmonellosis (excluding typhoid)

There were 7,154 cases of salmonellosis (not elsewhere classified) reported to NNDSS with symptom onset in 1999, which was a decrease of 9.5 per cent from 7,489 cases reported in 1998. The median age of cases in 1999 was 11 years (range 0–97) and 50.7 per cent of notified cases were male. The highest age-specific rate was 220.7 cases per 100,000 population in 0–4 year-old children (Figure 13). The highest notification rates were in the Northern Territory (184.6 per 100,000 population) and the lowest rates were reported from the Australian Capital Territory (20.7 per 100,000 population). The Kimberly Statistical Division had in excess of 300 cases per 100,000 population, which was comparable to previous years (Map 4). Reports of salmonellosis were greatest in the months January to March (Figure 14).

In Australia during 1999, the National Enteric Pathogen Surveillance Scheme (NEPSS) recorded 7,179 cases of non-typhoidal salmonellosis, and 15 outbreaks involving more than 10 cases, and 15 clusters of less than 10 cases (NEPSS 1999 annual report). The largest of these was an outbreak of 501 cases of *Salmonella* Typhimurium 135a associated with commercial orange juice. This outbreak occurred predominantly in South Australia, but cases were identified in neighbouring States.[30] A waterborne outbreak of *S.* Saintpaul was reported in March 1999 in 28 workers at a large construction site in Central Queensland.[31]

Salmonella Typhimurium was the most common serovar reported to NEPSS in 1999, with phage types 135, 135a and 9 being the most common. NEPSS recorded 201 cases of *Salmonella* Enteriditis phage type 4. In New South Wales, the Australian Capital Territory, Victoria and South Australia, there were a total of 88 cases of *S.* Enteriditis phage type 4.

Figure 13. Notification rate for salmonellosis, Australia, 1999, by age and sex

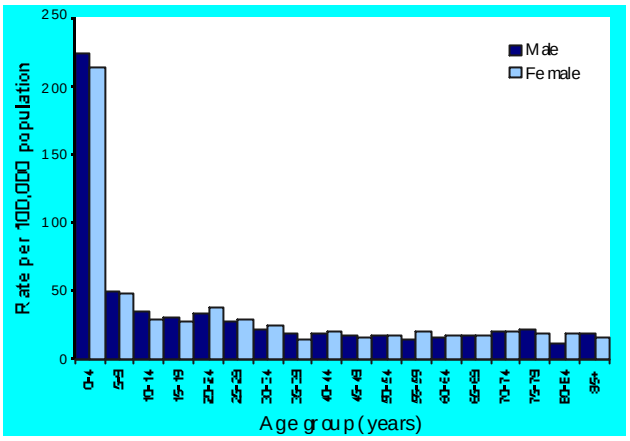
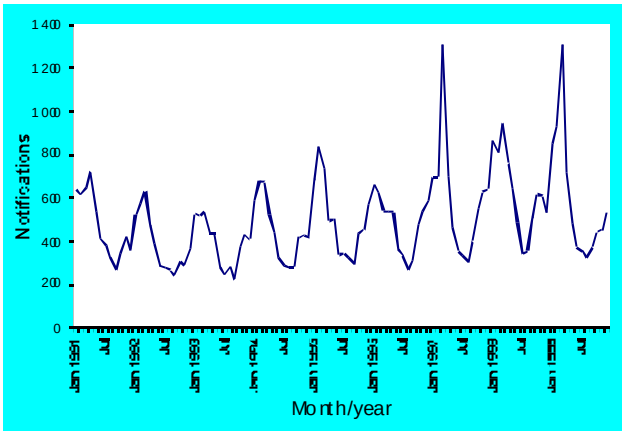
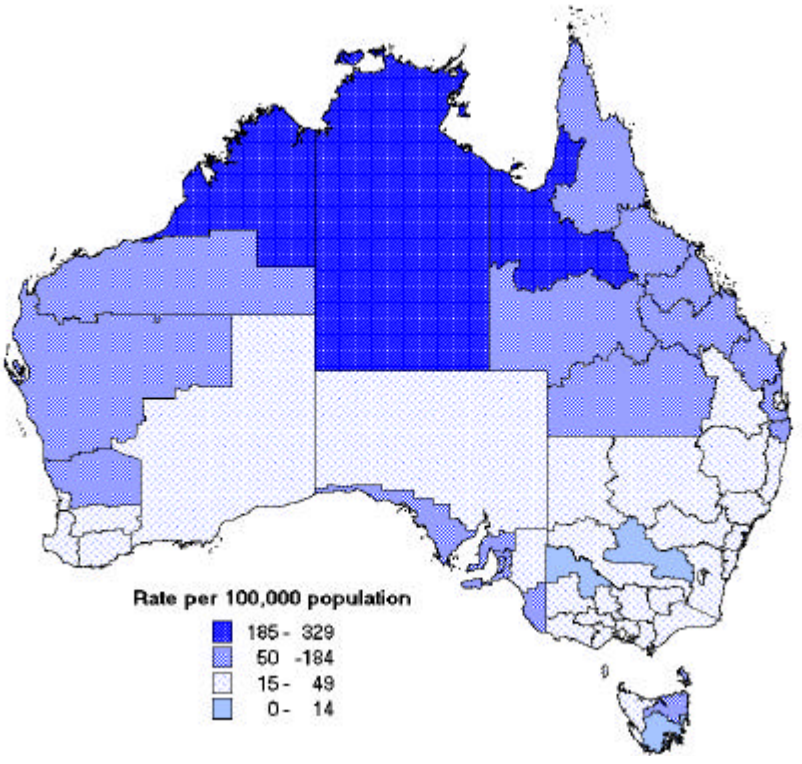


Figure 14. Notifications of salmonellosis, Australia, 1991 to 1999, by month of onset



Map 4. Salmonellosis notification rate by Statistical Division of residence



Of those cases where data on overseas travel was available, all had a history of recent overseas travel, and the majority had travelled to Indonesia.

Shigellosis

There were 547 cases of shigellosis reported to NNDSS with onset of symptoms in 1999, which was a 8 per cent decrease from 594 cases reported in 1998. The median age of cases in 1999 was 21 years (range 0–83 years) and 54.1 per cent of notified cases were female. The highest age-specific rate was 13.0 cases per 100,000 population in 0–4 year-old children (Figure 15). The highest notification rates were in the Northern Territory (57.5 per 100,000 population) and the lowest rates were reported from Tasmania (0.2 per 100,000 population). Shigellosis was not specifically notifiable from New South Wales. Cases were more commonly notified during the months of January to April (Figure 16).

A report of a *Shigella sonnei* outbreak in a long-term nursing centre was reported.[32] Thirteen cases of multi-drug resistant *S. sonnei* were found among staff and patients and the isolates were genetically indistinguishable. It is probable that transmission was person-to-person and that break-downs in the institutional infection control procedures were responsible.

Figure 15. Notification rate for shigellosis, Australia, 1999, by age and sex

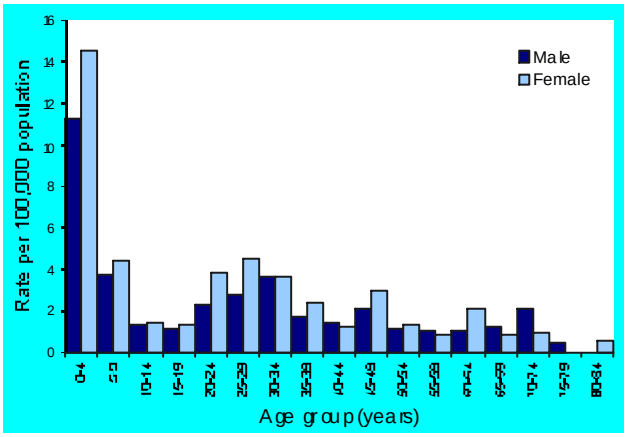
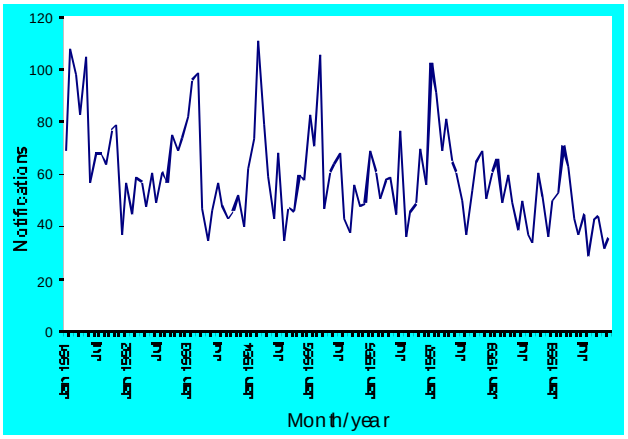


Figure 16. Notifications of shigellosis, Australia, 1991 to 1999, by month of onset



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

There were 43 cases of Shiga-like toxin producing *Escherichia coli*/Verotoxigenic *E. coli* (SLTEC/VTEC) reported to NNDSS with symptom onset in 1999, which was a 207 per cent increase from 14 cases reported in 1998. It should be noted however, that SLTEC/VTEC only became notifiable in August 1998, which may account for some of the increase in 1999. South Australia reported 90.7 per cent of cases, which reflects this State's policy of screening for toxin genes in faecal specimens (by PCR), from all cases of bloody diarrhoea. The median age of cases in 1999 was 33 years (range 1–77 years) and 37.2 per cent of notified cases were male. The highest age-specific rates were 0.5 per 100,000 population in 5–9 year-old children, and 0.9 per 100,000 population in 75–79 year-old people. SLTEC/VTEC was not specifically notifiable from Queensland or Western Australia.

Haemolytic uraemic syndrome

Infections with SLTEC/VTEC have the potential to cause severe and life-threatening illness including haemolytic uraemic syndrome (HUS). HUS is generally diagnosed on the basis of microangiopathic haemolytic anaemia, acute renal impairment and thrombocytopaenia (reduced platelet counts). Children aged less than 5 years are at increased risk of HUS. In an outbreak of HUS associated with the consumption of mettwurst in South Australia in 1994/1995 there was one death and 18 children required dialysis.[33]

There were 24 cases of HUS notified to NNDSS with symptom onset in 1999 (Table 1). States and Territories made this condition nationally notifiable in August 1998. New South Wales reported 11 cases and Victoria reported 8 cases. In New South Wales, 5 of the cases of HUS were considered to be a cluster. All presented with bloody diarrhoea, but no cultures were positive for VTEC. A common food source, minced beef, was postulated as the source of bacterial infection, but not proven.[34] In Victoria, 8 cases and 2 deaths were reported, however, they appeared to be sporadic cases.[35]

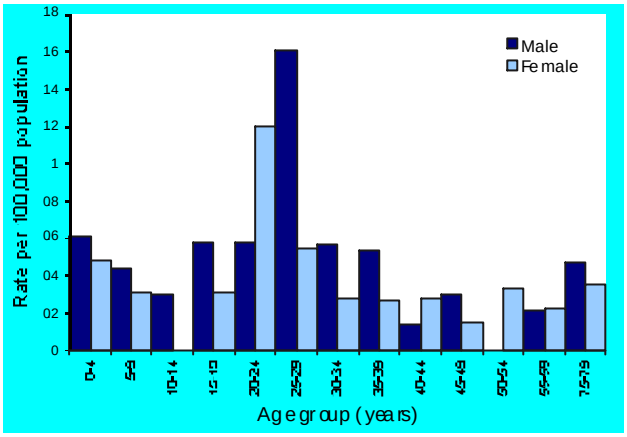
The median age of cases reported to NNDSS in 1999 was 4.5 years (range 0–70 years) and 54 per cent of notified cases were female. The highest age-specific rate was 0.9 cases per 100,000 population in 0–4 year-old children.

Typhoid

Most cases of typhoid in Australia occur in travellers returning to Australia from typhoid endemic countries. There were 72 cases of typhoid with symptom onset in 1999, which was a 12.5 per cent increase from 63 cases reported in 1998. The median age of cases reported to NNDSS in 1999 was 25.5 years (range 1–78 years) and 41.7 per cent of notified cases were female. The highest age-specific rate was 1.1 cases per 100,000 population in 25–29 year-old people (Figure 17). The highest notification rates were in New South Wales (0.6 per 100,000 population).

In June 1999, Australian health departments were notified of 12 cases of typhoid in people who had attended a Pacific Island cruise in May 1999 (Typhoid Epidemiology Working Group, unpublished report). The CDNA co-ordinated a national response to the outbreak. All 12 cases of typhoid had attended a tour on the Kokoda Trail in Papua New

Figure 17. Notification rate for typhoid, Australia, 1999, by age and sex



Guinea. Investigators found that typhoid illness in tour participants was associated with either contaminated coleslaw or drinking water. A mixed infection was suspected as 81 per cent of 159 tour participants experienced gastroenteritis following the tour.

Yersiniosis

There were 143 cases of yersiniosis reported to NNDSS with dates of symptom onset in 1999, which was a 25 per cent decrease from 190 cases reported in 1998. The median age of cases in 1999 was 19 years (range 0–86 years) and 56.6 per cent of notified cases were male. The highest age-specific rates were 3.8 cases per 100,000 population in 0–4 year-old children, and 1.1 per 100,000 population in 20–24 year-old people (Figure 18). The highest notification rates were in Queensland (2.9 per 100,000 population) and South Australia (1.2 per 100,000 population). Yersiniosis was not specifically notifiable in New South Wales. Cases were more commonly notified during January to March (Figure 19).

Quarantinable diseases

In Australia, the human diseases proclaimed to be quarantinable under the *Quarantine Act 1908* are cholera, plague, rabies, yellow fever, and 4 viral haemorrhagic fevers (Ebola, Marburg, Lassa and Crimean-Congo). Cholera, plague, yellow fever and the viral haemorrhagic fevers are of international public health significance with mandatory reporting to the WHO, under International Health Regulations (http://www.who.int/m/topics/international_health_regulations/en/index.html). Rabies is a disease of both human and animal quarantine importance in Australia. All States and Territories notify the quarantinable diseases to the NNDSS.

The only cases of quarantinable disease reported in Australia in 1999 were 3 cases of cholera. Two of these cases were imported, one from Indonesia and one from India. The other case occurred from local water sources in New South Wales. Two were *Vibrio cholerae* 01 classical Ogawa and one was 01 El Tor/Ogawa.

Cases of cholera continue to be reported in travellers returning from foreign countries, particularly from Asia. These cases demonstrate the importance of travellers consuming safe food and drink in areas where cholera is known to occur. In general, travellers should be aware of how to avoid the diseases which are commonly reported in many developing countries.

Although no cases of rabies or yellow fever were reported in Australia, worldwide these 2 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 be vaccinated with the yellow fever vaccine from an approved Australian vaccination centre. Information on quarantinable diseases can be found on the Department of Health and Ageing Website at: http://www.health.gov.au/pubhlth/consumer/index/index.html

Sexually transmitted infections

The infections classified as sexually transmissible for surveillance in the NNDSS are chancroid, chlamydial infection, donovanosis, gonococcal infection, lymphogranuloma venereum and syphilis.

States and Territory health departments follow NHMRC case definitions for the reporting of these conditions (Appendix 1d).

There are important infections commonly or usually spread by sexual contact, which are not subject to national surveillance through the NNDSS. These include genital herpes (herpes simplex virus type I and II), genital warts

Figure 18. Notification rate for yersiniosis, Australia, 1999, by age and sex

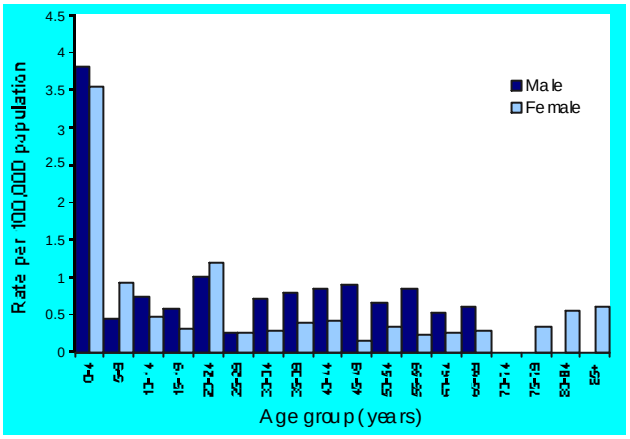
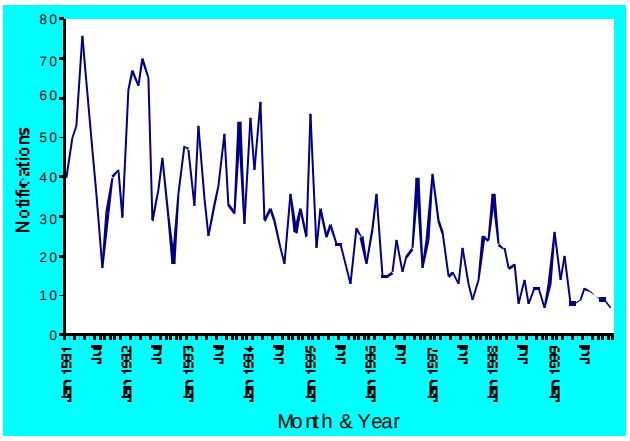


Figure 19. Notifications of yersiniosis, Australia, 1999, by month of onset



(human papilloma virus, several types), trichomoniasis and parasitic infestations such as pubic lice and scabies.

In addition to the sexually transmissible infections (STI) surveillance by the NNDSS, the Australian Gonococcal Surveillance Programme (AGSP), a national laboratory-based surveillance system, documents the antibiotic sensitivity of gonococcal isolates. The AGSP includes some clinical and demographic data. National data on HIV and AIDS are collected and reported separately by the National Centre in HIV Epidemiology and Clinical Research. The Centre also reports on trends in sexually transmissible infection notifications received via NNDSS. The full report for 1999 is available at www.med.unsw. edu.au/nchecr.

Chancroid

Chancroid is a bacterial infection causing genital ulcers. There have only been 11 cases reported to the NNDSS since 1991. No cases of chancroid were reported in Australia in 1999.

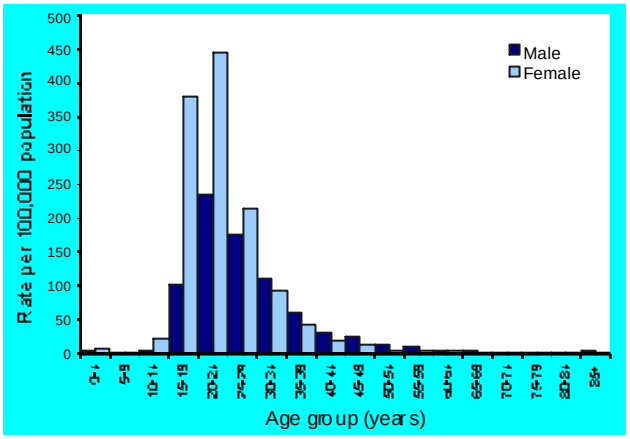
Chlamydial infection

Chlamydial infections were the most commonly reported STI and the third most commonly reported notifiable disease in Australia in 1999, when 14,082 notifications of chlamydial infection were reported to the NNDSS (Table 1). In New South Wales, reporting of genital chlamydial infection commenced in September 1998, so that the reporting for chlamydial infections was national for the first time in 1999. The inclusion of New South Wales data gives a more accurate estimate of the national prevalence than previous years. Chlamydial infections may be under-reported because of a high proportion of asymptomatic infections, particularly among women.[29] The recent advent of nucleic acid tests (NAT) for chlamydia may also explain increases in notification; up to 83 per cent (2,747 of 3,298) of chlamydial infections reported to the Laboratory Virology and Serology Reporting Scheme (LabVISE) were detected by nucleic-acid methods.

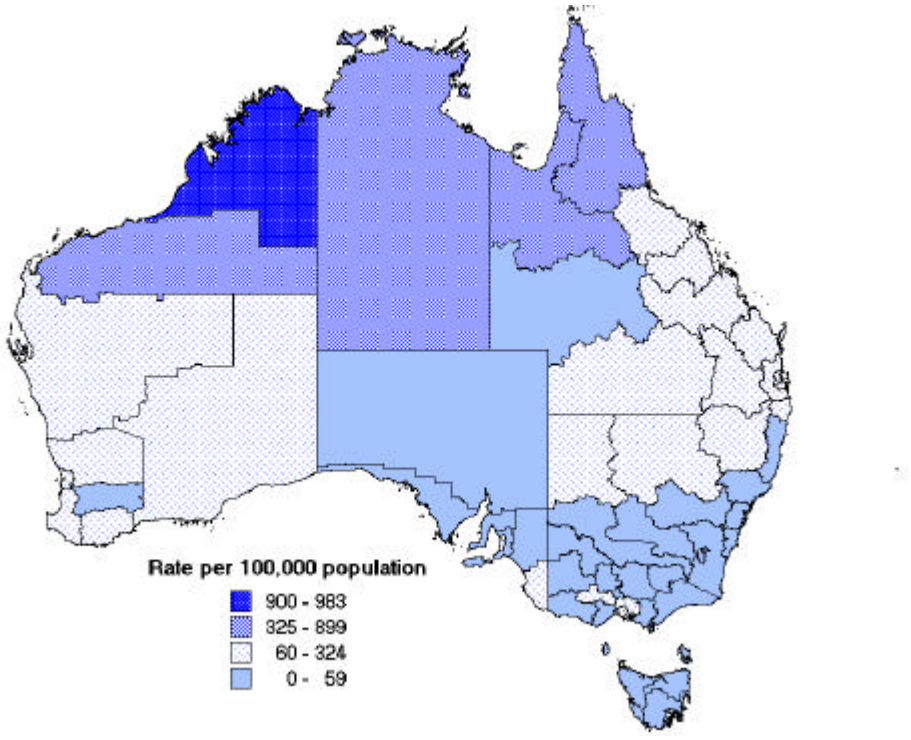
The majority (92%) of reported cases of chlamydia were in the 15–39 year age range. The notification rate for chlamydial infections in 1999 was 74.2 cases per 100,000 population, higher than the rate of 60.5 cases per 100,000 population reported in 1998.

The male to female ratio was 1:1.5. In both males and females the highest rates of disease were recorded for the 20–24 year age group (Figure 20). High rates of notification were reported from northern Australia, including rates over 400 per 100,000 population in the Northern Territory (Map 5). The National Centre in HIV Epidemiology and Clinical Research (NCHECR) reported rates of chlamydial disease in indigenous Australians from NNDSS data in their annual report. Based on data from the Northern Territory,

Figure 20. Notification rate for chlamydial infection, Australia, 1999, by age and sex



Map 5. Chlamydial infection notification rate by Statistical Division of residence



South Australia and Western Australia, which were the only jurisdictions to report indigenous status in more than half of notifications, the NCHECR estimated a rate of chlamydial infection among indigenous Australians of 882 per 100,000 population compared with a rate of 75 per 100,000 population in non-indigenous Australians.

Lymphogranuloma venereum

Lymphogranuloma venereum (LGV) is a sexually acquired chlamydial infection caused by certain serovars of *Chlamydia trachomatis*. The disease begins with a painless genital lesion and may progress to suppurating draining lymph nodes and the development of inguinal buboes. LGV is a common sexually transmitted infection especially in poorer communities in tropical and sub-tropical regions of the world. In Australia, there have only been 7 reports to the NNDSS since 1991 and none since 1995. There were no cases of lymphogranuloma venereum reported from any State or Territory in 1999.

Donovanosis

Donovanosis is a notifiable disease in all jurisdictions except South Australia. Donovanosis is a chronic genital ulcer disease that occurs in indigenous Australians in rural and remote communities. Notifications of donovanosis have fallen significantly over the past 10 years, and particularly since 1994 due to the introduction of more sensitive and acceptable testing methods and more effective treatment with azithromycin. Since 1994, the notification rate of donovanosis has fallen ten-fold from 1.1 per 100,000 population to 0.1 per 100,000 population. Donovanosis only became a notifiable disease in New South Wales in September 1998. A total of 16 notifications were received in 1999, all from the Northern Territory, Queensland or Western Australia. The male to female ratio was 1:7, a ratio consistent with that in 1998. Fifty percent of the notifications were in the 15–29 year age range.

Gonococcal infection

In 1999, a total of 5,676 notifications of gonococcal infection were received nationally (Table 1). The notification rate of 29.9 per 100,000 population continues a steady increase in notifications since 1994 (Figure 21).[36] This rate remains far below the very high rates recorded in the 1970s and early 1980s, which peaked at 84.4 per 100,000 population in 1982.[37] The number of notifications of gonococcal infection has increased over the past decade. The increase was due in part to an outbreak of gonorrhoea among men who have sex with men in Victoria and to increased testing as part of sexual health programs in Victoria and New South Wales.

There was a wide geographical variation in the rate of notification of gonococcal infection (Map 6). The highest rates of notification were from the Northern Territory (590 per 100,000 population) and from northern Statistical Divisions in Western Australia (Map 6). The male to female ratio of 2.2:1 was higher than in previous years. However, the notification rate for females aged 15 to 19 years was higher than for males in the same age group (Figure 22). The NCHECR reported rates of gonococcal disease in indigenous Australians, from NNDSS data in their annual report. Based on data from the Northern Territory, South Australia and Western Australia, which were the only jurisdictions to report indigenous status in more than half of notifications, the NCHECR estimated a rate of gonococcal infection among indigenous Australians at 1334 per 100,000 population compared with a rate of 17 per 100,000 population in non-indigenous Australians.

Figure 21. Trends in the national notification rate for gonococcal infections, Australia, 1991 to 1999

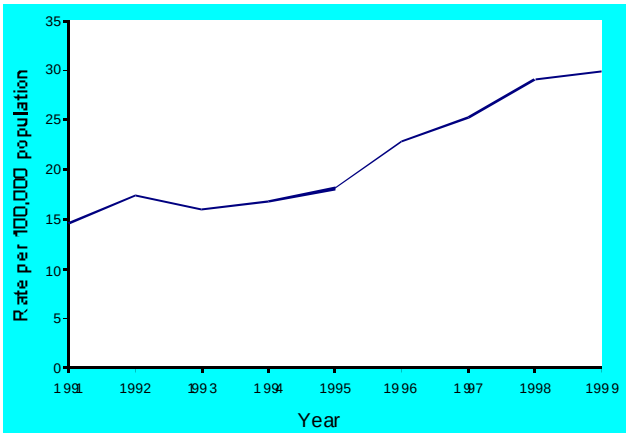
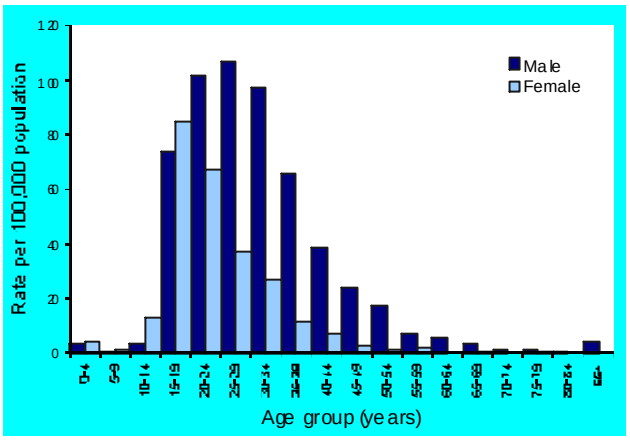


Figure 22. Notification rate for gonococcal infections, Australia, 1999, by age and sex



A survey of the antibiotic susceptibility of *Neisseria gonorrhoeae* by the AGSP on 3,740 isolates in 1999 has been published.[38] Antibiotic susceptibility patterns varied significantly between regions. Generally rates of resistance to penicillin and quinolone groups of antibiotics were higher in urban than in rural areas.

Syphilis

A total of 1,979 notifications of syphilis were received in 1999 (Table 1) with a rate of 10.4 per 100,000 population, representing a 16.9 per cent increase in the rate compared with 1998 (1,689 notifications and a rate of 9 per 100,000 population). This increase continues that seen in 1998 and reverses the trends seen since 1992. However, the rate remains lower than those seen in the 1980s. There was wide geographical variation in the notification rate (Table 2, Map 7). While most States and Territories show a slow decline in notification rates for syphilis, Queensland has shown an increase since 1997 (Figure 23). Subsequent information received from the Queensland Communicable Disease Unit suggests that the increase shown is due to poor case definition and the recording of follow-up syphilis

Map 6. Gonococcal infections notification rate by Statistical Division of residence

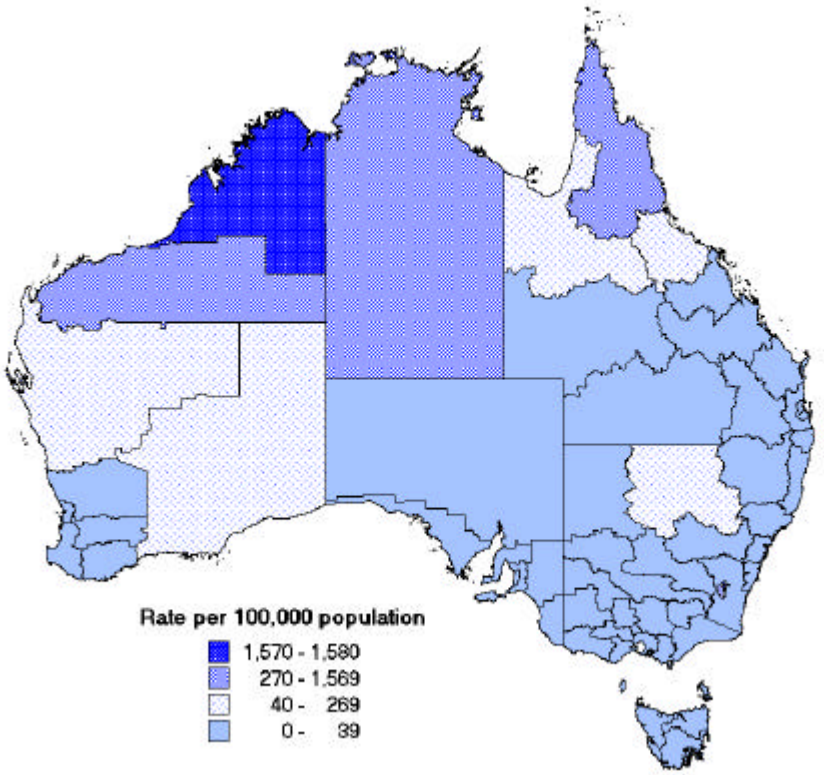


Figure 23. Notification rate for syphilis, New South Wales, Western Australia and Queensland, 1991 to 1999

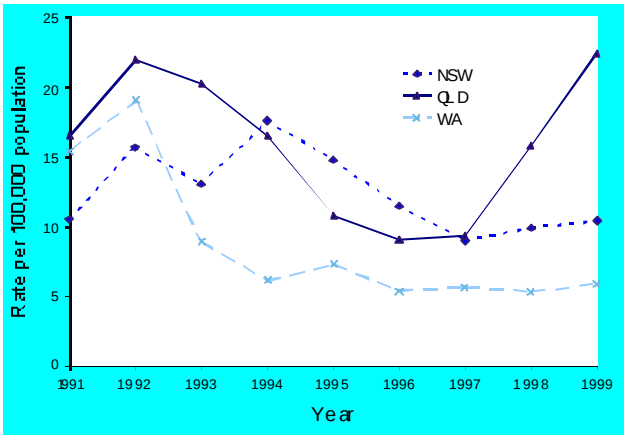
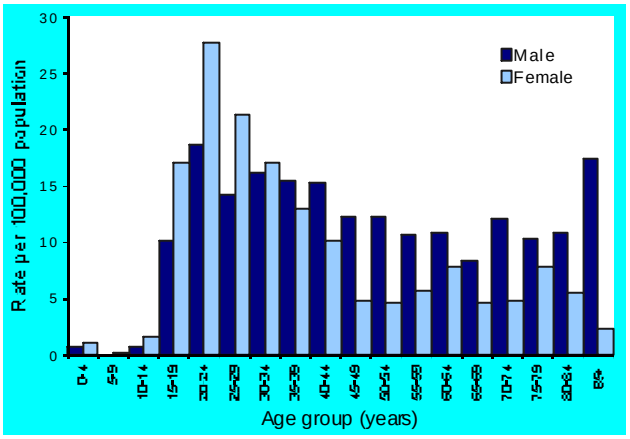


Figure 24. Notification rate for syphilis, Australia, 1999, by age and sex



serology results on the register. A review of Queensland syphilis registers was undertaken in July 2001. High notification rates continued to be reported from the Northern Territory and Western Australia but these are declining.

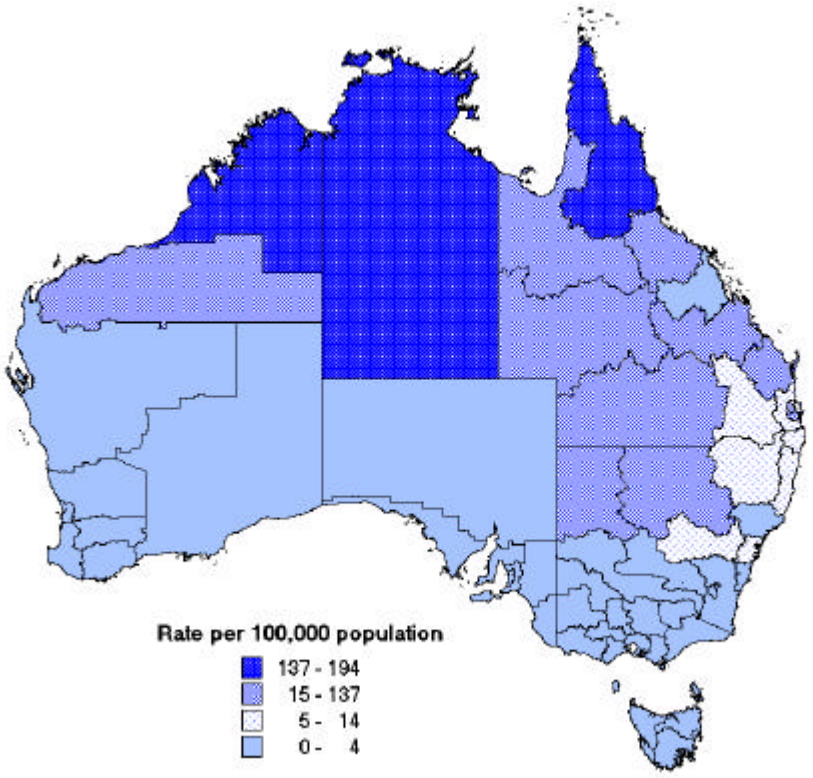
The male to female ratio for syphilis notifications was 1.1:1. Notification rates were higher among females than males in age groups younger than 34 years and higher in males than females in age groups older than 35 years (Figure 24). The NCHECR reported rates of syphilis in indigenous Australians based on NNDSS data in their annual report. Based on data from the Northern Territory, South Australia and Western Australia, which were the only jurisdictions to report indigenous status in more than half of notifications, the NCHECR estimated a rate of syphilis among indigenous Australians of 253 per 100,000 population compared with a rate of 3 per 100,000 population in non-indigenous Australians. There were 2 cases of congenital syphilis in 1999, both reported from New South Wales.

Vaccine preventable diseases

Introduction

This section summarises the national notification data for diseases targeted by the standard childhood vaccination schedule in 1999. The only change to the schedule in 1999 was the recommendation that DTPa (Diphtheria-Tetanus-acellular Pertussis) be used for all 5 infant doses. Previously this vaccine was only funded nationally for the 2 booster

Map 7. Syphilis notification rates by Statistical Division of residence



doses. Other diseases for which vaccines are licensed in Australia but which were not incorporated into the standard childhood schedule in 1999 (hepatitis A, hepatitis B, invasive pneumococcal disease, influenza, some serotypes of meningococcal disease, varicella and Q fever) are not described in this section. The 1999 influenza surveillance data, and investigations for polio and acute flaccid paralysis have been published in earlier editions of *CDI*.[39,40,41] Congenital rubella notifications for 1999 (5 notifications, one definite congenital rubella infection late in pregnancy) are described in the Seventh Annual Report of the Australian Paediatric Surveillance Unit.[42]

The third annual report of vaccination coverage estimates for children aged 12 months and the second annual report for children aged 24 months (using data extracted from the Australian Childhood Immunisation Register-ACIR) are also included in this section. A full description of the methodology used for calculating these estimates have been described previously.[43]

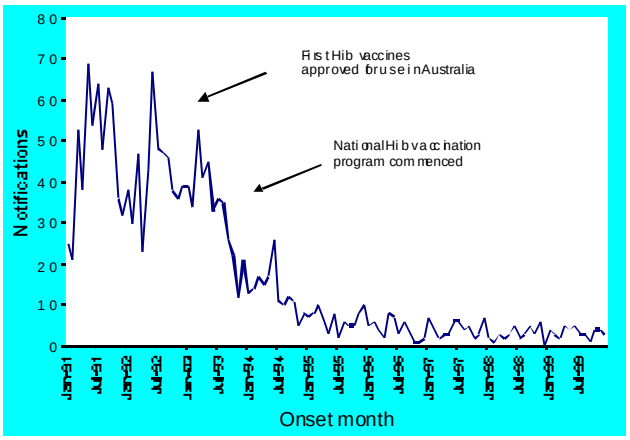
Diphtheria

There were no cases of diphtheria notified in 1999. Prior to this, the last known case occurred in 1992 and was notified in 1993.

Haemophilus influenzae type b

There were 40 notifications of *Haemophilus influenzae* type b (Hib) disease in 1999, five more than in 1998 but considerably less than in the pre-vaccine era (Figure 25). As in previous years, most notified cases (52.5%) were less than 5 years of age and the highest notification rates were in children less than 2 years of age. The notification rate for children 0 to 11 months of age was 3.6 per 100,000 population and for one-year-olds was 4.0 per 100,000 population. This compares with an overall notification rate of 0.2 per 100,000 population. There were slightly more females than males (male:female ratio 1:1.2). The Northern Territory had the highest notification rate (1.6 per 100,000 population, 3 cases) although most cases (25/40) were from New South Wales and Queensland.

Figure 25. Notifications of Hib, Australia, 1991 to 1999, by month of onset



Measles

There were 230 measles notifications in 1999, a rate of 1.2 per 100,000 population. This is the lowest annual rate for Australia since national surveillance began in 1991 (Figure 26).

Geographical distribution

All States and Territories except Victoria and the Northern Territory had their lowest ever annual notification rates of measles. In 1999, the Northern Territory reported 10 cases, nine more than in 1998, leading to a notification rate for 1999 which was at least double that of any other jurisdiction (Table 2). Although the Northern Territory had the highest rate, Victoria had by far the greatest proportion of cases (48%). Most of the Victorian cases (62/111, 56%) occurred during an outbreak in February/March (Figure 26). This outbreak had as its index case a returned traveller from Bali, and all except 10 of the 62 cases were young adults aged 18–30 years.[44] Two other smaller clusters of measles cases occurred in Victoria between August and October. The first was traced to a returned traveller arriving from London via Malaysia, while the second was associated with evacuees from East Timor.[35] Queensland experienced increased numbers of notifications in June and July while other jurisdictions recorded their lowest numbers for the year at that time.

Age and sex distribution

As in recent years, age-specific notification rates for measles were highest for 0–4 year-olds (6.3 per 100,000 population) especially those aged less than one year (15.4 per 100,000 population) and one year of age (7.6 per 100,000 population). However, rates for these age groups were considerably lower than in the past (Figure 27). Rates for 5–9 year-olds were also the lowest on record. In contrast, rates for older ages increased compared with those for the previous year — a reflection of the outbreak amongst young adults in Victoria in 1999. The most apparent rise was in the 20–24 year age group (rate: 4.0 per 100,000 population) which accounted for 20 per cent of the cases in 1999 (compared with less than 10 per cent in the previous 6 years). This age group had the second highest age-specific rate. As in past years there were similar numbers of male and female cases, with slightly more females than males in 1999 (male:female ratio 1:1.2).

Mumps

In 1999 there were 184 notifications of mumps, a rate of 1.0 per 100,000 population. This is similar to the number of notifications in the past 2 years. There were notifications from most age groups (Figure 28) with 50.8 per cent from people aged at least 15 years. As in previous years the highest notification rates were in the 5–9 year age group (2.7 per 100,000 population) and the 0–4 year age group (2.6 per 100,000 population, Figure 29). However rates in these age groups have been lower since 1995 while rates in people aged at least 15 years have been steadily increasing since 1993. This pattern was apparent even in New South Wales where only laboratory-confirmed cases are notifiable. In 1999, there was a secondary peak in notifications in the 25–29 year age group (1.4 per 100,000 population).

Overall, there were similar numbers of mumps notifications from males and females (male:female ratio 1.1:1), however, there were more notifications for males than females in the age groups most frequently reported. The rates were highest in the Australian Capital Territory and Western Australia (Table 1) while Victoria provided most of the notifications (39.7%) (Table 1). Notified cases occurred throughout the year, but peaked in May largely due to increased reports from Victoria at this time.

Figure 26. Notifications of measles, Australia, 1991 to 1999, by month of onset (and State/ Territory of residence)

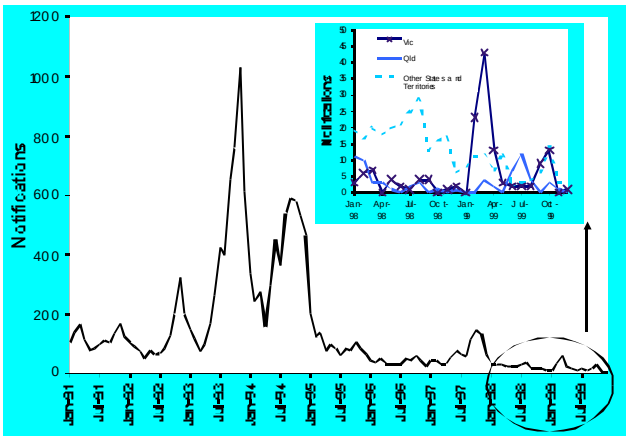


Figure 27. Notification rate for measles, Australia, 1996 to 1999, by age group and year of onset

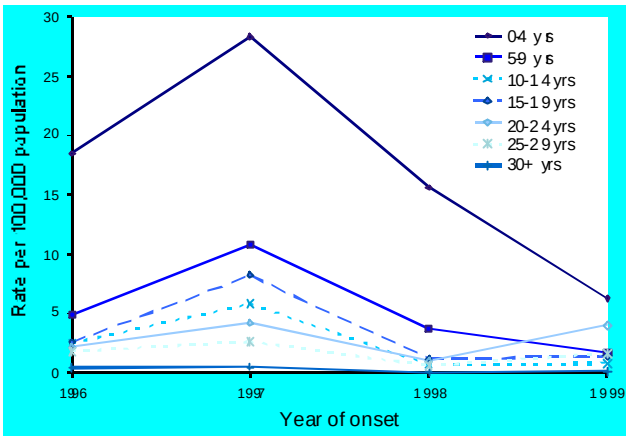


Figure 28. Mumps notification rate, Australia, 1999, by age and sex

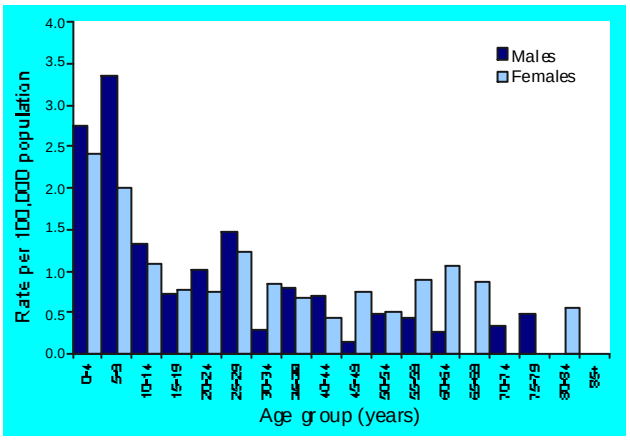


Figure 29. Notification rate for mumps, Australia, 1993 to 1999, by age group and year of onset

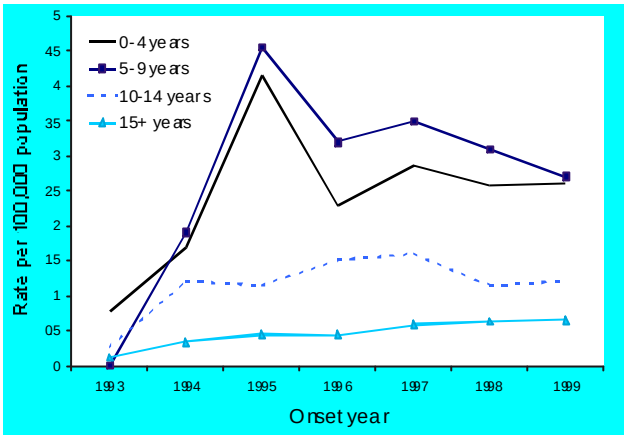
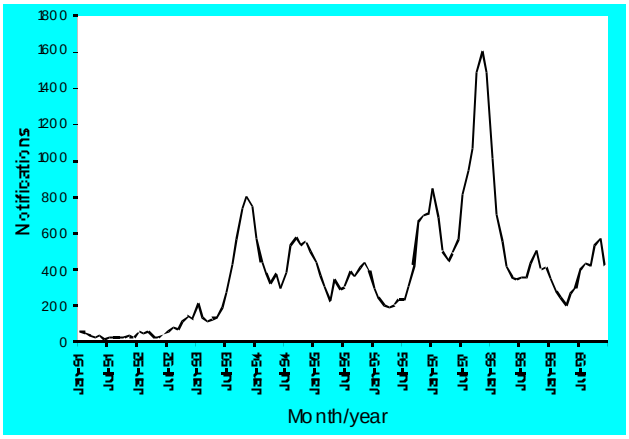


Figure 30. Notifications of pertussis, Australia, 1991 to 1999, by month of onset



Pertussis

There were 4,396 notified cases of pertussis in 1999, 1,430 fewer than in 1998. The annual notification rate of 23.2 per 100,000 population was the lowest recorded since 1992. The majority (63%) of the notifications occurred in the second half of the year when there were outbreaks in Tasmania, Western Australia, Queensland and Victoria. Notifications peaked in November 1999, when 572 cases were notified (Figure 30).

For the first time since the establishment of the current notification system, the 10–14 year age group had the highest notification rate of pertussis instead of infants aged less than one year (Figure 31). Children aged 1–4 years had the lowest rate, which is also in contrast to past years when rates were lowest in adults. The notification rate in 5–9 year-olds continued to decline, both overall and relative to all other age groups except those aged less than one year. In 1999, the rate for 5–9 year-olds was only marginally higher than the rate in adults and 1–4 year-olds.

Notification rates of pertussis varied considerably by geographic location (Map 8). At the State/Territory level, rates were highest in Tasmania (129.9 per 100,000 population) and lowest in the Northern Territory (only 2 cases notified, giving a rate of 1 per 100,000 population).

Map 8. Pertussis notification rates by Statistical Division of residence

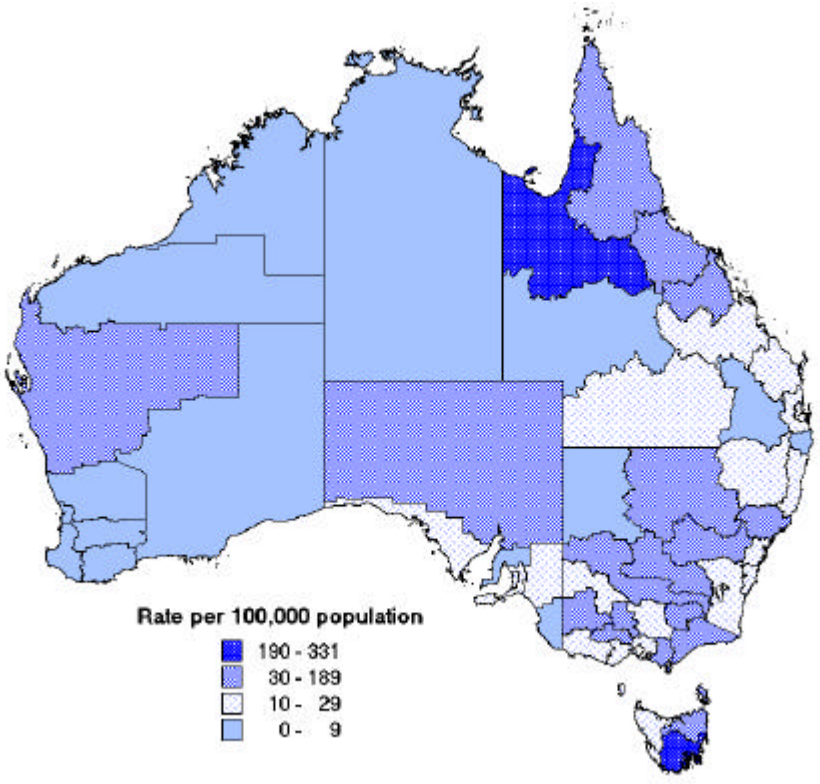
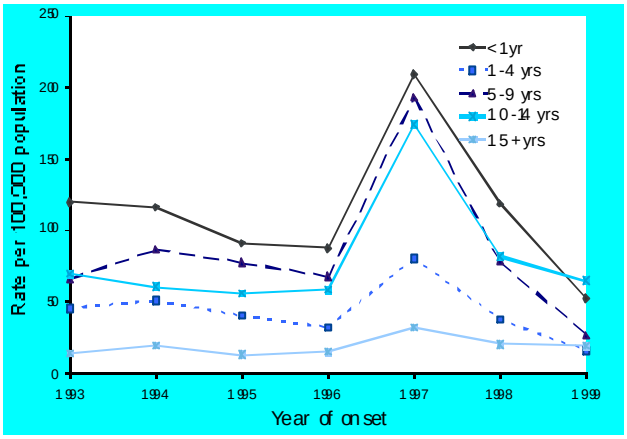


Figure 31. Notification rate for pertussis, Australia, 1993 to 1999, by age group and year of onset



Poliomyelitis

No cases of poliomyelitis were reported in 1999.

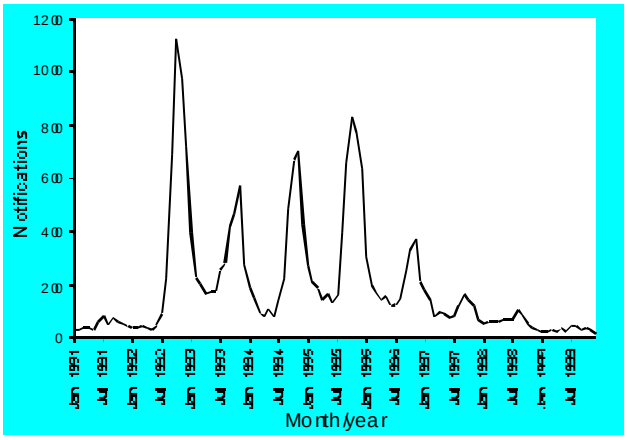
It is difficult to determine exactly when the last case of locally acquired poliomyelitis occurred in Australia. However, the last laboratory confirmed case was in 1967 and there were three clinically compatible cases notified in 1972 with no additional information currently available.[45] All cases notified since 1972 have been investigated further and this has led them to be re-classified as cases of vaccine-associated paralytic poliomyelitis (VAPP). The last known imported case of poliomyelitis was due to wild poliovirus type 1 in 1977.

Rubella

Secular and geographic distributions

Since 1995, annual numbers of rubella notifications have been declining (Figure 32). In 1999, there were 376 notifications, a notification rate of 2.0 per 100,000 population. This is half the number/rate of 1998, and the lowest on record both nationally and in each State and Territory. As in 1998, the highest number of notified cases occurred in August, which is slightly earlier than the expected seasonal increase in spring months. This peak was predominantly due to increased notifications from Victoria and Queensland who also contributed most (73.9%) of the notifications for the year. Despite most notifications coming from these 2 States, the highest notification rate was from the Australian Capital Territory (Table 2).

Figure 32. Notifications of rubella, Australia, 1991 to 1999, by month of onset

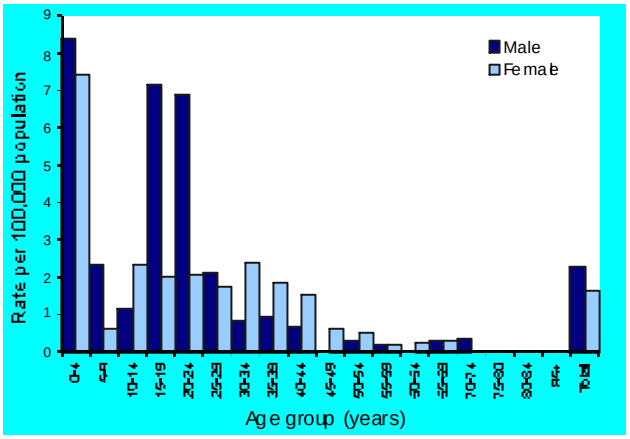


Age and sex distribution

In 1999, notification rates were highest for both males and females aged 0–4 years (rate 8.0 per 100,000 population, Figure 33), which is in contrast to the previous 6 years when males aged 15–24 years had the highest rate.[24] This altered distribution reflects a continued decline in rates for 15–24 year-old males, while rates for 0–4 year-olds remained constant between 1998 and 1999. Rates for young adult males have been decreasing in recent years due to the replacement of the schoolgirl rubella program with adolescent vaccination of both males and females between 1994 and 1998. Despite the lower rates for males aged 15–24 years, this group continued to contribute a significant proportion of the notifications (25.8%) and overall there were still more males than females notified with rubella (male:female ratio 1.4:1). Within the 0–4 year age group, the majority of cases (80.4%) were aged less than 2 years. Those aged less than one year had the highest rate (21.9 per 100,000 population) and were the only age group to show a marked increase in rates in 1999.

Notification rates in 1999 for women of childbearing age were the lowest on record; there were 82 cases, a rate of 1.9 per 100,000 population. The rate reductions were seen in each of the 5-year age groups between 15 and 44 years.

Figure 33. Notifications of rubella, Australia, 1999, by age and sex



Tetanus

In 1999 there were 2 cases of tetanus (one male and one female). Both cases were aged at least 60 years with one reported from Queensland and the other from New South Wales. This is the lowest number of cases reported for a year since the establishment of the current notification database in 1991.

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 1999 (Table 7). This trend was also evident in each State and Territory.

Vaccination coverage at 2 years of age was first reported in 1998. Coverage estimates for vaccines recommended at 12 months and 18 months of age were higher in 1999 compared with the previous year, as were the estimates for being fully vaccinated at 2 years of age (Table 8). However, only MMR coverage nationally and DTP coverage for the Northern Territory showed any trend upwards during 1999. 'Fully vaccinated' coverage levels were reported to be lower than estimates for individual vaccines. One likely factor is poor identification of children on immunisation encounter forms, which leads to difficulties matching new and existing vaccination records on the ACIR. It is important to note that in other countries such as the United Kingdom, 3 doses of DTP and Hib vaccine constitute full vaccination with these vaccines at 2 years of age.

In 1999, notification rates for measles, rubella and tetanus were the lowest on record. Rates for Hib infection also remained low, while pertussis rates were the lowest since before the epidemic of 1997. Although overall pertussis notification rates for Australia were the lowest since 1992, many temporally and geographically distinct outbreaks occurred in 1999, with adolescents aged 10–14 years emerging as the age group most at risk. Improved vaccination coverage for the first 4 doses of DTP vaccine and the inclusion of a fifth dose at 4 years of age (in 1994) have been associated with a more rapid decline in rates for ages less than 10 years old compared with those for 10–14 year-olds. The implications of this trend, now that a pertussis vaccine is available for ages 9 years and over, will be considered by a working party of the Australian Technical Advisory Committee on Immunisation (ATAGI) in 2001.

The record low notification rates for measles and rubella highlight the success of the Measles Control Campaign (MCC),[46] and the current vaccination program. The MCC actively targeted pre-school and primary school-aged children and significantly improved their immunity to both measles and rubella.[47] As a result, in 1999 Australia recorded the lowest ever notification rates for measles and rubella for children in these age groups. Importantly, improved rubella control has also led to the lowest rate of rubella amongst women of childbearing age with only one definite congenital rubella infection reported in 1999.[42]

With record low rates of measles, most clinically compatible cases are now likely to be due to other viral infections.[48] Hence, it is imperative that the recommendations for measles surveillance proposed in the National Measles Surveillance Strategy are introduced; laboratory confirmation should be sought on all sporadic clinical notifications and at least 2 cases during an outbreak.[49]

Clusters of measles cases continued to occur in 1999, mostly amongst young adults. Serosurveys have shown that some young adults may have low levels of measles immunity,[50] as they are too old to have been part of the two-dose MMR vaccination program (introduced in 1994) but have grown up in a period when exposure to wild measles virus was declining. A vaccination initiative to improve MMR coverage in this age group is currently under way.[51] In addition to reducing the incidence of measles, it is hoped that the initiative will impact on the burden of mumps in adults, which has also been increasing in recent years.

Table 7. Percentage of Australian children born in 1998 vaccinated according to data available on the Australian Childhood Immunisation Register. Estimate at one year of age

Birth date Vaccine group	1 January to 31 March 1998 % vaccinated	1 April to 30 June 1998 % vaccinated	1 July to 30 September 1998 % vaccinated	1 October to 31 December 1998 % vaccinated
DTP (3)	87.6	88.0	88.3	89.5
OPV (3)	87.3	87.9	88.3	89.5
Hib (2 or 3*)	87.4	87.7	87.9	88.9
Fully vaccinated	86.1	86.5	87.0	88.1

* Number of doses depends on the vaccine used

Table 8. Percentage of Australian children born in 1997 vaccinated according to data available on the Australian Childhood Immunisation Register. Estimate at 2 years of age

Birth date Vaccine	1 January to 31 March 1997 % vaccinated	1 April to 30 June 1997 % vaccinated	1 July to 30 September 1997 % vaccinated	1 October to 31 December 1997 % vaccinated
DTP (4)	82.8	83.8	82.8	83.6
OPV (4)	87.7	89.8	82.8	83.7
Hib (3 or 4*)	82.8	83.8	82.4	83.4
MMR (1)	87.8	88.7	89.0	89.7
Fully vaccinated	73.5	75.9	74.9	76.7

* Number of doses depends on the vaccine used

Vaccination coverage estimates from the ACIR continued to increase in 1999. During 1999, the impact of the General Practice Immunisation Incentives (GPII) Program, which began in July 1998, would have been expected to improve both reporting of vaccinations to the ACIR and vaccination delivery in general practice. Given the role of general practitioners as the largest single group of immunisation providers nationally, the GPII Program together with linking maternity and child-care allowance payments to vaccination uptake are expected to lead to continued improvement in vaccination coverage at 12 and 24 months. Improvements in the accuracy and timeliness of ACIR data compared to earlier years[52] should enable their use in documenting the vaccination status of notified cases of vaccine preventable diseases. This, together with continued efforts to improve the quality of NNDSS surveillance data will be important components of enhanced surveillance in the future.

Vectorborne diseases

Arthropod-borne viruses, which are able to replicate in arthropod vectors and in vertebrate hosts, are collectively referred to as arboviruses. The nationally notifiable vector-borne diseases include several arboviruses and malaria. Although there are over 70 types of arboviruses in Australia, only a small number cause disease in humans (Mackenzie, 1998).

The NNDSS collects information on 2 alpha viruses, Barmah Forest (BF) and Ross River (RR) viruses, and one flavivirus, dengue, as well as malaria and arboviruses (not elsewhere classified). This category includes infections with the flaviviruses Murray Valley encephalitis (MVE) virus, Kunjin virus, Japanese encephalitis (JE) virus, Kokobera virus and Stratford virus, as well as the alphavirus Sindbis.

In the States and Territories, data on human cases are supplemented by sentinel chicken surveillance (seroconversions to MVE and Kunjin viruses), animal surveillance (seroconversions to JE in pigs), vector data, virus isolations and meteorological data.

Alphavirus infections

Barmah Forest virus infection

Barmah Forest virus was first isolated from mosquitoes trapped in the Barmah Forest in Victoria in 1974. The first association with disease in humans was described in 1988. Subsequently epidemics of BF virus disease were reported from the Northern Territory (1992), Western Australia (1992–1993) and New South Wales (1995).[53] BF virus infection is characterised by polyarthritis, myalgia, rash, fever, lethargy and malaise and may cause a chronic disease in some patients.[54] *Aedes* and *Culex* mosquitoes spread the disease and marsupials are a suspected host. The Southern Oscillation Index, which is closely related to temperature and rainfall patterns in eastern Australia also appears to be linked to levels of BF virus disease.[55]

In 1999, 639 notifications of BF virus infection were reported, representing a slight increase above the number of cases reported in 1998. The highest rates were reported in the Northern Territory (9.3/100,000 population) and Queensland (8.8/100,000 population). Rates were very low in southern states; no cases were reported from South Australia (Map 9). The male to female ratio was 1.5:1. The highest rates of infection were in those aged 45–49 years (Figure 34). Peak notifications were in the period January to April and followed previously observed seasonal trends (Figure 35). The first reports of BF virus disease were reported from Tasmania in 1999.

Map 9. Barmah Forest virus infection notification rate by Statistical Division of residence

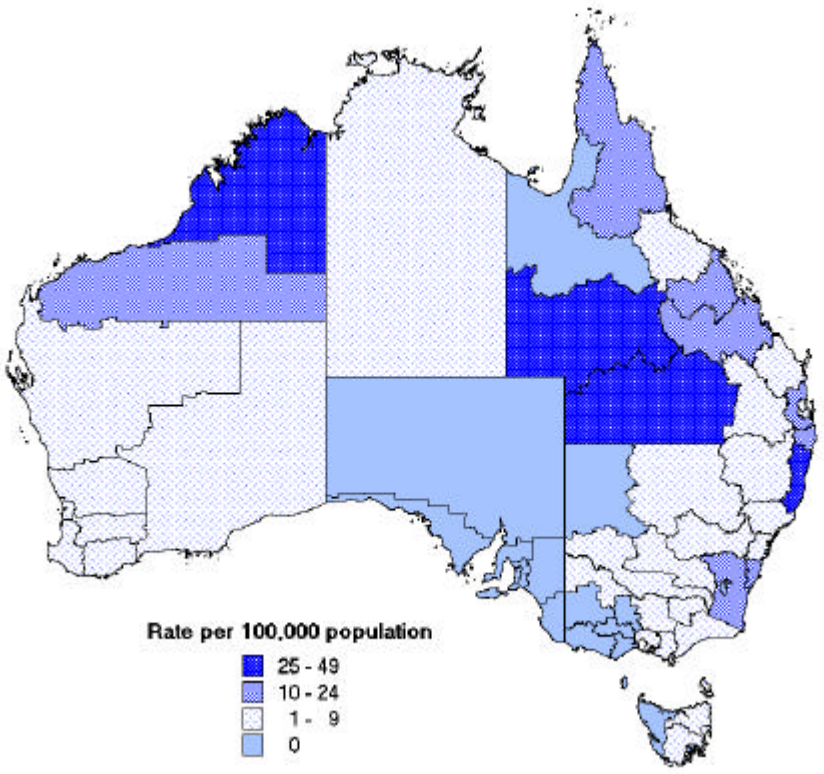


Figure 34. Notification rate for Barmah Forest virus infections, Australia, 1999, by age and sex

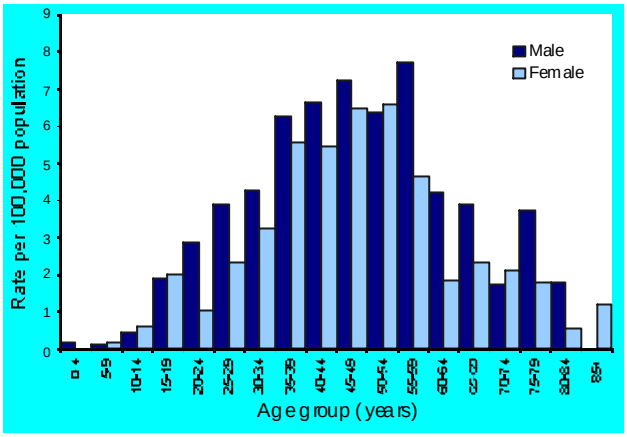
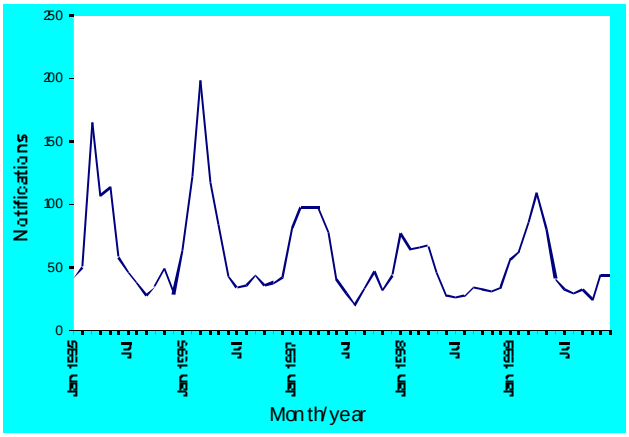


Figure 35. Notifications of Barmah Forest virus infections, Australia, 1995 to 1999, by month of onset

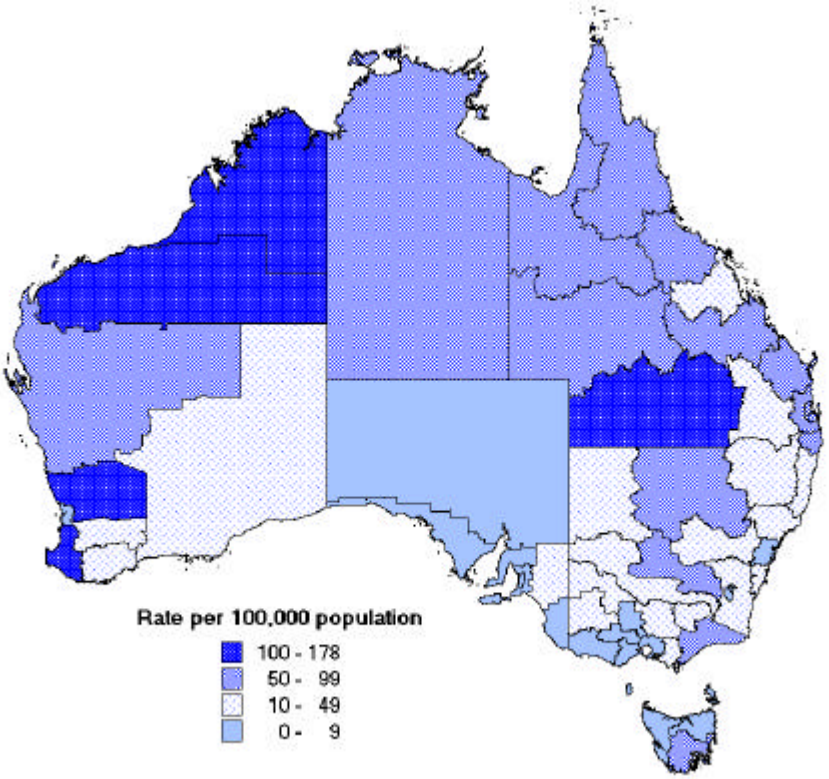


Ross River virus infection

Ross River virus is the most common arbovirus reported in Australia and the cause of the most arboviral disease. Sporadic cases occur throughout Australia. Epidemics, associated with heavy rainfall occur in temperate regions while transmission in tropical north-eastern Australia occurs throughout the year. Major outbreaks have been recorded in Western Australia (1991-1992 and 1995-1996), Victoria and South Australia (1993 and 1997), New South Wales (1996 and 1997) and Queensland (1996). Since 1991, more than half of all reports of RR virus have originated in Queensland. Recent evidence indicates that the virus may persist in desiccation-resistant eggs of the *Aedes* spp mosquito, which would explain the rapid onset of cases after heavy rain and flooding. Marsupials and horses have been implicated as hosts for the virus and flying foxes may be responsible for the wide spread dispersal of different genetic types of the virus.[53] Clinical RR virus disease occurs most commonly in adults, marked by arthralgia and myalgia (joint and muscle pain). True arthritis occurs in over 40 per cent of patients, while about 50 per cent of patients have a fever or rash.[57]

There were 4,416 notifications of RR virus infections in 1999, an increase from 17.0 to 23.3 cases per 100,000 population since 1998. Rates were highest in the Northern Territory (81.4/100,000 population), Queensland (65.7 per 100,000 population) and Western Australia (33.6/100,000 population) (Map 10). The male to female ratio was 1.1:1.

Map 10. Ross River virus infection notification rate by Statistical Division of residence



The highest rates were in the 35–39 year age group (Figure 36). Peak reporting was in the first and second quarters of the year (Figure 37).

Flavivirus infections

Dengue fever

Historical trends of dengue in Australia

Despite periodic epidemics of dengue fever since the 1980s, dengue virus is not endemic in Australia. The spread of dengue in Australia is limited to the range of the mosquito vector *Aedes aegypti*, which is limited to the Torres Strait Islands and north Queensland.[53]

Outbreaks of dengue in Australia have included a few cases of dengue type 1 in Cairns and the Torres Strait. An outbreak of more than 900 confirmed cases in Townsville and Charters Towers in 1992–1993 was caused by dengue type 2. In 1996–1997 another outbreak of dengue type 2 occurred in the Torres Strait. In 1997–1998 165 cases of dengue type 3 and 12 of dengue type 2 were reported from Cairns.[53]

Dengue haemorrhagic fever (DHF), first described in Australia in 1897[56] is a major complication arising from secondary infection with heterologous serotypes of the dengue virus which enhances viral uptake and replication. This complication, which primarily affects children in endemic countries, has a high fatality rate and is common in countries such as Thailand. Two cases of DHF have been reported in Australia, one in 1992 and another in 1997.[53] There is a concern that introduction of other dengue serotypes into northern Australia could increase the risk of dengue haemorrhagic fever.

Dengue occurrence in 1999

There were 131 notifications of dengue in 1999, a rate of 0.7 per 100,000 population. This was a significant reduction on the 1998 rate of 3.1 per 100,000 population. The highest rates were found in the Northern Territory (16.1 per 100,000 population) and Queensland (1.8 per 100,000 population). The male to female ratio was 1.2:1. The highest rate among men was in the 45–49 year age group and in the 25–29 year group for women. Notifications for the year peaked in the summer (first and fourth quarters of the year, Figure 38). While all cases of dengue reported from the Northern Territory were infected overseas, an outbreak of dengue in north Queensland in 1999 accounted for 45 cases or 73 per cent of the State's total.

Arbovirus infections not elsewhere classified (NEC)

In 1999 there were 62 cases of infections with arboviruses 'not elsewhere classified' reported (a rate of 0.3 per 100,000 population). This rate was similar to that found in 1998 (0.5 per 100,000 population). The cases reported in 1999 were predominantly from Victoria and the Northern Territory. The male to female ratio was 0.9:1. The highest rate for women was in the 50–54 year age group and for men in the 45–49 year age group. While not specifically identified in NNDSS, reports from individual States and Territories indicate that there were no reports of infection with Murray Valley encephalitis or Japanese encephalitis from any jurisdiction in 1999.

Figure 36. Notification rate for Ross River virus infections, Australia, 1999, by age and sex

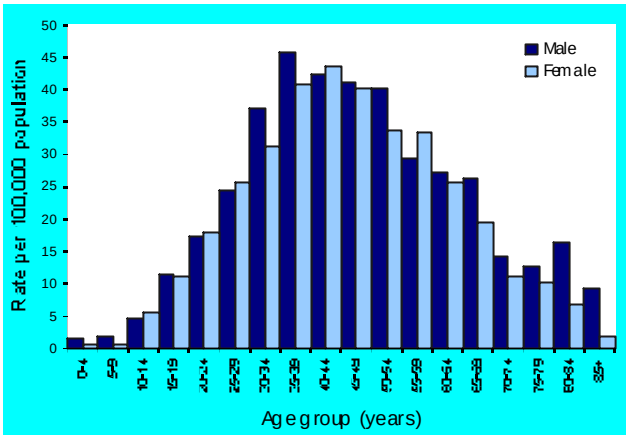


Figure 37. Notifications of Ross River virus infections, Australia, 1991 to 1999, by month of onset

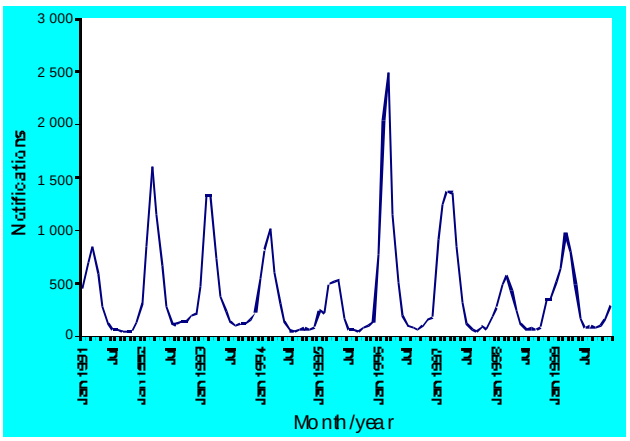
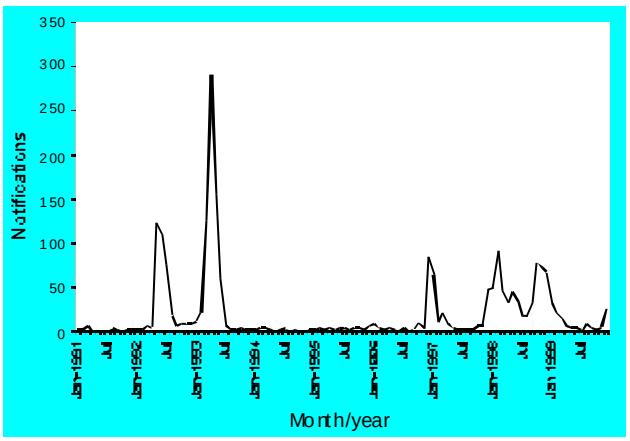


Figure 38. Notifications of dengue fever, Australia, 1991 to 1999, by month of onset



A report of the finding of the mosquito species *Culex gelidus* from the Northern Territory following the first detection of this species in Brisbane in 1999 has potentially important implications.[58] The report noted prolific breeding of this

species in wastewater around dairies, sewerage treatment facilities, abattoirs and piggeries. The mosquito is an important vector and amplification host for the virus causing Japanese encephalitis. Larval surveys are required to delineate the range of this mosquito before control programs are implemented.

Malaria

Australia has been free of endemic malaria since 1983. Sporadic cases are reported primarily among returning travellers in malaria endemic countries such as Indonesia and Papua New Guinea. The three requirements for malaria transmission exist in Australia: infected humans, mosquito vectors and suitable climate. Surveillance of malaria and the rapid entomological response to prevent infection of local *Anopheles* mosquitoes are important public health activities in northern Australia.[59]

In 1999 there were 724 cases of malaria reported to the NNDSS. Overall, the national rates remained stable compared with those reported in 1998 (3.8 per 100,000 population compared with 3.6 per 100,000 population). Among the jurisdictions, the highest rates were reported from the Northern Territory (34.7 per 100,000 population; an increase from 14.2 per 100,000 population in 1998), Queensland (8.7 per 100,000 population) and the Australian Capital Territory (7.0 per 100,000 population). The male to female ratio was 2.6:1. The peak rates were in the 25–29 year age group for men and in the 15–19 age group for women.

Malarial parasites were identified and reported in 588 (81%) of the cases. *Plasmodium vivax* was the most common isolate (358 cases, 61% of the total), followed by *P. falciparum* (193 cases, 26% of cases).

Information on overseas travel in cases of malaria was available from Victoria and the Northern Territory only. In Victoria, 72 per cent of the 81 cases reported had a history of recent travel in Papua New Guinea and/or Indonesia.[35] All Northern Territory malaria notifications were in people who had travelled to malaria endemic countries. In the Northern Territory cases the history of anti-malarial prophylaxis was also recorded. Among the 63 cases reported in 1999, 44 (70%) had not taken any prophylaxis, 4 (6%) had taken some and 15 (24%) had taken full courses of anti-malarial medications (data summarised from reports in *NT Communicable Diseases Bulletin* Vol 6).

Other vectorborne disease surveillance

AQIS exotic mosquito interceptions in 1999

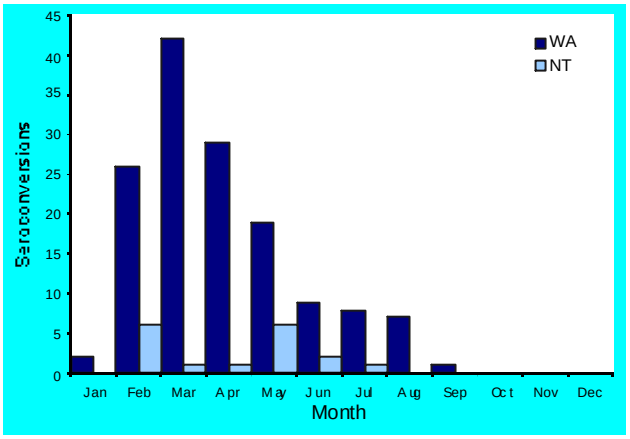
In 1999, the Australian Quarantine Inspection Service (AQIS) reported 30 interceptions of mosquitoes on various imported goods. Seventeen species of mosquitoes were identified, of which 11 species were considered unknown to Australia. These were 8 interceptions of *Culex* spp, one of *Aedes* spp, one of *Coquillettidia* spp and one of *Toxorhynchites* spp. These figures indicate a constant threat of importation of exotic mosquito species, some of which may be vectors for disease.

Sentinel Chicken Surveillance Programme

Sentinel chicken flocks are used to monitor flavivirus activity in Australia. The main viruses of concern are Murray Valley encephalitis (MVE) and Kunjin. In 1999, 26 flocks were maintained in the north of Western Australia, seven in the Northern Territory, 9 in New South Wales and 10 in Victoria. Flocks in Western Australia and the Northern Territory were tested year round and those in New South Wales and Victoria were tested only from November to March during the main risk season. Maps identifying the location of these flocks were published in *Commun Dis Intell* 1999;23:55 and bimonthly reports on seroconversions in sentinel chickens were published in *CDI* throughout 1999.

A summary of seroconversion to MVE virus in Western Australia and the Northern Territory sentinel chicken flocks in 1999 is shown in Figure 39. The peak months of seroconversion are between February and August. There were only small numbers of seroconversions to Kunjin virus in the same flocks. There were no seroconversions to any flavivirus among sentinel chicken flocks in New South Wales or Victoria in 1999.

Figure 39. Seroconversions to Murray Valley encephalitis virus in sentinel chickens, Western Australia and the Northern Territory, 1999



Zoonoses

Zoonoses are diseases of humans acquired from an animal source. Although there are many recognised zoonoses in Australia, only 5 zoonotic infections are reported at the national level. All notifiable zoonoses have epidemic potential and are associated with certain occupations. Zoonotic infection may present with non-specific clinical symptoms and a definitive diagnosis depends on appropriate laboratory investigations.

Brucellosis, leptospirosis and Q fever infections were nationally notifiable in 1999. In New South Wales neither hydatid infection nor ornithosis were notifiable diseases and ornithosis was not notifiable in Queensland. Zoonotic diseases in Australia are not found in all jurisdictions; the Northern Territory has never reported a case of Q fever and has only reported a single case of hydatid (in 1994).

A total of 1,001 notifiable zoonotic infection cases were received by NNDSS in 1999, which accounted for 1.1 per cent of all the notifications. Most notifiable zoonotic infections were reported from Queensland (569, 57%) and New South Wales (222, 22%). Queensland had the highest notification rates for Q fever (8.5 per 100,000 population), leptospirosis (6.2 per 100,000 population) and brucellosis

(1.4 per 100,000 population), and Victoria had for the highest notification rates for ornithosis (1.4 per 100,000 population) and hydatid infection (0.4 per 100,000 population).

Brucellosis

Brucella are small aerobic gram-negative bacilli. Human brucellosis is caused by any of 4 species: *Brucella melitensis* (primarily from goats, sheep, and camels), *Brucella abortus* (from cattle), *Brucella suis* (from pigs) and *Brucella canis* (from dogs).

Brucellosis is transmitted from *Brucella*-infected animals to humans by direct contact with blood, tissues and urine of infected animals. Infection is through breaks in the skin or through consumption of contaminated animal products, such as milk and meat. Airborne transmission from animal to humans is also possible. Zoonotic organisms may be transmitted from human to human via blood transfusion and bone marrow transplantation, through the placenta, during breast-feeding, and during sexual activity.

Brucellosis is still a significant public health problem in some geographical areas in Australia. There were 52 notifications of brucellosis in 1999, giving a notification rate of 0.3 per 100,000 population which was an increase from 1998 (43 cases; 0.2 per 100,000 population). The national notification rate has been stable since 1991.

Most notifications (33, 63.4%) occurred between August and November. The majority of the brucellosis cases were males (48/52, 92.3%), with the overall male to female ratio of 12:1 reflecting occupational risk. The age-specific rates peaked in the 25–29 years male age group at 1.3 per 100,000 population.

Queensland reported 94.2 per cent (49/52) of all cases, with the other 3 cases from Victoria. The highest rates of disease were reported in the Central West (97.9 per 100,000 population) and the South West (31.1 per 100,000 population) Statistical Divisions of Queensland. A study has shown a high frequency of *B. suis* infections in Queensland, especially among men who hunt and slaughter feral pigs.[60]

Hydatid disease

Hydatid disease is caused by the larval stage of the tapeworm *Echinococcus granulosus*. Hydatid cysts are prevalent in areas where livestock is raised in association with dogs. In 1999, hydatid infection was notifiable for all States and Territories in Australia, except New South Wales.

There were a total of 29 hydatid disease notifications in 1999, the lowest since 1991; giving an annual notification rate of 0.2 per 100,000 population (Table 2). Notifications of hydatid infection were from Victoria (17 cases, 59% of the national total), Queensland (5 cases), Western Australia (3 cases), South Australia (3 cases) and Tasmania (1 case). The highest rates of disease were reported in the South West Statistical Division of Queensland (3./100,000 population), followed by the Midland Statistical Division of Western Australia (1.9 per 100,000 population).

Of the 29 notifications, 13 were in men, 14 were in women and two were in persons of unknown gender. The male to female ratio of disease was 0.9:1. Hydatid infections commonly occurred in the 45–64 year age range. The highest age-specific rates were in women aged 60–64 years (1.1 per 100,000 population) and in men aged 45–49 years (0.6 per 100,000 population).

Hydatid disease is distributed widely in rural Australia. In urban dwellers it is more common among the overseas born who would probably have acquired the infection overseas.[61] Disease in the Australian born occurs typically in rural settings where humans become infected by the ingestion of eggs passed in the faeces of dogs, dingoes or foxes. Wallabies, wombats, feral pigs, sheep and kangaroos are all intermediate hosts that act as reservoirs of the disease. Dogs and foxes, infected by feeding off the offal or other remains of these animals, can carry the disease into rural communities, or to the periphery of urban settlements.[62] Because the symptoms of hydatid disease usually occur only in the advanced stages of disease, and the infection may remain quiescent for many years, hydatid disease is thought to be under-reported in Australia.[61]

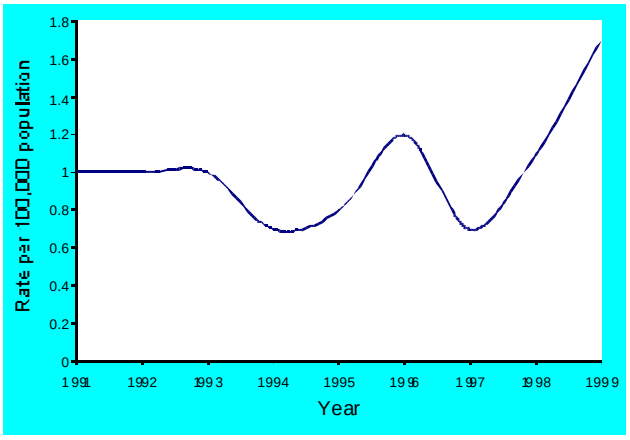
Leptospirosis

Leptospirosis is a zoonotic disease transmitted by wild and domestic animals caused by spirochetes of the *Leptospira* genus. The source of infection is soil or water contaminated with the urine of domestic or wild animals. Farmers, veterinarians and abattoir workers are at an increased risk of infection. Infections may be asymptomatic and the clinical manifestations of the disease are highly variable including fever, myalgia, meningitis, rash, haemolytic anaemia, and jaundice.[29] Leptospirosis occurs in all parts of Australia with the highest incidence in Queensland, where a laboratory based notification system has been in place since 1988.

There were 318 notifications of leptospirosis in Australia in 1999, a 68 per cent increase in notifications compared with 1998. The notification rate rose from 1.1 per 100,000 population in 1998 to 1.7 per 100,000 population in 1999 (Figure 40). The increased notification was mainly caused by an outbreak in January in Queensland, which consisted of 184 cases, half of whom required hospitalisation. This followed a period of heavy rainfall and flooding, and an increase in rodent population. At least 14 different serovars causing disease were isolated in this outbreak.[63]

In 1999, Queensland reported 69 per cent of all notifications (218 cases), 57 (18%) cases were from New South Wales and 29 (9%) cases were from Victoria. The highest rates of disease were localised to the Far North (19.2 per 100,000

Figure 40. Trends in national notification rate for leptospirosis, Australia, 1991 to 1999



population) and the South West (11.6 per 100,000 population) of Queensland, and the Western District (10.1 per 100,000 population) of Victoria.

The seasonal trend showed notifications were higher in summer months and reached a peak in March and April (Figure 41). Ninety-one per cent of all notifications were male with a male to female ratio of 10:1. The most frequent age for disease onset was from 20–59 years.

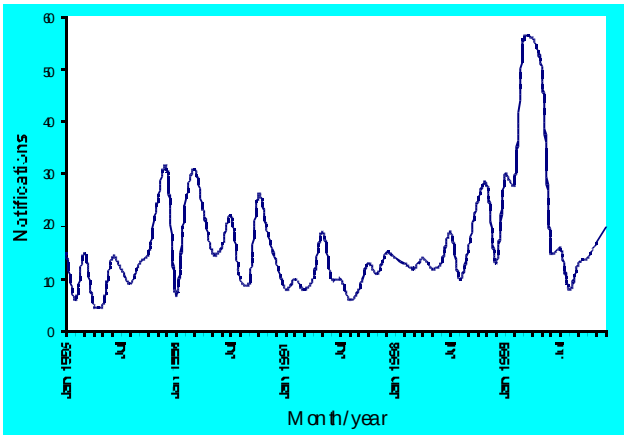
Ornithosis

Ornithosis, also known as psittacosis, is an acute generalised infection with *Chlamydia psittaci* and is commonly associated with exposure to pet birds, particularly parrots.

Eighty-four notifications of ornithosis were reported in 1999, an increase compared with 1998 (64 cases). The national notification rate was 0.9 per 100,000 population. In 1999 ornithosis was not a notifiable diseases in New South Wales or Queensland. Of the 84 cases reported in this year, 66 (79%) were from Victoria, 49 (58.3%) were male and 35 (41.7%) female, and the male to female ratio of disease was 1.4:1. The highest age-specific rates were reported in the 45–49 years age group for women (1.2 per 100,000 population) and in the 50–54 years age group for men (1.8 per 100,000 population).

Reported rates of ornithosis are highest in the older age groups, which may reflect increased investigation, and laboratory testing for atypical community acquired pneumonia in this group. Previously reported outbreaks have been associated with aviaries, pet shops or poultry processing plants, although an outbreak investigation in rural Victoria in 1995 showed no association with direct bird handling but rather lawn mowing and gardening in areas with high numbers of native birds.[64] Shedding of *C. psittaci* into the environment by sick birds and subsequent inhalation of aerosolised dust and bird excreta was postulated as the mechanism of human infection.

Figure 41. Notifications of leptospirosis, Australia, 1991 to 1999, by month of onset



Q fever

Q fever is a rickettsial illness caused by *Coxiella burnetii*. Livestock, such as sheep, cattle, goats, cats, dogs, some wild animals (bandicoots and many species of feral rodents), birds and ticks are natural reservoirs. Outbreaks have occurred in occupational groups working with animals, including stockyard workers, meat packing and rendering workers, abattoir and dairy workers, and medical and veterinary research facility workers.

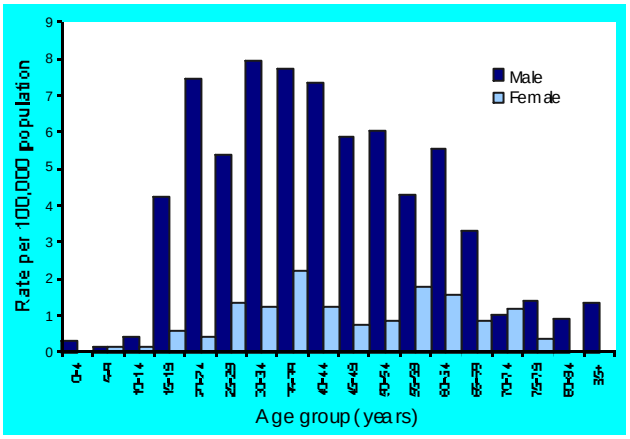
Transmission is usually through airborne dissemination of the organism in dust particles but also via direct contact with contaminated material, ingestion of contaminated placentas or ingestion of milk. Ticks may also be involved in transmission of the organism. Cases have occurred in individuals with no direct contact with contaminated animals and their bodily fluids, but such cases have resided downwind from areas that are contaminated.

In 1999, 518 notifications of Q fever were reported with an annual notification rate of 2.7 per 100,000 population, a slight decrease from 3.0 per 100,000 population reported in 1998. Queensland and New South Wales each accounted for 57 per cent and 32 per cent of all the cases for the year, respectively. The highest notification rates for Statistical Divisions were localised to the South West (155.6 per 100,000 population) and the Central West (130.6 per 100,000 population) of Queensland (Map 11).

The highest age-specific notification rates were in the 35–39 year age groups for males (9.5 per 100,000 population) and in the 40–44 year age groups for women (2.8 per 100,000 population, Figure 42). Males accounted for 79 per cent of all the notifications, and male to female ratio was 3.8:1.

Q fever is still the most important of all zoonotic diseases in terms of reported numbers of cases in Australia. The true prevalence of the disease is likely to be under-estimated. A recent study found that 27 per cent of Australian abattoir staff tested positive for Q fever infection.[65] An effective vaccine is available in Australia for people who are at high-risk.[66]

Figure 42. Notifications of Q fever, Australia, 1999, by age and sex

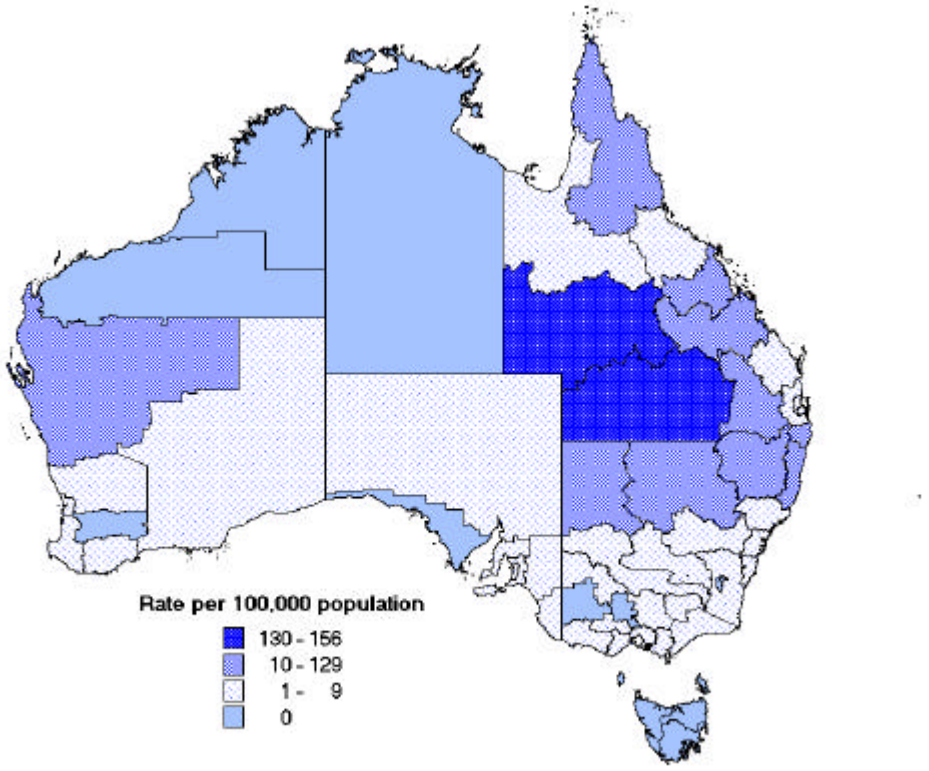


Other bacterial infections

Legionellosis

Legionellosis is an acute bacterial infection with two clinical manifestations: Legionnaire's disease associated with pneumonia and Pontiac fever, which is generally self-limiting. Legionellosis describes a group of diseases caused

Map 11. Q fever notifications by Statistical Division of residence



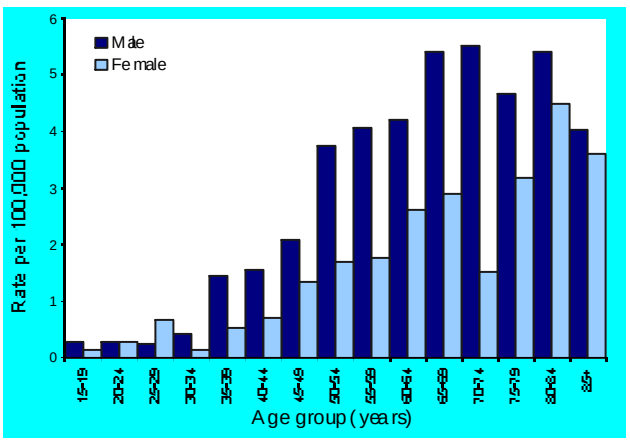
by various species of *Legionella* as well as the pneumonia of classical Legionnaire's disease caused by *Legionella pneumophila*.

L. pneumophila occurs in water sources with a wide range of temperatures, pH and dissolved oxygen contents. Despite chlorination, the bacteria proliferate in cooling towers and water systems depending on favourable temperatures, sediment accumulation, and commensal microflora. Inhalation of aerosols generated by air-conditioning, nebulisers, humidifiers and showerheads is the major mode of transmission. Age, chronic lung disease, immunosuppression and cigarette smoking have been identified as important risk factors for legionellosis.[67] *L. longbeachae* has been recognised for some years as a frequent cause of *Legionella* pneumonia in Australia.[68,69] *L. longbeachae* has been isolated from a large proportion of potting mixtures in Australia, suggesting this route of exposure may be important in the epidemiology of sporadic legionellosis in Australia. [70]

Legionellosis is notifiable in all States and Territories of Australia, and includes notifications of infections caused by all *Legionella* species. There were 249 notifications of legionellosis in 1999 resulting in a notification rate of 1.3 per 100,000 population compared with 1.4/100,000 population in 1998. The rates were highest in South Australia (4.2 per 100,000 population), Western Australia (2.3 per 100,000 population) and the Northern Territory (2.1 per 100,000 population, Table 2). Men accounted for 63 per cent of reported cases giving a male to female ratio of 1.7:1. Cases were recorded in age groups from 15 to 85 with a peak in the 50–54 year age group. Persons aged more than 60 years made up 47 per cent of all cases (Figure 43).

Most cases of legionellosis were sporadic, though Victoria reported 2 small clusters, one associated with a private spa and the other with contaminated cooling towers[35] (Kirk, 2000). Increased reporting of legionellosis in recent years may reflect the easier diagnosis using the urinary antigen test.[71] Data on the isolated species were available for 132 of the cases. Of these 113 (86%) were identified as *L. longbeachae* and 19 (14%) as *L. pneumophila. L. Pneumophila* isolates were only identified in Queensland (14) and South Australia (5). New South Wales only reported *L. Longbeachae. Legionella* species information was not available from other jurisdictions.

Figure 43. Legionellosis notification rate, Australia, 1999, by age and sex



Leprosy

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

There were 6 cases of leprosy notified nationally in 1999 compared with only 3 cases in 1998. Three of the 1999 cases occurred in Western Australia with one each in New South Wales, Queensland and Victoria. Of the 6 cases, three were male and three were female. The age range was 15–39 years.

Invasive meningococcal disease

Neisseria meningitidis is the cause of outbreaks of meningitis worldwide accounting for at least 500,000 cases and 50,000 deaths per annum. A pandemic in the sub-Saharan African 'meningitis belt', which began in 1996, has resulted in at least 300,000 cases to date and many thousands of deaths. An on-going epidemic of meningococcal disease in New Zealand since 1990 peaked at 13.3 cases per 100,000 population in 1997 (Martin, 2001, Communicable Disease Control Conference, April 2001, Abstract 2).

In Australia, there were 568 notifications of meningococcal disease nationally in 1999; a rate of 3.0 per 100,000 population compared with 2.4 per 100,000 population in 1998. Of the total, 365 (64%) cases were culture-confirmed. Of these 212 (58%) were serogroup B, 143 (39%) were serogroup C, 5 (1.5%) were serogroup W135 and 5 (1.5%) were serogroup Y. A pattern of seasonal variation in meningococcal infection notifications continued, with the greatest number of cases occurring in late winter or early spring (Figure 44). The distribution of notifications by age shows the highest peak in children aged 0–4 years and an additional peak in the 15–24 year age group. Overall the male to female ratio was 1.3:1 (Figure 45).

The Australian Meningococcal Surveillance Programme report for 1999[72] reported the phenotype and antibiotic susceptibility of *Neisseria meningitidis* from invasive cases of meningococcal disease. Of the 368 isolates, 90 per cent of the isolates were either serogroup B or C. Serogroup B predominated in all States and Territories. Serogroup C isolates increased in Victoria and phenotype C-2a:P1.2 was the most frequently isolated phenotype. This phenotype was isolated infrequently before 1999. Another new Australian serogroup C phenotype C:2aP1.4(7) was isolated in New South Wales and Victoria. About three-quarters of all isolates showed decreased susceptibility to penicillin and three had reduced susceptibility to rifampicin. Case fatality rates were 9.4 per cent of culture-positive cases, with a higher mortality noted among cases with serogroup C disease.

Enhanced surveillance for invasive meningococcal disease commenced in Queensland in 1999.[73] The Queensland model includes probable cases for the first time, defined as a petechial or purpuric rash, isolation of *N. meningitidis* from a throat swab or an epidemiological link to a confirmed case. Enhanced surveillance has demonstrated a need to promote the use of parenteral antibiotics by GPs on suspicion of meningococcal disease and a need to encourage more timely reporting of cases to health authorities.

Figure 44. Notifications of invasive meningococcal disease, Australia, 1991 to 1999, by month of onset

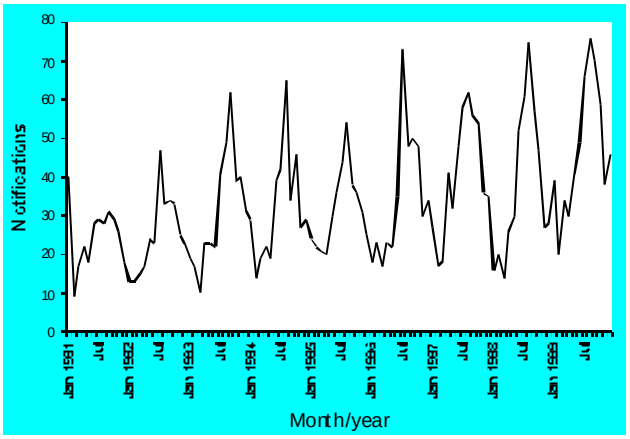
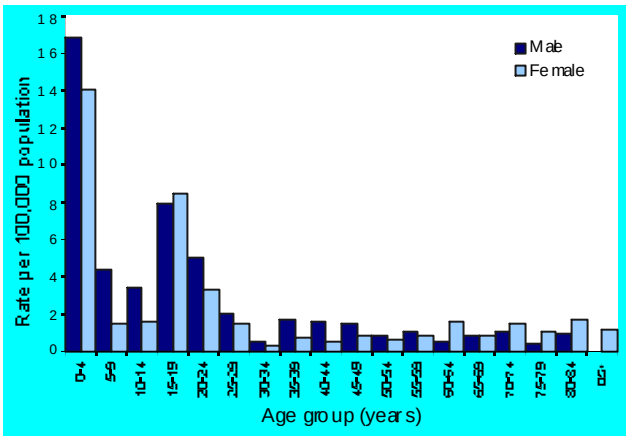


Figure 45. Notification rate for invasive meningococcal disease, Australia, 1999, by age and sex



Victoria reported a marked increase in invasive meningococcal disease in 1999, with a doubling in the notification rate in the 15–19 year age group. There was a large increase in the proportion of cases caused by serogroup C from 13 per cent in 1998 to 31 per cent in 1999.[35] Serogroup B remained the dominant serogroup in children aged 0–4 years in all jurisdictions.

Tuberculosis

There are three national surveillance systems through which tuberculosis (TB) notifications are handled. The NNDSS provides the timeliest information on national TB notifications, but consists mainly of demographic information. The National Mycobacterial Surveillance System (NMSS), a surveillance system dedicated to tuberculosis and atypical mycobacterial infections, produces an annual report on TB notifications[74] with detailed information on risk factors, diagnostic methods, drug therapy and relapse status. The Australian Mycobacterial Reference Laboratory Network (MRLN) maintains national data on drug susceptibility profiles, site of disease, age, sex and laboratory method of diagnosis for all mycobacterial isolates. These data are published annually in conjunction with the NMSS surveillance report.[75]

In 1999, 1,153 TB notifications were reported nationally and the reporting rate was 6.1 per 100,000 population. This is consistent with rates since 1994. The highest rate was in the Northern Territory (51.8/100,000); this was inflated by a large number of cases diagnosed in East Timorese refugees in Darwin in September 1999 (see 1999 TB report following). There was little difference in notification rates between males and females with males accounting for just over 50 per cent of notifications. Some increases in jurisdictions such as Victoria were due to cases detected among refugees in Australian 'Safe Havens' program during the Kosovo and East Timor crises. Data from Victoria showed that 98 per cent of TB notifications were in people born outside Australia and that 35 per cent of cases were born in South East Asia. The 1999 TB report indicates that there are very low rates of tuberculosis in the non-indigenous Australian born population and that this rate is continuing to decline.

Other communicable disease surveillance

LabVISE

The Laboratory Virology and Serology (LabVISE) Reporting Scheme is a passive surveillance scheme based on voluntary reports of infectious agents contributed by sentinel virology and serology laboratories around Australia, to the Commonwealth Department of Health and Aged Care. In 1999, reports from the scheme were analysed and published monthly 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 persons they infect. LabVISE records information on some infectious agents that are not reported by other surveillance schemes. The scheme currently holds over 500,000 records collected since 1982.

In 1999, there were 26,452 reports to LabVISE, contributed by 15 laboratories representing every State and Territory. This was a small increase on the number of reports in 1998 (26,359). Although there were no contributing laboratories contributing directly to LabVISE in either the Northern Territory or the Australian Capital Territory, samples from these jurisdictions were included in the reports from reference laboratories (Table 9). LabVISE reporting is not equally distributed across Australia; indeed the Northern Territory has the highest number of reports per 100,000 population while some southern States are relatively poorly represented in the data set.

The breakdown of reports is shown in Table 9. Of the 26,452 reports received, 19,531 (74%) were of viral infections and 6,921 (26%) were bacterial, spirochaetes, fungal, protozoan or helminthic infections. Among reports of viral infection, ortho/paramyxoviruses (including influenza A and B, para-influenza and RSV viruses) made up 32 per cent of reports, and reports of Herpes viruses (including Herpes, CMV, Varicella-zoster and Epstein-Barr virus) constituted 26 per cent (Figure 46). Among reports of non-viral infections, Chlamydia made up nearly half of all reports (49%).

In 1999/2000, an evaluation highlighted a number of weaknesses of the LabVISE scheme, which prevent the optimal utilisation of the collected data. These were the lack of clear objectives, the inability to collect population-based data and the reduction in the number of participating laboratories.

Advances in technology and improvements to laboratory systems have made the prospect of data acquisition direct from laboratory achievable. The use of such technology would facilitate reporting procedures for laboratories, improve the quality and timeliness of data and enhance the capacity of the scheme to collect additional data. Improved analysis and dissemination of the information generated by LabVISE would further enhance the scheme. The evaluation recommended three options of which the Public Health Laboratory Network (PHLN) endorsed the following: 'that LabVISE be retained and developed as a broad based surveillance scheme with clear objectives and that a feasibility study be performed to assess additional uses of laboratory generated data and the possibility of real time transfer of these data direct to public health units and the Commonwealth Department of Health and Aged Care. Such a feasibility study should also examine safeguards for confidentiality and what additional resources may be required for implementation.' These developments to LabVISE will commence in 2002.

Additional reports related to pathogens under surveillance by LabVISE

The Rotavirus Surveillance Programme 1999-2000[76]

Rotavirus surveillance began in July 1999 with the formation of the National Rotavirus Reference Centre, a collaborative laboratory-based initiative. Between June 1999 and May 2000, 1,126 rotavirus specimens from children hospitalised with acute diarrhoea were typed. The common serotypes G1-G4 were represented with serotype G1 being the most common isolate from the whole country. The program reported the first isolates of serotype G9, not previously found in Australia and accounting for 10 per cent of typable strains. The emergence of this new serotype has implications for the rotavirus vaccination strategy, which targets serotypes G1-G4.

Norwalk-like virus outbreaks in 1999[77]

Three outbreaks of gastroenteritis in nursing homes in Brisbane in 1999 were demonstrated to be associated with the presence of Norwalk-like virus (NLV). These findings have implications for infection control procedures, particularly in institutional settings. This, and other reports based on PCR diagnosis, of NLV incidents in Australia and overseas, demonstrate that NLV represents a significant but previously unrecognised cause of gastroenteritis.

Figure 46. LabVISE reports, 1999 (total)

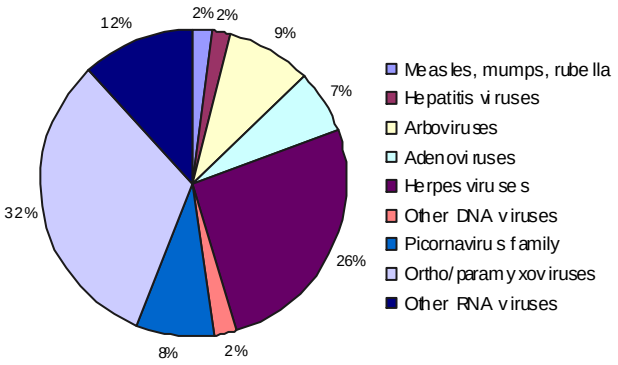


Table 9. Infectious agents reported to LabVISE, 1999

Organism Type	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Measles virus	0	4	2	3	9	1	117	36	172
Mumps virus	0	0	0	0	2	0	6	50	58
Rubella	1	7	0	100	9	2	11	15	145
Hepatitis A virus	0	9	30	101	43	2	10	180	375
Hepatitis D virus	0	0	0	3	4	0	0	1	8
Hepatitis E virus	0	0	0	1	0	0	0	0	1
Ross River virus	0	42	65	875	28	3	44	366	1,423
Barmah Forest virus	1	8	9	123	0	0	4	35	180
Dengue virus	1	1	13	3	1	1	0	68	88
MVE virus	0	0	0	0	0	0	0	2	2
JE virus	0	0	0	0	0	0	0	1	1
Kunjin virus	0	0	1	0	0	0	0	4	5
Flaviviruses (unspecified)	0	0	3	22	0	0	2	0	27
Adenoviruses	10	213	7	45	217	6	328	479	1,305
Herpes viruses	15	425	52	1,671	1,006	17	902	997	5,085
Other DNA viruses	3	3	1	59	26	14	201	167	474
Picornavirus family	17	590	36	25	47	2	168	745	1,630
Ortho/paramyxoviruses	108	1,570	24	596	481	38	1,494	1,921	6,232
Other RNA viruses	56	895	6	4	204	32	503	620	2,320
Chlamydia trachomatis	61	373	205	1,226	326	17	101	981	3,290
Chlamydia pneumoniae	0	0	0	0	0	0	0	2	2
Chlamydia psittaci	0	0	0	0	0	0	68	10	78
Chlamydia spp	0	8	0	12	0	0	1	0	21
Mycoplasma spp	0	94	6	364	82	3	486	95	1,130
Coxiella burnetti	6	18	1	144	2	0	27	23	221
Rickettsia spp	0	0	0	0	0	1	3	14	18
Streptococcus group A	0	15	69	280	1	0	3	0	368
Brucella species	0	2	0	7	0	0	2	0	11
Bordetella pertussis	1	30	1	443	1	2	329	38	845
Legionella pneumophila	0	6	0	0	3	0	2	6	17
Legionella longbeachae	0	1	0	0	12	0	0	38	51
Yersinia entercolitica	1	8	0	1	0	0	0	0	10
Fungi	0	9	0	0	0	0	0	0	9
Leptospira spp	0	3	0	40	0	0	1	12	56
Treponema pallidium	1	49	431	280	0	0	1	12	774
Protozoa	1	3	0	2	1	1	2	6	16
Echinococcus granulosus	0	0	0	0	0	0	0	4	4
Total	283	4,386	962	6,430	2,505	142	4,816	6,929	26,452

Enterovirus 71 outbreak in Western Australia

A report on an outbreak of hand, foot and mouth disease caused by enterovirus 71 in Western Australia in 1999 associated with severe neurological disease, was recently published.[78] Fourteen children with enterovirus 71 were identified, of whom four developed long-term neurological sequelae. Several large epidemics of enterovirus 71 infection in young children have occurred in South East Asia, including a large outbreak of 129,106 cases of hand, foot and mouth disease in Taiwan in 1998.[79]

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 on a number of conditions each week. The aim of ASPREN is to provide an indicator of the burden of disease in the primary care setting and to detect trends in consultation rates.

There were approximately 120 general practitioners participating in the scheme from all States and Territories in

1999. Approximately 75 per cent of these were located in metropolitan areas and the remainder were in rural areas. Between 7,000 and 8,000 consultations were recorded each week.

In 1999, 7 conditions related to communicable diseases and environmental health were reported. These were influenza, rubella, measles, chickenpox, and gastroenteritis. Case definitions for these conditions were published in *Commun Dis Intell* 1999;24:7-8. In total there were 323,417 consultations in the sentinel practices reported to ASPREN of which 5,938 were of communicable diseases. The majority of communicable diseases reported were gastroenteritis (3,227 presentations, 47% of the total) and influenza (2,106 presentations, 31%, Figure 47). The weekly reporting of gastroenteritis and influenza as a rate per 1,000 consultations is shown in Figures 48 and 49 respectively. While presentations with symptoms of gastroenteritis were highest in the warmer months (weeks 40 to 52), influenza-like illnesses peaked in the winter months (week 34).

National Influenza Surveillance

This summary is based on the 'Annual report of the National Influenza Surveillance Scheme 1999'.[41]

Influenza surveillance is based on 3 systems: laboratory diagnosis, including virus isolation and serology by laboratories participating in LabVISE; consultation rates for clinically diagnosed influenza illness by sentinel general practitioners; and the absenteeism data of workers from a national employer.

A total of 3,247 reports were received by LabVISE, with 2,681 for influenza A and 386 for influenza B. The ratio of influenza A to influenza B was 7.4:1. Total influenza reports showed a baseline until May, a small peak in June and a second higher and broader peak from late June to early September. The greatest number of reports were recorded in the 15–44 year age group and the male to female ratio approached 1:1. The expected predominance of influenza reports amongst the elderly was not seen in the collected surveillance data. The ASPREN and New South Wales sentinel general practitioner schemes showed a peak of GP attendances for influenza-like illnesses from mid-May to September 1999. Comparison of ASPREN and LabVISE data showed a similar pattern, with the trends in ASPREN data followed about 2 weeks later by similar patterns in LabVISE data. Absenteeism surveillance showed the highest levels in August and September for absences of more than 3 days.

Independent influenza surveillance programs showed an earlier peak on the east coast of Australia than in the west, with an overall ratio of influenza A to B of 5:1. Most isolates were A Sydney 5/97 H3N2-like viruses. The WHO Collaborating Centre for Reference and Research on Influenza performed analysis on 813 isolates in 1999. This represented 25 per cent of the total influenza reports received through LabVISE. Of these, 683 were influenza A and 130 influenza B. The majority of the influenza A strains were H3N2 subtype, closely related to the A/Sydney/5/97 vaccine strain. Approximately 20 per cent reacted more strongly with a recent isolate A/Moscow/10/99. However, there was no evidence of substantial antigenic drift among the influenza A (H3N2) isolates. The three H1N1 isolates found showed significant antigenic changes from the vaccine A/Beijing/262/95 and were closely related to a new variant A/New Caledonia/20/99. Influenza B isolates remained closely related to the vaccine reference strain B/Beijing/184/93. The pattern of influenza in Australia was similar to that seen in most parts of the world in 1999. The level of influenza was lower than many regions in which more severe outbreaks occurred, such as New Caledonia and New Zealand.

Figure 47. ASPREN communicable disease surveillance presentations to GPs, 1999

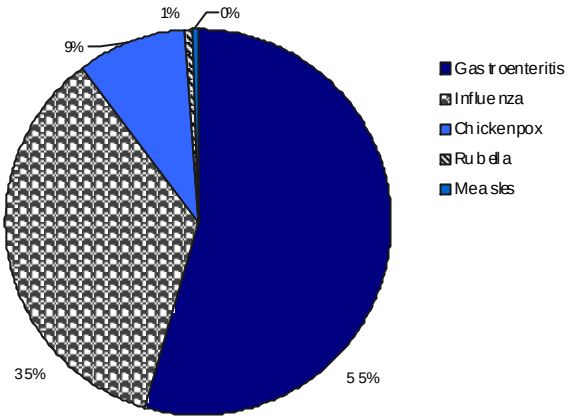


Figure 48. ASPREN consultations for gastroenteritis, 1999

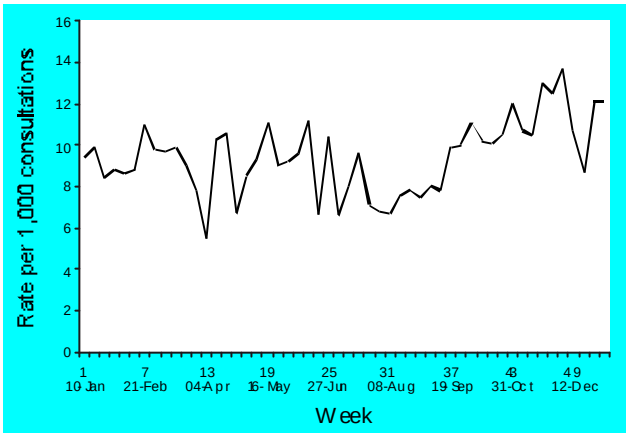
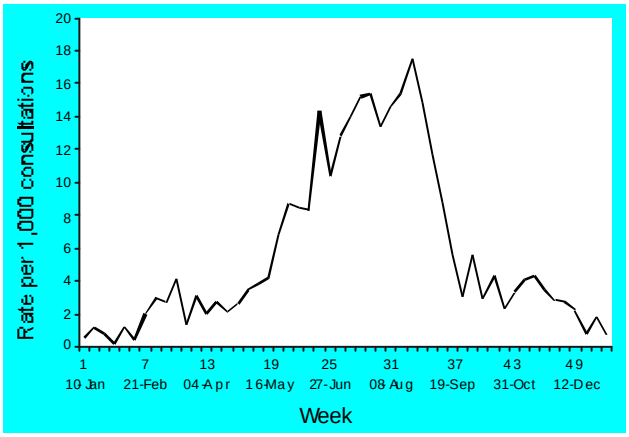


Figure 49. ASPREN presentations of influenza-like illness, 1999



Antibiotic resistance in Australia

In 1999, the major event in this field was the publication of the report of the Joint Expert Technical Advisory Committee on Antibiotic Resistance.[80] This committee was appointed in April 1998 to examine the issue of the use of antibiotics in food-producing animals and the implications for antibiotic resistant bacteria in humans. The committee was charged with developing evidence-based recommendations for the appropriate future management of antibiotic use in food-producing animals.

The following are key extracts from the JETACAR report.

Scientific background

JETACAR reviewed internationally available information on the nature of antibiotics, and the molecular basis of bacterial resistance. The bacteria known to be involved in the transfer from food-producing animals, and those recognised as the cause of medical conditions of most concern in relation to treatment failure due to antibiotic resistance, were also identified. The report identified priority medical problems potentially arising from, or exacerbated by, the use of antibiotics in livestock production. The benefits of antibiotic use in animals were also reviewed and alternatives canvassed. Focusing on Australian information, the committee reviewed current regulatory controls and use patterns of antibiotics in humans and animals and antibiotic resistance patterns in humans. Few data on antibiotic resistance were available for animal isolates in Australia. There were many gaps in the data available and in the current scientific knowledge of the mechanisms involved.

Assessment of evidence

JETACAR attempted to provide an evidenced-based hazard characterisation for antibiotic use in food-producing animals, a framework for the future development of risk assessment methodology for individual drugs, and the basis for the development of an integrated antibiotic-resistance management strategy.

Overall conclusion

JETACAR considered all aspects of the occurrence of antibiotic resistance and its importance in human and veterinary medicine. The committee agreed there was evidence for the emergence of resistant bacteria in humans and animals following antibiotic use, the spread of resistant animal bacteria to humans, the transfer of antibiotic resistance genes from animal bacteria to human pathogens, and resistant strains of animal bacteria causing human disease.

Resistance management program and recommendations

Based on the scientific findings outlined above, and the 4 factors that influence emergence and spread of antibiotic-resistant bacteria (antibiotic load, antibiotic regimen, bacterial load and prevalence of resistant bacteria), JETACAR developed an antibiotic resistance management program that focuses simultaneously on human and animal use of antibiotics in Australia. The proposed program is a co-ordinated multidisciplinary approach with 5 key elements, as follows: regulatory controls; monitoring and surveillance; infection prevention strategies and hygienic measures; education; and further research. The basics of this 'five point plan' are equally applicable to human and veterinary medicine, as well as other areas of antibiotic use. All 5 elements of the program must be implemented together if there is to be any chance of reversing the trend towards increasing antibiotic resistance. In addition, further recommendations are included on communication of the issues surrounding antibiotic resistance management to stakeholders and the general public. The overall coordination of the strategy is covered in recommendations 20 and 21. Finally a recommendation was made that a working group convened by the DHAC develop a fully coordinated resistance management plan for human antibiotics. The plan so developed should be incorporated into the recommended functions of the Working Party on Antibiotics or its successor.

CJD in Australia 1999

This summary is based on report from The Australian National CJD Registry, The University of Melbourne – update to January 2000.

The Australian National Creutzfeldt-Jakob Disease Registry was established in 1993 in response to 4 CJD deaths attributed to cadaveric-derived human pituitary hormone treatment for infertility or short stature. The work of the Registry was expanded to include monitoring both health-care acquired CJD and transmissible spongiform encephalopathies (TSE), both sporadic and familial, in Australia.

Australia is free of animal forms of prion disease, such as bovine spongiform encephalopathy (BSE) and scrapie. At the end of 1999 there were 482 cases on the Registry. These were 208 definite cases, 144 probable cases and 114 incomplete cases (cases positive in an immunoassay but not finally classified). While there has been a doubling of the average incidence to one case per million (1988–1999) compared with 1970–1987, this reflects better case ascertainment due to improved recognition, confirmation and reporting. The composition of cases on the Registry is 91.9 per cent sporadic, 5.7 per cent familial and 2.4 per cent iatrogenic.

Appendices

Appendix 1a. Case definitions and ICD-10 code for notifiable diseases reported to NNDSS in 1999, gastrointestinal diseases

Disease	Case definition (NHMRC 1994)	ICD-10 code(s)
Botulism	A clinically compatible illness (diplopia, blurred vision, muscle weakness, paralysis or bulbar palsy) with a history of exposure to a probable food source in the absence of a contaminated wound AND one of the following: isolation of *Clostridium botulinum* from faeces or other clinical specimens OR detection of *C. botulinum* toxin in serum, faeces or probable food source OR epidemiological linkage to other cases of confirmed foodborne botulism	AO5.1
Campylobacteriosis	Isolation of *Campylobacter* species from a clinical specimen	AO4.5
Haemolytic uraemic syndrome (HUS)*	Acute microangiopathic anaemia on peripheral blood smear AND acute renal impairment AND/OR thrombocytopaenia	D59.3
Hepatitis A	Anti-HAV IgM positive in the absence of recent vaccination OR demonstration of a clinical case of hepatitis (jaundice and/or elevated aminotransferase levels) without a non-infectious cause AND epidemiologically linked to a serologically confirmed case	B15
Hepatitis E*	A person who demonstrates anti-HEV IgM in sera collected less than 4 weeks after onset of acute hepatitis OR IgG seroconversion in paired sera OR HEV identified by nucleic acid test OR HEV identified by EM on stool OR a hepatitis-like illness in the absence of other causes of hepatitis and detection of antibodies to HEV	B17.2
Listeriosis	Isolation of *Listeria monocytogenes* from a site which is normally sterile, including foetal gastrointestinal contents	A32
Salmonellosis	Isolation of *Salmonella* species (excluding *S. typhi*) from any clinical specimen	AO2
Shigellosis	Isolation of *Shigella* species from any clinical specimen	AO3
SLTEC, VTEC*	A person with bloody diarrhoea or HUS from whom, in a clinical specimen: Shiga-toxin producing *E. coli* (SLTEC) are isolated OR isolation of Shiga-toxin from an *E. coli* isolate OR identification of the gene associated with the production of Shiga-toxin in *E. coli*	A4.1, A4.4
Typhoid	Isolation of *Salmonella typhi* or *S. parathphi* serotype A, B, or C from any clinical specimen	AO1.0
Yersiniosis	Isolation of *Yersinia enterocolitica* or *Y. pseudotuberculosis* from blood or faeces OR detection of circulating antigen by ELISA or agglutination test OR positive *Yersinia* serology in the presence of clinical compatible illness	AO4.6

All definitions from Surveillance Case Definitions, National Health and Medical Research Council, March 1994, except those marked * which are draft summary definitions from the Communicable Diseases Network Australia (January 2001). Some Australian States and Territories have their own case definitions for some diseases, which may vary from those shown here.

Appendix 1b. Case definitions and ICD-10 code for notifiable diseases reported to NNDSS in 1999, bloodborne diseases

Disease	Case definition (NHMRC 1994)	ICD-10 code(s)
Hepatitis B (incident)	Demonstration of documented seroconversion to HBV	B16
Hepatitis B (unspecified)	HBsAg positive AND *either*: anti-HBcIgM positive OR demonstration of a clinical illness consistent with acute viral hepatitis (jaundice, elevated aminotransferases)	B18.0, B18.1
Hepatitis C (incident)	Demonstration of documented seroconversion to hepatitis C	B17.1
Hepatitis C (unspecified)	Demonstration of anti-hepatitis C positive or hepatitis C PCR positive AND a clinical illness consistent with acute viral hepatitis AND is not an acute case of hepatitis A, B, or D.	B18.2
Hepatitis D*	Positive for anti-HDV or HDV Ag or seroconversion or rise in IgG in serum or liver AND HBSAG OR anti-HBc negative	B17.0, B16.1, B18.0
Hepatitis (NEC)	Any other viral hepatitis not classified here	B17.8

All definitions from Surveillance Case Definitions, National Health and Medical Research Council, March 1994, except those marked * which are draft summary definitions from the Communicable Diseases Network Australia (January 2001). Some Australian States and Territories have their own case definitions for some diseases, which may vary from those shown here.

Appendix 1c. Case definitions and ICD-10 code for notifiable diseases reported to NNDSS in 1999, quarantinable diseases

Disease	Case definition (NHMRC 1994)	ICD-10 code(s)
Cholera	An illness characterised by diarrhoea and/or vomiting AND isolation of toxigenic *Vibrio cholerae* serogroup O1 or O139 from a clinical sample	AO0
Plague	A four-fold or greater change in serum antibody titre for *Yersinia pestis* OR isolation of *Yersinia pestis* from a clinical specimen	A20
Rabies	Clinically compatible neurological illness AND either detection of rabies viral antigens in tissue OR isolation of rabies virus from saliva, skin snips, CSF or neural tissue	A82
Viral haemorrhagic fever	Sudden or insidious onset of fever, nausea, vomiting, diarrhoea, multifocal haemorrhages and shock. An appropriate travel history to an endemic country is supportive of diagnosis AND one of the following: demonstration of specific IgM antibody by ELISA, IFA or Western blot OR isolation of the virus in cell culture OR demonstration of viral antigen in a tissue specimen to Ebola virus, Lassa fever virus, Marburg virus or Crimean Congo virus.	A96, A98, A99
Yellow fever	A clinically compatible illness AND demonstration of yellow fever virus, antigen or genome in any clinical specimen OR a four-fold or greater change in serum antibody titre to yellow fever virus, OR a single elevated yellow fever specific IgM antibody titre, where cross-reaction with other flaviviruses has been ruled out and the patient has not received yellow fever vaccine during the previous 2 months	A95

Appendix 1d. Case definitions and ICD-10 code for notifiable diseases reported to NNDSS in 1999, sexually transmissible infections

Disease	Case definition (NHMRC 1994)	ICD-10 code(s)
Chancroid	Isolation of *Haemophilus ducreyi* from a clinical specimen OR a clinically compatible illness characterised by painful genital ulceration and inflammatory inguinal adenopathy, where syphilis, granuloma inguinale and herpes simplex have been excluded OR a clinically compatible illness in a patient who is epidemiologically linked to a laboratory confirmed case	A57
Chlamydial infection	Isolation of *Chlamydia trachomatis* from a clinical (genital) specimen OR demonstration of *Chlamydia trachomatis* in a clinical (genital) specimen by antigen detection methods	A56
Donovanosis	Demonstration of intracytoplasmic Donovan bodies on Wright or Giemsa stained smears or biopsies of clinical specimens OR a clinically compatible illness characterised by usually painless, beefy red, granulomatous or ulcerative lesions with rolled edges and a tendency to form scar tissue, where syphilis has been excluded	A58
Gonococcal infection	Isolation of *Neisseria gonorrhoeae* from a clinical specimen	A54
Lymphogranuloma venereum	Isolation of *Chlamydia trachomatis* serotype L1, L2 or L3 from a clinical specimen OR demonstration (by immunofluorescence) of inclusion bodies in leucocytes aspirated from an inguinal lymph node (bubo) OR a positive serological test for lymphogranuloma venereum strain of *Chlamydia trachomatis* in the presence of a clinically compatible illness (one or more tender, fluctuant inguinal lymph nodes or characteristic proctogenital lesions)	A55
Syphilis	A compatible clinical illness or past history AND demonstration of *Treponema pallidum* by darkfield, fluorescent antibody or equivalent microscopic methods OR reactive treponemal tests (eg: FTA-ABS, TPHA)	A50, A51, A52

Appendix 1e. Case definitions and ICD-10 code for notifiable diseases reported to NNDSS in 1999, vectorborne

Disease	Case definition (NHMRC 1994)	ICD-10 code(s)
Arbovirus infection (NEC)	Demonstration of a four-fold or greater change in serum antibody titres between acute and convalescent-phase serum specimens obtained at least 2 weeks apart and preferably conducted in parallel at the same laboratory OR demonstration of specific IgM antibodies in CSF or acute phase serum OR isolation of virus from blood, CSF or tissue specimens	A92, A93, A94
Barmah Forest virus infection	Demonstration of above criteria for Barmah Forest virus	A92.8
Ross River virus infection	Demonstration of criteria for Arbovirus infection for Ross River virus	B33.1
Dengue	Demonstration of above criteria for dengue virus (all types)	A90
Malaria	Demonstration of malaria parasites (*Plasmodium* species) in a blood film	B50, B51, B52, B53

Appendix 1f. Case definitions and ICD-10 code for notifiable diseases reported to NNDSS in 1999, vaccine preventable diseases

Disease	Case definition (NHMRC 1994)	ICD-10 code(s)
Diphtheria	Isolation of toxigenic *Corynebacterium diphtheriae* AND pharyngitis and/or laryngitis (with or without a membrane OR toxic (cardiac or neurological) symptoms	A36
Haemophilus influenzae type B	An invasive clinically compatible illness (meningitis, epiglottitis, cellulitis, septic arthritis, osteomyelitis, pneumonia, pericarditis or septicaemia) AND either the isolation of *Haemophilus influenzae* type b (Hib) from blood OR detection of Hib antigen (in a clinical case) OR detection of Gram-negative bacteria where the organism fails to grow in a clinical case	A41.3, GO0.0, JO5.1
Measles	An illness characterised by all the following features: a generalised maculopapular rash lasting three or more days AND a fever (at least 38°C if measured) AND cough or coryza or conjunctivitis or Koplik spots OR Demonstration of measles specific IgM antibody OR A four-fold or greater change in measles antibody titre between acute and convalescent-phase sera obtained at least 2 weeks apart, with tests preferably conducted in parallel at the same laboratory OR Isolation of the measles virus from a clinical specimen OR A clinically compatible case epidemiologically related to another case	BO5
Mumps	Isolation of mumps virus from a clinical specimen OR 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 2 days or more without other apparent cause)	B26
Pertussis	Isolation of *Bordetella pertussis* from a clinical specimen OR elevated *Bordetella pertussis* -specific IgA in serum or *B. pertussis* antigen in a nasopharyngeal specimen using immunofluorescence with a history of clinically compatible illness	A37
Poliomyelitis	Acute onset of a flaccid paralysis of one or more limbs with decreased or absent tendon reflexes in the affected limbs without other apparent cause, without sensory or cognitive loss	A80
Rubella	A generalised maculopapular rash and fever AND one or more of: arthralgia/arthritis OR lymphadenopathy OR conjunctivitis AND an epidemiological link to a confirmed case OR demonstration of rubella-specific IgM antibody, except following immunisation OR a four-fold or greater change in rubella antibody titre between acute and convalescent-phase sera obtained at least 2 weeks apart	BO6
Tetanus	A clinically compatible illness without other apparent cause, with or without a history of injury and with or without laboratory evidence of the organism or its toxin	A33

Appendix 1g. Case definitions and ICD-10 code for notifiable diseases reported to NNDSS in 1999, zoonoses

Disease	Case definition (NHMRC 1994)	ICD-10 code(s)
Brucellosis	Isolation of *Brucella* species from a clinical specimen OR a four-fold or greater change in *Brucella* agglutination titres or complement-fixation titres between acute and convalescent-phase serum samples at least 2 weeks apart with the tests preferably conducted in parallel at the same laboratory	A23
Hydatid infection	Positive serological test for infection with *Echinococcus granulosus* in a patient with clinical, radiological or sonographic evidence of hydatid disease OR identification of *Echinococcus granulosus* in cyst fluid or sputum OR immunoelectrophoresis demonstrating arc 5 or three or more arcs	A28
Leptospirosis	Isolation of *Leptospira* species from clinical specimens OR a four-fold or greater change in *Leptospira* agglutination titres or complement-fixation titres between acute and convalescent-phase serum samples at least 2 weeks apart with the tests preferably conducted in parallel at the same laboratory OR demonstration of leptospiral antigen in a clinical specimen OR a single raised *Leptospira* agglutination titre with a clinically compatible illness	A27
Ornithosis (Psittacosis)*	A clinically compatible illness (fever, headache, myalgia, dry cough, pneumonia) AND a four-fold or greater rise in serum antibody titres to *Chlamydia psittaci* between acute and convalescent phase sera OR detection of *C. psittaci* by nucleic acid test OR a single high titre of IgG to *C. psittaci* after the onset of a clinically compatible illness and where other diseases are excluded	A70
Q fever	A four-fold or greater change in serum (CF) antibody titre to phase II antigen of *Coxiella Burnetti* OR a four-fold or greater change in ELISA antibody titre to phase I or II antigens of *C. Burnetti* OR an IgM fluorescent antibody titre of at least 1:160 during convalescent phase of the illness (ie: 10 days or more after onset)	A78

All definitions from Surveillance Case Definitions, National Health and Medical Research Council, March 1994, except those marked * which are draft summary definitions from the Communicable Diseases Network Australia (January 2001). Some Australian States and Territories have their own case definitions for some diseases, which may vary from those shown here.

Appendix 1h. Case definitions and ICD-10 code for notifiable diseases reported to NNDSS in 1999, other bacterial infections

Disease	Case definition (NHMRC 1994)	ICD-10 code(s)
Legionellosis	A clinically compatible illness (fever, cough or pneumonia) AND at least one of the following: isolation of *Legionella* species from lung tissues, respiratory secretions, pleural fluid, blood or other tissues OR demonstration of *Legionella* species antigens in lung tissue, respiratory secretions or pleural fluid OR a four-fold or greater rise in (IFA) titre against *Legionella* species to at least 128, between acute and convalescent phase sera OR a stable high *Legionella* titre (at least 512) in convalescent phase serum	A48.1
Leprosy	Enlarged dermal nerves with associated sensory loss OR demonstration of acid-fast bacilli (in a skin smear or biopsy specimen) OR a histological picture compatible with leprosy in a biopsy specimen	A30
Meningococcal infection	Isolation of *Neisseria meningitidis* from a normally sterile site OR detection of meningococcal antigen in joints, blood or CSF OR detection of Gram-negative intracellular diplococci in blood or CSF	A39
Tuberculosis	Isolation of *Mycobacterium tuberculosis, Mycobacterium bovis,* or *Mycobacterium africanum* from a clinical specimen OR demonstration of acid-fast bacilli in a clinical specimen or in a histopathological lesion, when culture is not available, in a person with signs or symptoms compatible with tuberculosis OR evidence of resolution of disease where treatment with two or more anti-tuberculosis medications have been prescribed and follow-up has been instigated	A15, A16, A17, A18, A19

Appendix 2. Years from which diseases became notifiable in different jurisdictions in Australia*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA
Bloodborne								
Hepatitis B (incident)	1996	1993	1993	1991	1993	1993	1993	1996
Hepatitis B (unspecified)	1991	1991	NN	1991	1991	1991	1991	1991
Hepatitis C (incident)	1995	1993	1995	NN	1993	1995	1997	1996
Hepatitis C (unspecified)	1991	1991	1991	1991	1994	1991	1991	1993
Hepatitis D	1999	1999	1999	1999	1999	1999	1999	NN
Hepatitis (NEC)	1991	1991	1991	1991	1991	1991	1991	NN
Gastrointestinal diseases								
Botulism	1992	1998	1998	1998	1993	1992	1992	NN
Campylobacteriosis	1991	NN	1991	1991	1991	1991	1991	1991
Haemolytic uraemic syndrome	1999	1999	1999	1999	1999	1999	1999	1999
Hepatitis A	1991	1991	1991	1991	1991	1991	1991	1991
Hepatitis E	1999	1999	1999	1999	1999	1999	1999	NN
Listeriosis	1991	1991	1993	1991	1993	1991	1991	1991
Salmonellosis (NEC)	1991	1991	1991	1991	1991	1991	1991	1991
Shigellosis	1991	NN	1991	1991	1991	1991	1991	1991
SLTEC, VTEC	1999	1999	1999	NN	1999	1999	1999	NN
Typhoid [1]	1991	1991	1991	1991	1991	1991	1991	1991
Yersiniosis (NEC)	1993	NN	1991	1991	1991	1991	1991	1991

Appendix 2. (continued) Years from which diseases became notifiable in different jurisdictions in Australia*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA
Quarantinable								
Cholera	1991	1991	1991	1991	1991	1991	1991	1991
Plague	1991	1991	1991	1991	1991	1991	1991	1991
Rabies	1993	1997	1991	1991	1991	1991	1991	1991
Viral haemorrhagic fever	1993	1991	1991	1991	1991	1991	1991	1991
Yellow fever	1991	1991	1991	1991	1991	1991	1991	1991
Sexually transmissible								
Chancroid	1991	1994	1991	1991	1997	1993	1991	1991
Chlamydial	1993	1991	1991	1991	1993	1991	1991	1994
Donovanosis	1991	1999	1991	1991	NN	1993	1991	1991
Gonococcal infection[2]	1991	1993	1991	1991	1991	1991	1991	1991
Lymphogranuloma venereum	1991	1997	1991	1991	1997	1992	1991	NN
Syphilis	1991	1991	1991	1991	1991	1991	1991	1991
Vaccine preventable								
Diphtheria	1991	1991	1991	1991	1991	1991	1991	1991
Haemophilus influenzae type b	1993	1991	1991	1991	1993	1993	1993	1994
Measles	1991	1991	1991	1991	1991	1991	1991	1991
Mumps	1992	1992	1995	1997	1994	1995	1992	1994
Pertussis	1991	1991	1991	1991	1991	1991	1991	1991
Poliomyelitis	1991	1991	1991	1991	1991	1991	1991	1991
Rubella	1991	1991	1993	1991	1993	1995	1992	1994
Tetanus	1991	1991	1991	1994	1991	1991	1991	1991
Vectorborne								
Arbovirus infection (NEC)[3]	1997	1997	1997	1997	1997	1997	1997	1991
Barmah Forest virus infection	1995	1995	1997	1995	1995	1995	1995	1996
Dengue	1993	1991	1991	1991	1991	1991	1991	1991
Malaria	1991	1991	1991	1991	1991	1991	1991	1991
Ross River virus infection	1993	1993	1991	1991	1993	1993	1991	1991
Zoonoses								
Brucellosis	1991	1991	1991	1991	1991	1991	1991	1991
Hydatid Infection	1991	NN	1991	1991	1991	1991	1991	1991
Leptospirosis	1991	1991	1991	1991	1991	1991	1991	1991
Ornithosis	1991	NN	1991	NN	1991	1991	1991	1991
Q fever	1991	1991	1991	1991	1991	1991	1991	1991
Other bacterial infections								
Legionellosis	1991	1991	1991	1991	1991	1991	1991	1991
Leprosy	1991	1991	1991	1991	1991	1991	1991	1991
Meningococcal infection	1991	1991	1991	1991	1991	1991	1991	1991
Tuberculosis	1991	1991	1991	1991	1991	1991	1991	1991

* 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.

NN Not notifiable in 1999

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 RR, dengue and BF.

Appendix 3. Completeness of data received in NNDSS from States and Territories in 1999

Field	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total
No. missing age	0	21	41	3	1	0	172	18	256
% complete for age	100	99.9	98.9	100	100	100	99.2	99.8	99.7
No. missing sex	2	334	21	8	0	0	414	1	780
% complete for sex	99.8	98.6	99.4	100	100	100	98	100	99.1

Appendix 4. Population totals for States and Territories, 1999*

State	Population
ACT	313,346
NSW	6,411,680
NT	192,882
Qld	3,512,356
SA	1,493,074
Tas	470,261
Vic	4,712,173
WA	1,861,016
Australia	18,966,788

* Based on Australian Bureau of Statistics mid-year population estimates

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Australian notifiable diseases 2001

Nationally consistent notification of infectious diseases provides data on disease distribution across all States and Territories. These data provide a basis for the development of public health policy, a mechanism for the development of response to communicable disease outbreaks of national significance and basic information relating to the development and implementation of a communicable disease control policy. The following list shows the communicable diseases that have been nationally endorsed by the Communicable Diseases Network Australia.

Australian nationally notifiable diseases

Acquired immunodeficiency syndrome (AIDS)

Anthrax

Arboviruses — not elsewhere classified (NEC)

Australian bat lyssavirus

Barmah Forest virus

Botulism (foodborne)

Brucellosis

Campylobacteriosis

Chlamydia trachomatis

Cholera

Cryptosporidiosis

Dengue virus

Diphtheria

Donovanosis

Gonococcal infection

Haemolytic uraemic syndrome (HUS)

Haemophilus influenzae type b (HIB) (invasive only)

Hepatitis A

Hepatitis B incident

Hepatitis B unspecified

Hepatitis C incident and unspecified

Hepatitis D

Hepatitis E

Hepatitis – not elsewhere classified (NEC)

Human immunodeficiency (HIV) infection

Influenza (laboratory-confirmed)

Japanese encephalitis virus

Kunjin virus

Legionellosis

Leprosy

Leptospirosis

Listeriosis

Lyssavirus – not elsewhere classified (NEC)Malaria

Measles

Meningococcal infection

Mumps

Murray Valley encephalitis virus

Ornithosis (psittacosis)

Pertussis (whooping cough)

Plague

Poliomyelitis

Pneumococcal infection (invasive)

Q fever

Rabies

Ross River virus

Rubella

- congenital rubella

Salmonellosis (including paratyphoid)

Shigellosis

Shiga toxin-producing *Escherichia coli*/verotoxigenic *E. coli* (SLTEC/VTEC)

Syphilis

- congenital syphilis

Tetanus

Tuberculosis

Typhoid

Viral haemorrhagic fevers (quarantinable)

Yellow fever

Australian State/Territory notifiable communicable diseases, 2001

In addition to the list of nationally notifiable diseases, each State and Territory in Australia has its own list of notifiable diseases. The diseases that are additional to those on the national register are listed below for each State/Territory.

Australian Capital Territory

Chancroid

Equine morbillivirus (Hendra virus) infection

Giardiasis

Lymphogranuloma Venereum

Yersiniosis

New South Wales

Adverse event following immunisation

Chancroid

Foodborne illness in 2 or more related cases

Gastroenteritis among people of any age, in an institution (eg. among persons in educational or residential institutions)

Lymphogranuloma Venereum

Typhus (epidemic)

Northern Territory

Acute post-streptococcal glomerulonephritis

Acute rheumatic fever

Adverse event following immunisation

Amoebiasis

Atypical mycobacterial disease

Chancroid

Chlamydial conjunctivitis

Echinococcosis (hydatid disease)

Human T-cell lymphotropic virus

Lymphogranuloma Venereum

Melioidosis

Rotavirus infection

Smallpox

Trichomoniasis

Thrombotic thrombocytopaenia purpura

Typhus (all forms)

Vibrio food poisoning

Water or foodborne diseases in 2 or more related cases

Yersiniosis

Queensland

Acute flaccid paralysis

Adverse event following immunisation

Atypical mycobacterial disease

Bunyavirus infections (not included in arbovirus NEC)

Chancroid

Ciguatera poisoning

Echinococcosis (hydatid disease)

Elevated lead levels

Equine morbillivirus (Hendra virus) infection

Foodborne or waterborne disease in 2 or more related cases

Lymphogranuloma venereum

Melioidosis

Yersiniosis

South Australia

Atypical mycobacterial disease

Echinococcosis (hydatid disease)

Yersiniosis

Tasmania

Amoebiasis

Chancroid

Echinococcosis (hydatid disease)

Giardiasis

Lymphogranuloma venereum

Mycobacterial infection

Rickettsial infection (including Flinders Island spotted fever and others)

Suspected cases of food or waterborne illness

Taeniasis

Vancomycin resistant enterococci

Vibrio infection

Yersiniosis

Victoria

Food and waterborne illness in 2 or more related cases

Giardiasis

Western Australia

Amoebiasis

Amoebic meningitis

Chancroid

Echinococcosis (hydatid disease)

Giardiasis

Melioidosis

Methicilin-resistant *Staphyloccocus aureus* infection

Relapsing fever

Scarlet fever

Schistosomiasis (Bilharzia)

Typhus (Rickettsial infection)

Yersiniosis

Transmissible spongiform encephalopathies in Australia

Alison Boyd, Ashley Fletcher, James S Lee, Victoria Lewis, Colin L Masters, Steven J Collins

Abstract

The Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR) commenced surveillance in September 1993 as part of the Commonwealth's response to 4 cases of pituitary hormone (gonadotrophin)-associated Creutzfeldt-Jakob disease (CJD). With the passage of time, the Registry has become responsible for ascertaining all human transmissible spongiform encephalopathies (TSE; also known as prion diseases) within Australia since 1970. Included in the spectrum of diseases monitored are classical (sporadic, genetic, and health care acquired) CJD, and variant CJD (vCJD), first reported in 1996 in the United Kingdom. Variant CJD has not yet been diagnosed in Australia. Final classification of persons with suspected human prion disease is based upon all available clinical, investigational and pathological information. Ascertainment methods are diverse and include prompted, half-yearly personal communications from neurologists and neuropathologists, death certificate searches, and morbidity separation coding searches of major hospital, and State and Territory databases. More recently, referral for diagnostic CSF 14-3-3 protein testing (performed by the ANCJDR) has considerably increased prospective notifications of suspect cases. As at September 2001 there were 460 cases on the register; 237 definite cases, 168 probable and 55 incomplete cases awaiting final classification. ***Commun Dis Intell*** **2001;25:248-253.**

Keywords: Transmissible spongiform encephalopathies; TSE; Creutzfeldt-Jakob disease; CJD; vCJD

Introduction

Transmissible spongiform encephalopathies (TSE) constitute a group of invariably fatal neurodegenerative diseases which affect both animals and humans. In 1998 Prusiner reviewed TSE[1] summarising developments since the 1950s, emphasising events over the last 2 decades. Bovine spongiform encephalopathy (BSE; 'mad cow' disease) and scrapie are the most common animal forms while Creutzfeldt-Jakob disease (CJD) is the most common human phenotype. Variant CJD (vCJD) is believed to be linked to the consumption of BSE prion protein-contaminated beef or beef products. This association was first positively identified in United Kingdom (UK) cattle in 1986. Variant CJD is characterised clinically by a younger age at death (mean 29 years), prominent psychiatric presenting symptoms, and longer illness duration. Classical CJD typically presents as a rapidly progressive dementia in older persons, often associated with myoclonus and ataxia. Other forms of human TSE include Gerstmann-Sträussler-Scheinker disease (GSS), fatal familial insomnia (FFI) and Kuru, with the first two almost invariably associated with mutations in the prion protein gene (PRNP) on chromosome 20. Kuru occurs exclusively in a restricted geographical region in the Eastern Highlands of Papua New Guinea (PNG) and is related to the practice of ritualistic endo-cannibalism as a mourning rite of deceased relatives. The disease has almost disappeared since the cessation of cannibalism in the 1950s.

Neuropathologically, varying combinations of microvacuolation of the gray matter, astrocytic gliosis, neuronal loss and immunodetectable deposits of prion protein are characteristic changes of TSE. Disease pathogenesis appears intimately linked to expression of an abnormal protease-resistant conformation of the prion protein (PrP^{res}), although precise mechanistic details of how this isoform arises from the normal mammalian prion protein (PrP^{c}) and consequent pathophysiological steps remain to be elucidated. The infectious unit or particle (termed prion) carries no nucleic acid and appears predominantly, if not exclusively, composed of PrP^{res}. Expression of PrP^{c} is obligatory for disease development and successful transmission, with PrP^{res} thought to serve as a template facilitating further conversion of the wild type prion protein, PrP^{c}, into the disease causing isoform. Early transmission studies in the 1960s established transmissibility, however TSE are not considered classical infectious diseases.

The Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR) was established in September 1993 in response to the recognition of 4 probable human pituitary hormone (hPG) related CJD deaths. The original Registry's objective was detailed scrutiny for further health care acquired CJD cases over the restricted period 1 January 1988 to 31 December 1997. This 10-year epoch was intended to encompass the highest likely period surrounding the 4 index cases. However, following the commissioned inquiry into the use of human pituitary hormones under the Australian Human Pituitary Hormone Program (AHPHP), reporting in 1994,[2] recommendations were adopted to expand ANCJDR activities, including retrospective case ascertainment to 1 January 1970. Prospective monitoring to ensure complete ascertainment of any further occurrences of health care acquired CJD has required the Registry to continue to

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evaluate all persons suspected to be manifesting a TSE. CJD is not a notifiable disease in Australia.

The most common form of human prion disease is sporadic CJD which by definition is without apparent cause. This form constitutes between 85 to 90 per cent of all human TSEs, and typically death occurs after a median illness duration of 4 months. Less commonly, the CJD phenotype can occur as a result of a mutation in the PRNP gene (genetic form), or as a consequence of horizontal transmission, which in Western societies to date is invariably from inadvertent contamination through health care provision (health care acquired CJD). Sources of health care acquired CJD transmission[3] have included cornea[4] and dura mater transplants,[5] as well as treatment with cadaveric pituitary hormones.[6,7] Genetic forms of human TSE account for approximately 12–14 per cent of all cases when systematic PRNP genotyping is undertaken,[8] while health care acquired CJD is very rare with only 10 cases so far recognised in Australia. Variant CJD represents a new strain of human prion disease and based on a range of data, most likely occurred as a consequence of BSE crossing the species barrier from cattle to man via the consumption of contaminated meat products. Australia remains free of BSE which is now recognised in a number of European countries, and most recently Japan.

As of September 2001, the ANCJDR had classified 405 persons who had died from clinically likely (probable) or definite prion disease, with a further 55 incomplete (suspect) cases requiring further investigation before final classification is possible. These incomplete cases are either awaiting postmortem neuropathological examination or additional clinical details.

Classification methods and case ascertainment

Classification of human prion diseases is based on internationally accepted criteria (Table 1),[9,10] with minor modifications. In brief, definite cases are those neuropathologically confirmed, preferably with the presence of PrP^{res} demonstrated in the brain, by immunochemical methods. Probable or clinically likely patients with CJD manifest a combination of rapidly progressive dementia with any two of the following features: myoclonus; visual or cerebellar dysfunction; pyramidal or extrapyramidal dysfunction; or akinetic mutism. Variant CJD is distinguishable from classical CJD clinically and neuropathologically. Since 1997, a positive 14-3-3 result has been the most useful premortem investigation whereas prior to 1997 an EEG showing typical periodic discharges was used as an aid in

Table 1. Classification criteria for classical and variant CJD

	Definite	Probable
Classical CJD	• Neuropathologically confirmed *supplemented by* • Immunochemical confirmation of PrP^{res}	• Progressive dementia <2 years *Investigations* • Typical EEG *and/or* • Positive CSF 14-3-3 protein test At least 2 out of 4 of the clinical features: • Myoclonus • Visual or cerebellar dysfunction • Pyramidal/extrapyramidal dysfunction • Akinetic mutism
Variant CJD	• Progressive neuropsychiatric disorder *and* • Neuropathological confirmation of diagnosis of vCJD	• Progressive neuropsychiatric disorder without family history of prion disease • Duration of illness >six months *and* 4 out of 5 of the clinical features: • Early psychiatric symptoms • Persistent paraesthesia or dysaesthesia • Ataxia • Myoclonus or chorea • Dementia *Investigations* • Abnormal but non-typical EEG for CJD *and* • Bilateral pulvinar high signal on MRI *or* • A positive tonsil biopsy

diagnosis.[11] The ANCJDR does not utilise the possible classification. Cases unable to fulfill the above criteria are excluded with one exception: a single health care acquired case due to cadaveric pituitary growth hormone was classified as a possible case because of concomitant medical problems militating against the probable classification.

The various mechanisms by which the ANCJDR ascertains cases of suspected prion disease are summarised in Table 2. The most frequent method of primary case ascertainment has been personal communications by medical practitioners, usually by telephone or correspondence, particularly neurologists and pathologists. At the inception of the Registry, all neurologists and neuropathologists within Australia were asked to inform the ANCJDR of any cases encountered during their entire professional practice. As a safeguard, reply-paid mail-outs are posted to these two important specialist groups semi-annually, prompting reporting of recently deceased or prospective cases.

Table 2. First sources of case ascertainment

Method	Per cent
Personal communications, including	66.5
Neurologists (includes mail-out reply cards)	(40.1)
Neuropathologists (includes mail-out reply cards)	(17.5)
Pituitary Hormones Task Force	(3.7)
Family	(4.7)
CJD Counselling Service	0.5
Death certificates	23.3
Hospital and health department searches	10.2
Hospital medical records	(8.2)
Health department search	(2.0)
Total	100

Death certificate searches through the Australian Health Index have proven to be of benefit in the retrospective ascertainment of cases. A search for death certificates in 1995 revealed 188 cases specifically coded to CJD. Unfortunately, death certificate searches for the epoch 1970 to 1979 have been impracticable due to the Australian Bureau of Statistics previously discarding identifying information and the lack of a standardised disease coding system across Australia. The ANCJDR routinely performs annual searches for CJD-coded death certificates through the Australian Institute of Health and Welfare (AIHW). The prospective notification mechanisms of the ANCJDR usually facilitate detection of patients approximately 18 months prior to death certificate notification.

The most time-consuming method of case ascertainment has been a nation-wide search of the health information departments of all university-affiliated teaching hospitals seeking all separations specifically coded to CJD and pre-senile dementia according to the ICD-8 and 9, and more recently the CJD specific ICD-9 CM code. In addition, searches have been performed within each State and Territory health information coding system using the CJD specific codes, providing similar but not identical information to that obtained from the hospitals. The reasons for search discrepancies vary but have included coding or data entry errors. Specific searches of private hospital data were not undertaken.

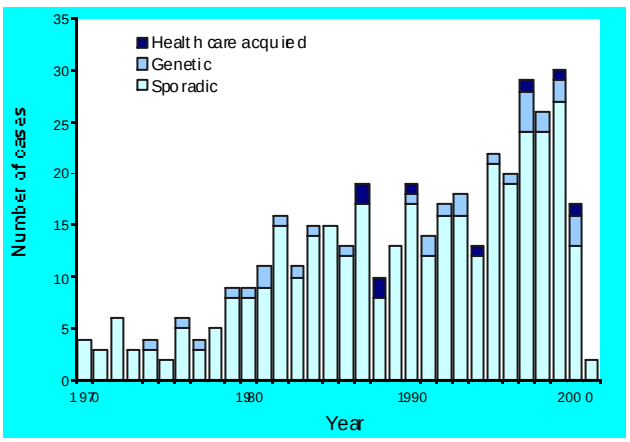
The introduction of an assay for the CSF 14-3-3 protein (a non-specific marker of neuronal degeneration) has over the last few years significantly increased the number of prospective referrals of suspect CJD cases to the Registry.[12] This test has shown 90 per cent sensitivity and specificity in appropriately evaluated patients with suspect CJD.[13] However, acknowledging the small but definite false positive and negative rates of this non-specific test, final inclusion on the register depends on review of all available clinical information. 14-3-3 negative cases are followed-up nine months after testing as a safeguard against a false negative result. Patients ultimately classified as CJD through this notification mechanism are currently included under neurologists' personal communications.

The ANCJDR records predominant occupation following consideration of lifetime employment history. The Australian Standard Classification of Occupations (ASCO)[14] was used for classification. Groups of interest include farmers subsumed under group 1 as managers/administrators and butchers/abattoir workers under group 4. The ANCJDR has included a home duties/childcare group, not recognised by ASCO. Disparity between the ANCJDR data classification of predominant occupation versus census data hinders comparative analysis. Australian Bureau of Statistics (ABS)[15] data were used to generate disease incidence rates, as well as age adjusted data.

Results

There has been a steady increase in the annual incidence of CJD since 1970, with probable plateauing during the late 1990s (Figure). When 'incomplete' cases are taken into account, the 1988 to 1999 average annual incidence is 1.1 cases per million (0.6 prior to 1988). Using probable and definite cases alone for the same time period the annual incidence is 1.0 case per million (0.6 prior to 1988). The

Figure. Classification of deaths from transmissible spongiform encephalopathies



increase for definite and probable cases has been reasonably uniform across Australia with absolute numbers reflecting regional population distributions: New South Wales (including the Australian Capital Territory) 139; the Northern Territory 1; Queensland 69; South Australia 48; Tasmania 4; Victoria 105; and Western Australia 39.

Acknowledging their salient demographic similarities, definite and probable cases have been aggregated for statistical analysis. The overall composition of probable and definite cases is 90.4 per cent sporadic, 7.4 per cent genetic and 2.2 per cent health care acquired. Analysis by gender reveals 173 males and 193 females with sporadic CJD, 13 males and 17 females with genetic TSE, and 4 males and 5 females with health care acquired disease. Age at death for sporadic CJD typically occurs between 56–75 years (mean 65.6 years), with an average duration of illness of 6.5 months. Age at death in genetic CJD ranges from 20 years through to 82 years, with the average age at death 56.8 years, and the mean duration of illness 17.6 months. The health care acquired cases include the 4 recipients of gonadotrophin (one probable and three pathologically proven), the single possible case arising from human growth hormone, and five cases related to the implantation of Lyodura (dura mater) grafts (three probable and two definite). Age at death for health care acquired CJD ranges from 26–62 years, with an average age at death 42.3 years. The average duration of illness is 7.8 months.

Analysis of cases by occupation (summarised in Table 3), reveals no groups to be over-represented. A closer look at the spectrum of health care workers developing CJD reveal all cases but one to be sporadic CJD; a radiologist; a dental surgeon; a registered nurse, and three general practitioners. One general practitioner was confirmed with genetic CJD. None of the health care workers are known to have had professional contact with CJD patients. Specifically, the nurse worked in a rural hospital where no known CJD cases had been recorded. A second nurse was a recipient of human derived pituitary gonadotrophin. Fifteen cases have no recorded occupation and one health care acquired case is known never to have worked.

After age adjustment, sub-group comparison by country of birth reveals 68.3 per cent of the general population to be Australian born whereas 66.0 per cent of ANCJDR cases were born in Australia. There is also no excess of cases amongst any immigrant sub-group.

Discussion

Although the catalyst for formal national surveillance of TSE in Australia was to monitor further instances of human pituitary hormone-related CJD, the activities of the ANCJDR have broadened to include vigilance for vCJD. The additional health care acquired cases recognised (as of September 2001) have been as a consequence of dura mater grafts with the most recent death occurring in 2000. One of these patients had an incubation period of approximately 17 years, equivalent to the longest seen so far for this type of iatrogenic transmission. It has now been approximately 10 years since the last death from gonadotrophin-related CJD in Australia, raising the likelihood that no further cases of this type of transmission will be seen domestically in contrast to continuing growth-hormone related cases occurring in other countries such as France.[3] Nevertheless, given the known potential for unpredictable and lengthy incubation periods, continued vigilance for many more years is required.

Over the 30 year surveillance period, the incidence of human TSE in Australia has increased considerably. This

Table 3. Cases by standard occupation

Occupation group	Cases on Register		Working Australian population
	n	%	%
1. Managers and administrators	55	13.6	9.4
2. Professionals	39	9.6	11.3
Medical professionals	4		
Health professionals	1		
Nurse professionals	1		
3. Para-professionals	16	4.0	4.8
Enrolled nurse	1		
4. Trades persons	40	9.9	12.2
5. Clerks	32	7.9	13.4
6. Salespersons and personal service workers	23	5.7	12.6
7. Plant and machine operators, and drivers	29	7.2	5.6
8. Labourers and related workers	51	12.6	12.0
9. Home duties/childcare	94	23.2	18.7
Total	379	93.7	100

phenomenon is analogous to the experience of other long-term comprehensive national CJD surveillance programs and is thought to reflect better case ascertainment stemming from improved recognition, confirmation and reporting. There has been a relative plateauing in annual incidence since the late 1990s, suggesting current ascertainment methods have been optimised. A combination of factors (including the diagnostic CSF 14-3-3 protein test, PRNP genotyping and enhanced post-mortem rates) have contributed to this improved classification and identification of CJD cases across Australia. These investigations have especially increased the ascertainment of non-typical and familial cases. Nevertheless, our non-systematic approach to PRNP genotyping is likely to have contributed to the lower percentage of genetic cases compared with international CJD surveillance programs utilising systematic genetic analysis.

Excessive temporo-spatial grouping of apparently sporadic CJD cases is occasionally seen,[16] and the ANCJDR has undertaken a comprehensive investigation of such a cluster in an Australian rural city which is the subject of a separate report.

At the time of writing, vCJD has not been diagnosed in Australia. Acknowledging the likely zoonotic basis of vCJD from BSE, Australia's continued verified freedom from the latter and the relatively small number of variant cases so far in the UK compared to the total number of BSE affected cattle, the likelihood of occurrence is low but not zero. This is especially so given the extensive flux of citizens between Australia and the UK over the past 2 decades with the possibility of 'importing' vCJD. For a number of reasons, particularly its increased heat stability and the presence of protease-resistant prion protein in lymphoreticular organs,[17] vCJD is believed to constitute a greater public health risk than its classical counterpart. Therefore, any vCJD occurrence in Australia will pose similar public health and safety issues as in the UK. Hence, the low but potential risk of vCJD arising in Australia mandates continued careful monitoring to enable early detection, and safeguard as much as possible against untoward outcomes, especially inadvertent health care related transmission.

The true significance of occupation is tentative and further research will be needed to elucidate potential risks. A case-control study of risk factors for sporadic CJD has been undertaken by the ANCJDR with the results already reported in detail.[18] Importantly, the risk of sporadic CJD progressively increased with the number of surgical treatments to a maximum of three procedures (odds ratio 2.13, 95% CI 1.34-3.41)) and did not appear related to site or complexity of the procedure. There was also an increased risk of CJD with employment or residence on a farm ($p<0.001$) or market garden ($p=0.002$) for periods longer than 10 years ($p=0.002$). These findings raise interesting possibilities with regards to under-recognised or potentially novel transmission events but await validation from further studies.

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Australia announces new measures for imported beef products

Fiona J Brooke

On 18 July 2001, the Australian Government announced a new requirement that all beef and beef products imported into Australia should certify their bovine spongiform encephalopathy (BSE) free status. This policy announcement follows on from, and effectively implements, the new food standard recently agreed by the Australian New Zealand Food Standards Council that requires all beef products sold for human consumption in Australia be derived from BSE-free animals.

The new certification regime has been developed from a detailed assessment of the risks posed to Australia from imported beef.

Australia has been monitoring the growing epidemic of BSE in Europe. In the last 12 months Germany, Spain, Italy, the Czech Republic, Greece, and Slovakia have all notified their first cases of indigenous BSE. Japan is the first non-European country with proven BSE.[1] A little more than 12 months ago these countries were declaring that their herds were free of BSE. The implementation of new testing requirements by the European Commission has resulted in a growing recognition that BSE is a far more widespread problem than initially acknowledged.

Attention has also turned to the widespread export of potentially infective meat and bone meal (MBM) from the United Kingdom and other European countries. As a result of these exports, it appears that the threat of BSE is now potentially global — which has certainly been borne out with the report of BSE in Japan.

Whilst the exact tonnage exported is not available, the United Nations Food and Agriculture Organization has estimated that in excess of 170,000 tonnes of MBM was exported from the United Kingdom and other parts of Europe world-wide from 1990-1996. It is likely that many of the countries importing MBM over this period may not have had effective BSE controls and surveillance programs in place. Consequently, due to the external challenge from possibly BSE-infected MBM, BSE may have been amplified in cattle herds within some of these countries to varying extents and to date escaped detection.

In order to assess the different range of risk exposures, countries wishing to export beef and beef products to Australia will need to apply to the Australia New Zealand Food Authority (ANZFA) and supply information on their exposure to different BSE risk factors and the range of control measures that have been in place to prevent the amplification of BSE within that country. Countries will be assessed by ANZFA and placed into one of the following categories:

Category A (certification required) — beef and beef products from these countries are regarded as posing a negligible risk to human health.

Category B (certification required) — these countries, while not reporting cases of BSE, may have been exposed to high risk factors, such as the importation of high-risk meat and bone meal.

Category C (certification required) — countries in this category are known to have considerable exposure to BSE risk materials, but have not reported indigenous cases of BSE.

Category D – These countries have reported cases of indigenous BSE in their herds. Beef and beef products from countries in this category pose the highest level of risk and will be refused entry to Australia.

This policy will be monitored to ensure it reflects any changes in the science on BSE. For example, should a validated test become available for the detection of specified risk material in beef end products, such a test may be adopted and incorporated into Australia's risk management protocols.

Additional details on the certification measures and the assessment process can be obtained from the following websites: http://www.health.gov.au/pubhlth/strateg/bse/response.htm and http://www.anzfa.gov.au/mediareleases publications/mediareleases/mediareleases2001/australia announcesnewmeasurestoprotectpublicfromeffectsofbse/index.cfm.

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Tuberculosis notifications in Australia, 1999

Paul Roche, Angela Merianos and the National TB Advisory Committee (Ral Antic (Chair), John Carnie, Amanda Christensen, Justin Waring, Anastasios Konstantinos, Vicki Krause, Mark Hurwitz, Avner Misrachi, Ivan Bastian) for the Communicable Diseases Network Australia

Abstract

Australia has one of the lowest incidence of tuberculosis in the world. The crude annual notification rate for tuberculosis (TB) has remained stable at between 5 and 6 per 100,000 population since 1991. In 1999, there were a total of 1,159 TB notifications in Australia of which 1,117 were new TB cases, and 42 were relapsed cases. The corresponding annual notification rate for new and relapsed TB was 5.9 and 0.2 per 100,000 population respectively. People born overseas accounted for 83 per cent of the notified cases. TB notification rates remain highest among overseas-born residents from high prevalence countries, and indigenous Australians. The lowest rates of disease are in the non-indigenous, Australian born population and data from the last 7 years indicate that the rate of tuberculosis in this population is continuing to fall. *Commun Dis Intell* 2001;25:254-260.

Keywords: tuberculosis; TB; surveillance

Introduction

There were 8.4 million new cases of tuberculosis (TB) worldwide in 1999, an increase from 8 million in 1997 due mainly to increases in the incidence of TB in the African countries most affected by the HIV/AIDS epidemic.[1] In 1998, 3.6 million cases (45% of all estimated TB cases) were reported to the World Health Organization (WHO) Global Surveillance Programme by 189 countries.[2]

The HIV pandemic continues to fuel the TB epidemic in many regions of the world, especially Asia and sub-Saharan Africa. An estimated 8 per cent of the global incident TB cases in 1997 occurred in people co-infected with HIV. While the global case fatality rate for TB was 23 per cent in 1997, case fatality rates for TB exceeded 50 per cent in African countries with high prevalence rates of HIV.[3]

The global burden of TB has been further exacerbated by poverty, natural disasters, conflict and political instability, all of which have served to thwart the development of health services in many countries, or have lead to a progressive erosion in existing health infrastructure. Human migration, so often the consequence of these events, has created a social context in which the delivery of effective drug treatment is further compromised. Poorly supervised and inadequately treated TB is the basis for the emergent problem of multi-drug resistant TB (MDR-TB). The current state of drug resistant TB in Australia is described in the following report in this issue of *CDI*.[4]

In response to the TB epidemic, the WHO has advocated Directly Observed Therapy Short-course (DOTS) as the most effective method of TB treatment. DOTS programs were accessible to 43 per cent of the world's population in 1998.[2] In Australia 3 States and Territories had implemented DOTS methods in their TB programs in 1999, which covered in total 37 per cent of the Australian population. In other Australian States, TB treatment programs appropriate to the local epidemiology and health infrastructures were implemented, which included the follow-up of all cases.

More than half of the world's TB occurs in South-east Asia and the Western Pacific regions, with Australia straddling both these regions. Despite this, Australia has maintained a low and stable rate of TB through effective pre-migration screening and the activities of specialised, multi-disciplinary TB services in the States and Territories.

The National Mycobacterial Surveillance System (NMSS), established in 1991, has monitored trends in the national rates of active TB over the last 8 years. Future enhancements to the national TB surveillance system will serve to better inform policy makers, public health practitioners and clinicians on the outcomes achieved from TB control efforts.

Methods

Notifications of TB are reported to State and Territory health authorities throughout the year, collated on an annual basis and sent to the NMSS at the Department of Health and Aged Care, Canberra, in computerised format. All reports are de-identified beforehand. Core data fields are shared with the National Notifiable Diseases Surveillance System (NNDSS). Variables reported in this core set include a unique identifier for each notification, disease code (to differentiate *Mycobacterium tuberculosis* infections from atypical mycobacteria infections), postcode of residence, date of birth, sex, dates of disease onset and report, indigenous status, and confirmation status of the report. A supplementary data set includes indigenous status, country of birth, length of residence in Australia for overseas born persons, pathogen species, principal site of disease, methods of diagnosis, antimicrobial therapy initiated at the time of notification, past BCG vaccination, HIV status and classification of TB as new or relapsed disease.

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The case definitions used in 1999 are described below:

Tuberculosis (new case)

- A case which has been confirmed by the identification of *Mycobacterium tuberculosis* (or *M. africanum* or *M. bovis*) by culture; or
- a case which has been diagnosed to be active clinically and which has been accepted as such by the State or Territory Director of Tuberculosis/Program Manager.

Tuberculosis (relapsed case)

- A case of active tuberculosis diagnosed again (bacteriologically, radiologically or clinically) having been considered inactive or quiescent following previous full treatment (as deemed appropriate by the State or Territory Tuberculosis Director/Program Manager).

Mortality data for tuberculosis and denominator population data for the calculation of rates were obtained from the Australian Bureau of Statistics (ABS). Denominator data for age and sex are based on mid-year population estimates for 1999. Resident population by indigenous status and country of birth was based on estimates of the relevant populations as at 30 June 1999.

Results

Notification rates – new and relapsed cases

In 1999, 1,159 cases of active tuberculosis were notified nationally (6.11 per 100,000). Of these 1,117 (96.4%) were new cases and 42 (3.6%) were relapsed cases. The corresponding crude annual incidence rate was 5.89 per 100,000 for new cases and 0.22 per 100,000 for relapsed cases (Table 1 and Figure 1).

Crude incidence rates vary widely between jurisdictions (Table 2). Since 1991, rates of TB have been less than 5 per 100,000 in Tasmania, Queensland, South Australia and Western Australia. In the Australian Capital Territory rates have been less than 5 per 100,000 for all years except 1992, 1995 and 1999. The two most populous States, Victoria and

Figure 1. Incidence rates per 100,000 population for new tuberculosis notifications, Australia, 1948 to1999

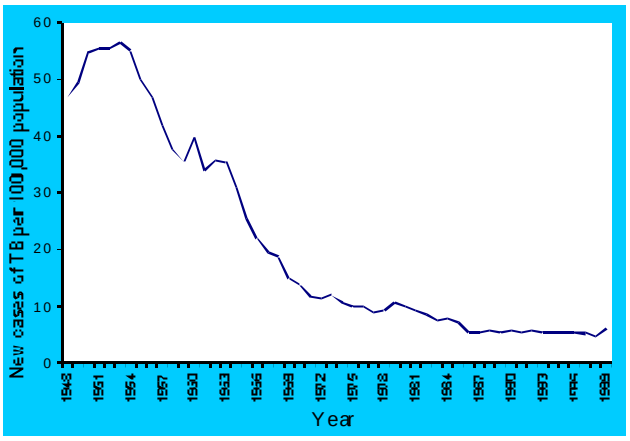


Figure 2. Notifications rates for new cases of tuberculosis, Australia, 1997 to 1999, by State and Territory

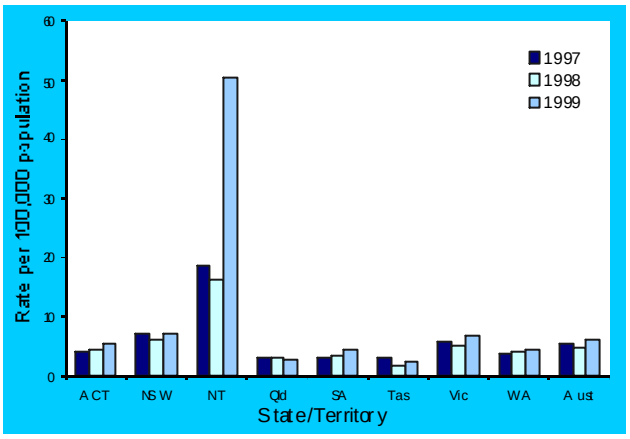


Table 1. Notifications of new and relapsed cases of tuberculosis, and rates per 100,000 population, Australia, 1986 to 1999, by year

Year	New cases		Relapsed cases		Total cases	
	Number	Rate	Number	Rate	Number	Rate
1986	863	5.39	43	0.27	906	5.66
1987	868	5.34	39	0.24	907	5.58
1988	925	5.60	29	0.18	954	5.77
1989	902	5.36	50	0.30	952	5.66
1990	979	5.74	37	0.22	1,016	5.95
1991	903	5.22	47	0.27	950	5.50
1992	983	5.62	28	0.16	1,011	5.78
1993	944	5.35	47	0.27	991	5.61
1994	996	5.58	61	0.34	1,057	5.93
1995	988	5.47	50	0.28	1,038	5.75
1996	983	5.37	54	0.29	1,037	5.66
1997	954	5.15	47	0.25	1,001	5.40
1998	884	4.72	39	0.21	923	4.92
1999	1,117	5.89	42	0.22	1,159	6.11

New South Wales, have reported intermediate rates of between 5 and 8 per 100,000 since 1991, and the Northern Territory has reported rates in excess of 15 per 100,000 over the same time period. In 1999 all States and Territories with the exception of Queensland showed an increase in the rate of TB notifications (Figure 2). The Northern Territory had the largest increase, from 16.3 to 50.3 per 100,000, due to the large number of cases of tuberculosis among East Timorese refugees who were evacuated to Darwin in the latter part of 1999.

Age and sex

In 1999, sex was reported in all but one case. Information on age was available in over 99 per cent of cases with age data missing for only 3 cases (Table 3). Males accounted for 585 (50.5%) and females for 573 (49.5%) of the notifications with the corresponding incidence rate being 6.2 and 6.0 per 100,000 population respectively. There were 23 cases of tuberculosis notified in children under the age of 5, with a corresponding rate of 1.8 per 100,000 population. All notifications of relapsed TB were in persons aged over 30 years and 50 per cent were male.

Principal sites of disease

A principal site of disease was reported for all but 16 cases of new TB and all but one case of relapsed TB. Of the new cases, 735 (65.8%) had pulmonary and 117 (10.5%) had nodal disease (Table 4). Thirty-seven per cent (283 cases) of the new pulmonary cases were smear-positive.[1]

Table 2. Notifications of new and relapsed cases of tuberculosis and rates per 100,000 population, Australia, 1999, by State and Territory

State/Territory	New cases		Relapsed cases		Total cases	
	Number	Rate	Number	Rate	Number	Rate
Australian Capital Territory	16	5.11	1	0.32	17	5.43
New South Wales	444	6.92	25	0.39	469	7.31
Northern Territory	96	49.77	1	0.52	97	50.29
Queensland	86	2.45	7	0.20	93	2.65
South Australia	66	4.42	3	0.20	69	4.62
Tasmania	11	2.34	0	0.00	11	2.34
Victoria	314	6.66	5	0.11	319	6.77
Western Australia	84	4.51	0	0.00	84	4.51
Total	1,117	5.89	42	0.22	1,159	6.11

Table 3. Notifications of tuberculosis and rates per 100,000 population, Australia, 1999, by age group and sex

Age group (years)	Males		Females		Total	
	Number	Rate	Number	Rate	Number	Rate
0-4	13	2.0	10	1.6	23	1.8
5-9	4	0.6	4	0.6	8	0.6
10-14	6	0.9	8	1.2	14	1.1
15-19	21	3.1	27	4.2	48	3.6
20-24	52	7.5	44	6.6	96	7.1
25-29	64	8.6	80	10.9	144	9.7
30-34	53	7.5	71	10.0	124	8.8
35-39	37	4.9	63	8.3	100	6.6
40-44	50	7.1	43	6.0	93	6.5
45-49	39	5.9	31	4.7	70	5.3
50-54	27	4.4	24	4.0	51	4.2
55-59	32	6.9	25	5.5	57	6.2
60-64	27	7.1	25	6.6	52	6.9
65-69	31	9.3	25	7.2	56	8.3
70-74	36	12.4	32	9.7	68	11.0
75-79	46	21.6	21	7.4	67	13.5
80-84	27	24.4	22	12.4	49	17.0
85+	17	22.9	17	10.2	35*	14.5
Unknown	3		1		4	
TOTAL	585	6.2	573	6.0	1,159	6.1

* The gender of one case in the age group > 85 years was not identified

The rate of pulmonary tuberculosis in non-indigenous Australian-born persons was 0.9 per 100,000 compared to 13.6 per 100,000 in the overseas-born and 6.6 per 100,000 in indigenous Australians. The rate of extra-pulmonary tuberculosis in non-indigenous Australian-born persons was 0.18 per 100,000 compared to 5.1 per 100,000 in overseas born persons and 1.2 per 100,000 in indigenous Australians.

BCG status

BCG vaccination status was known in 376 (32%) of the 1159 TB notifications. Of these 92 (24.5%, [8% of total cases]) reported a history of BCG and 284 (75.5%, [24% of total cases]) had not received a BCG.

Antimicrobial therapy

An initial drug therapy comprised of the 4-drug combination of isoniazid, rifampicin pyrazinamide and ethambutol was given to 970 (81%) of patients.

HIV status

Information on HIV status was provided in only 110 (9.5%) of notified cases of TB. Of these, only 4 were positive.

Country of birth

The majority (955 cases, 82% of the total) of TB notifications was in people born overseas. The number of new TB cases reported in the Australian-born and overseas-born populations was 183 (16.4%) and 923 (82.6%) respectively. Eleven cases (0.9%) of new TB cases were in people whose country of birth was not recorded. The corresponding rate of new TB disease in the Australian-born and overseas-born populations was 1.2 and 20.6 per 100,000 population respectively (Figure 3).

The incidence rates of all TB notifications (new and relapsed) per 100,000 overseas-born resident populations in Australia, are shown in Figure 4. The highest rates of TB were in Australians born in Indonesia (142 cases; 229.4 cases per 100,000); Vietnam (124 cases; 71.4 per 100,000); the Philippines (85 cases; 70.3 per 100,000); China (82 cases; 51.4 per 100,000); and India (74 cases; 71.2 per 100,000). Together these countries accounted for 507 (53%) notifications in the overseas-born cases. The rates of TB per 100,000 overseas-born resident population in Australia for 1998 is presented together with WHO case incidence rates for TB in the country of origin for the same year (Table 5).

The age and sex distributions of Australian-born and overseas-born TB incidence rates are shown in Figure 5. The overseas-born population shows high age specific rates in both young adults and the elderly, whereas in the Australian-born population, there is a gradual increase in age-specific rates with advancing age.

Indigenous status

Indigenous status was reported for 1152 (99.6%) of all notifications of TB. All but one of the Australian-born TB cases had their indigenous status reported. Indigenous Australians accounted for 34 TB cases in 1999, of which 3

Figure 3. Incidence rates of tuberculosis, new disease in non-indigenous Australian and overseas born, 1991 to 1999

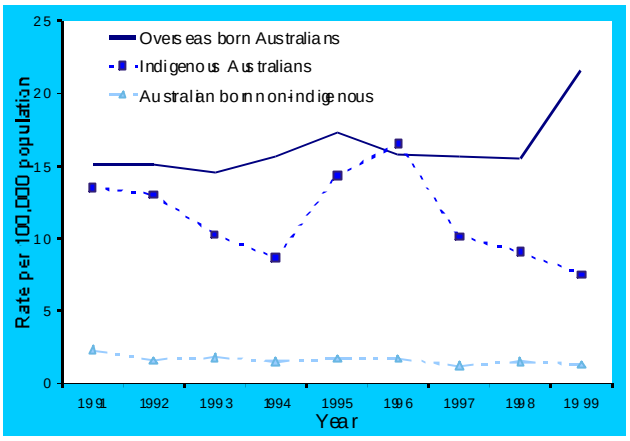


Figure 4. Incidence rates by country of birth, per 100,000 resident population in Australia, 1999

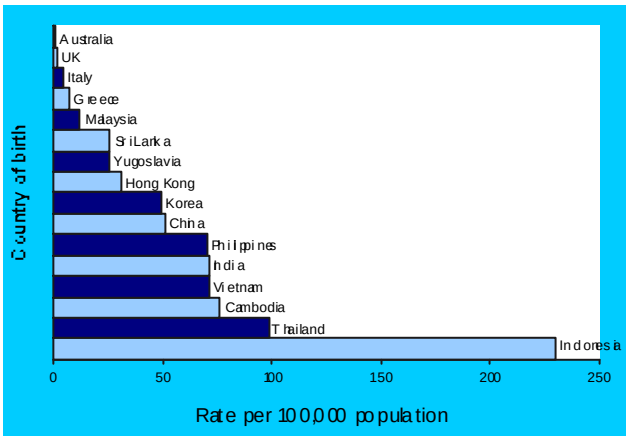


Table 4. Notifications of new and relapsed cases of tuberculosis in Australia, 1999, by site of disease

Site	New cases	Relapsed cases	Total cases	Total %
Pulmonary	735	32	767	66.2
Pleural	30	0	30	2.6
Lymph nodes	117	4	121	10.4
Bone/Joint	26	1	27	2.3
Genitourinary	37	0	37	3.2
Miliary	17	1	18	1.6
Meningeal	13	0	13	1.1
Peritoneal	14	0	14	1.2
Unspecified	128	4	132	11.4

Table 5. Tuberculosis notifications, Australia, 1999. Number and estimated rates per 100,000 for selected countries of birth *

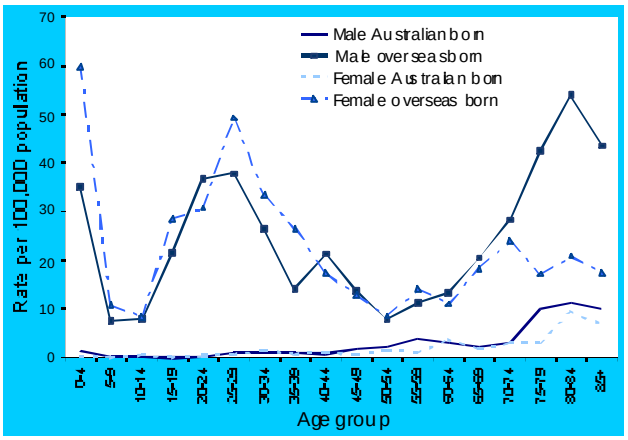
Country of birth	New cases	Relapsed cases	Total cases	Estimated Australian resident population by country of birth 1999	Rate per 100,000 population in Australia, by country of birth*	WHO incidence rate (per 100,000) for country, 1999
Indonesia (including East Timor)	141	1	142	61,900	229.4	107.1 (1995)
Vietnam	123	1	124	173,600	71.4	111.0
Philippines	81	4	85	120,800	70.3	294.5
China	75	7	82	159,400	51.4	33.7
India	74	0	74	103,900	71.2	118.3
Yugoslavia (NFD)	53	1	54	207,600	26.0	39.3
United Kingdom	22	5	27	1,215,000	2.2	10.1
Somalia	25	1	26	N/a	-	40.3
Thailand	20	2	22	22,327	98.5	51.2
South Korea	17	3	20	40,200	49.8	57.3
Cambodia	18	0	18	23,711	75.9	148.6
Hong Kong	16	0	16	50,800	31.5	111.7
Ethiopia	14	0	14	N/a	-	97.4
Sri Lanka	14	0	14	54,800	25.5	35.7
Italy	12	0	12	245,200	4.9	8.5
Greece	11	0	11	142,200	7.7	7.3
Malaysia	11	0	11	92,300	11.9	64.4
Total overseas born	923	32	955	4,482,014	21.3	-
Australian born	183	10	193	14,484,774	1.3	-
Unknown COB	11	0	11	-	-	-
Total	1,117	42	1,159	18,966,788	6.1	-

* Rates per 100,000 resident population should be interpreted with caution as many of the cases are in visitors to Australia who are not included in the census data

NFD 'Not further defined'

N/a Not available.

Figure 5. Age and sex-specific tuberculosis incidence rates in Australian-born and overseas-born individuals, per 100,000 resident population, 1999



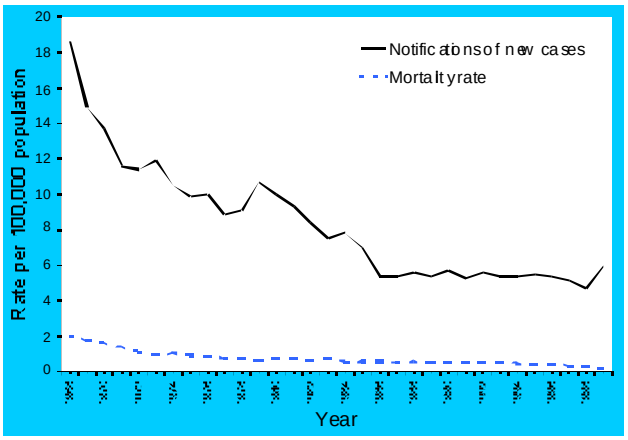
were relapses and 31 were new cases of TB. Fifteen (44%) notifications of TB in indigenous Australians were reported from the Northern Territory. The Australian Capital Territory reported no indigenous cases. The TB incidence rate was 8.3 per 100,000 indigenous population. The TB incidence rate in the Australian born, non-indigenous population was 1.1 per 100,000 population.

Among indigenous Australians with TB notified in 1999, 19 were male and 15 female. Ten (29%) of the notifications in indigenous people were aged over 60 years, and 2 cases (6%) were aged less than 14 years. The age and sex incidence rates for indigenous and non-indigenous Australian-born persons are given in Table 6. The age-specific rates for indigenous Australians show a similar pattern to non-indigenous Australians, with an increase in TB with increasing age.

Mortality

In 1999, the Australian Bureau of Statistics reported 29 deaths for which TB was the underlying cause. The crude mortality rate was 0.18 per 100,000, which is the same as the lowest rate reported for TB in 30 years and lower than 1998 (0.33 per 100,000 population, Figure 6). These were 18 (62%) male deaths and 11 (38%) female deaths from TB. Twenty-seven (93%) deaths occurred in persons aged 50 years or more, and two TB deaths were registered in persons under 50 years of age.

Figure 6. Incidence rates of new TB and crude TB mortality rates, Australia, 1968 to 1999



Discussion

Australia continues to report one of the lowest TB incidence rates in the world. Other developed countries that have reported incidence rates of less than 5 per 100,000 or less in 1998 include Sweden, Malta and Norway.[2] From 1986 to 1997 annual incidence rates for TB in Australia stabilised at between 5 and 6 per 100,000 and in 1998 dropped below 5 per 100,000 population.[5-12] In 1999, the crude rate has risen to 6.1 per 100,000. A major reason for an increase in cases of tuberculosis in Australia in 1999 was the significant numbers of cases of tuberculosis found among refugees from Kosovo and East Timor given temporary resident visas under the 'Safe Havens' programs. The notifications from the Northern Territory included 61 cases among East Timorese evacuated to Darwin in September 1999,[13] boosting the Northern Territory caseload to 97 cases compared with the 1998 total of 31 cases. Significant numbers of Kosovar refugees given temporary residence in Victoria and New South Wales contributed to increased TB notifications in these States.[14,15] Since the ABS could not provide an estimate of the Australian resident population of people born in East Timor or Kosovo, the cases in nationals from these locations were included under Indonesia and Yugoslavia respectively. The rates per 100,000 in these communities rose from 73.9 to 229.4 for Indonesian-born Australian residents and from 5.4 to 22.6 for Yugoslavian-born residents (Table 5).

In 1999, 53 per cent of all TB notifications in overseas-born Australians were in those born in India, Indonesia, China, the Philippines or Vietnam. These 5 high-burden countries account for more than half of all new TB cases notified annually.[2] While the proportion of overseas-born cases represented in annual TB notifications in Australia has increased over the last decade, the national incidence rates of TB have not. In 1986, 60 per cent of TB notifications were overseas-born, compared to 70 per cent in 1990, 75 per cent in 1996, 77 per cent in 1998 and 83 per cent in 1999. For all years, with the exception of 1995, rates in the overseas-born have been between 15 and 17 per 100,000, but rose in 1999 to 21.3 per 100,000. In the Australian-born population there has been a decline in the proportion of all TB notifications as well as a progressive decline in incidence rates, from 2.8 per 100,000 in 1986 to 1.3 per 100,000 in 1999.

Over the last 7 years, rates of TB have been significantly higher in indigenous Australians compared to the non-indigenous, Australian-born population. Reporting accurately on trends in indigenous Australians has been made difficult by the shifts in the census denominator estimates for these populations and the inconsistent reporting of indigenous status by some jurisdictions. Among the risk factors for TB in indigenous Australians are poor socio-

Table 6. Notifications of tuberculosis and rate per 100,000, Australia, 1999, by age and indigenous status

Age group (years)	Indigenous Australians		Non-indigenous Australian-born	
	Number	Rate	Number	Rate
0-4	1	1.8	9	0.7
5-9	0	0.0	1	0.1
10-14	1	2.0	3	0.3
15-19	0	0.0	0	0.0
20-24	2	5.5	1	0.1
25-29	3	8.4	8	0.7
30-34	3	9.7	10	1.0
35-39	1	3.7	8	0.8
40-44	2	9.2	7	0.7
45-49	5	29.5	7	0.8
50-54	1	8.0	13	1.7
55-59	3	34.5	11	1.9
60-64	6	97.4	10	2.1
65-69	1	21.9	8	1.8
70-74	1	36.7	12	2.9
75+	4	123.8	50	7.0
Total	34	8.3	158	1.1

* One case among Australian born did not have indigenous status reported

economic status, diabetes, renal disease, smoking, and alcohol abuse and poor nutrition.[16]

There are few indications that the global TB threat is abating and there is evidence that incidence is increasing in some countries due to HIV co-infection. These findings reinforce the need for all nations to remain vigilant. Having a surveillance system in place that can accurately report on trends, and important changes in the epidemiology of TB, alerts public health authorities and policy makers to emerging problems and appropriate action. In spite of our gains in the control of TB, Australia's geographical location within the WHO Western Pacific Region that includes 7 high burden countries, some with close links to Australia, means that we too must continue to invest in TB prevention and control.

Acknowledgments

The members of the Communicable Diseases Network Australia are thanked for their co-operation with this surveillance initiative, together with the State and Territory Directors of Tuberculosis, and other health department personnel in the States and Territories who are involved in compiling the individual data sets.

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Retirement of David Dawson

Richard Lumb, Infectious Diseases Laboratories, Institute of Medical and Veterinary Science, SA

The publication of the Australian Tuberculosis Laboratory data for 1998 and 1999 in the current issue of *Communicable Diseases Intelligence* (*CDI*) marks the final contribution from Mr David Dawson, the Chief Scientist at the Queensland Diagnostic and Reference Laboratory for Mycobacterial Diseases at The Prince Charles Hospital, Brisbane.

David's leadership has been a crucial element in the reporting of tuberculosis (TB) laboratory data in Australia. In 1986, the Commonwealth Department of Health, Housing and Community Services ceased maintaining detailed national statistics on TB. David, as the then Convenor of the Special Interest Group in Mycobacteria within the Australian Society for Microbiology, believed that this action was a retrograde step and organised for laboratory data from the five State reference laboratories in Australia to be collated on an annual basis to report details of new bacteriologically positive cases of tuberculosis. These data were the only detailed source of information available in Australia until the publication of Tuberculosis Notification rates in *CDI* in 1992. A timely commentary 'Tuberculosis in Australia: an unfinished fight' in the *Medical Journal of Australia* (*Med J Aust* 1991:154) by David reiterated the need for accurate, comprehensive, and up-to-date statistics as part of an effective Tuberculosis Control Program. The State and Territory TB Directors, and others, were crying out for the same action, and finally, the surveillance system for tuberculosis was revised under the auspices of the Communicable Diseases Network Australia.

David has also been at the forefront in the laboratory diagnosis of mycobacterial diseases. He has published widely, particularly in investigations of atypical mycobacteria, and was among the first to identify and characterise clinical strains of *Mycobacterium haemophilum*. His knowledge of mycobacteria is encyclopaedic, and many scientists owe much to David for his enthusiasm for the topic and his readiness to impart that knowledge. His laboratory is part of the World Health Organization's Supranational Reference Laboratory Network and is also a WHO Collaborating Centre in Tuberculosis Bacteriology in recognition of his expertise in tuberculosis.

On behalf of the Australian Mycobacterial Laboratory Network, the Special Interest Group in Mycobacteria, and all your colleagues in Australia and overseas, we wish you all the best in your retirement, and many happy days at the beach house.

Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 1998-1999

Report of the Australian Mycobacterium Reference Laboratory Network

David Dawson, WHO Collaborating Centre in Tuberculosis Bacteriology, Queensland Health Pathology Services

Abstract

The Australian Mycobacterium Reference Laboratory Network collected and analysed laboratory data on new diagnoses of infection with *Mycobacterium tuberculosis* complex in 1998 and 1999. Totals of 700 and 760 cases were identified, representing annual reporting rates of 3.7 and 4.0 cases of laboratory confirmed tuberculosis (TB) per 100,000 population in the years 1998 and 1999 respectively. Australia's TB reporting rates have varied little in the past decade, ranging from 3.7 to 4.1 cases per 100,000 population. Reporting rates vary between States, reflecting differences in the distribution of persons in 'high-risk' categories for TB. The male:female ratio decreased to almost 1:1. The median age for males with culture-confirmed TB is in the 45-49 age group; for females, themedian is in the 35-39 age group. Pulmonary disease was diagnosed in 63 per cent of cases whereas disease of lymph nodes accounted for 21 per cent of all cases. Children have the lowest rates of culture-confirmed TB; males in the older age groups have the highest rates. Microscopy was positive for 60 per cent of culture-positive sputa, and for approximately 45 per cent of bronchoscopy specimens. The frequency of multi-drug resistance (less than 1%) was slightly lower than in previous years. *Commun Dis Intell* 2001;25:261-265.

Keywords: Mycobacterium *tuberculosis complex, laboratory network, tuberculosis, TB, drug resistance*

Introduction

Data from the World Health Organization (WHO) show that Australia's notification rate for tuberculosis (TB) — around 5 cases per 100,000 population — is among the lowest in the world.[1] There remains, however, an undeniable need for surveillance among Australia's population in order to ensure the continued efficacy of our national TB program. Furthermore, reports from WHO point to the huge TB burden in certain neighbouring countries: they reflect the difficulties in managing TB in the presence of high background rates of tuberculous infection when health care systems are fragmented and under-funded.[2] Drug resistance and co-infection with HIV are additional obstacles to TB control in developing countries.[3]

Data pertaining to TB in Australia come from 2 sources. Since 1991, the National Mycobacterial Surveillance System (NMSS) of the Communicable Diseases Network Australia New Zealand has provided statistics on cases reported to public health authorities in Australia's States and Territories.[4] The Australian Tuberculosis Reporting Scheme has been conducted by the Mycobacterium Reference Laboratory Network (MRLN) since 1986.[5] Whereas a proportion of cases registered with NMSS will have been identified through clinical and epidemiological criteria alone, the statistics compiled by the MRLN relate to diagnoses made by isolation of *Mycobacterium tuberculosis* complex. This report deals with laboratory diagnoses made in the years 1998 and 1999.

Methods

The data are based on patients submitting clinical samples from which *Mycobacterium tuberculosis* complex (MTBC, but excluding the BCG strain) are grown on culture. Due to the specialised nature of TB bacteriology, it can be assumed that the 5 laboratories that comprise the MRLN account for almost all, if not all, of the bacteriological diagnoses in Australia. Comparable bacteriological procedures are used in the reference laboratories. Relapse patients, that is, those previously diagnosed, treated and considered cured, were included in these data because laboratories cannot usually differentiate them from new cases. Temporary visitors to Australia are also included.

For each new laboratory diagnosis the following information was collected:

- demographic: patient identifier, age, sex, HIV status and state of residence
- specimen: type, site of collection, date of collection and microscopy result, and
- isolate: species of mycobacterium and results of drug susceptibility tests.

Data for 1998 and 1999 from contributing laboratories were submitted in standard format to the scheme co-ordinator for collation and analysis. Duplicate entries (as indicated by identical patient identifier and age) were deleted before analysis. Rates were calculated using the respective mid-year estimates of the population supplied by the Australian Bureau of Statistics.

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The nature of the first clinical sample that yielded an isolate of MTBC was used to record the nominal site of disease for individual cases. Culture-positive specimens collected at bronchoscopy, as well as gastric washings, were taken to identify cases of pulmonary disease. In cases of multi-site disease, provided a sputum sample was culture-positive, these cases were included among those listed as having pulmonary disease: the most significant category for public health purposes. Although many patients were known to have isolates from more than one body site, such data are of doubtful value for the laboratory-based report and were not collated. Similarly, it is not always possible to accurately categorise cases of miliary and disseminated disease from data available to laboratories.

Results

Total reports and distribution by State

Totals of 700 and 760 cases were recorded in 1998 and 1999 respectively. These figures represent annual rates of 3.7 and 4.0 cases of laboratory-confirmed TB per 100,000 population. The distribution of cases by state of residence is shown in Table 1. State-specific annual reporting rates varied from less than one (Tasmania, 1999) to 11.6 per 100,000 (Northern Territory, 1998). It should be noted that the data from Victoria includes 37 cases identified among Timorese refugees.

Causative organism

The large majority of cases were due to *M. tuberculosis*. However, in 1998 there were 5 isolates of *M. bovis* and two of *M. africanum*. In 1999, there were 4 isolates of *M. bovis* and three of *M. africanum* .

Distribution by gender, age and site of disease

Full information for gender, age and site of disease was submitted for 688 of the 700 cases recorded in 1998, and for 756 of the 760 cases recorded in 1999. Figure 1 shows the distribution of the 1444 cases by age group and gender. The overall male:female ratio was 0.96:1 in 1998 and 1.15:1 in 1999. For the 2 years combined it was 1.06:1. In both years, the median age group for all cases was 40-44 years. The median age group for males was 45-49 whereas that for females was 40-44 years. Age and gender specific rates varied from less than one per 100,000 population in children younger than 15 years to almost 30 per 100,000 per year in males over 80 years of age (data not shown). Nine cases were children younger than 10 years. Four children had disease in pulmonary sites, four had lymph node infections, and one had disease in a knee joint. There were no culture-confirmed cases of tuberculous meningitis in children. (**Editor's note:** There were 13 cases of tuberculous meningitis reported in the preceding TB Annual report, 1999 (pp 254-259). All but 2 of these were in adults. Of the two cases in children, one was culture-confirmed and one was diagnosed by microscopy.)

Figure 2 shows the distribution of 1,444 cases by site of disease and sex. Pulmonary infection was demonstrated in 63 per cent of the total cases (male:female ratio 1.3:1). Several patients with pulmonary disease were also proven to have disease in other body sites. Twenty-one per cent had disease of lymph nodes identified (male:female ratio 0.5:1). There were also 60 cases of pleural disease (male:female ratio 1.6:1) and 50 cases of disease in genito-urinary sites (male:female ratio 1.0:1).

Association with HIV

The reference laboratories reported no isolates of MTBC from persons known to be HIV+. For the majority of patients, however, HIV status was not recorded.

Figure 1. MTBC isolates, 1998 to 1999, by age group and sex

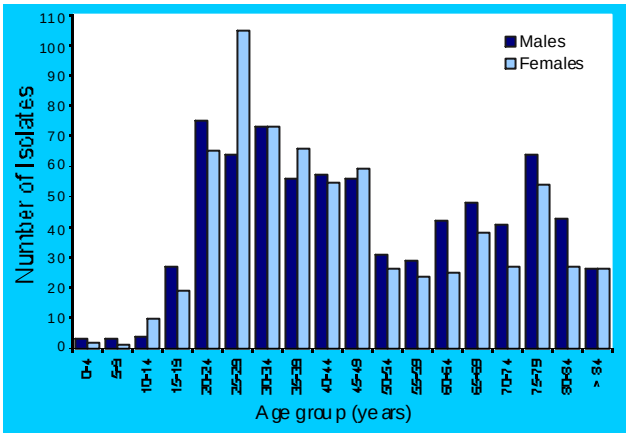
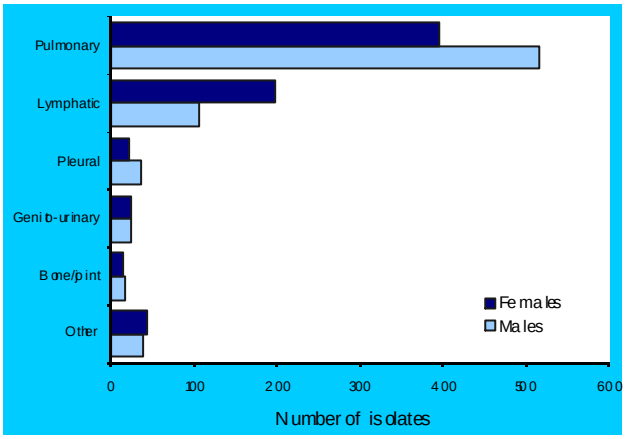


Table 1. MTBC isolates in Australia, 1996 to 1999, cases and rates per 100,000 population, by State or Territory

State	1999		1998		1997[1]		1996[1]	
	n	Rate	n	Rate	n	Rate	n	Rate
New South Wales[2]	291	4.3	289	4.4	329	5.0	341	5.3
Victoria	261	5.5	192	4.1	193	4.2	214	4.7
Queensland	75	2.1	85	2.5	74	2.2	90	2.7
Western Australia	64	3.4	66	3.6	51	2.8	51	2.9
South Australia	46	3.1	40	2.7	39	2.6	28	1.9
Tasmania	2	0.4	6	1.3	8	1.8	3	0.6
Northern Territory	21	10.9	22	11.6	28	15.0	23	12.6
Total	760	4.0	700	3.7	722	3.9	750	4.1

1. Data from previous reports of the MRLN.
2. Data for the Australian Capital Territory are included with those from New South Wales.

Figure 2. MTBC isolates, Australia, 1998 to 1999, by site and sex



Microscopy results

Acid-fast microscopy (AFM) results were available for 677 and 753 samples in 1998 and 1999 respectively (Table 2). In 1998, 60.3 per cent of 300 sputum samples that were positive on culture were also positive by AFM; the corresponding figures for 1999 were 60.9 per cent of 396 sputum samples. Bronchoscopy samples provided 87 diagnoses in 1998 in which AFM was positive for 41 (47.1%) infections. In 1999, 40 (43.5%) of 92 bronchoscopy collections were positive on smear.

In vitro drug susceptibility

In 1998, 699 of 700 isolates, and in 1999 all 760 isolates, were tested for *in vitro* susceptibility to the 4 drugs recommended for standard treatment of TB in Australia, i.e., isoniazid (H), rifampicin (R), ethambutol (E) and pyrazinamide (Z).[6] A total of 68 isolates (9.7% of the total) in 1998 were resistant to at least one of the standard compounds; the corresponding figure for 1999 was 59 (7.8%). The frequency of resistance to H, R, E and Z, alone or in combination, is shown in Table 3. Resistance to H and/or R was recorded in 65 isolates (9.2% of total) in 1998 and in 54 isolates (7.1%) in 1999. Resistance to both H and R ('multi-drug resistant TB', MDR-TB) was demonstrated in 6 isolates (0.9% of total) in 1998 and 4 (0.5%) isolates in 1999 (Table 4). All of the MDR isolates were *M. tuberculosis*. Eight of the 10 MDR isolates came from pulmonary sites; two were from cervical lymph nodes. Five patients were diagnosed from sputum samples, of which three were

Table 2. Frequency of positive microscopy in specimens yielding MTBC on culture in 1998 and 1999

	1999			1998		
	No	Smear positive	%	No	Smear positive	%
All specimens	753	368	48.9	677	303	44.7
Sputum	396	241	60.9	300	181	60.3
Bronchoscopy	92	40	43.5	87	41	47.1

Table 3. *In vitro* resistance of isolates to the standard anti-tuberculosis drugs, Australia, 1996 to 1999

	1999		1998		1997[1]		1996	
	No	%[2]	No	%[2]	No	%[2]	No	%[2]
Isoniazid (H)	52	6.8	63	9.9	48	6.6	73	9.7
Rifampicin (R)	6	0.8	8	1.1	15	2.1	16	2.1
Ethambutol (E)	2	0.3	4	0.6	4	0.6	2	0.3
Pyrazinamide[3] (Z)	6	0.8	9	1.3	24	3.3	18	2.3

1. Data taken from previous publications of the MRLN.
2. Percentage of strains tested which were resistant to drug alone or in combination with other drugs
3. All strains of *M. bovis* are naturally resistant to pyrazinamide; 5 *M. bovis* were identified in 1998; 4 *M. bovis* were identified in 1999.

Table 4. Drug resistance patterns in MDR strains, Australia, 1996 to 1999

Resistance pattern (standard drugs)[1]	No of isolates			
	1999	1998	1997	1996
H + R only	2	2	6	10
H + R + E	1	1	1	1
H + R + Z	1	2	5	4
H + R + E + Z	0	1	2	0

1. H = isoniazid; R = rifampicin; E = ethambutol; Z = pyrazinamide

positive by AFM. Six of 37 isolates from Timorese patients were resistant to H; none were MDR. Five isolates identified as *M tuberculosis* were recorded as resistant to Z alone. In addition to the standard drugs, streptomycin (S) was tested against the majority of isolates showing resistance to H and/or R as well as 348 isolates that were fully-susceptible to the standard drugs. Approximately 30 per cent of strains resistant to H and/or R are also resistant to S and a further 4 per cent of otherwise susceptible strains are resistant to S.

Discussion

The data for 1998-99 confirm the continuing stable situation for bacteriologically-confirmed TB in Australia: annual reporting rates of approximately 4 cases per 100,000 per year have been recorded for the past 10 years. The pathophysiology of TB dictates that not all cases reported from clinical sources will be confirmed by laboratories. This situation will apply even though an appropriate clinical sample was submitted to a competent laboratory for microbiological testing. Data from NMSS showed that 923 cases of tuberculosis were notified by State and Territory jurisdictions during 1998; reference laboratories confirmed only 700 cases in that year. This finding is in keeping with previous comparative data,[7] and suggest that in Australia, at present, only around 75 per cent of cases reported to NMSS are confirmed by culture. The Public Health Laboratory Network's definition of a laboratory-proven case of TB allows for detection of specific nucleic acid in clinical samples, in the absence of confirmation by a positive culture. Wider use of nucleic acid amplification assays, and a better appreciation of the clinical utility of such tests, may bring a reduction in the numerical disparity between clinical and laboratory diagnoses. Future reports from the MRLN will include diagnoses made by nucleic acid amplification tests.

The annual incidence rates in the various States and Territories range from below 2 per 100,000 in Tasmania to more than 11 per 100,000 in the Northern Territory (Table 1). Current data show only minor deviations from those in previous years. As would be expected, when compared to laboratory-derived data, the NMSS reports provide relatively similar state-specific differences in case numbers and reporting rates — which can be taken as validation of the parallel reporting processes. The differences in rates between States and Territories are almost certainly due to peculiarities in the demography of high-risk subgroups, rather than local differences in the risk of acquiring tuberculous infection.

Our previous report made mention of incomplete data relating to age, sex and site of disease.[7] The ascertainment level for these data is much improved; 688 of 700 cases in 1998, and 756 of 760 cases in 1999. Cases of active disease are distributed unevenly across age groups (Figure 1). The distributions shown in Figure 1 are almost identical to those in previous reports, and generally agree with statistics from notified cases. NMSS data show significantly more cases in young children, e.g. 28 reports in children under 10 years were notified to NMSS, whereas only 3 cases were confirmed by culture. This discrepancy can be explained by the fact that diagnosis of TB in young children is difficult, and is often based on clinical and epidemiological findings, rather than definitive laboratory results.[8]

There are signs that the overall male:female ratio – which has previously been reported as around 1.3:1 – is becoming closer to 1:1. For the current reporting period, there were almost equivalent numbers of males and females (1.06:1). NMSS data for 1988 generally agree with this observation. Our laboratory data show clear differences in sex-based distribution within the various disease categories. Pulmonary and pleural TB is more likely to be found in males, whereas the distribution of lymphatic TB is heavily skewed to females (male:female ratio, 0.5:1). Earlier studies by the MRLN have consistently found more cases of lymph node disease in females.

Pulmonary infection was demonstrated in 63 per cent of patients with culture-proven TB. The majority of patients who did not have pulmonary TB had disease of the lymph nodes (21 per cent of the total). In 1996-97 corresponding figures were 64 per cent and 19 per cent. The NMSS report for 1998 gives figures of 59.3 and 21.5 per cent for pulmonary and lymph node infection, respectively.[4]

Our data illustrate the continuing value of microscopy as a diagnostic tool (Table 2). Almost half of all samples that grew MTBC on culture were found positive on smear. Among patients with pulmonary disease, 60 per cent of the culture-positive sputum specimens were also positive by AFM. United States authorities report an equivalent statistic for AFM in that country.[9] We found that microscopy was less sensitive on bronchoscopy samples, detecting around 45 per cent of culture-positive samples. The 1998 report from NMSS indicated that only 32 per cent of pulmonary cases were smear-positive.[4] While this might suggest under-reporting of microscopy results in NMSS, it should be noted that the denominator in the NMSS statistic would include:

(i) diagnoses made from histology, and

(ii) diagnoses made on clinical findings alone.

The median age group for females with TB (40-44) is higher than in 1997 when it was 35-39. The median age group for males is unchanged at 45-49 years. NMSS data has shown different age distributions of Australian-born patients compared to counterparts born overseas, with the former generally being older than the latter.[4] Persons born outside Australia now account for three-quarters of Australian TB notifications and provide the majority of cases in the young and middle age groups.

Although there were no culture-proven cases listed as associated with HIV, this is almost certainly not the true picture. Reference laboratory databases are unlikely to show HIV status for patients under investigation for TB. NMSS reported 4 cases of HIV associated TB in 1998.[4]

This report provides recent data on *in vitro* drug resistance among isolates from Australian patients with TB. To date, such information is not available through NMSS. We found that 9.7 per cent and 7.8 per cent of isolates during 1998 and 1999 respectively, had in vitro resistance to at least one of the standard anti-TB drugs, H, R, E or Z. The corresponding figures for previous years were: 1997 (9%); 1996 (11%); 1995 (9%); 1994 (7%). Resistance to one or both of H and R — the most effective anti-tuberculosis compounds — was detected in 9.2 per cent and 7.1 per cent of isolates in 1998 and 1999 respectively. Corresponding figures for previous years were: 1997 (6.9%); 1996 (9.9%); 1995 (7.5%); 1994 (6.1%). We found 6 (0.9%) isolates in 1998 and 4 (0.5%) in 1999 that were resistant to H and R, i.e., were MDR. The

corresponding figures for previous years were: 1997 (1.9%); 1996 (2%); 1995 (0.7%); 1994 (0.3%). Our findings thus show no significant temporal changes in the prevalence of drug resistance among Australian isolates.

Laboratories do not have the information necessary to stratify patients on the basis of prior anti-tuberculosis therapy. It is therefore not possible to categorise resistance as one of 'primary' or 'acquired'. The supplementary dataset now established for cases notified to NMSS has the potential to allow more precise correlation of drug resistance with a patient's country of birth and treatment history. Such data are required by the Global Project on Anti-tuberculosis Drug Resistance Surveillance. Anecdotal evidence suggests that acquired drug resistance is rarely seen in patients receiving treatment in Australia. Furthermore, drug resistance is almost always linked to persons born outside Australia, or to the Australian-born who acquire tuberculous infection when travelling overseas (Dawson, unpublished). This observation, combined with the demonstrated low prevalence of drug resistance, justifies the conclusion that transmission of a drug-resistant strain of MTBC is an uncommon event in this country. Outbreaks of drug-resistant TB are unquestionably rare in Australia.

Molecular studies of resistant isolates of MTBC have defined the specific mutations that confer their resistance to the anti-tuberculosis drugs.[10] These studies have provided insight into the mechanisms of drug-resistance and through correlation with MIC values have confirmed earlier reports of strains with 'low-level' and 'high-level' resistance to H.[11] A significant proportion of H-resistant isolates have MIC values of 0.1 mcg/ml, while others are resistant at 0.4 mcg/ml. The clinical implication of these findings have not been proven, although the argument that 'low-level' resistance is likely to respond to standard therapy with H, would seem tenable. All Australian reference laboratories are now testing for resistance at both levels of H. Future reports will attempt to provide more detailed data on this topic.

Acknowledgements

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

Queensland Health Pathology Services, The Prince Charles Hospital, Chermside, Queensland.

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

Victorian Infectious Diseases Reference Laboratory, North Melbourne, Victoria.

Institute of Medical and Veterinary Sciences, Adelaide, South Australia.

Western Australian Centre for Pathology and Medical Research, The Queen Elizabeth II Medical Centre, Nedlands, Western Australia.

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Networking for health protection:
the Communicable Diseases Network Australia

Peter Lindenmayer, CDNA Secretariat

The Communicable Diseases Network Australia (CDNA) has been an active participant in communicable disease activities (under a number of different names) for over a decade. However, because much of its work is behind the scenes and away from the public eye, not many people outside the Network know very much about CDNA, what it does or who is involved. This article is intended to fill in some of this background, and to provide an update on CDNA's recent activities.

Background

What is now CDNA started life in 1989 as the 'Australian Communicable Disease Control Network', a joint initiative of the National Health and Medical Research Council (NHMRC) and the Australian Health Ministers' Advisory Council (AHMAC). AHMAC anticipated[1] that the Network would be a means to ensure improved national co-ordination in the areas of:

- data collection and analysis;
- surveillance;
- training;
- research; and
- policy development.

With the inclusion of New Zealand as an active member, the Network adopted the title Communicable Diseases Network Australia New Zealand (CDNANZ), and was known by this name for many years. In April 2001 it became the Communicable Diseases Network Australia. The new title better reflects the fact that the Network is funded and supported by Australian jurisdictions, and its primary focus and responsibilities are in relation to Australia. However, CDNA continues to recognise the importance of international communication and collaboration on communicable disease matters. New Zealand remains a valued member and the Network is actively developing its links with other regional neighbours.

With the formation of the National Public Health Partnership (NPHP) in 1997, CDNA became one of the key committees reporting to the NPHP (and thence to AHMAC), linking the Network more strongly with the broader public health field.

Membership

CDNA's membership consists of the heads of communicable disease control units in all Australian State and Territory health authorities, the Director of the Surveillance Section of the Commonwealth Department of Health and Ageing (DHA) formerly the Department of Health and Aged Care, plus a number of representatives of other agencies with expertise in communicable disease matters (Table 1). During the last year CDNA was very pleased to welcome 2 new member organisations — OzFoodNet, represented by co-ordinator Martyn Kirk, and the Secretariat of the Pacific Community (SPC), represented by Dr Yvan Souares. Although at this stage Papua New Guinea and East Timor are not members of the Network, CDNA liaises with health authorities there on communicable disease matters.

Activities

The original concept of the Network envisaged a body with its own specialist staff seconded from government departments and other agencies, providing a direct capacity to undertake the tasks assigned to it. For various reasons this aspect of the Network did not develop, and instead DHA (and its predecessors) has provided secretariat services and other support where this was considered appropriate — and as resources allowed. One consequence has been that many of those original tasks outlined by AHMAC have been undertaken by other bodies on behalf of CDNA. For example, national communicable diseases surveillance for most notifiable diseases is undertaken by DHA's Surveillance Section, with other agencies such as the National Centre for Immunisation and Research of Vaccine Preventable Diseases and the National Centre for HIV Epidemiology and Clinical Research also contributing. Similarly, the ANU's National Centre for Epidemiology and Population Health (NCEPH) has initiated the Master of Applied Epidemiology (MAE) program with strong ongoing support from CDNA.

Another consequence of the absence of a dedicated specialist staff has been that CDNA representatives (or their staff) themselves undertake much of the immediate work of the Network, sometimes a heavy burden for already busy communicable diseases managers and units, and at times a significant limiting factor for the Network's activities.

The best known of CDNA activities are the regular fortnightly national teleconferences, attended by all members (often with other colleagues and staff sitting in). The teleconferences commence with a series of 'status reports', in which each member organisation provides a brief report on notable developments in its area of responsibility. These reports allow members to keep abreast of the rapidly changing picture of communicable diseases across the country and the region, to seek advice on handling some of the more difficult issues that arise, and to co-ordinate responses across jurisdictions and other agencies. Many of these reports refer to evolving investigations which may have significant health, economic or social consequences. Confidentiality is therefore an important issue and the Network operates on the principle that any information provided to members remains in the 'ownership' of the organisation providing it, and should only be used by others for relevant public health purposes.

In addition, the Network holds 'special teleconferences' when events demand more immediate responses or to consider a matter in more depth. Some policy discussion

Table 1. Current membership of CDNA

Current CDNA members	
Chair: Dr Greg Stewart, Acting Chief Health Officer, New South Wales Former Chair: Dr Shirley Bowen (Chief Health Officer, Australian Capital Territory, 2000-2001)	
Jurisdictional members	
Dr Eddie O'Brien	Australian Capital Territory
Dr Jeremy McAnulty	New South Wales
Dr Vicki Krause	Northern Territory
Dr Robert Hall	South Australia
Dr Avner Misrachi	Tasmania
Dr Linda Selvey	Queensland
Dr Graham Tallis	Victoria
Dr Gary Dowse	Western Australia
Dr Angela Merianos	Commonwealth (Surveillance Section, Department of Health and Ageing)
Dr Douglas Lush	New Zealand
Dr Yvan Souares	Secretariat of the Pacific Community
Non-jurisdictional members	
Ms Mary Beers	National Centre for Epidemiology and Population Health
Dr Chris Bunn	Department of Agriculture, Fisheries and Forestry — Australia
Professor Margaret Burgess	National Centre for Immunisation Research and Surveillance (of Vaccine Preventable Diseases)
Dr Scott Crerar	Australia New Zealand Food Authority
Professor Lyn Gilbert	Australian Society for Microbiology
Mr Gerard Fitzsimmons	Australian Institute of Health and Welfare
Dr David Smith	PHLN Representative
Professor John Kaldor	National Centre in HIV Epidemiology and Clinical Research
Mr Martyn Kirk	OzFoodNet
Professor Aileen Plant	Curtin University, Western Australia
Sqdn Ldr Anne-Marie Pope	Australian Defence Force

and development also occurs at (both regular and special) teleconferences, but the Network usually holds at least two face-to-face meetings each year to provide a better opportunity to do this in-depth work.

Committees

When work of a more specialised nature is required, this is often assigned to one of a number of standing committees or short term working groups, which generally bring in other people with particular expertise in the fields of interest. Committees and working groups that have operated during 2001 are listed in Table 2.

Recent achievements

The nature of much of its work means that the achievements of CDNA are not always visible or easily measurable. Those closely involved will certainly recognise that outcomes such as earlier or more appropriate responses to outbreaks, better policies on communicable disease management, improved co-ordination of preventive activities, among others, do make an important contribution to public health objectives — containing the spread of disease, reducing morbidity and the burden of communicable diseases on the acute system and maintaining productivity levels.

Although these achievements may not often be quantified, the fact that each fortnight around 20 busy members make it a priority to join a one hour teleconference is one indication of the value of these activities. Even with the development of

Table 2. CDNA committees

CDNA Committees operating during 2001
Communicable Diseases Control Conference Organising Committee
Hepatitis C Surveillance Committee (now Viral Hepatitis Surveillance Committee)
Infection Control Guidelines Steering Committee
Influenza Pandemic Planning Committee
Meningococcal Disease Control Guidelines Committee
National Arbovirus Advisory Committee
National Enteric Pathogens Surveillance System Steering Committee
National Tuberculosis Advisory Committee
Pneumococcal Working Party (co-auspiced with ATAGI[2])
Public Health Laboratory Network
STI Surveillance Committee

specialised e-mail listservers and Internet sites, the teleconferences seem to remain a most effective way to keep a high level of common understanding among a far-flung and varied group of people in the field.

Conclusively demonstrating the benefits of these connections is generally more difficult, but the recent anthrax scare and other bioterrorism issues did provide an example of the effectiveness of these types of networks. During the days and weeks following the first anthrax cases in the United States and the subsequent rash of threats, CNDA's jurisdictional members communicated regularly via teleconferences and e-mail, keeping all jurisdictions up to date on developments and sharing drafts of incident management protocols tailored to meet Australia's needs. New Zealand was also included in this process. CDNA's Public Health Laboratory Network (PHLN) members were an integral part of the response, ensuring that key laboratories all around the country were aware of the most appropriate methods for testing suspect materials and facilitating specialist training where this was required. These communications were in turn linked to higher level decision-makers within each health authority.

As a result of this work, a relatively consistent national response to these incidents was developed, and all jurisdictions were able to share and benefit from work done elsewhere. Mobilising such systems in emergencies depends heavily on having effective infrastructure already in place. A really effective infrastructure consists not only of appropriate hardware, training and systems, but also requires a common understanding of issues, knowledge of and respect for particular areas of expertise, mutual trust and established relationships — all of which are fostered by Networks such as CDNA and PHLN.

As well as creating and supporting this type of infrastructure, there are also more tangible examples of CDNA's work, many of them the product of committees that draw in a wider circle of expertise. Among the recent achievements this year have been:

Improvement to the National Notifiable Diseases Surveillance System

The National Notifiable Diseases Surveillance System (NNDSS) is operated by the Surveillance Section of the Department of Health and Ageing on behalf of the Network. Although this has been running for some years, following agreement by all Australian jurisdictional CDNA members in late 2000, for the first time Australia now has a list of nationally notifiable diseases that are reported on by all state and territory health authorities. Standard case definitions, reporting systems and access protocols are now being finalised. Web-based systems allow wide access to summary NNDSS information, while researchers are able to obtain more detailed data with the approval of the jurisdictions, which retain ownership of their material.

A number of these diseases (such as tuberculosis, invasive pneumococcal disease and incident hepatitis C) have also been identified as priorities for the development of enhanced surveillance systems that will allow a better understanding of the factors associated with disease spread and the impact of control measures such as vaccination.

Communicable Diseases Control Conference

Held in Canberra at the beginning of April 2001, the Communicable Diseases Control Conference was well attended and highly rated by participants for topical content and general value. Key note speakers included the Minister for Health and Aged Care, the Commonwealth's Chief Medical Officer, international experts such as Professor Roy Anderson (modelling and planning for BSE and TSEs), Dr Diana Martin (Meningococcal disease) and Dr Allan Hogue (risk assessment of foodborne and waterborne diseases) and many excellent local presenters. The conference provided a valuable opportunity for information exchange and discussion across a range of disciplines and regions, and there was a clear consensus that CDNA should hold another conference in 2 years. (For those with long term diaries, this will again be held in conjunction with NCEPH's MAE Conference in Canberra, probably late March 2003.)

Publication of *Guidelines for the early clinical and public health management of meningococcal disease*

Meningococcal disease is of significant (and arguably growing) concern to the general public; it is a disease that needs careful public health management. The new guidelines are a major revision of the NHMRC's 1996 meningococcal guidelines, and were completed by a CDNA Committee following wide national and international consultation.

Development of guidelines on Australian bat lyssavirus

Although there have only been two human cases of Australian bat lyssavirus since 1996, the existence of lyssavirus in Australian bat populations and the apparent 100 per cent fatality rate of human infections means that the disease poses a potential threat to the Australian population. With input from an expert committee, CDNA has developed information brochures for medical practitioners, veterinarians and the general public on the prevention and management of lyssavirus infection.

Revision of *Infection control guidelines in the health care setting*

The Infection Control Guidelines Steering Committee of CDNA has been working on the revision and expansion of this major document, which will provide an updated set of standards on infection control for the health sector. The work has involved detailed research on international standards, public consultation, the evaluation of numerous submissions from interested groups, and joint activities with other expert committees and individuals. Needless to say, the evolving picture of emerging diseases, particularly transmissible spongiform encephalopathies, has meant that the Committee's work has had to incorporate the latest technical findings and consider the complex public policy issues that result from these developments. It is hoped that the final version of the guidelines will be available by early 2002.

Public health management of communicable diseases among asylum seekers

Significant numbers of people seeking asylum in Australia are accommodated in detention centres located in a number of different States and Territories. The nature of the entry and country of origin of these people often raises communicable disease issues that differ from those of other entrants to the country. Until recently, different Australian jurisdictions had varying protocols with respect to the control of communicable diseases in these detention environments. CDNA has provided a forum for the discussion and

alignment of these policies, with a view to protecting the health of both the detainees and the Australian community. Discussions with the Department of Immigration and Multicultural Affairs have occurred on how the objectives and standards proposed by CDNA can be best met in the widely varying circumstances in which detainees are held.

Formulation of a National TB Strategic Plan

The National TB Strategic Plan was developed by CDNA's National Tuberculosis Advisory Committee. It identifies updated priorities and strategies to ensure continued improvement in Australia's control of TB, and provides linkage with regional and global programs under WHO's auspices. Australia has achieved TB levels that are among the lowest in the world and most programs operate very effectively, but in this elimination phase of the disease it is important to ensure that strategic directions are identified and efforts are maintained.

Laboratory initiatives

The PHLN, the largest of the committees reporting through CDNA, continues to be very active in a number of areas important to laboratory aspects of communicable disease control. PHLN meets by teleconference each month and plays a key role in maintaining and improving the level and co-ordination of the laboratory work that is an essential underpinning for effective surveillance activities. For example, its ongoing program of revising laboratory case definitions helps ensure that new techniques are evaluated and brought into standard use when supported by expert opinion.

PHLN's Laboratory Infection Containment Project (funded by NPHP) is designed to enhance current safeguards against the spread of infectious micro-organisms from laboratories. The project recently completed its first stage, an overview report of the national situation and relevant international developments, and stage two will involve consultation with a broad range of industry stakeholders. PHLN has also been involved in an assessment of the economics of public health laboratory functions in all jurisdictions.

Internal functioning

Over the past 12 months CDNA has taken a number of steps to improve its effectiveness. In conjunction with the Department of Health and Ageing (which provides the secretariat function), it instituted changes in the secretariat that enable more focussed support for Network members and functions. This has also clarified the primary role of the secretariat in supporting the Network as a whole.

In addition, CDNA has commenced a strategic planning process that is intended to provide a better understanding of the changing context of public health communicable disease control, and the ways in which the Network can best contribute. This work has included in-depth interviews with a range of significant stakeholders and consideration of models from overseas. The Network will use this information to continue to develop, retaining those essential core elements while evolving in response to changing needs.

Over the decade since CDNA was established there has been a renewed appreciation of the actual and potential impact of communicable diseases on health status and economic activity. Although the model for the Network originally envisaged by AHMAC did not eventuate, in most of the areas set out in the original charter, CDNA has made important contributions. Data collection is more comprehensive; surveillance is more sophisticated; the MAE program (in particular) is providing high quality training; and the range and quality of relevant policies has been enhanced. Only in the area of research has CDNA not taken an active role, and it is arguable that this was not appropriate or realistic in the first place. As CDNA's strategic planning process is finalised, and with other developments in the field, the Network and the public health system as a whole needs to consider how effectively the present structure of the Network can serve the emerging biological and organisational environments of the future.

References

1. Minutes, first meeting of the Interim Board of Management of the Australian Communicable Diseases Control Network, 8 December 1990.
2. The Australian Technical Advisory Group on Immunisation (ATAGI) reports to the Commonwealth Minister for Health.

New Chairs of CDNA and PHLN

Dr Shirley Bowen steps down from her position as CDNA Chair at the termination of her two year appointment at the end of November 2001. Shirley will also be leaving her position as the Australian Capital Territory's Chief Health Officer to take up a new appointment in Western Australia. The National Public Health Partnership has appointed Dr Greg Stewart, Acting Chief Health Officer in New South Wales, as the new Chair of CDNA.

Professor Lyn Gilbert has resigned from the Chair of PHLN, and has been succeeded by Dr David Smith. Lyn will remain on PHLN, and will continue to be the Australian Society for Microbiology representative on CDNA.

The Network offers its sincere thanks to Dr Bowen for her work as Chair over the last two years, and welcomes its new members

OzFoodNet: enhancing foodborne disease surveillance across Australia

Quarterly report, April-June 2001

Martyn Kirk for the OzFoodNet Working Group[1]

Introduction

OzFoodNet is a collaborative network conducting applied research into foodborne disease. It was established by the Commonwealth Department of Health and Ageing, formerly the Department of Health and Aged Care.[1] During the second quarter of 2001, OzFoodNet prepared several protocols for national studies investigating the burden and causes of foodborne disease. OzFoodNet epidemiologists assisted with investigations into several outbreaks of foodborne illness, including a major outbreak of *Salmonella* Bovismorbificans 32 associated with iceberg lettuce, and Australia's first outbreak of multi-drug resistant *Salmonella* Typhimurium Definitive Type (DT) 104.

This second quarterly report of OzFoodNet summarises the reporting of foodborne disease in the six States of Australia during the second quarter of 2001.[2] During this time, the Australian Capital Territory and the Northern Territory participated as observers in OzFoodNet, and we have not included data from these jurisdictions unless specified.

Notifications in the second quarter

In this report we have used the date that the health department received notifications, unless specified otherwise.

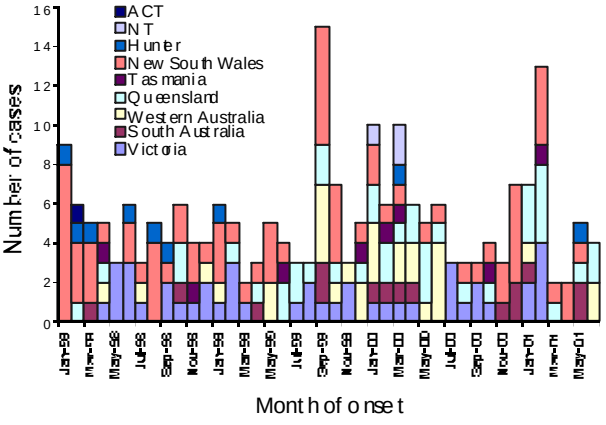
During the quarter, OzFoodNet sites reported 3,551 notifications of campylobacteriosis (excluding New South Wales). The median ages of cases ranged between 24–32 years. All States reported that the male to female ratio of cases was approximately 1:1 except South Australia (1.4:1). The Tasmanian Health Department reported a large increase in the rate of *Campylobacter* infections in the northern part of Tasmania, although no source was identified. The South Australian Department of Human Services investigated one point source outbreak of *Campylobacter* infection associated with a restaurant meal.

OzFoodNet sites reported a total of 1,672 cases of salmonellosis during the second quarter and identified the source of 10 *Salmonella* outbreaks. The median ages of reported cases in all States ranged from 15–24 years old, except for Queensland where the median age was 9 years. OzFoodNet sites reported that *Salmonella* Typhimurium (phage types 64, 135 and 126), Virchow (8 and 36var1) and Enteritidis were the most common serovars (Table 1). The majority of these *Salmonella* Enteritidis infections were acquired overseas.

The Tasmanian OzFoodNet Site reported that *Salmonella* Mississippi — an endemic serovar in this State — was the most common *Salmonella* infection for the quarter. Tasmania reported one household cluster of S. Mississippi, although no source was identified. Both Queensland and New South Wales OzFoodNet sites reported that *Salmonella* Birkenhead was common, which relate to an endemic focus of this serovar in southeast Queensland and northern New South Wales. During the quarter, State and Territory Health Departments commenced investigations into a variety of *Salmonella* serovars, including: Bovismorbificans (phage types 32 and 14), Muenchen, Typhimurium (phage types 126, 135 and 104) and Virchow (phage type 8).

State health departments received 11 notifications of listeriosis during the second quarter of 2001, which compared to 22 for the same quarter in 2000. Median ages for cases ranged from 38.5 to 70 years. South Australia reported one foetal infection in an infant of 34 weeks gestation. The mother had previously consumed soft cheese. There is little seasonality to notifications of *Listeria* infection (Figure). Despite the small numbers of cases of

Figure. Notifications of listeriosis, Australia, 1998 to June 2001, by States and Territories, and month of onset



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The full membership of the OzFoodNet Working Group is listed at the end of this report.

The OzFoodNet Working Group is (in alphabetical order):

Rosie Ashbolt (Tas), Meredith Caelli (Hunter PHU), Scott Crerar (ANZFA), Craig Dalton (Hunter PHU), Rod Givney (SA), Joy Gregory (Vic), Gillian Hall (NCEPH), Brigid Hardy (AFFA), Geoff Hogg (MDU), Rebecca Hundy (SA), Martyn Kirk (ANZFA), Vanessa Madden (Tas), Ian McKay (DHA), Lynn Meuleners (WA), Geoff Millard (ACT), David Peacock (NT), Nittita Prasopa-Plaizier (Vic), Paul Roche (DHA), Russell Stafford (Qld), Nola Tomaska (NCEPH), Leanne Unicomb (Hunter PHU), Craig Williams (ANZFA)

Table 1. Top five *Salmonella* infections reported to OzFoodNet sites, April to June 2001, by date of receipt

OzFoodNet site	Top five *Salmonella* infections, by type	Number of cases				
		2nd quarter 2001	2nd quarter 2000	YTD 2001	Total 2000	Ratio[1]
Queensland	Virchow 8	59	66	123	126	0.89
	Saintpaul	40	90	106	132	0.44
	Bovismorbificans 32	36	0	36	1	-
	Birkenhead	33	21	96	55	1.57
	Typhimurium 126	27	1	35	2	27.0
Hunter	Typhimurium 64	4	3	7	14	1.33
	Typhimurium 44	2	0	5	0	-
	Typhimurium 170	2	0	3	1	-
	Typhimurium 135	1	3	7	10	0.33
	Enteritidis	1	1	1	1	1.00
New South Wales	Typhimurium 135	34	34	25	138	1.00
	Typhimurium 9	29	47	30	115	0.61
	Birkenhead	18	17	12	77	1.05
	Typhimurium 64	17	21	55	53	0.81
	Stanley	10	5	11	56	2.00
South Australia	Typhimurium 126	29	1	74	4	29.00
	Typhimurium 9	14	7	36	28	2.00
	Typhimurium 64	9	4	22	20	2.25
	Typhimurium 108	5	3	6	9	1.70
	Virchow	5	2	15	1	2.50
Tasmania	Mississippi	31	19	86	69	1.60
	Typhimurium 9	5	4	9	21	1.30
	Enteritidis 4	2	3	2	5	0.67
	Enteritidis 1	2	1	2	1	2.00
	Typhimurium 135	2	1	2	5	2.00
Western Australia	Typhimurium 135	5	59	49	68	0.08
	Typhimurium 22	3	2	5	2	1.50
	Typhimurium 4	3	7	18	13	0.43
	Typhimurium 8	3	0	4	1	-
	Bovismorbificans	2	0	7	0	-
Victoria	Typhimurium 9	33	55	89	186	0.60
	Typhimurium 135	20	21	63	71	0.95
	Typhimurium 4	17	2	54	37	8.50
	Virchow 36var1	11	10	20	19	1.10
	Virchow 34	8	18	24	60	0.44

1. Ratio of number of cases reported in second quarter 2001 to second quarter 2000

listeriosis notified to individual jurisdictions, there is potential for cross-border or national outbreaks.

OzFoodNet sites reported 10 cases of shiga toxin producing *E. coli* infections during the quarter, with South Australia and Queensland each reporting four cases. Investigators did not identify any sources and all cases appeared sporadic. The median ages ranged from 14 to 71 years.

Foodborne disease outbreaks

During the second quarter of 2001, OzFoodNet sites reported 16 outbreaks that were potentially related to food (Table 2). These outbreaks affected approximately 224 people, of which 24 were hospitalised and one died. The Victorian Department of Human Services identified helva imported from Turkey, as the source of the first Australian outbreak of multi-drug resistant *Salmonella* Typhimurium DT 104.* This serovar is a significant problem for countries in the northern hemisphere, due to its propensity to affect many different types of animals, and the antibiotic resistance of the serovar.[3] The Queensland Department of Health identified iceberg lettuce that was contaminated during processing as the source of an outbreak of *Salmonella* Bovismorbificans 32 that affected people across Queensland.

Table 2. Outbreaks reported by OzFoodNet sites, April to June 2001

State	Month of outbreak	Setting	Agent responsible	Number exposed	Number affected	Responsible vehicles
Hunter	June	Take-away pizza shop	Unknown	Unknown	4	Pizza
	May	Kebab shop	Unknown	Unknown	2	Suspected chicken kebab
	May	Supermarket	Unknown	Unknown	3	Suspected BBQ chicken
	May	Take-away pizza shop	Unknown	Unknown	8	Pizza
Qld	June	Caterer	Suspected Norwalk	14	10	Unknown
	June	Home	Ciguatera	3	3	Barracuda (Sphyraena jello)
	June	Hotel	*S.* Montevideo	Unknown	8	Unknown
	May	Take away restaurant	*S.* Bovismorbificans 32	Unknown	36	Commercially processed iceberg lettuce
SA	June	Café	*S.* Zanzibar	Unknown	2	Suspected chicken dish
	June (investigations still continuing)	Community	*S.* Typhimurium 126	Unknown	44 to end of June; 72 to 10th August 2001	Chicken products
	May	Restaurant	Campylobacter jejuni	13	10	Unknown
Tas	April	Household	*S.* Mississippi	7	7	Unknown
	April	Household	*S.* Typhimurium 9	6	6	Suspected duck eggs
Vic	April	School Camp	*S.* Typhimurium 9	55	29	Unknown
	June	Community	*S.* Typhimurium DT104	Unknown	23 (20 Vic; 2 NSW;1 Qld)	Turkish Helva
WA	June	Restaurant	*S.* Typhimurium 64	~40	29	Fried icecream

Applied research

The OzFoodNet collaboration has achieved some important goals during the first six months of 2001, which included:

- development of a National survey of diarrhoeal disease through the National Centre for Epidemiology and Population Health;
- development of protocols for four national case control studies to examine risk factors for campylobacteriosis, listeriosis, *Salmonella* Enteritidis, and shiga-toxin producing *E. coli*;
- development of a national outbreak register for foodborne disease;
- communicating about clusters of foodborne diseases, through a fortnightly cluster report;
- comparing molecular typing methods for *Campylobacter*;
- development of a national survey of pathology laboratories;
- assisting with the investigation of several important clusters and outbreaks of foodborne disease that have crossed jurisdictional boundaries;
- communication with international agencies involved in similar international work; and
- formation of an important forum for discussing issues relating to foodborne disease.

There are many important areas of foodborne disease surveillance, which OzFoodNet cannot adequately address in this first phase of work. Some examples of this further work include: estimating the cost of foodborne disease, determining the fraction of foodborne disease that is notified to health authorities, novel means of detecting outbreaks, risk factors for other foodborne pathogens, etc. OzFoodNet is currently developing a paper on the potential future of ongoing collaborative work to improve our understanding of foodborne disease.

References

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2. O'Grady K, Powling J, Tan A, Vulcanis M, Lightfoot D, Gregory J, et al. Salmonella Typhimurium Dt104 — Australia, Europe. ProMed Mail, 22 August 2001, www.promedmail.org (Accessed 12 November 2001).
3. Crerar SK, Nicholls TJ, Barton MD. Multi-resistant Salmonella Typhimurium DT104 — implications for animal industries and the veterinary profession. *Aust Vet J*1999;77:170–1.

Erratum

The Table 1 headings of the OzFoodNet report in the August issue of *CDI* were incorrect. The corrected table is reprinted below. *CDI* apologises for this error.

Table 1. Top five serovar of *Salmonella* notified to OzFoodNet sites, January to March 2001, by date of onset

State		First quarter 2001	First quarter 2000	Total 2000	% change
New South Wales	Typhimurium 135	52	28	121	85.7
	Typhimurium 135a	28	3	13	833.3
	Typhimurium 9	38	44	136	-13.6
	Typhimurium 64	31	33	76	-6.1
	Birkenhead	26	23	76	13.0
Hunter PHU	Typhimurium 135a	7	0	0	
	Typhimurium 135	5	2	10	150.0
	Typhimurium 64	3	5	13	-40.0
	Typhimurium 9	3	2	3	50.0
	Typhimurium 44	2	0	0	
Queensland	Birkenhead	62	35	97	77.1
	Saintpaul	61	44	185	38.6
	Virchow 8	48	66	175	-27.3
	Typhimurium 135	42	57	127	-26.3
	Aberdeen	27	25	58	8.0
South Australia	Typhimurium 9	19	8	28	137.5
	Typhimurium 135	19	1	3	1800.0
	Typhimurium 126	15	0	4	
	Typhimurium 64	13	4	20	225.0
	Chester	9	6	18	50.0
Tasmania	Mississippi	56	36	73	55.6
	Typhimurium 9	3	7	22	-57.1
	Bovismorbificans	1	0	1	
	Enteriditis 1	1	1	1	0.0
	Meunchen	1	0	0	
Victoria[1]	Typhimurium 9	51	58	178	-12.1
	Typhimurium 135	43	23	68	87.0
	Typhimurium 4	37	7	38	428.6
	Virchow 34	16	37	60	-56.8
	Typhimurium 170	15	9	36	66.7

[1] Victorian data reported by date of receipt at the Victorian Department of Human Services.

Surveillance of antibiotic resistance in *Neisseria gonorrhoeae* in the WHO Western Pacific Region, 2000

The WHO Western Pacific Gonococcal antimicrobial Surveillance Programme[1]

Abstract

A long-term program of surveillance of antimicrobial resistance in *Neisseria gonorrhoeae* isolated in the World Health Organization's Western Pacific Region (WHO WPR GASP) continued in 2000. About 11,000 gonococci were examined in 15 focal points. Widespread resistance to the penicillin group of antibiotics was confirmed. Resistance to quinolone antibiotics, already widely dispersed, increased further with a shift to higher levels of resistance in many centres. Gonococci with decreased susceptibility to third generation cephalosporins were observed in 5 centres. Spectinomycin resistance was infrequently encountered. Options for cheap and effective treatment of gonorrhoea in the WPR are increasingly limited. *Commun Dis Intell* 2001;25:274-277.

Keywords: surveillance; Neisseria gonorrhoeae*; antimicrobial resistance; gonorrhoea; antibiotics; quinolones; penicillins; spectinomycin; cephalosporins*

Introduction

Early and successful antibiotic treatment of gonococcal infection is important not only for the individual patient but is also a significant factor in control of disease and the prevention of complications. The ability of *Neisseria gonorrhoeae* to become resistant to cheap and effective antibiotics is well recognised and has significantly compromised both individual and public health management of gonorrhoea. Because treatment of gonorrhoea is best given as single dose treatment on initial diagnosis, standardised treatment schedules have been established. However, antibiotic resistance in the gonococcus can arise quickly. It is therefore important to have available accurate data on antimicrobial resistance in the gonococcus in order to guide selection of an appropriate antibiotic treatment. Antibiotic resistance in gonococci often spreads rapidly between countries, and infected travellers often present for treatment in countries distant from the place of contact. Thus for a number of reasons it is important to have available regional as well as local data on antibiotic resistance.

The WHO Western Pacific Region (WPR) Gonococcal Antimicrobial Surveillance Programme (GASP) is a continuing program of susceptibility surveillance in the Region and has published surveillance data annually since 1992.[1] The Region has an unfortunate history of development of antimicrobial resistance in gonococci with penicillin, spectinomycin and quinolone resistant *N. gonorrhoeae* all appearing in and spreading beyond the WPR. This communication provides an analysis of surveillance of antimicrobial resistance in *N. gonorrhoeae* in the WHO WPR in 2000.

Methods

The methods used by the WHO WPR GASP have been published[2] and provide full details of the source of isolates, sample populations, laboratory test methods and quality assurance programs used to generate data. These methods were unaltered in 2000. Most isolates were collected from symptomatic STD clinic patients. As a guide to the interpretation of the following data, a WHO expert committee has recommended that treatment regimens be altered once resistance to a particular antibiotic reaches 5 per cent.[3]

Results and discussion

About 10,500 gonococcal isolates were examined in 15 participating countries (listed in the acknowledgments) in 2000.

Resistance to the *penicillins* remained widespread by both chromosomal (CMRNG) and plasmid mediated mechanisms (penicillinase producing *N. gonorrhoeae* — PPNG). Table 1 provides details of CMRNG, PPNG and/or total penicillin resistance in 15 WPR focal points. Very high proportions of combined forms of penicillin resistance (CMRNG + PPNG) were recorded in Korea (91%), the Philippines (89%), China (80%), Brunei (63%), Singapore (58%), Hong Kong SAR (54%), and Vietnam (48%). With the exception of the Hong Kong SAR and Vietnam where penicillin resistance declined somewhat, these proportions approximated those found in preceding years. Apart from New Caledonia (no penicillin resistance), Papua New Guinea (36.5% of strains penicillin resistance), and Tonga (no penicillin resistance), data from some Pacific Island states were unavailable this year. With the exception of Papua New Guinea, low levels of penicillin resistant were observed in these countries in past years. Other participants submitting data in 2000 (Australia, Japan and New Zealand) had proportions of penicillin resistance between 8 and 28 per cent. Malaysia had a high proportion of isolates showing resistance in a small sample.

Resistance to the *quinolone* antibiotics remained a major problem in many parts of the WPR and the situation

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Table 1. Penicillin sensitivity of strains of *Neisseria gonorrhoeae* isolated in 15 countries in the WHO WPR, 2000

Country	No. tested	PPNG		CMRNG		All Pen R	
		No.	%	No.	%	No.	%
Australia	3,468	302	8.7	377	10.8	679	19.5
Brunei	59					37	63.0
China	1,007	344	34.1	464	46.0	808	80.1
Fiji	756	25	3.3	3	0.3	28	3.6
Hong Kong SAR	2,743	292	10.6	1,191	43.4	1,483	54.0
Japan	213	1	0.5	59	27.6	60	28.1
Korea	190	119	62.6	54	28.4	173	91.1
Malaysia	12	9	75.0	1	8.3	10	82.6
New Caledonia	74	0		0		0	0.0
New Zealand	694	20	2.9	35	5.0	55	7.9
Papua New Guinea	224	77	34.3	5	2.2	82	36.5
Philippines	290	259	89.3	0	0.0	259	89.3
Singapore	635	349	55.0	18	2.8	367	57.8
Tonga	50	0	0.0	0	0.0	0	0.0
Vietnam	157	74	47.1	1	0.7	75	47.8

PPNG Penicillinase producing *N. gonorrhoeae*
CMRNG Chromosomally mediated resistance in *N. gonorrhoeae*

deteriorated further in 2000. Data from 12 WPR countries are shown in Table 2 and quinolone resistant strains (QRNG) are divided into 'less susceptible' and 'resistant' categories on the basis of susceptibility determinations. Eleven of 12 WPR countries which examined isolates for quinolone resistance detected QRNG in 2000. High proportions of QRNG were detected in China, Hong Kong, the Philippines, Japan and Vietnam maintaining a situation observed in previous reports. In the above countries and also in Korea and Australia, the proportion of strains fully resistant to quinolone antibiotics increased while the proportion of less sensitive isolates decreased, i.e. there was again an upward shift in overall levels of resistance. In Hong Kong the percentage of 'resistant' QRNG has increased from about 50 per cent in 1998 to about 66 per cent in 1999 to 80 per cent in 2000 and in China quinolone resistance rates again increased markedly in an expanded sample. A shift to higher MICs in Japan saw the proportion of 'resistant' QRNG there increase from about 23 per cent in 1999 to 40 per cent in 2000. In Brunei the proportion of QRNG was essentially unchanged from previous years.

An ominous trend was the presence of a small number of isolates with altered susceptibility to third generation cephalosporins. These strains were seen in Singapore, Brunei, China, Australia and New Zealand. Because of methodological differences in testing, MIC values are not directly comparable between centres, but values ranged up to 0.25 mg/L. Third generation cephalosporins are crucially important agents in the treatment of gonorrhoea as resistance to other agents accelerates.

A small number of spectinomycin resistant strains were found in China, Papua New Guinea, Vietnam, Brunei and Korea. Only very occasional strains resistant to this injectable antibiotic have been found in recent WPR surveys.

Although tetracyclines are not a recommended treatment for gonorrhoea, these agents are widely used and readily available in the WPR. One particular type of plasmid-mediated resistance gives rise to high-level tetracycline resistance (TRNG). About 6,900 gonococci were examined for high-level tetracycline resistance in 12 WPR countries in 2000 (Table 3). High proportions of TRNG isolates were again prominent in Malaysia, Brunei, Singapore, Vietnam, China and Papua New Guinea ranging between 25 and 70 per cent. In other countries the proportions of TRNG ranged between 0.5 and 11 per cent of strains examined. The proportion of TRNG has increased significantly in China in recent years from around 3 per cent in 1998 to nearly 15 per cent in 1999 to 25 per cent in 2000.

The data recorded in 2000 in the WPR indicate that further increases in gonococcal resistance to antibiotics have occurred. Resistance to the penicillins is so widespread that any contemplated use of this group of antibiotics would require prior validation of likely efficacy. A similar requirement would now seem to be appropriate for quinolone antibiotics given the extensive resistance

Table 2. Quinolone resistance in strains of *Neisseria gonorrhoeae* isolated in 12 countries in the WHO WPR, 2000

Country	No. tested	Less susceptible		Resistant	
		No.	%	No.	%
Australia	3,468	334	9.6	285	8.2
Brunei	60	3	5.0	7	12.0
China	1,007	141	14.0	858	85.2
Hong Kong SAR	2,743	459	16.7	2,180	79.5
Japan	213	65	30.5	86	40.0
Korea	190	122	64.2	50	26.3
Malaysia	12	0		0	
New Zealand	694	14	2.0	16	2.3
Papua New Guinea	224	0	0.0	2	0.9
Philippines	290	4	1.4	110	37.9
Singapore	635	39	6.1	121	19.0
Vietnam	157	18	11.5	67	42.7

Table 3. High level tetracycline resistancein strains of *Neisseria gonorrhoeae* isolated in 11 countries in the WHO WPR in 2000

Country	No. tested	No. TRNG	% TRNG
Australia	3,468	318	9.2
Brunei	33	24	72.0
China	1,007	260	25.8
Japan	213	1	0.5
Korea	190	5	2.6
Malaysia	12	3	25.0
New Zealand	694	23	3.3
Papua New Guinea	224	75	33.5
Philippines	290	33	11.4
Singapore	635	453	71.3
Vietnam	155	69	44.5

revealed in this and earlier surveys. Finding suitable alternative treatments is difficult given the cost of suitable antibiotics. The recognition of gonococci with altered susceptibility to third generation cephalosporins is also a matter of concern. Although treatment failure attributable to resistance to this antibiotic is yet to be confirmed, there will be considerable and continuing interest in the number of strains which show this altered susceptibility in future surveys and also in the MICs associated with this phenomenon.

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Letter to the Editor

Increasing notification rates for invasive meningococcal disease (IMD) in Australia (*Commun Dis Intell* 2001;25: 126-129) justify consideration of new immunisation strategies including the use of meningococcal serogroup C conjugate (MCC) vaccine. Such a strategy has been deployed with great effect in the United Kingdom and the Republic of Ireland over recent years.

The juxtaposition of two statements in your editorial 'a 25 per cent increase in serogroup B disease across all age groups in the United Kingdom' and 'this observation supports a hypothesis that serogroup replacement may be an important factor in the epidemiology of meningococcal disease' suggests that there is concern that the MCC vaccination campaign in the UK has been in some way responsible for serogroup replacement and increasing IMD. There is no such concern. There is no evidence that serogroup replacement is occurring.

According to Dr Ed Kaczmarski from the Public Health Laboratory Service (PHLS) and the Meningococcal Reference Unit, careful surveillance for evidence of serogroup replacement is in place and so far there is no evidence of this occurring. Concerns that the prevention of serogroup C disease by vaccination might result in a capsular switch to serogroup B remain entirely speculative.[1]

To date there have been 1,204 cases in all age groups of IMD due to serogroup B notified to the PHLS this year compared with a total of 1,645 in 2000.[2] For serogroup C the numbers are 255 and 712 respectively, although the cases now are largely in age groups which have not been vaccinated. Archival data can be viewed showing the dramatic decline in serogroup C disease in England and Wales,[3] and in Scotland.[4]

In the Republic of Ireland an overall 28 per cent decrease in IMD notifications occurred in 2000 to 2001 in comparison with the previous year.[5] The incidence of group B IMD dropped by 14.4 per cent to 6.5 per 100,000 population in 2000 to 2001 from 7.6 per 100,000 population in 1999 to 2000 and there are no indications to date that the incidence of IMD due to non-B, non-C serogroups is increasing.

Any epidemiological or molecular evidence of 'vacuum filling', capsular switching or serogroup replacement will appear rapidly in the public domain. Meningococcal serogroup B and other serogroup vaccines are under development for the purpose of building on the successes of existing meningococcal immunisation programmes. The reader's attention should be drawn to a forthcoming editorial by John Tapsall to appear in the Journal of Paediatrics and Child Health which addresses the Australian situation more particularly and the forthcoming Proceedings of the 41st ICAAC which will address the global imperatives for meningococcal immunisation.

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An outbreak of *Salmonella* Typhimurium phage type 99 linked to a hotel buffet in Victoria

Jane Greig,[1,2] Karin Lalor,[1] Catherine Ferreira,[1] Eileen McCormick [3]

Salmonella Typhimurium phage type 99 is a relatively rare serovar in Australia. The National Enteric Pathogen Surveillance Scheme reported only 16 cases Australia-wide in 2000, and 19, 4, 3,1 and 1 for the 5 preceding years. Victoria contributed 4, 7, 1, 0, 0 and 0 cases for these years respectively. From 15 to 28 June 2001 the Victorian Department of Human Services was notified of 4 cases of *S.* Typhimurium 99; all 4 resided in the same rural region of Victoria. Earlier in the year there was a notification of *S.* Typhimurium 99 in a man who lived in the same region and who had become ill after burying 6 sheep that had recently died with *S.* Typhimurium 99 infections at his workplace, a feedlot.

The Department initiated an investigation into the cluster of *S.* Typhimurium 99 cases. During interviews it was found that one case had eaten at a local hotel buffet in the 3 days

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before illness onset. A number of others who had eaten from the hotel buffet on the same evening, 6 June 2001, had also subsequently become ill with gastrointestinal symptoms. Lamb's fry was the suspected source of illness. Further investigation revealed that 6 out of 10 people contacted had eaten lamb's fry. All of the six who consumed lambs's fry were subsequently ill with diarrhoea.

A study was undertaken of patrons eating from the hotel buffet on the evening of 6 June. A standard questionnaire was administered by phone, seeking information on illness history, consumption of buffet foods according to the list provided by the hotel, and any other meals bought and/or eaten away from home in the first 2 weeks of June. A suspected case was defined as a person who ate at the hotel on the evening of 6 June 2001 and became ill with gastrointestinal illness (vomiting and/or diarrhoea i.e. three or more loose bowel motions in a 24 hour period) in the 5 days following. A case was confirmed if *S.* Typhimurium 99 was isolated from a faecal specimen. Both suspected and confirmed cases were included in the analysis.

The exact number of patrons who attended the hotel on the evening of 6 June 2001 could not be established, but the regular buffet caters for approximately 80 and there were 7 bookings for a total of 92 people listed for 6 June. Six of the booking groups totalling 83 people were contacted. Interviews were conducted with 78 patrons, revealing 18 suspected cases in addition to the confirmed index case. As there was a delay in identifying suspected cases no further faecal samples were obtained. However, it was determined that one of the 18 suspected cases was actually a confirmed case because he was part of the June cluster of 4 *S.* Typhimurium 99 cases. Therefore the overall attack rate of suspected and confirmed cases was 24 per cent. All cases reported diarrhoea, followed by nausea (79%), and lethargy (63%). Vomiting was not common (21%). Over half of the cases visited a doctor (53%), and two were admitted to hospital (10%). The median incubation period was 25 hours (range 16-68). The median age of cases was 45 years (range 7-72) and 58 per cent were male.

Of 31 food or beverage items from the hotel buffet or menu, there was a strong association between illness and consumption of the lamb's fry with bacon and onion in gravy (RR 8.44; 95% CI 3.13 – 22.77; p value < 0.001). There was no statistical association between illness and consuming food from any other local establishment.

The hotel was inspected for general food handling techniques, food storage, and cooking procedures for the lamb's fry dish. No hotel staff were ill either before or after the buffet dinner. The inspection revealed inadequate recording of coolroom, freezer, delivery, cooking and reheating temperatures. Potential problems during the preparation of the lamb's fry dish included: possible lack of refrigeration after frying; addition of meat juices to gravy during preparation; unknown temperature of combined dish of lamb's fry with bacon and onion in gravy after reheating; unknown holding temperature of the dish in a Bain Marie during self service.

The supply chain of the lamb's fry was followed as far as possible through hotel suppliers and meat processors. The meat processor buys animals from all over Australia, but occasionally obtains animals from the feedlot where the *S.* Typhimurium 99 case notified in February worked. There was no way to determine when animals were traded between these two companies, so a link between the source of infection for the earlier case and the current cohort is only speculative. All of the lamb's fry was consumed on 6 June, so liver samples from the same batch could not be tested. Lamb meat and offal samples from the hotel, its direct supplier, and the meat processing company two steps further back in the supply line were all negative on microbiological culture tests. Powdered gravy mix from the hotel also tested negative.

It was not possible to determine whether the lamb liver was contaminated with *S.* Typhimurium 99 before reaching the hotel. *Salmonella* contaminated raw meat or offal will not pose a health risk if adequately handled, stored and cooked. Education has been provided at the premises encompassing storage, cooking, post-cooking temperature control, physical handling of products, cross contamination, and personal hygiene.

Acknowledgement

Thanks to local government Environmental Health Officers Vanessa Crow and Greg Andrews for assistance with the investigation.

Invasive meningococcal disease and HIV coinfection

Deborah L Couldwell

Abstract

Three cases of meningococcal disease which occurred over a 3 year period in HIV-infected people living in the Wentworth Health Area of Sydney, Australia, are described. None of the 3 had ever received antiretroviral therapy which may have contributed to development of invasive meningococcal disease. *Commun Dis Intell* 2001;25:278-279.

Keywords: meningococcal; HIV; Neisseria meningitidis

Introduction

Neisseria meningitidis commonly colonises the human nasopharynx. In a small proportion of subjects, acquisition progresses rapidly to invasive disease, resulting in bacteraemia and/or meningitis. Although the risk of development of invasive disease is thought to be largely determined by the virulence of the meningococcal strain, environmental and host factors also contribute. These factors include age, concomitant upper respiratory tract infection, cigarette smoking, and host immune function.[1]

Numerous encapsulated bacteria cause sepsis at increased rates in HIV-infected individuals; higher rates of mortality also occur.[2] The commonly involved pathogens vary with geographic location as well as patient risk factors. Although there have been a number of reports of meningococcal disease in HIV-infected patients,[3,4,5] an increased risk in HIV-infected people has not been demonstrated.[6,7] However, a population-based study of sporadic meningococcal disease from Atlanta in the United States identified immune compromise due to conditions including HIV-infection in two-thirds of affected adults over 24 years of age.

Background and case descriptions

In 1996, an outbreak of meningococcal disease due to *N. meningitidis* serotype C:2a:P1.5 originated in the Wentworth Health Area of western Sydney, Australia.[8] Following the initial cluster of cases, which occurred in a background of sporadic disease predominantly due to serogroup B infections,[9] an increase of this disease has been reported in the region.

The annual reporting rate from July 1997 to June 2000 was 6.4 per 100,000 population (a total of 60 cases), with resultant mortality in 5.0 per cent and severe morbidity in 8.3 per cent of cases (Population Health Unit Wentworth Area Health Service, personal communication). Three of those affected were HIV-infected adults, and in these cases outcome was uniformly poor, with death in 2 cases, and severe morbidity in the third. The two different strains of *N. meningitidis* involved in infections of HIV-infected patients were unrelated to the local outbreak strain, and no contact with other cases of meningococcal disease or among the cases was discovered.

Case 1

A 40-year-old homosexual man was diagnosed with HIV infection in 1996. He had no history of HIV-related disease and had never received antiretroviral treatment. He smoked 30 cigarettes daily. At a routine clinic visit in December 1997 his CD4 cell count was 399 x 10^6/L (reference range 420-1410 x 10^6/L), with HIV viral load 764 copies per ml. One week later, he was admitted to hospital with meningism, septic shock and a typical meningococcal rash. Despite appropriate treatment, he developed acute renal failure and peripheral ischaemia resulting in partial lower limb amputations. The organism isolated was *N. meningitidis* serotype C: NT: P1.5. Following his eventual recovery, he commenced antiretroviral treatment with zidovudine, lamivudine and saquinavir. He has persistent mild chronic renal failure and by March 2001 his CD4 cell count had increased to 528 x 10^6/L (reference range 380-1390 x 106/L), with an undetectable HIV viral load (<50 copies/ml).

Case 2

In early 1998, a 33-year-old man accompanied by his wife presented to a different hospital with a one day history of productive cough, headache, vomiting, arthralgia and weakness. There was a history of weight loss over the preceding few months. He had been seen by his general practitioner the previous day, diagnosed with sinusitis and started on an unknown antibiotic. At presentation, he had signs of cerebral irritation, but no rash, and over the following 2 hours, he experienced rapid neurological deterioration. CSF examination confirmed bacterial meningitis, treatment with ceftriaxone was then instituted, but he died the following day. *N. meningitidis* grew on blood culture, and was later identified as a fully antibiotic sensitive strain of serotype C: NT: P1.5. HIV antibody result was positive, but no potential high risk factors for HIV infection were identified. History of cigarette smoking was not recorded.

Case 3

The 50-year-old homosexual man had been HIV antibody positive since 1991. In September 1999 he had an episode

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of single dermatomal Herpes zoster but had no other history of HIV-related disease. He was a heavy smoker and lived alone. Over the preceding 2 years his CD4 cell count had fallen from 720 to 420 x 10^6/L (reference range 380-1390 x 10^6/L). HIV viral load had been consistently over 100,000 copies per ml during this time but he had never commenced antiretroviral treatment. One evening in early 2000 acquaintances noticed that he was unwell with flu-like symptoms, and the next day he was found deceased at home. A post-mortem examination revealed meningitis as the cause of death with *N. meningitidis* isolated from a meningeal swab. The strain was later identified as serotype B: 4: P1,15.

Discussion

From July 1997 to June 2000, 3 cases of meningococcal disease occurred in HIV-infected people in this region, who thereby accounted for 5 per cent of a total of 60 cases. The local population prevalence of HIV infection over the same period was estimated to be between 55 and 73 per 100,000 population,[10] suggesting an increased incidence of meningococcal disease in people with HIV infection over this time period.

The overall mortality rate of meningococcal disease in the region was 5 per cent, however, the mortality rate in those cases with HIV coinfection was 67 per cent. Severe morbidity occurred in another 8.3 per cent of total cases, including the remaining case with HIV coinfection. Thus, meningococcal disease was uniformly severe in those with concomitant HIV infection, although, the number of affected patients was small.

These 3 cases occurred in adults outside the age groups with the highest susceptibility to meningococcal disease, but smoking was a contributing factor in at least two cases. A factor common to all 3 cases was untreated HIV infection. In one case, the CD4 cell count prior to the development of meningococcal disease was mildly depleted. In another, there was evidence of deteriorating immune function over the preceding 2 years with falling CD4 cell counts and development of symptomatic disease. CD4 cell count was not known for one patient. Profound depletion of CD4 cells is well correlated with susceptibility to disease caused by certain opportunistic pathogens. However, increased susceptibility to bacterial pathogens occurs throughout the course of HIV infection and is related to aberrant immune responses as well as to loss of CD4 cells. Treatment of HIV infection with highly active antiretroviral drugs leads to progressive immune reconstitution[11] which could confer protection against invasive bacterial infections.

These cases may implicate untreated HIV infection as a cofactor in the development of invasive disease following acquisition of *Neisseria meningitidis*. The outcome of meningococcal disease may be worse in those with HIV coinfection, but case numbers are too small to draw a definite conclusion.

Acknowledgements

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Erratum

Definitions of meningococcal disease in use in Australia

The table describing definitions of meningococcal disease in use in Australia in the editorial 'Meningococcal disease' (*Commun Dis Intell* 2001;25:26-129) contained inaccuracies in the definitions in use in Victoria and Queensland.

In Victoria, cases with a clinically compatible illness in whom other diseases have been excluded are included as clinical meningococcal cases.

In Queensland, meningococcal cases with strong clinical evidence are included as 'probable' cases.

We are grateful to Priscilla Robinson, Communicable Diseases Section, Department of Human Services, Victoria and Dr Linda Selvey, Communicable Diseases Unit, Queensland Health, for these corrections

An exercise in communication:-analysis of calls to a meningococcal disease hotline

Justine D Ward,[1] Brad D McCall,[1] Sarah G Cherian[1]

Abstract

We describe our experience with a hotline which was set up to deal with enquiries relating to a secondary school mass vaccination campaign against meningococcal disease. Three thousand, three hundred calls were received over 6 days, mostly from the general public but also from contacts of the school and health practitioners. The hotline served as an important means of providing consistent advice and reassurance to the public and reduced the burden of calls to hospitals and public health units. *Commun Dis Intell* 2001;25:280-281.

Keywords: Meningococcal, outbreak, telephone hotline

Introduction

A mass vaccination and chemoprophylaxis intervention was conducted at a secondary school in Brisbane from 17 to 21 August 2001 in response to 2 cases of serogroup C meningococcal disease. The polysaccharide vaccine and antibiotics (ciprofloxacin) were provided to staff, students and selected football contacts. Both cases died, the first on 5 August 2001 and the second on 21 August 2001 after the mass intervention had commenced. These cases occurred at a time when other cases and deaths from meningococcal disease were being reported in the media.

A community hotline was established within hours of the decision to undertake the intervention at the school. The aim of the hotline was to provide a means of assistance to the school vaccination team and address any community concerns about the intervention or meningococcal disease. Six telephone lines were established and the hotline was open for 6 days, from 17 to 22 August 2001. The hotline was staffed by hospital and community health nurses, with supervision by a trained medical officer. Staff were briefed at the beginning of each shift. The hotline remained open for 24 hours following the death of the second case.

Both the cases and the intervention were extensively reported in the media during the time that the hotline was open and the hotline number was made available through the media. However, on 19 August 2001 a widely circulated newspaper suggested that the public could ring the hotline if they wanted advice regarding a person with symptoms of the disease. This was contrary to the information provided by Queensland Health.

Methods

Staff collected the name and phone number of each caller, and recorded a brief comment as to the reason for the call. At the end of each shift, the data sheets were collected and counted. Every tenth case was selected and categorised according to a coding system (Table 1).

The authors all served as medical supervisors on different shifts, and their qualitative assessment of the experience is also provided. Call costs, fax and postage costs and staff wages (including time and half and double time rates but no on-costs) were used to calculate an estimate of the costs of running the hotline.

Results

There was a total of 3,300 calls to the hotline and the number of calls per hour tended to increase over the six day period (Table). The total cost was estimated to be $23,600, of which $ 2,600 was phone and fax costs and $21,000 was staff costs. There was a noticeable increase in the frequency of calls around the time of news bulletins.

Calls from contacts or relatives of the school cases (33%) and other cases (8%) made up the highest proportion of calls on the first evening after the campaign started but decreased thereafter. Over 50 per cent of calls were general enquiries about the disease or the vaccine. The third most frequently asked question was to request advice about the management of a sick person (16.2%), particularly on days 3 and 4. Nearly 13 per cent of calls were from students, teachers or footballers connected with the cases or contacts of this group.

The proportion of callers seeking information on the vaccine increased on the last day. Information regarding the vaccine and the reasons why Queensland Health was not recommending vaccination for the general public was given to callers. Some callers accepted this advice but many stated that they would be seeking vaccination from their general practitioners anyway and some callers expressed anger that the vaccine was not being provided to the general public or to contacts outside the school.

Comments

The hotline served as an important means of providing advice and dealing with the considerable public concern generated by the occurrence of the cases and the reporting in the media. The provision of consistent advice and reassurance to the general public was an important focus of the communication strategy in a similar incident in the United Kingdom in which mass vaccination was carried out at a school.[1] The experience of an increase in demand

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Table. Summary of call numbers and reasons for calling

Date	Hours of operation	No. of calls	Calls/ hour/ day	Coded reasons for calling (%)								
				1	2	3	4	5	6	7	8	9
17/8	6pm to 10pm	125	31.3	4 (33)		3 (25)	1 (8)	1 (8)				3 (25)
18/8	6am to 10pm	555	34.7	7 (13)	6 (11)	18 (33)	12 (22)	2 (4)		2 (4)	2 (4)	6 (11)
19/8	6am to 10pm	896	56.1	9 (10)	24 (27)	21 (24)	20 (22)	5 (6)		4 (4)	2 (2)	4 (4)
20/8	6am to 8pm	773	55.2	6 (8)	16 (21)	18 (23)	16 (21)	4 (5)	1 (1)	5 (6)	3 (4	8 (10)
21/8	8.30am to 5pm	427	50.2	4 (10)	4 (10)	18 (43)	7 (17)	2 (5)	2 (5)		2 (5)	3 (7)
22/8	8.30am to 5pm	524	61.7	3 (6)	3 (6)	19 (37)	18 (35)	1 (2)	2 (4)	1 (2)		5 (10)
	Total	3.300		3 (10.1)	53 (16.2)	97 (29.7)	74 (22.6)	15 (4.6)	5 (1.5)	12 (3.7)	9 (2.8)	29 (8.9)

1. Contact or relative of student/teacher/footballer
2. Advice on sick person
3. General advice on meningococcal disease plus or minus vaccine information
4. Information re vaccine specifically
5. Contact of another meningococcal case
6. Going to social or athletics meet at the school
7. Enquiry from health professional
8. Eligible student/teacher/footballer (enquiry re intervention or side-effects of prophylaxis)
9. Unclear

following television and newspaper coverage has been reported by other help line services.[1,2]

The hotline ran over a weekend when regular medical services were not routinely available to most people and only hospital Emergency Departments were available to field calls from the public. Hospitals were instructed to forward calls to the hotline and it was reported that this service was welcomed by hospital staff. Public Health Units and hospitals still received large numbers of calls and would likely have been overwhelmed if the hotline had not been available.

Inappropriate calls to the hotline might have been avoided if the media had not suggested that the hotline be called for concerns about management of sick people. It is more appropriate that people seek advice regarding an acutely ill person from their general practitioner or nearest hospital.

Just under 5 per cent of calls concerned other suspected cases of meningococcal disease. Therefore, it is important that the medical officer supervising the hotline be briefed on all recent confirmed or suspected cases. Although health service providers received direct information about the campaign, the hotline also served as an additional means of communication with this group.

The establishment of a hotline to deal with community concerns is a valid procedure in such a setting of high community concern especially when fuelled by media reporting. However, the costs involved mean that such interventions should be considered only when the community concern is such that additional reliable methods to the usual media releases of providing consistent information to the public and dealing with misinformation are required.

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Exotic *Aedes* Mosquitoes : Onshore detection and elimination in Darwin, Northern Territory

Peter I Whelan,[1] Richard C Russell,[2] Gwenda Hayes,[1] Garry Tucker,[3] Graeme Goodwin[3]

Introduction

The Medical Entomology Branch (MEB) of the Territory Health Services (THS) and the Australian Quarantine Inspection Service (AQIS) have coordinated and separate programs to detect and eliminate exotic mosquitoes imported into Darwin. AQIS searches all overseas vessels and cargo for exotic mosquitoes and conducts elimination procedures on positive or potential receptacles on vessels or within 400m of a port area. Both AQIS and MEB operate egg traps (ovitrap) detection programs continuously in the Darwin Port area. The ovitraps are checked weekly (AQIS) or fortnightly (MEB) for eggs and larvae, and eggs are reared to late instar larvae for identification. The AQIS onshore program is inside a 400m area of port facilities while the MEB programs are within port areas, in nearby suburban and industrial areas, as well as in other vulnerable sites for importation or establishment throughout Darwin.[1,2]

The Northern Territory is free of the 2 principal vectors of dengue, although there have been numerous instances of both *Aedes aegypti* and *Aedes albopictus* imported into Darwin.[2,3] On each occasion the importations have been quickly detected and eliminated by established procedures.[4] The most recent detections have been in cargo on or recently offloaded from overseas vessels.[2] On only two previous occasions since 1978 have exotic *Aedes* eggs or larvae been detected in onshore ovitraps and none have been detected in other onshore receptacles. This paper describes the most recent onshore detection of an exotic *Aedes* species, and discusses the identity of the species, the elimination procedures taken and future precautions and procedures.

Detection

On the afternoon of the 14 June 2001 an AQIS officer notified MEB of the detection of possible exotic *Aedes* mosquito larvae reared from an ovitrap in the Fort Hill wharf area of Darwin. Three third instar larvae were submitted to the MEB on 14 June 2001 and subsequently one fourth instar larvae on 18 June 2001. The larvae had been reared from egg paddles from two separate ovitraps within 100m of each other in the Fort Hill Wharf industrial area close to a forested escarpment below the city area of Darwin.

The first larvae were initially identified by the MEB as probably *Aedes scutellaris,* an exotic New Guinea and Torres Strait species, but they were possibly *Ae. albopictus,* which is exotic to Australia and a potential vector of dengue and other arboviruses.[5] There was uncertainty with the identification because the larvae were at an earlier growth stage than normally required for positive identification and differed from a key identification character in a published description.[6] Although *Ae. scutellaris* larvae cannot be distinguished from *Aedes katherinensis*, a tropical north Australian species, this species is absent from the Darwin locality and thus was ruled out of contention because of the location and other circumstances surrounding the ovitrap recoveries. The importation was treated as *Ae. albopictus* until the identity could be resolved.

Surveillance and eradication measures

The importation of adult exotic mosquitoes and subsequent detection of eggs in onshore egg traps raised the possibility of the establishment of an exotic *Aedes* mosquito species in the Darwin port area. It was decided that immediate surveys and precautionary elimination measures were required to determine the current situation and ensure the importation was eliminated.

It was agreed that AQIS would have principal responsibility in this situation, with responsibility for larval survey and control, public advice and landholder liaison. AQIS is nominally responsible for surveys and elimination of exotic mosquitoes within a 400m quarantine zone around port facilities. The MEB was to provide scientific advice, assist with larval surveys and increased surveillance, and carry out adult mosquito control.

The target and risk area was assessed as the area between Stokes Hill and Fort Hill wharves. This included various industrial and port facilities adjacent to the shoreline, a steep forested escarpment fronting the wharf area, and the grounds of Government House at the top of the escarpment overlooking Fort Hill wharf.

AQIS notified nearby premises and the public of the detection and planned control measures. A quarantine hold was placed over movement of all potential water holding receptacles such as tyres, in the target area. Further advice was given to various premises during a search for any water containing receptacles. The MEB carried out adult mosquito control by fogging bioresmethrin throughout the target area between 5.30pm and 6.30pm on the 15 June 2001.

The receptacle survey was carried out within a 400m zone around the positive ovitraps over 2 days after the fogging,

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including a survey of vessels in the port area, the Port Authority premises, and Government House. The survey located all receptacles holding or with evidence of recently containing water.

All water-holding receptacles were sampled for larvae. Sixteen separate premises were surveyed and 42 receptacles with water were located. Eleven of these receptacles contained mosquito larvae. Receptacles with water and larvae were found primarily adjacent to the positive ovitrap location in the Port facilities area and at Government House. One receptacle without larvae included an underground concrete section of a conveyor belt facility containing several thousand litres of rainwater. All larvae were collected alive and reared to fourth instar larvae in the MEB facilities. All larvae recovered were native species with four of the receptacles containing *Aedes notoscriptus* alone, while the remaining seven contained either alone or in combination, *Culex annulirostris*, *Culex quinquefasciatus* and *Culex halifaxii*.

All receptacles with water or evidence of recent flooding were sprayed with the insecticide deltamethrin to the point of run off, with no spraying carried out until the initial receptacle survey was completed. A follow up receptacle survey will be carried out 3 days after the first rains of the wet season (possibly in October or November 2001).

Over 2 days during the receptacle survey, carbon dioxide-baited light traps were set on the Dilia floating hotel (Olympia) recently transferred from East Arm port, in the grounds of Government House, in the inner city area adjacent to the escarpment overlooking the port, and near each ovitrap positive position. The MEB set 6 additional ovitraps within and outside the port area. AQIS set an additional 6 ovitraps within the port area. The increased ovitrap surveillance was maintained for 2 months. There was no further detection of exotic *Aedes* in this period.

Discussion

The ovitraps were set three weeks prior to the notification and collected 2 weeks prior to the notification, indicating that the eggs were laid between this period. A Papuan vessel moored at Darwin about three weeks prior to the detection had pooling in a canvas tarpaulin on board but no larvae were detected in the water. However, inspection was delayed for a number of days after mooring. The water was dark and hindered inspection for larvae in the water. The water was discarded and the tarpaulin was treated with insecticide.

The Olympia was moored nearby at Fort Hill wharf and was considered a potential, although unlikely, source of the mosquitoes. This vessel had numerous *Cx. quinquefasciatus* larvae detected, and one *Ae. aegypti* adult was found flying on board when it first arrived in Darwin from Dili, East Timor, in early January. While no *Aedes* larvae were detected then, the *Culex* breeding site was below floorboards decking and there was the possibility that *Aedes* larvae were present prior to treatment in early January. On 5 January 2001, while moored at East Arm port, this vessel was treated with adulticides (with chlorine and the residual insecticide deltamethrin) to destroy potential breeding sites.

The larvae from the ovitraps were examined further (by RCR) and it was concluded that the larvae could have been either *Ae. scutellaris* or *Ae. albopictus*, but did not fully conform to published keys or descriptions of either species although they were more consistent with the former.

There is a slight possibility that the larvae were *Ae. katherinensis*. However, its absence from the greater Darwin area, the failure to detect this species in ovitraps in Darwin for over 25 years, and the occurrence of this species only in very low numbers in remote localities away from potential importation routes, indicates that the larvae were unlikely to have been this species.

There is also a possibility that the larvae were an undescribed species of the *Aedes scutellaris* taxomomic group from East Timor. *Aedes scutellaris* itself has a Papuan distribution and has not been recorded from Timor, although *Ae. albopictus* has been collected there,[6,7] but there are many members of the group from a wide area of the islands of South East Asia and the Pacific. It is possible that a member of the group does exist on Timor, and suspect *Ae. albopictus,* particularly from Timor, should be scrutinised carefully.

Following this episode, an adult male mosquito of the *Aedes scutellaris* group collected in Timor (D McGinn and H Standfast, personal communication) has been determined (by author RCR) on its genitalia characters, to be *Aedes alorensis* (previously known only from Alor to the north of Timor) or a local Timor species of the group very similar to *Ae. alorensis*. Adult females of *Ae. alorensis* are very similar to *Ae. katherinensis,* and *Ae. alorensis* larvae have never been described, but the male genitalia are quite distinctive and readily distinguished from those of *Ae. katherinensis* and *Ae. scutellaris*.

This is the third record of an ovitrap positive for exotic mosquitoes in Darwin. The previous records include one of *Ae. albopictus* in January 1989 in the wharf area at Frances Bay, which is approximately one kilometre east of Fort Hill wharf, and one of *Ae. aegypti* in May 2000 at the Hudson Creek wharf area approximately 9 kilometres east of Fort Hill wharf. On each occasion adult fogging and increased ovitrap surveillance and receptacle survey and treatment were carried out and no evidence of establishment was found.

The last importation of *Ae. albopictus* occurred on 31 January 2001 at Frances Bay, associated with the tray of a damaged vehicle offloaded from a vessel that had recently arrived from Dili, East Timor. Live adults were observed flying from the vehicle. Immediate adult fogging operations and receptacle insecticide treatments were carried out. Increased ovitrap surveillance and receptacle surveys failed to detect any establishment. This importation was unlikely to have been a source of the current detection.

This is the second record of ?*Ae. scutellaris* importation into Darwin. The previous importation was in vehicle tyres on board a general cargo vessel from Jakarta on 25 May 2000. On that occasion the larvae were examined (by authors PIW and RCR) who concluded that they were probably *Ae. scutellaris* although the identification could not be confirmed with certainty and there was some possibility that the larvae could have been the related species *Ae. malayensis* which occurs on Java and nearby areas. *Aedes scutellaris* has not been recorded from Jakarta, the vessel's last port of call, but the ship stops regularly at Dili, East Timor, most recently in March 2000. It is possible that the larvae found 2 months later in May 2000 were from eggs laid during a stop at Dili. *Aedes alorensis* was not considered at that time as the

larvae are not described and there were no records from Timor.

The conclusion is that the eggs in the ovitraps probably originated from one or two females flying from the Papuan vessel which was in port around the time of probable egg laying. The identity of the larvae remains undecided but is ?*Ae. scutellaris*. However, because of the minor differences in described larval morphological characters and the variability of some of these determining characteristics, there is a possibility that the larvae were *Ae. albopictus,* another of the *Aedes scutellaris* group such as *Ae. malayensis, Ae. katherinensis*, *Ae. alorensis* or an undescribed similar member of the *Aedes scutellaris* group from Timor. Adults (females and males) are required for a determination of species.

It is recommended that quarantine procedures be modified to collect larvae or pupae alive from receptacles on vessels from Timor arriving in Darwin, and that the larvae and pupae be link reared to adults in secure premises to confirm any identification. It is also recommended that a critical review be undertaken of the larval descriptions of *Ae. albopictus* and relevant members of the *Aedes scutellaris* group, particularly with respect to early as well as fourth instars.

The interception of this importation is evidence that the ovitrap surveillance procedures to detect exotic mosquitoes are working well in Darwin. Although there have been a few risk situations with cargo, in general the vessel and cargo inspections do detect importations, and their detection has allowed timely eradication procedures.[2] This detection and eradication is at odds from instances in other parts of the world.[8] A considerable amount of the success of these procedures is due to the good cooperation and liaison between AQIS and the MEB/THS. However, some of these arrangements have not been formalised, particularly in regards to requirements for countering the establishment of an exotic species after importation, and the responsibilities and methodologies for large-scale eradication measures. It is strongly recommended that protocols and procedures for such arrangements should be discussed, agreed and formalised as soon as possible, for Darwin and other 'at-risk' international ports in Australia.

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Short report: prevalence of markers of exposure to Q fever in rural central Queensland

Roscoe Taylor,[1] Ian Hunter ,[1] Richard Tan[2]

Despite consistently high rates of Q fever in parts of rural Queensland and inland northern New South Wales,[1,2] little is published about the prevalence of immune markers indicating prior exposure to the infection amongst rural communities.

The Central Queensland Rural Division of General Practice conducted Q fever awareness-raising campaigns amongst rural communities, culminating in several successful screening and vaccination programs during 2001.[3]

Participants were self-selected and came from a wide range of occupations. Owing to the nature of the awareness campaigns, most resided and/or worked on farming properties, or had some other field or sale yard exposure to the agricultural industry. Abattoir workers were not part of

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the program. Table 1 shows participants' age, sex, and duration of residence on farms.

A widely-used meatworks industry screening questionnaire[4] was adapted to collect data on age, sex, participant's occupation, length of residency on a farm property, risk factors for exposure, and previous history of Q fever disease or vaccination. Immune markers of exposure were assessed using both skin testing to detect cell-mediated immunity, and serology (CFT followed by confirmatory EIA in borderline cases to detect the presence of IgG antibodies against phase II antigens of *Coxiella burnetti*). Skin testing was carried out by trained practitioners, including two of the authors, in accordance with standard pre-vaccination testing procedures,[5] and results were read 7 days later.

For the purpose of this analysis, equivocal or borderline test results were regarded as negative, and persons with either a positive serology result, or a positive skin test or both were regarded as immune 'positives'.

Of 272 people presenting for screening, skin and serology results were available for 265. The remainder were excluded from analysis. Forty-nine people (18.5%) had evidence of prior exposure to Q fever, and 196 went on to receive vaccination. Epi Info version 1.1 was used to generate univariate statistics for Q fever risk factors (Table 2).

There was little difference in the mean age of positive compared with negative subjects (Table 1). Males were significantly more likely to be immune than females (odds ratio 3.39, 95% CI = 1.44 – 8.26). Adjustment for a modest but statistically significant difference between the 2 groups in the mean number of years spent on a property (38.7 years for males vs 33.1 for females) using logistic regression produced an odds ratio of 3.11 (95% CI 1.31 – 7.41). The increased risk for males reflects differences in degree of exposure through occupational and other activities.

As would be expected given the nature of rural life, most participants reported multiple risk factors in the form of

Table 1. Age, sex and duration of farm residence

	N	Age (years)			Ever resided on a farm	
		Range	Mean	Median	%	Mean no. years
Female	94	12 - 79	44.1	44.5	86.1	33.1
Male	171	14 - 78	46.3	45.0	89.5	38.7
All Persons	265	12 - 79	45.5	45.0	88.3	36.8

Table 2. Univariate analysis of risk factors for Q fever exposure

Exposure/activity	Yes		No		OR	(95 % CI)
	Positive*	Negative†	Positive*	Negative†		
Ever lived on property	44	190	5	25	1.16	(0.40 - 4.09)
Sex of respondent (M = exposed)	41	130	8	86	3.39	(1.44 - 8.26)
Feedlot work	11	40	38	176	1.27	(0.56 - 2.86)
Stock/farm work	44	187	5	29	1.36	(0.48 - 4.77)
Tannery work	0	1	49	215	Not calculable	
Dressing kangaroo carcass and/or dressing the pelt	6	17	43	199	1.63	(0.50 - 4.66)
Collecting sheep/cattle manure for the garden	24	108	25	108	0.96	(0.49 - 1.87)
Private slaughter of sheep or goats	9	46	40	170	0.83	(0.35 - 1.94)
Shearing	3	10	46	206	1.34	(0.23 - 5.49)
Animal husbandry	27	95	22	121	1.56	(0.80 - 3.05)
Livestock buying	18	69	31	147	1.24	(0.62 - 2.47)
Animal transport	28	105	21	111	1.41	(0.72 - 2.76)
Milking cows or goats	21	77	28	139	1.35	(0.69 - 2.66)
Other	12	68	37	148	0.71	(0.33 - 1.51)

* Testing positive to serology, skin or both.

† Testing negative to both serology and skin.

activities leading to contact with animals or animal products. No particular risk factor was significant on univariate analysis.

Only 15 of 47 (31.9%) skin and/or blood test-positive subjects for whom data were available, responded positively to the question, 'Do you recall having an illness, possibly lasting 7 days or more, that commenced suddenly with fever, chills, profuse sweating, muscle and joint pains, severe headache and fatigue?'. Given problems with illness recollection, the variable nature of presentations due to Q fever, and lack of specificity, this question appears to offer little assistance to community-based programs in determining who will test positive, with a positive predictive value of 0.25.

The proportion of subjects with either a positive skin or blood test, or both, are shown in Table 3 to demonstrate the importance of skin testing in the assessment of prior exposure to Q fever.[6] Seroprevalence studies that have not utilised skin testing should be interpreted with caution, as according to our data such surveys may underestimate exposure by at least 50 per cent. A range of factors (e.g. the age distribution of the sample, and the recency of exposure) would contribute to variations in the relative proportion of positive skin to serology results. It is also important to highlight the relevance of skin testing during education of the many general practitioners currently taking an interest in offering vaccination to their patients. The announcement of a National Q Fever Management Program[7] targeting high-risk groups in the meat and livestock industries, has led to increased awareness and demand for vaccination in many sectors, particularly amongst rural communities. Coupled with this has been an increase in requests for vaccination training by doctors.

In Queensland, abattoir workers and shearers may access free vaccination programs from November 2001. Very recently the Federal Government also announced that the Program will be expanded to include livestock workers, although this group will not attract the same level of subsidy.[8] This is a welcome addition given that in central west and south-west Queensland (which have the highest Q fever notification rates in Queensland),[9] it is people working on the land and those who are associated with the livestock industry who account for the majority of notifications, rather than abattoir workers.[10,11] Our finding that exposure to Q fever is quite common amongst this rural group is supported by preliminary screening results from more broadly-based campaigns in other rural communities suggesting the prevalence of exposure may be even higher in south-west Queensland than Central Queensland.[12,13]

These data provide additional support for implementation of the recommendation by Garner et al in 1997,[1] to extend Q fever vaccination programs into rural communities in geographic areas of high incidence in Australia.

Table 3. Serology and Skin Test results

Only skin +ve n (%)	Only serology +ve n (%)	Both skin and blood +ve n (%)	Total n (%)
24 (49.0)	7 (14.3)	18 (36.7)	49 (100)

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Communicable Diseases Surveillance

Presentation of NNDSS data

With the move to a quarterly reporting system in *Communicable Diseases Intelligence*, the summary tables have changed to fall in line with a quarterly report. Table 2 presents 'date of notification' data, which is 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 unit. Table 3 presents the notification rate of Diseases by State or Territory for the current reporting quarter.

Table 2 now includes the following summary columns: current quarter totals, totals for the previous quarter; total for the same quarter in the previous year; a 5-year mean for the same quarter, the year to date total for each disease, the mean of the last 5 years year to date totals and the ratio of the current quarter to the mean of to the mean of the second quarter for the last 5 years.

Notifiable diseases 2001

The Communicable Diseases Network Australia has revised the list of diseases that are reportable to the NNDSS. All jurisdictions are working towards reporting against the new national list. Transmission of a dataset consistent with the new list will depend upon changes to public health legislation and IT system development. The following new diseases have been added to the NNDSS database: anthrax, Murray Valley encephalitis, Kunjin virus infection, cryptosporidiosis, influenza (laboratory-confirmed), Australian bat lyssavirus infection and invasive pneumococcal disease (laboratory-confirmed). Data on the following diseases will no longer be collected: chancroid, hydatid disease, lymphogranuloma venereum, non-TB mycobacterial infections, and yersiniosis.

Highlights for 3rd quarter, 2001

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 who have formed a Data Management Network. This additional information has enabled the reporting of more informative highlights each month.*

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia, and the CDI *Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme. 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 compared with the 5-year third quarter mean. Disease notifications above or below the 5-year mean, plus- or minus- two standard deviations are marked with an asterisk. These and other disease trends are commented on below.

As this report comments on notifications in winter and spring, the focus is on those diseases with a winter/spring peak, namely: influenza; pertussis; meningococcal disease; and rotavirus infections.

Gastrointestinal diseases

Campylobacteriosis

Notifications of gastrointestinal disease caused by *Campylobacter* species continued to increase in this quarter. Four thousand one hundred and fifteen notifications were reported nationally which is significantly above the range of 5-years' data.

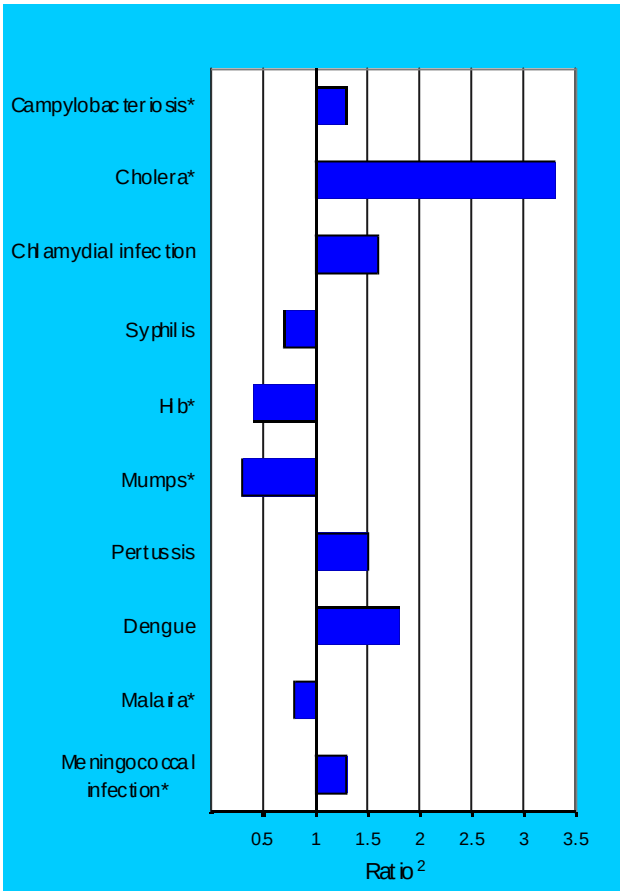
Cryptosporidiosis

Notifications were received in this quarter from all jurisdictions except Tasmania. There were 858 notifications in 2001 to the end of the third quarter. These numbers indicate that cryptosporidiosis may become the third most common gastrointestinal illness reported to the NNDSS after infections with *Campylobacter* and *Salmonella*. In the previous quarter, we commented on several outbreaks in the summer months associated with swimming pools. In this quarter, 5 linked cases associated with consumption of unpasteurised milk were reported from Queensland. Of the 5 cases, 3 were hospitalised. Cryptosporidiosis infection associated with consumption of unpasteurised products has been previously described. These products are probably contaminated with cow manure.[1]

Hepatitis A

National notifications of hepatitis A continued to fall in this quarter. Dr Jeffrey Hanna from Queensland Health

Figure 1. Selected[1] diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 July to 30 September 2001 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 plus- or minus- two standard deviations.

reported: 'There has been a marked decline in notifications of hepatitis A throughout 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 north Queensland in 1999, 34 in 2000, and 11 cases in the first 9 months of this year. The last case in an indigenous person was in June 2000. The majority of cases in 2000 and 2001 have been acquired abroad, particularly in Papua New Guinea, emphasising the importance of vaccination prior to either travel or work abroad.'

Hepatitis A in Australia has declined significantly over the past 30 years although levels in indigenous communities have remained high. In Australia, 3 patterns of hepatitis A epidemiology are recognised.[2] These are large, slowly evolving community-wide outbreaks, occurring at intervals of 5 years or more. They affect susceptible individuals exposed to intense levels of transmission within their groups and are a potential source for infection for the wider community. Settings for community-wide outbreaks include child care centres and pre-schools, schools and residential facilities for the intellectually disabled, networks of men who have sex with men and networks of injecting drug users. Secondly, sporadic cases of hepatitis A may occur in people without obvious risk factors although some may be associated with overseas travel or travel to indigenous communities. Thirdly, point-source outbreaks of hepatitis A may occur from exposure to contaminated food or water and/or infected food-handler. These are relatively rare in Australia. The last major point-source outbreak of hepatitis A arose from contaminated oysters in New South Wales in 1997.[3]

Vaccination against hepatitis A in Australia is recommended for travellers to endemic areas, visitors to remote indigenous communities, child-care and pre-school personnel, the intellectually disabled and their carers, health care workers, sewerage workers, men who have sex with men, injecting drug users, patients with chronic liver disease, haemophiliacs who may have received pooled plasma concentrates and food handlers.[2]

Salmonellosis

Twenty-two cases of *Salmonella* Typhimurium 99 were detected in a large outbreak of gastroenteritis at a Melbourne restaurant in July. Illness was associated with the consumption of a meal consisting of eye fillet of beef, onions and potatoes with 2 sauces. All food samples were negative and the specific source of contamination remains unknown. (Information supplied by Kerry-Ann O'Grady, Department of Human Services, Victoria.)

Increased notifications of infection with *Salmonella* Stanley were reported from all States and Territories in Australia in the third quarter 2001. Seventy cases were reported up to the end of September, of whom 20 appeared to have acquired the infection outside Australia. Of cases acquired within Australia, about half reported consuming a brand of peanuts imported from China. A product recall was initiated and case reports have waned. Outbreaks of *Salmonella* Stanley associated with the same brand of peanuts were recently reported from Canada and the United Kingdom. Pulsed field gel electrophoresis indicated that isolates from peanuts and human cases shared the same distinctive profile across the 3 countries.*Salmonella* Newport was also isolated from the peanuts in Australia but a link to human cases has not yet been established. In Canada the peanuts also were contaminated with *Salmonella* Lexington. (Information supplied by Martyn Kirk, OzFoodNet.)

Quarantinable diseases

Cholera

Two cases of cholera were reported in the third quarter 2001; one each from South Australia and Victoria. A third case with an onset in the second quarter and reported in the third quarter, was reported from New South Wales. Because of our analysis of data by date of onset, this case does not appear in this quarters' reporting in Table 2. All cases were acquired overseas: the 2 cases reported in the third quarter were associated with travel to Bali and in both cases ice used in drinks is suspected as the source of the infection. Since January 2001, 6 travellers to Bali have contracted cholera: 2 from Japan; 1 each from France and New Zealand, and the 2 Australian cases. All 6 were *V. cholerae* serogroup 01, serotype Ogawa.

The case from New South Wales appears to have acquired cholera in Hong Kong (serogroup 01 El Tor, serotype Inaba).

Yellow fever

A single suspect case of yellow fever was reported from Victoria, but the notification was subsequently withdrawn as the clinical and laboratory evidence for yellow fever was not conclusive. There have been no cases of yellow fever reported in Australia since the inception of the NNDSS in 1991. A summary of the case from Dr Priscilla Robinson, Department of Human Services, Victoria follows.

'A 53-year-old woman was notified to the Victorian Department of Human Services in September with a provisional diagnosis of yellow fever. She had recently returned from Peru and Chile. She had been vaccinated against hepatitis A and B and typhoid, and boosted for tetanus, and although she had asked about yellow fever vaccination had been told that it was unnecessary. While away she drank only bottled water, wore loose clothing and used insect repellent. Despite these precautions, she recalled being bitten by mosquitoes. On arrival at Melbourne, she was identified by the Australian Quarantine Inspection Service as a person who had been in a yellow fever area who was not protected by vaccination and given a screening letter. She became unwell during the journey home with an acute febrile illness consisting of headaches, shivers and epistaxis (unusual for her), consistent with possible early symptoms of yellow fever. She felt worse during the evening and presented to the local hospital for assessment and treatment, presenting the screening letter to the on-call medical officer. She developed upper respiratory symptoms (sore throat and loss of voice), with minimal sputum. Management of her illness was transferred to an infectious disease specialist, in conjunction with her GP. The diagnosis was not laboratory confirmed. Serological screening for flaviviruses was PCR and antigen negative. Acute phase sera was equivocal for yellow fever total antibody, however there were no changes in titres in serology taken 8 days later. The preliminary diagnosis of yellow fever was rejected by the physician and reference laboratory. The cause of her illness remains unknown.'

Sexually transmitted infections

Notifications of chlamydial infections and gonococcal infections increased in the third quarter 2001. An increase in the number of notifications of donovanosis was also noted; this is probably related to more active case detection as part of the Donovanosis Eradication Plan described in the last issue of *CDI*.

An apparent decline in notifications of syphilis should be interpreted with caution as syphilis notifications from Queensland are still undergoing a process of validation.

Vaccine preventable diseases

Notifications of *Haemophilus influenzae* type b and mumps were significantly below the range of 5-years' data for the third quarter 2001.

Influenza

National surveillance data consisting of laboratory reports through LabVISE and national and State-based sentinel general practice schemes have been reported weekly on the *Communicable Diseases Australia* Website (http://www.health.gov.au/pubhlth/cdi/ozflu/flucurr.htm). The peak of the influenza season in temperate regions of Australia appears to have passed and surveillance in New South Wales, Victoria and Western Australia has ceased.

Nationally, the 2001 influenza season was mild and numbers of laboratory-diagnosed cases as well as presentations of influenza-like illness to general practices were lower than in the 2000 season (Figure 2). Circulating strains were well matched by those included in the 2001 influenza vaccine.

The Northern Territory was a notable exception to this picture. The Northern Territory reported large increases in notifications of influenza-like illness from August 2001. In tropical regions of Australia, bimodal peaks of influenza activity in March/April and September/October are the usual pattern, however 21 geographically diverse cases were recorded in 2 weeks largely in indigenous people, which inflated the rate of influenza to record levels (Figure 3).

Measles

Sixteen sporadic cases of measles were reported during the third quarter 2001. At least 4 of the 16 cases were acquired overseas.

Figure 2. National consultation rates of influenza-like illness in ASPREN and sentinel GP practices, 2000 and 2001, by week

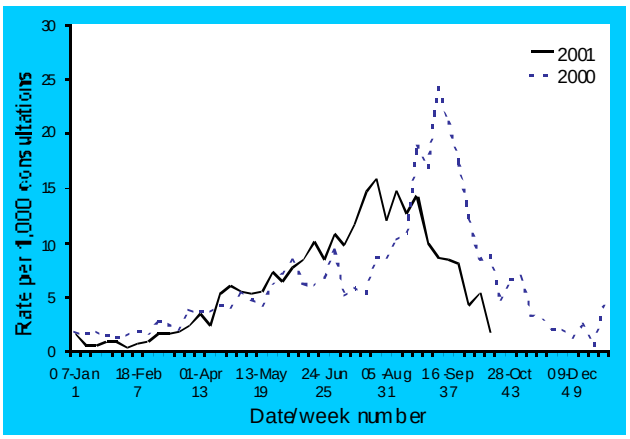
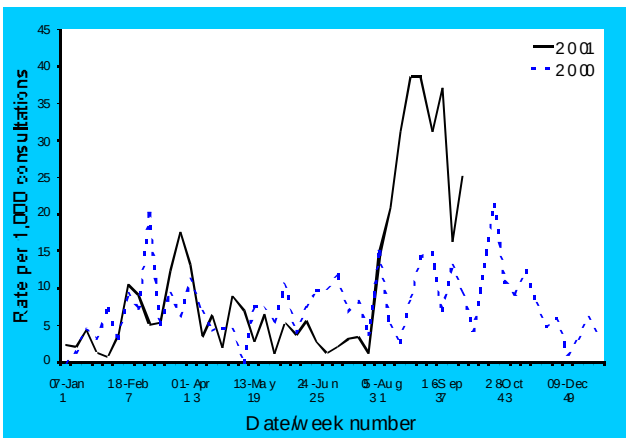


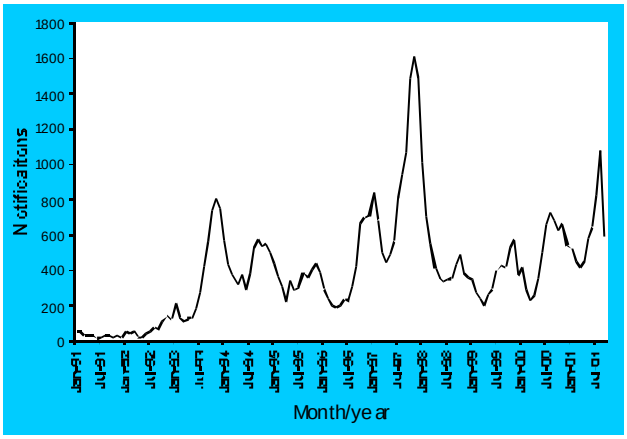
Figure 3. Consultation rates of influenza-like illness in sentinel GP practices, Northern Territory, 2000 and 2001, by week



Pertussis

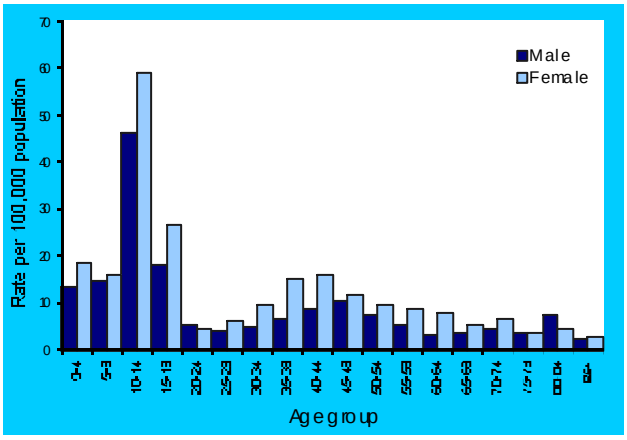
In keeping with the seasonal peaks of disease activity seen in spring, notifications of pertussis increased in this quarter. Monthly notifications in May, June and August were the highest recorded on the NNDSS for those months (Figure 4). Should this notification rate continue 2001 is tracking towards a peak in the 3 to 5 year cycle of pertussis, which is a recognised feature of pertussis epidemiology.[4]

Figure 4. Notification of pertussis, Australia, 1991 to 2001, by month of onset



Immunisation against pertussis in Australia consists of 5 doses given at 2, 4, 6 and 18 months and 4 years of age. Currently 88.3 per cent of 2 year-olds in Australia are age-appropriately immunised (ACIR data, see Tables this issue). As a consequence the peak notification rate for pertussis is now found among young adolescents (aged 10–14). In the third quarter 2001, 696 (28%) of all notifications of pertussis were from this age group (Figure 5). In addition 2,069 cases (83%) were aged 10 years or more and only 109 (4%) were in infants aged less than one year. However, it is of some concern that 13 per cent of cases are aged 1–9 years, confirming the importance of the 5th dose at 4–5 years and the need to raise coverage rates further. The need for an adolescent and adult vaccination program is being considered by the Australian Technical Advisory Group on Immunisation.

Figure 5. Notification rate of pertussis, Australia, 1 July to 30 September 2001, by age group and sex



Despite high levels of vaccination, pertussis has increased in a number of countries since 1997. This has prompted investigations into the evolution of variants of *Bordetella pertussis*. Mooi and colleagues have observed antigenic divergence between vaccine strains and clinical isolates of *Bordetella pertussis*, specifically in the surface-associated protein pertactin and the pertussis toxin.[5] Replacement of vaccine with non-vaccine strains as a result of herd immunity has not yet had any measurable effect on pertussis vaccine efficacy, but surveillance of variant strains of the bacteria may be important for the control of pertussis in the future.

Several recent studies have examined the importance of pertussis as a cause of prolonged coughing in adults and adolescents. A recent study in Canada suggests that up to 20 per cent of prolonged coughing is associated with laboratory evidence of pertussis infection. The nature of that evidence is controversial: in this study only 2.3 per cent of symptomatic cases were laboratory confirmed pertussis (by culture, PCR or a four-fold increase in pertussis antibody), while the remainder were diagnosed on the basis of a single high pertussis antibody titre.[6]

Since it is well established that adolescents and adults are frequently the source of pertussis infection for infants and children, and adolescents now have the highest rates of disease, vaccination of adolescents with acellular pertussis vaccines has been instituted in France, Germany and Canada. It remains to be seen how this will impact on the epidemiology of pertussis.

Vectorborne diseases

Dengue

An increase in the number of notifications of dengue was noted in this quarter. A number of these were imported cases from Samoa. A large outbreak of dengue strain 1 has been continuing through most of 2001, involving the Pacific Island nations of French Polynesia, Samoa and American Samoa, New Caledonia and Tokelau. In French Polynesia 33,000 cases have been reported with 1200 hospitalisations, 500 cases of dengue haemorrhagic fever and 8 deaths.

Other bacterial infections

Meningococcal infection

A series of small outbreaks of meningococcal disease affected several jurisdictions in the third quarter of 2001. National notifications of meningococcal disease in August (93 cases) was the highest monthly total ever recorded in the NNDSS. Notifications for meningococcal disease increase in spring; however the number of notifications has been increasing since the inception of the NNDSS in 1991 (Figure 6). This quarter's total was above the range of the last 5-years' data for the third quarter. The notification rates of cases in the third quarter show the highest rates in the 0–4 and 15–19 year age groups (Figure 7). The increase in case numbers is partly due to the wider availability of non-culture based diagnostic techniques including serology (meningococcal IgM) and nucleic acid tests. Some jurisdictions also now report probable cases of meningococcal disease.

Figure 6. Notifications of meningococcal disease, Australia, 1991 to 2001, by month of onset

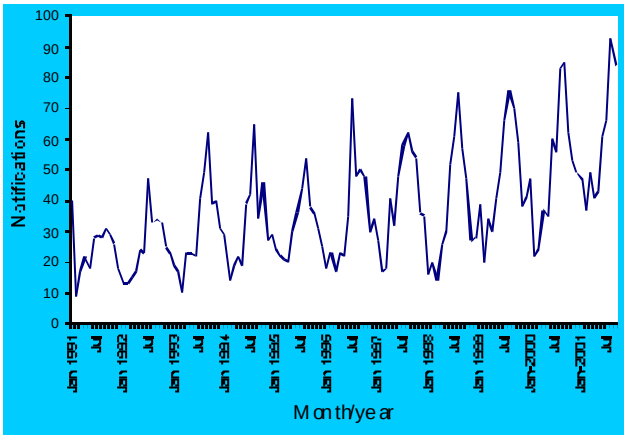
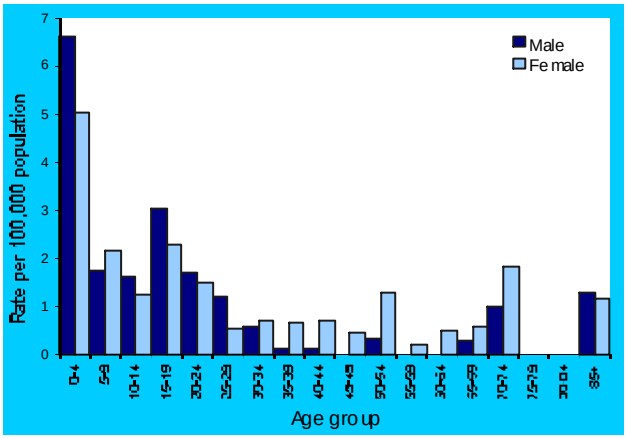


Figure 7. Notification rate of meningococcal disease, Australia, 1 July to 30 September 2001, by age group and sex



Non-culture based techniques currently account for approximately 27 per cent of all diagnoses of invasive meningococcal disease.[7]

Further information on meningococcal disease in this quarter in Queensland and Victoria were provided by Dr Linda Selvey and Dr Patricia Robinson respectively.

In Queensland there were 49 cases of invasive meningococcal disease in the third quarter (including 2 probable cases) compared to 48 cases for the first 2 quarters (including 4 probable cases). This is consistent with the expected seasonal increase. Of the cases in the third quarter, 24 were serogroup B infections (49%), 15 serogroup C infections (31%) and 2 serogroup Y infections (5%). This compares to 54 per cent serogroup B, 10 per cent serogroup C and 4 per cent serogroup Y in the first 2 quarters of the year. There were 5 deaths in the period, giving a crude case fatality rate of 10 per cent. Four deaths were caused by group C infection, the other being by group B. The peak incidence was in August, with only 8 cases being notified in September. In August there was a cluster of 2 cases in a boarding school, both of whom died. All of the students and many staff at this school were vaccinated in response to this cluster. There were no other clusters in the period.

In Victoria there were 114 cases of meningococcal disease notified to the Victorian Department of Human Services between the beginning of January and the end of September 2001. Of these, 35 were confirmed as serogroup B (22 by culture and 13 by PCR) and 45 as serogroup C (30 by culture and 15 by PCR). Of the remaining 34 cases, one was confirmed as serogroup Y. It was not possible to group 2 cases, and 31 were clinically diagnosed. The age distribution of serogroup B and C disease shows notable differences, with 54 per cent of serogroup B cases being under 15 compared to 33 per cent of serogroup C cases. Of the 25 cases who were aged 30 years or more, only 4 were confirmed as serogroup B disease compared to 15 serogroup C, and in this age group the case fatality rate was 20 per cent compared with 3-5 per cent in younger cases. In this quarter, the case fatality rate was 6 per cent for serogroup B, and almost double that rate (11%) for serogroup C cases, while the overall case fatality rate was 7 per cent.

Other non-notifiable diseases

Mycobacterium ulcerans

Kerry-Ann O'Grady supplied the following information on *Mycobacterium ulcerans* cases in Victoria. Eleven PCR-confirmed cases of cutaneous *Mycobacterium ulcerans* infection (Bairnsdale /Daintree/ Buruli Ulcer) were detected in residents or frequent visitors of a Victorian coastal town. Environmental sampling revealed one PCR-positive site in the area, an irrigation dam at the local golf course, although its relationship to the cases is unknown. A Victorian research team has been funded to investigate possible sources of infection and modes of transmission.

This is the latest of several outbreaks of *Mycobacterium ulcerans* infection near Melbourne in the past decade. This environmental mycobacterium causes chronic progressive skin ulcers and occurs in soil and in swamp waters; cases of human infection occur by the contamination of small cuts or abrasions of the skin.[8] The outbreaks of disease in Australia appear to be associated with disturbance of swamp waters or contamination of dams or irrigation systems with swamp water.[9] Environmental studies using PCR have recently supported this hypothesis.[10]

Mycobacterium ulcerans infections are an increasingly significant public health problem in at least 31 countries in Africa, the Western Pacific, Asia and South America. In January 1998 the World Health Organization launched the Global Buruli Ulcer Initiative to coordinate global research and control efforts into this disease. Further information can be accessed at: http://www.who.int/m/topics/buruli_ulcer/en/index.html.

LabVISE

There were 6256 reports to LabVISE from 13 laboratories in the third quarter of 2001. Data are included from PathCentre, Western Australia, which have been excluded from previous reports due to technical problems. Previously unpublished PathCentre data for the period October 2000 to June 2001 are shown in Table 6.

In the third quarter 2001, there were reports of viral infection and 1,775 reports of bacteria and other microorganisms. The largest number of reports among viruses were for respiratory syncytial virus (1,146 reports), the influenza

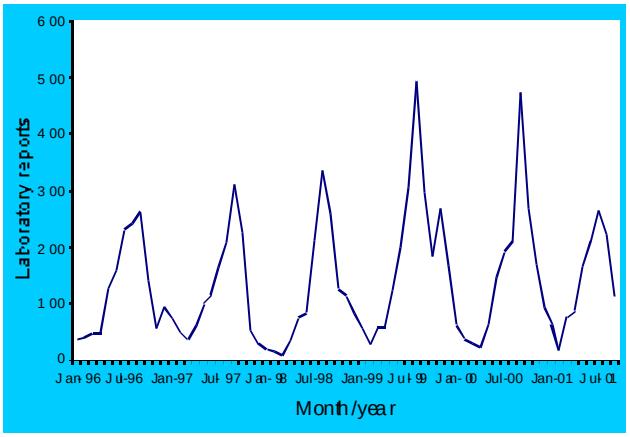
viruses (total 540 reports), and rotavirus (601 reports). Among the bacterial isolates the largest numbers of reports were of *Chlamydia* spp (639 reports) and *Bordetella pertussis* (439 reports).

Rotavirus

Rotavirus is a major cause of diarrhoea in children and may also be important as a cause of viral diarrhoea in the elderly. Rotavirus infections are typically increased in winter months (May to September in Australia). LabVISE rotavirus data (1996 to 2001) by month of laboratory report are shown in Figure 8. The Australian Rotavirus Surveillance Program collects important information on circulating rotavirus serotypes in Australia.[11]

During 2001 the Northern Territory experienced a large outbreak of rotavirus disease; the largest since surveillance for the disease was commenced in 1994.The outbreak commenced in April 2001 in Alice Springs and spread rapidly across the Northern Territory and into western Queensland. Children under 5 years of age made up 95 per cent of cases and notification rates were approximately 5 times higher in indigenous persons compared with non-indigenous people. The predominant strain isolated was G9, which has only been identified in the Northern Territory since 1999.[12]

Figure 8. Laboratory reports of rotavirus, Australia, 1995 to September 2001, by month of report



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Tables

There were 24,064 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 July and 30 September 2001 (Table 2). Figure 1 illustrates, for selected diseases, the third quarter 2001 totals as ratios to the mean of the third quarters for the previous 5 years. A summary of diseases currently being reported by each jurisdiction is provided in Table 1. The notification rate of diseases per 100,000 population for each State or Territory is presented in Table 3.

There were 6256 reports received by the *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 July to 30 September 2001 (Tables 4 and 5).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 27-30 to 35-39, ending 30 September 2001, are included in this issue of *Communicable Diseases Intelligence* (Table 7).

Table 1. Reporting of notifiable diseases by jurisdiction

Disease	Data received from:*
Bloodborne	
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions except NT
Hepatitis C (incident)	All jurisdictions except Qld
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except NSW
Cryptosporidiosis	All jurisdictions except Tas
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	
Cholera	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions except SA
Gonococcal infection	All jurisdictions
Syphilis	All jurisdictions
Vaccine preventable	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza	All jurisdictions except Tas
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease	All jurisdictions except SA and Tas
Poliomyelitis	All jurisdictions
Rubella	All jurisdictions
Tetanus	All jurisdictions
Vectorborne	
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
Ornithosis	All jurisdictions
Other lyssaviruses (NEC)	All jurisdictions
Q Fever	All jurisdictions
Other	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
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, 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 July to 30 September 2001, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 3rd quarter 2001[1]	Total 2nd quarter 2001[1]	Total 3rd quarter 2000[1]	Last 5 years mean 3rd quarter	Year to date 2001	Last 5 years YTD mean	Ratio[1]
Bloodborne															
Hepatitis B (incident)	0	9	1	12	3	9	39	10	83	94	143	79	273	228	1.0
Hepatitis B (unspecified)	16	893	NN	188	62	6	557	116	1,838	1,497	2,438	1,919	4,822	5,551	1.0
Hepatitis C (incident)	3	27	0	NN	12	0	20	4	66	69	141	75	211	216	0.9
Hepatitis C (unspecified)	62	1,489	52	743	194	97	1,476	347	4,422	3,869	4,998	4,559	12,620	13,666	1.0
Hepatitis D	0	1	0	0	0	0	1	0	2	6	5	5	13	11	0.4
Gastrointestinal															
Botulism	0	0	0	0	0	0	0	0	0	1	0	0	2	0	0.0
Campylobacteriosis[2]	103	-	73	1,065	675	155	1,336	708	4,115	3,629	3,433	3,111	11,138	8,988	1.3
Cryptosporidiosis	3	23	20	59	10	NDR	88	15	218	385	0	N/A	656	N/A	N/A
Haemolytic uraemic syndrome	0	0	0	0	1	0	0	0	1	0	1	2	3	9	0.6
Hepatitis A	4	56	11	30	5	0	22	14	142	115	155	425	353	1,625	0.3
Hepatitis E	0	1	0	0	0	0	0	0	1	2	0	0	4	4	2.5
Listeriosis	1	1	0	4	2	1	1	0	10	6	9	16	39	50	0.6
Salmonellosis	16	257	68	267	131	10	199	162	1,110	1,550	935	1,028	4,840	5,093	1.1
Shigellosis	0	24	23	18	5	2	26	28	126	122	35	127	364	471	1.0
SLTEC,VTEC[3]	0	0	0	3	4	0	0	0	7	6	10	4	29	19	1.8
Typhoid	0	9	0	1	2	0	3	4	19	10	16	16	62	58	1.2
Quarantinable															
Cholera	0	0	0	0	1	0	1	0	2	0	0	1	2	3	3.3
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															
Chlamydial infection	93	1,048	336	1,234	319	114	992	607	4,643	4,294	4,531	3,060	13,833	9,078	1.6
Donovanosis	0	0	7	1	NN	0	0	3	11	8	1	8	21	24	1.3
Gonococcal infection[4]	9	264	394	272	26	6	247	265	1,483	1,392	1,454	1,244	4,332	3,991	1.2
Syphilis[5]	2	141	110	8	4	5	5	33	308	264	534	455	850	1,296	0.7

Table 2 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 July to 30 September 2001, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 3rd quarter 2001[1]	Total 2nd quarter 2001[1]	Total 3rd quarter 2000[1]	Last 5 years mean 3rd quarter	Year to date 2001	Last 5 years YTD mean	Ratio†
Vaccine preventable															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0.0
Haemophilus influenzae type b	0	3	0	0	1	0	0	0	4	14	12	11	23	32	0.4
Influenza‡	7	127	38	355	84	NDR	160	162	933	24	NDR	N/A	969	N/A	N/A
Measles	0	9	0	3	1	0	2	1	16	12	21	102	98	277	0.2
Mumps	0	4	1	0	4	1	3	5	18	30	66	52	79	142	0.3
Pertussis	20	1,190	36	402	577	41	202	28	2,496	1,284	2,070	1,647	4,997	4,034	1.5
Pneumococcal disease‡	6	168	32	170	NDR	NDR	132	62	570	326	NDR	N/A	983	N/A	N/A
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Rubella§	0	9	0	43	1	0	6	1	66	40	82	274	161	756	0.2
Tetanus	0	0	0	0	0	0	0	0	0	0	0	1	1	4	0.0
Vectorborne															
Arbovirus infection NEC	0	1	1	1	0	0	4	0	7	14	3	4	30	45	1.6
Barmah Forest virus infection	0	50	10	71	0	0	2	5	138	446	113	98	908	550	1.4
Dengue	7	13	8	4	1	1	0	3	37	50	10	20	120	160	1.8
Japanese encephalitis	0	0	0	0	0	0	0	0	0	0	NDR	N/A	0	N/A	N/A
Kunjin virus infection	0	0	0	0	0	0	0	0	0	0	NDR	N/A	0	N/A	N/A
Malaria	5	34	12	61	14	0	14	8	148	148	238	188	526	646	0.8
Murray Valley encephalitis	0	0	0	0	0	0	0	0	0	0	NDR	N/A	2	N/A	N/A
Ross River virus infection	0	30	13	83	3	1	9	12	154	1,130	222	235	2,861	4,717	0.7
Zoonoses															
Anthrax‡	0	0	0	0	NN	0	0	0	0	0	NDR	N/A	0	N/A	N/A
Australian bat lyssavirus‡	0	0	0	0	0	0	0	0	0	0	NDR	N/A	0	N/A	N/A
Brucellosis	0	0	0	3	0	0	0	0	3	2	10	14	11	27	0.2
Leptospirosis	0	19	0	15	0	0	4	0	38	60	37	37	168	163	1.0
Other lyssavirus (NEC‡	0	0	0	0	0	0	0	0	0	0	NDR	N/A	0	N/A	N/A
Ornithosis	0	11	1	0	3	0	11	0	26	23	22	13	78	46	2.1
Q fever	1	33	0	81	3	1	13	3	135	150	137	138	454	413	1.0

Table 2 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 July to 30 September 2001, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 3rd quarter 2001[1]	Total 2nd quarter 2001[1]	Total 3rd quarter 2000[1]	Last 5 years mean 3rd quarter	Year to date 2001	Last 5 years YTD mean	Ratio†
Other															
Legionellosis	1	14	0	9	5	3	12	10	54	76	65	44	195	202	1.2
Leprosy	0	1	0	0	0	0	0	0	1	0	0	2	2	6	0.5
Meningococcal infection	3	79	3	43	10	12	48	40	243	143	213	193	514	376	1.3
Tuberculosis	1	45	13	7	20	2	67	15	170	125	262	261	458	770	0.7
Total	363	6,083	1,263	5,370	2,143	437	5,704	2,371	24,064	21,432	22,512	19,464	66,237	63,915	1.2

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 NSW 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, Qld, SA, Vic and WA: 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 unit.

† Ratio = ratio of current month total to mean of last 5 years calculated as described above.

‡ Notifiable from January 2001 only.

NA Not calculated as only notifiable for under 5 years.

NDR No data received.

NN. Not Notifiable

NEC Not Elsewhere Classified.

- Elsewhere Classified.

Table 3. Notification rates of diseases by State or Territory, 1 July to 30 September 2001. (Rate per 100,000 population)

Disease[1]	State or Territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Bloodborne									
Hepatitis B (incident)	0.0	0.6	2.0	1.3	0.8	7.7	3.3	2.1	1.7
Hepatitis B (unspecified)	20.5	54.9	NN	20.9	16.5	5.1	46.4	24.5	38.5
Hepatitis C (incident)	3.8	1.7	0.0	NN	3.2	0.0	1.7	0.8	1.7
Hepatitis C (unspecified)	79.4	91.6	106.0	82.6	41.1	82.5	123.2	73.2	91.8
Hepatitis D	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Gastrointestinal									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacterosis[2]	131.9	-	148.8	118.4	179.9	131.9	111.4	149.3	128.9
Cryptosporidiosis	3.8	1.4	40.8	6.6	2.7	NDR	7.3	3.2	4.6
Haemolytic uraemic syndrome	0.0	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.0
Hepatitis A	5.1	3.4	22.4	3.3	1.3	0.0	1.8	3.0	2.9
Hepatitis E	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Listeriosis	1.3	0.1	0.0	0.4	0.5	0.9	0.1	0.0	0.2
Salmonellosis	20.5	15.8	138.6	29.7	34.9	8.5	16.6	34.2	23.0
Shigellosis	0.0	1.5	46.9	2.0	1.3	1.7	2.2	5.9	2.6
SLTEC,VTEC[3]	0.0	0.0	0.0	0.3	1.1	0.0	0.0	0.0	0.1
Typhoid	0.0	0.6	0.0	0.1	0.5	0.0	0.3	0.8	0.4
Quarantinable									
Cholera	0.0	0.0	0.0	0.0	0.3	0.0	0.1	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									
Chlamydial infection	119.1	64.5	684.7	148.3	85.0	97.0	82.7	128.0	100.5
Donovanosis	0.0	0.0	14.3	0.1	NN	0.0	0.0	0.6	0.2
Gonococcal infection[4]	11.5	16.2	802.9	30.2	6.9	5.1	20.6	55.9	30.8
Syphilis[5]	2.6	8.7	224.1	0.9	1.1	4.3	0.4	7.0	6.4
Vaccine preventable									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.2	0.0	0.0	0.3	0.0	0.0	0.0	0.1
Influenza	9.0	7.8	77.4	39.5	22.4	NDR	13.3	34.2	19.8
Measles	0.0	0.6	0.0	0.3	0.3	0.0	0.2	0.2	0.3
Mumps	0.0	0.2	2.0	0.0	1.1	0.9	0.3	1.1	0.4
Pertussis	25.6	73.2	73.4	44.7	153.8	34.9	16.8	5.9	51.8
Pneumococcal disease	7.7	10.3	65.2	18.9	NDR	NDR	11.0	13.1	13.2
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella[6]	0.0	0.6	0.0	5.4	0.3	0.0	0.5	0.2	1.4
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Vectorborne									
Arbovirus infection NEC	0.0	0.1	2.0	0.1	0.0	0.0	0.3	0.0	0.1
Barmah Forest virus infection	0.0	3.1	20.4	7.9	0.0	0.0	0.2	1.1	2.9
Dengue	9.0	0.8	16.3	0.4	0.3	0.9	0.0	0.6	0.8
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	0.0
Malaria	6.4	2.1	24.5	6.8	3.7	0.0	1.2	1.7	3.1
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	1.8	26.5	9.6	0.8	0.9	0.8	2.5	3.2

Table 3 (continued). Notification rates of diseases by State or Territory, 1 July to 30 September 2001. (Rate per 100,000 population)

Disease[1]	State or Territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
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	1.2	0.0	1.7	0.0	0.0	0.3	0.0	0.8
Other lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.7	2.0	0.0	0.8	0.0	0.9	0.0	0.5
Q fever	1.3	2.0	0.0	9.0	0.8	0.9	1.1	0.6	2.8
Other									
Legionellosis	1.3	0.9	0.0	1.0	1.3	2.6	1.0	2.1	1.1
Leprosy	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection	3.8	4.9	6.1	5.3	2.7	10.2	4.0	8.4	5.0
Tuberculosis	1.3	2.8	26.5	0.8	5.3	1.7	5.6	3.2	3.5

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, Qld, South Australia , Vic and Western Australia: includes gonococcal neonatal ophthalmia.
5. Includes congenital syphilis.
6. Includes congenital rubella.

NDR No data received.
NN Not Notifiable
NEC Not Elsewhere Classified.
- Elsewhere Classified.

Table 4. Virology and serology laboratory reports by laboratories for the reporting period 1 July to 30 September 2001[1]

State or Territory	Laboratory	July 2001	August 2001	September 2001	Total this period
Australian Capital Territory	The Canberra Hospital	-	-	-	-
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	180	189	40	409
	New Children's Hospital, Westmead	169	119	87	375
New South Wales	Repatriation General Hospital, Concord	-	-	-	0
	Royal Prince Alfred Hospital, Camperdown	79	64	23	166
	South West Area Pathology Service, Liverpool	206	165	48	419
Queensland	Queensland Medical Laboratory, West End	199	321	400	920
	Townsville General Hospital	13	4	-	17
South Australia	Institute of Medical and Veterinary Science, Adelaide	268	766	842	1,876
Tasmania	Northern Tasmanian Pathology Service, Launceston	36	26	8	70
	Royal Hobart Hospital, Hobart	-	-	-	0
Victoria	Monash Medical Centre, Melbourne	-	-	-	0
	Rickettsia Reference Laboratory, Geelong*	6	-	-	6
	Royal Children's Hospital, Melbourne	150	37	26	213
	Victorian Infectious Diseases Reference Laboratory, Fairfield	160	142	98	400
Western Australia	PathCentre Virology, Perth	405	437	-	842
	Princess Margaret Hospital, Perth	324	156	-	480
	Western Diagnostic Pathology	36	27	-	63
Total		2,231	2,453	1,572	6,256

1. The complete list of laboratories reporting for the 12 months, January to December 2001, will appear in every report from January 2000 regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.

- Nil reports

* The Rickettsia Reference Laboratory, Geelong has recently joined the LabVISE scheme as a contributing laboratory. The first reports for this laboratory were received in September 2001 and are published in this issue of *CDI* for the first time.

Table 5. Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 July to 30 September 2001, and total reports for the year[2]

	State or Territory[1]								This period 2001	This period 2000	Year to date 2001[3]	Year to date 2000
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	-	-	-	2	-	-	3	2	7	12	101	38
Mumps virus	-	1	-	-	1	-	-	2	4	9	31	40
Rubella virus	1	2	-	15	2	-	-	-	20	10	50	33
Hepatitis viruses												
Hepatitis A virus	-	3	2	11	2	-	1	2	21	27	65	121
Hepatitis D virus	-	-	-	1	2	-	2	1	6	3	9	6
Arboviruses												
Ross River virus	-	1	4	16	2	-	-	11	34	34	821	1,099
Barmah Forest virus	-	4	1	16	-	-	-	1	22	17	249	121
Dengue type 4	-	-	2	-	-	-	-	-	2		3	
Dengue not typed	-	-	19	-	-	-	-	13	32	3	186	167
Murray Valley encephalitis virus	-	-	1	-	-	-	-	-	1	1	7	19
Flavivirus (unspecified)	-	-	1	1	-	-	4	-	6	2	21	39
Adenoviruses												
Adenovirus type 1	-	-	-	-	-	-	1	-	1	2	3	6
Adenovirus type 2	-	-	-	-	-	-	1	-	1	1	3	7
Adenovirus type 7	-	-	-	-	-	-	1	-	1	1	10	5
Adenovirus type 8	-	-	-	-	-	3	1	-	4	1	18	1
Adenovirus type 40	-	-	2	-	-	-	-	6	8	13	43	82
Adenovirus not typed/pending	2	94	1	9	104	10	29	32	281	210	734	756
Herpes viruses												
Cytomegalovirus	-	58	2	16	134	2	44	6	262	252	900	873
Varicella-zoster virus	3	34	7	97	43	1	66	95	346	221	1,258	970
Epstein-Barr virus	-	17	8	134	148	-	14	63	384	259	1,316	1,466
Molluscum contagiosum	-	-	-	-	-	-	-	1	1		11	9
Other DNA viruses												
Contagious pustular dermatitis	-	-	-	-	-	-	-	1	1	1	4	7
Poxvirus group not typed	-	-	-	-	-	-	2	-	2		2	
Parvovirus	-	2	2	14	46	-	11	21	96	81	248	248
Picornavirus family												
Coxsackievirus A16	1	-	-	-	-	-	-	-	1	3	3	6
Echovirus type 5	-	-	-	-	1	-	-	-	1		1	
Echovirus type 9	-	13	-	-	-	-	-	-	13	2	71	5
Echovirus type 13	-	5	-	-	-	-	-	-	5		17	
Echovirus type 18	-	1	-	-	-	-	-	-	1		6	
Echovirus type 30	-	3	-	-	1	-	-	-	4	7	28	114
Poliovirus type 1 (unchar)	-	7	-	-	-	-	-	-	7	7	16	11
Poliovirus type 2 (unchar)	-	4	-	-	-	-	-	-	4	1	13	4
Poliovirus type 3 (unchar)	-	5	-	-	-	-	-	-	5	2	7	5
Rhinovirus (all types)	-	69	-	-	3	-	2	18	92	71	293	283
Enterovirus type 71 (BCR)	-	-	-	-	-	-	-	2	2		24	
Enterovirus not typed/pending	-	8	6	2	2	6	34	117	175	84	577	667
Picorna virus not typed	-	-	-	-	-	-	6	-	6	2	6	2

Table 5 (continued). Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 July to 30 September 2001, and total reports for the year[2]

	State or Territory[1]								This period 2001	This period 2000	Year to date 2001[3]	Year to date 2000
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Ortho/paramyxoviruses												
Influenza A virus	7	127	1	49	174	-	19	83	460	677	621	1,035
Influenza A virus H1N1	-	-	-	-	-	-	15	-	15		15	
Influenza A virus H3N2	-	-	-	-	-	-	5	-	5	6	5	7
Influenza B virus	1	10	1	-	32	-	6	10	60	336	121	433
Parainfluenza virus type 1	-	1	-	-	13	-	-	2	16	40	34	220
Parainfluenza virus type 2	-	1	-	-	15	-	-	1	17	10	42	31
Parainfluenza virus type 3	-	44	-	9	144	-	6	79	282	129	492	239
Parainfluenza virus typing pending	-	2	-	-	-	-	1	-	3		3	1
Respiratory syncytial virus	3	387	-	28	219	37	126	346	1,146	1,283	2,368	2,568
Other RNA viruses												
HTLV-1	-	-	-	-	1	-	-	3	4	2	17	5
Rotavirus	1	268	2	1	135	3	32	159	601	874	1,225	1,240
Norwalk agent	-	-	-	-	-	-	13	-	13	13	124	17
Other												
Chlamydia trachomatis - A-K	-	-	-	-	-	-	-	1	1	478	1	2,226
Chlamydia trachomatis not typed	12	103	22	147	181	5	1	168	639		2,605	
Chlamydia pneumoniae	-	-	-	1	-	-	-	1	2	34	7	36
Chlamydia psittaci	-	-	2	-	-	-	8	2	12	17	58	67
Mycoplasma pneumoniae	-	21	5	34	64	10	60	38	232	151	632	450
Coxiella Burnetti (Q fever)	1	5	-	11	4	-	14	6	41	22	129	54
Rickettsia - Spotted fever group	-	-	-	-	-	1	-	-	7	3	114	21
Rickettsia spp - other	-	-	-	-	-	-	-	1	1	3	4	9
Streptococcus group A	-	2	7	55	-	-	15	-	79	67	271	248
Yersinia enterocolitica	-	1	-	-	-	-	-	-	1	1	3	9
Brucella species	-	-	-	1	-	-	-	-	1	1	3	5
Bordetella pertussis	8	46	4	40	305	2	31	3	439	174	899	449
Legionella pneumophila	-	-	-	-	-	-	18	-	18	18	48	33
Legionella longbeachae	-	-	1	-	1	-	-	6	8	8	19	43
Legionella species	-	-	-	-	-	-	5	-	5	1	12	2
Cryptococcus species	-	-	-	1	8	-	-	-	9	3	34	11
Leptospira species	-	1	-	2	3	-	-	-	6	9	33	42
Treponema pallidum	-	25	69	71	86	-	-	10	261	195	938	562
Toxoplasma gondii	-	2	-	-	2	2	2	-	8	4	24	12
Echinococcus granulosus	-	-	-	-	3	-	-	2	5	2	21	16
Total	40	1,377	172	784	1,881	82	599	1,315	6,256	5,900	18,077	17,301

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 6. Summary of data sent to LabVISE from Path Centre, Western Australia, October 2000 to June 2001

	2000			2001						
Organism	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	Jun	Total
Adenovirus not typed	13	22	12	14	23	17	19	16	11	147
Adenovirus type 1	0	0	0	0	0	0	1	0	0	1
Adenovirus type 40	0	2	2	0	6	7	11	2	13	43
Adenovirus type 41	0	0	0	2	0	0	0	0	0	2
Influenza A virus	74	50	10	9	1	1	2	2	6	155
Influenza B virus	12	6	2	6	3	1	2	4	5	41
Parainfluenza virus type 1	0	0	0	3	0	0	0	0	0	3
Parainfluenza virus type 2	0	1	0	0	0	0	0	0	1	2
Parainfluenza virus type 3	9	10	17	12	11	4	1	8	11	83
Parainfluenza virus type 4	0	0	0	0	0	0	0	2	0	2
Respiratory syncytial virus	16	12	2	8	14	21	3	18	18	112
Rhinovirus (all types)	17	13	10	1	15	16	12	15	16	115
Mycoplasma pneumoniae	9	19	11	16	10	4	4	20	12	105
Chlamydia psittaci	0	1	1	0	3	1	0	1	1	8
Mumps virus	4	3	1	2	1	2	8	1	2	24
Epstein-Barr virus	15	23	21	38	25	30	9	16	15	192
Varicella-Zoster virus	49	72	84	52	47	70	43	50	61	528
Herpes type 6	0	1	1	0	0	1	0	0	0	3
Coxiella Burnetti	8	2	1	2	3	6	2	3	0	27
Rickettsia, other	0	1	3	1	0	0	0	1		6
Enteroviruses - not typed	22	31	48	25	29	79	42	69	91	436
Molluscum contagiosum	1	1	0	0	2	3	1	2	2	12
ORF virus	0	0	0	0	1	0	0	2	0	3
Measles virus	0	0	0	0	0	3	0	2	0	5
Rubella virus	0	1	1	0	1	1	2	1	0	7
Hepatitis A virus	2	2	3	2	0	1	1		1	12
Chlamydia trachomatis not typed	108	133	116	79	121	149	93	176	126	1101
Papovavirus group	0	1	0	0	0	0	0	1	1	3
Cytomegalovirus	11	4	8	2	3	7	18	0	0	53
Rotavirus	3	4	1	0	3	13	8	27	61	120
Calicivirus	0	0	0	0	0	0	1	0	0	1
Enterovirus type 71 (BCR)	0	0	1	0	0	0	1	0	0	2
Parvovirus	12	15	19	15	29	10	8	10	24	142
HTLV-1	2	0	2	1	1	3	2	1	0	12
Barmah Forest Virus	4	4	6	3	3	11	4	4	3	42
Dengue type 2	0	0	0	0	0	0	0	0	1	1
Dengue type 3	0	0	0	3	0	0	0	0	1	4

Table 6. (continued) Summary of data sent to LabVISE from Path Centre, Western Australia. November 2000 to June 2001

	2000			2001						
Organism	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	Jun	Total
Dengue type 4	0	0	0	0	0	0	0	0	1	1
MVE virus	1	0	0	1	1	7	1	0	0	11
Ross River virus	0	8	8	25	28	31	19	21	4	144
Dengue	3	2	4	6	6	60	26	0	19	126
Kunjin virus	0	0	0	0	1	4	2	2	2	11
Bordetella species	0	1	0	3	0	6	2	1	0	13
Bordetella pertussis	0	4	8	2	0	1	0	1	3	19
Legionella pneumophila	0	1	1	0	1	1	0	1	0	5
Legionella longbeachae	2	1	3	1	0	3	1	2	2	15
Leptospira species	1	2	1	0	0	0	1	0	0	5
Schistosoma species	6	4	5	4	1	10	2	4	5	41
Total	404	457	413	338	393	584	352	486	519	3946

Table 7. Australian Sentinel Practice Research Network reports, weeks 27-30 to 35-39, 2001

Week number	27-30		31-34		35-39	
Ending on	29 July 2001		26 August 2001		30 September 2001	
Doctors reporting	207		228		285	
Total encounters	24,716		27,865		34,900	
Condition	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	317	12.8	373	13.4	279	8.0
Influenza with culture	3	0.1	6	0.2	2	0.1
Chickenpox	66	2.7	60	2.2	76	2.2
Shingles	27	1.1	33	1.2	52	1.5

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia. The system coordinates 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 *2000;24:6-7.*

LabVISE is a sentinel reporting scheme. Currently 17 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 *2000;24:10.*

ASPREN currently comprises about 120 general practitioners from throughout the country, not all of whom report each week. Between 7,000 and 8,000 consultations are reported each week, with special attention to 12 conditions chosen for sentinel surveillance in 2001. Communicable Diseases Intelligence *reports the consultation rates for four of these. For further information, including case definitions, see* Commun Dis Intell *2001;25:106.*

Additional reports

Gonococcal surveillance

John Tapsall, The Prince of Wales Hospital, Randwick, NSW, 2031 for the Australian Gonococcal Surveillance Programme.

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various States and Territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5 per cent or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatment.[1] *Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as TRNG. Tetracyclines are however, not a recommended therapy for gonorrhoea in Australia. Comparability of data is achieved by means of a standardised system of testing and a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented.*

Reporting period 1 April to 30 June 2001

The AGSP laboratories examined a total of 858 isolates in this quarter, a lower number than in the same period in the past 2 years. About 45 per cent of this total was from New South Wales, 17 per cent from Victoria, 13 per cent from Queensland, 12 per cent from the Northern Territory, 9 per cent from Western Australia and 3 per cent from South Australia. Isolates from other centres were few.

Penicillins

Figure 1 shows the proportions of gonococci fully sensitive (MIC ≤ 0.03 mg/L), less sensitive (MIC 0.06 – 1 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 resistant by chromosomal mechanisms fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

In this quarter about 22 per cent of all isolates were penicillin resistant by one or more mechanisms — 7 per cent PPNG and 15 per cent by chromosomal mechanisms (CMRNG). The proportion of penicillin resistant strains ranged from 4 per cent in the Northern Territory to 36 per cent in South Australia.

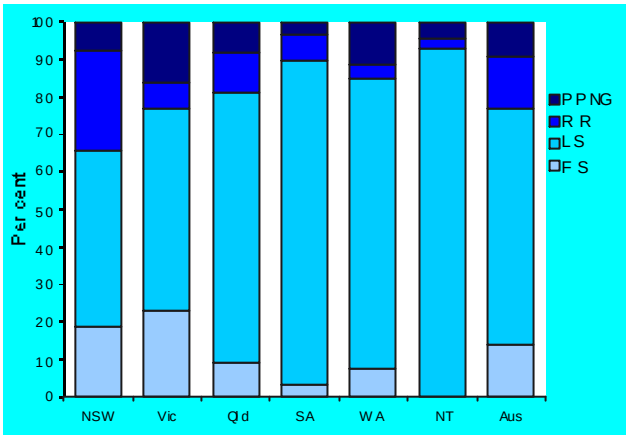
The number of PPNG isolates across Australia (58) was lower in this quarter than in the corresponding period in 2000 (74). The highest proportion of PPNG was found in isolates from Victoria (13%) and Queensland (10%). PPNG were present in all jurisdictions including 2 (1.9%) in the Northern Territory. South and South East Asian countries were the main source of external acquisition, but included an isolate acquired in Ethiopia. Local acquisition was prominent in Victoria and New South Wales.

More isolates were resistant to the penicillins by separate chromosomal mechanisms (128). These CMRNG were especially prominent in South Australia (29% of isolates there), New South Wales (22%), Victoria (13%) and Queensland (11%). Two CMRNG isolates were detected in the Northern Territory.

Ceftriaxone

Low numbers of isolates with decreased susceptibility to ceftriaxone were present in the Northern Territory and Western Australia.

Figure 1. Categorisation of gonococci isolated, Australia, 1 April to 30 June 2001, by penicillin susceptibility and by 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*

Spectinomycin

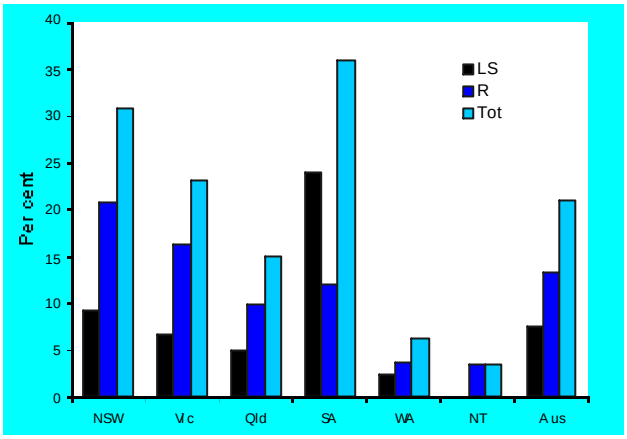
All isolates were susceptible to this injectable agent.

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 (Figure 2).

The total number (165) of all QRNG was again high but lower in this quarter than in the previous quarter (197) and the corresponding period in 2000 (183). QRNG were 19 per cent of all strains examined and this proportion was little changed from the second quarter of 2000. QRNG were again widely distributed. High proportions were maintained in South Australia (25%), New South Wales (28%), Queensland (18%) and Western Australia (13%). A marked decrease in the number of QRNG was seen in Victoria where the 12 QRNG represented 9 per cent of isolates. In the corresponding period of 2000, 25 per cent of Victorian

Figure 2. Distribution of *N. Gonorrhoeae* showing quinolone resistance, Australia, 1 April to 30 June 2001



LS QRNG Ciprofloxacin MICs 0.06 – 0.5 mg/L
R QRNG Ciprofloxacin MICs ≥1 mg/L

strains were QRNG and the proportion was 22 per cent in the previous quarter. In both New South Wales and now Victoria there has been a significant decrease in the number of 'less sensitive' QRNG in recent quarters. However, in New South Wales and also in Queensland, the proportion of isolation of 'resistant' QRNG has accelerated. Ninety-three of the New South Wales (24%), 21 (16%) of the Queensland and 7 (22%) of the South Australian gonococci exhibited high level resistance (MIC ciprofloxacin ≥1 mg/L). In Victoria this number declined to 9 from the 23 seen in the first quarter of 2001. Higher level QRNG were also seen in the Northern Territory and Western Australia. Local acquisition was again prominent and MICs ranged up to 16mg/L. The majority of QRNG (137 of 165, 83%) are now in the high level range compared with 64 per cent in this category the previous quarter and 40 per cent in the same period last year.

High level tetracycline resistance (TRNG)

The number (56) and proportion (6.5%) of TRNG detected continued to decline. TRNG represented 14 per cent of isolates from Queensland, 10 per cent of those from Victoria, 5 per cent from New South Wales, 4 per cent from Western Australia and 2 per cent from the Northern Territory.

Reference

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

Australian encephalitis: Sentinel Chicken Surveillance Programme

Sentinel chicken flocks are used to monitor flavivirus activity in Australia. The main viruses of concern are Murray Valley encephalitis (MVE) and Kunjin which cause the potentially fatal disease encephalitis, in humans. Currently 30 flocks are maintained in the north of Western Australia, 9 in the Northern Territory, 12 in New South Wales and 10 in Victoria. The flocks in Western Australia and the Northern Territory are tested year round but those in New South Wales and Victoria are tested only from November to March, during the main risk season.

Results are coordinated by the Arbovirus Laboratory in Perth and reported bimonthly. For more information and details of the location of sentinel chicken sites see Commun Dis Intell *2000;24:8-9.*

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July/August 2001

Sentinel chicken serology was carried out for 21 of the 30 flocks in Western Australia in July and August 2001. The number of seroconversions to flaviviruses have decreased in the north of Western Australia but MVE and Kunjin virus activity was still detected in both the Kimberley and Pilbara regions. In July there were 4 seroconversions from the Kimberley: 1 MVE from Kununurra; 2 KUN from Halls Creek; 1 Flavivirus from Kalumburu and 1 MVE seroconversion from Karratha in the Pilbara. In August there were no seroconversions from the Kimberley but 5 were recorded from the Pilbara: One FLAVI positive was reported from Port Hedland, 1 MVE seroconversion from both Harding Dam and Marble Bar and 2 seroconversions (1 FLAVI and 1 KUN) from Paraburdoo. A number of these seroconversions have yet to be confirmed.

All of the Western Australian flocks will be replaced in September 2001.

Serum samples from all 8 Northern Territory sentinel chicken flocks were tested at the University of Western Australia in July 2001 and samples from 5 flocks were tested in August 2001. There was only one new seroconversion to MVE virus in the Alice Springs flock in July.

The State Health Departments provide funding for the sentinel chicken surveillance programs in Western Australia and the Northern Territory.

September/October 2001

Sentinel chicken serology was carried out for 25 of the 30 flocks in Western Australia in September and October 2001. There was only one seroconversion to MVE virus from Marble Bar in the Pilbara region. The majority of the Western Australian flocks were replaced in September 2001.

Serum samples from all 8 Northern Territory sentinel chicken flocks were tested at the University of Western Australia in September 2001 and samples from 3 flocks were tested in October 2001. There were no new seroconversions to flaviviruses during this period.

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 and related Diseases 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: (02) 9332 4648. Facsimile: (02) 9332 1837.

HIV and AIDS diagnoses and deaths following AIDS reported for 1 April to 30 June 2001, as reported to 30 September 2001, are included in this issue of Communicable Diseases Intelligence *(Tables 8 and 9).*

Table 8. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 April to 30 June 2001, by sex and State or Territory of diagnosis

										Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2001	This period 2000	Year to date 2001	Year to date 2000
HIV diagnoses	Female	0	7	0	4	2	0	3	1	17	21	42	42
	Male	0	68	1	19	5	0	43	3	139	169	303	366
	Sex not reported	0	2	0	0	0	0	0	0	2	0	2	0
	Total [1]	0	77	1	23	7	0	46	4	158	191	348	410
AIDS diagnoses	Female	0	0	0	0	0	0	0	0	0	5	2	13
	Male	0	6	0	2	0	0	9	3	20	45	44	115
	Total [1]	0	6	0	2	0	0	9	3	20	50	47	128
AIDS deaths	Female	0	0	0	1	0	0	0	0	1	2	3	5
	Male	0	3	0	3	1	0	4	1	12	35	24	64
	Total [1]	0	3	0	4	1	0	4	1	13	37	27	69

Table 9. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 30 September 2001, by sex and State or Territory

		State or Territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	27	655	10	173	66	5	237	124	1,297
	Male	228	11,398	110	2,121	707	80	4,115	959	19,718
	Sex not reported	0	245	0	0	0	0	24	0	269
	Total[1]	255	12,319	120	2,301	773	85	4,391	1,089	21,333
AIDS diagnoses	Female	9	202	0	50	25	3	73	27	389
	Male	87	4,757	37	867	352	45	1,709	363	8,217
	Total[1]	96	4,971	37	919	377	48	1,791	392	8,631
AIDS deaths	Female	4	115	0	34	16	2	51	17	239
	Male	68	3,258	24	585	235	29	1,306	256	5,761
	Total[1]	72	3,381	24	621	251	31	1,364	274	6,018

1. Persons whose sex was reported as transgender are included in the totals

Childhood Immunisation Coverage

Tables 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 age 12 months for the cohort born between 1 April to 30 June 2000 and at 24 months of age for the cohort born between 1 April to 30 June 1999 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.*

Table 10. Percentage of children immunised at 1 year of age, preliminary results by disease and State for the birth cohort 1 April to 30 June 2000; assessment date 30 September 2001.

	State or Territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,088	21,807	931	12,274	4,414	1,460	15,071	6,090	63,135
Diphtheria, Tetanus, Pertussis (%)	93.2	91.4	90.3	92.5	92.4	91.8	92.6	90.4	91.9
Poliomyelitis (%)	93.2	91.4	90.0	92.4	92.4	91.8	92.6	90.3	91.8
Haemophilus influenzae type b (%)	95.3	94.1	94.2	94.6	95.0	94.8	95.1	93.8	94.5
Fully immunised (%)	92.7	90.7	89.3	91.8	91.6	91.0	92.0	89.5	91.2
Change in fully immunised since last quarter (%)	0.0	+0.2	+0.1	-0.2	-0.6	-0.6	-0.1	-0.7	-0.1

Table 11. Proportion of children immunised at 2 years of age, preliminary results by disease and State for the birth cohort 1 April to 30 June 1999; assessment date 30 September 2001 [1]

	State or Territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,106	21,732	907	12,375	4,629	1,567	15,088	6,480	63,884
Diphtheria, Tetanus, Pertussis (%)	89.2	89.2	82.4	91.0	91.1	90.2	90.4	88.6	89.8
Poliomyelitis (%)	94.3	93.1	94.5	94.1	95.2	94.2	94.4	93.4	93.9
Haemophilus influenzae type b (%)	95.2	94.9	91.8	95.0	96.4	95.2	95.8	94.6	95.2
Measles, Mumps, Rubella (%)	93.3	92.3	93.2	93.3	95.1	93.9	93.5	92.8	93.1
Fully immunised (%)[2]	86.6	85.7	79.8	88.6	89.1	88.7	87.5	86.0	87.0
Change in fully immunised since last quarter (%)	-1.2	+3.8	-0.2	+0.3	+0.5	+0.1	+1.1	+2.9	+1.9

1. The 12 months age data for this cohort was published in *Commun Dis Intell* 2000;24:324.
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.

Acknowledgment: These figures were provided by the Health Insurance Commission (HIC), to specifications provided by the Commonwealth Department of Health and Aged Care. For further information on these figures or data on the Australian Childhood Immunisation Register please contact the Immunisation Section of the HIC: Telephone 02 6124 6607.

Overseas briefs

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.

Hepatitis B infection in Scottish hospital

Source: The Guardian Online, 24 August 2001 (edited)

Health officials are trying to trace 350 hospital patients following the death of a 79-year-old man who caught hepatitis B from a surgeon at Queen Margaret Hospital in Dunfermline, Fife. A hospital surgeon was identified as the source of the infection. A second patient also contracted the disease from the doctor and is recovering. There were calls for hospital infection control strategies to be urgently reviewed after it emerged that the surgeon, with 27 years experience, had been immunised against hepatitis B and had undergone tests that indicated that the immunisation had worked.

Preliminary DNA testing suggest all 3 (isolates) of the virus are extremely similar and given that the surgeon was involved in both the patients' operations within the right time period for the incubation of the virus, both patients may have caught the hepatitis B virus from the surgeon. The 79-year-old patient contracted the virus last autumn after a gall bladder operation and died in February 2001.

This incident is unusual in that the surgeon had followed all the currently recommended safety procedures and was permitted to operate, yet remained infectious. A larger incident of the same kind occurred in another European country, where a surgeon who had been repeatedly vaccinated against hepatitis B but never showed seroconversion turned out to be a carrier. A retrospective survey showed that at least 28 patients (out of nearly 2,000) operated on by this surgeon over several years had contracted hepatitis B and 11 were shown to be the same strain as the surgeon's.

Hepatitis B in Guangdong Province, China

Source Philadelphia Inquirer, Associated Press report, 23 August 2001 (edited)

Shanghai: The use of dirty needles in injections and acupuncture has helped give the southern province of Guangdong one of the highest rates of hepatitis B infection in the world. Blood samples taken from patients during hospital visits show that 10 million people — 75 per cent of the province's population — have had the potentially lethal disease. Early surveys indicated that two thirds of China's 1.26 billion people had been infected, compared with about one in 20 Americans.

About 60 per cent of those who have had the disease caught it during childhood, usually during routine vaccinations. Mothers may also infect their children during birth or while breastfeeding. Most of those infected with hepatitis B survive. But in some cases, the virus continues to attack the liver, causing cirrhosis and cancer. These diseases kill about 300,000 people in China each year, about 80 per cent of whom had hepatitis B.

Experts also blame an illegal trade in needles that have been inadequately cleaned and repackaged. They also say that there are increasing reports of infection from acupuncture, a traditional Chinese remedy in which dozens of needles may be stuck into the skin. Effective vaccinations against hepatitis B exist and are now required for children in the United States. But at $US25, they are too expensive for most Chinese and are not covered by national health insurance.

Poliomyelitis — Dominican Republic — visitor advice

Source: MMWR 50(39);855-6. Public Health Dispatch: 5 October 2001 (edited)

From 12 July 2000 to 18 September 2001, a total of 21 cases of poliomyelitis (including 2 fatal cases) were reported from the Caribbean island of Hispaniola, divided between Haiti and the Dominican Republic. In the Dominican Republic, 13 of 168 reported cases of acute flaccid paralysis (AFP) were confirmed as polio by isolation of poliovirus type 1 from either patients or their healthy contacts. The median age of the patients was 3 years (range: 9 months 14 years). None were vaccinated adequately. The most recent confirmed case-patient in the Dominican Republic had paralysis onset on 25 January 2001.

In Haiti, 8 of 40 AFP cases were confirmed virologically; seven of the confirmed cases occurred during January to July 2001. The median age of the patients was 7 years (range: 2–12 years). One patient had received at least 3 doses of oral poliovirus vaccine (OPV). The most recent confirmed case occurred in Haiti and the patient had paralysis onset on 12 July 2001. Currently 18 AFP cases from the Dominican Republic and three from Haiti are pending final classification.

This outbreak was the first in the Americas since 1991 and was associated with the circulation of a type 1 OPV-derived virus, having substitutions affecting 1.8 per cent to 4.1 per cent of nucleotides encoding the major capsid protein (VP1). The circulating vaccine-derived poliovirus associated with the outbreak recovered the capacity to cause paralytic disease and widespread person-to-person transmission and was biologically indistinguishable from type 1 wild poliovirus. Contemporary vaccine-derived poliovirus isolates from persons with AFP in other countries of the Americas are more closely related (>99.5% VP1 sequence similarity) to the respective OPV strains, are unrelated to the Hispaniola outbreak viruses, and show no evidence of extensive person-to-person transmission. The outbreak in Hispaniola occurred in areas of very low OPV coverage.

In response to the outbreak, health authorities in both countries conducted house-to-house vaccination with OPV. In December 2000, and February and April 2001, 3 rounds of mass vaccination campaigns were conducted in the Dominican Republic. In each round, approximately 1.2 million OPV doses were administered to an estimated

population of 1.1 million children aged <5 years. Haiti conducted 2 rounds of mass vaccination in February and March 2001.

References

CDC. Outbreak of poliomyelitis-Dominican Republic and Haiti, 2000. MMWR 2000;49: 1094103.

CDC. Outbreak of poliomyelitis-Dominican Republic and Haiti, 20002001.MMWR 2001;50:1478.

CDC. Poliomyelitis prevention in the United States: updated recommendations of the Advisory Committee on Immunisation Practices (ACIP). MMWR 2000;49 (no.RR-5).

Imported poliomyelitis, Bulgaria

Source: Eurosurveillance Weekly, Issue 45, 8 November 2001 (edited)

In May this year, *Eurosurveillance Weekly* reported on the occurrence of 2 cases of poliomyelitis in Bulgaria.[1] The patients — 2 children of Romany origin — were infected with a wild poliovirus closely related to a strain isolated from India in July 2000. After the virus had been identified, Bulgaria's health ministry initiated contact tracing, screening of children at high risk (children from particularly vulnerable communities or living in nearby areas), a retrospective review of records, intensified surveillance for acute flaccid paralysis, and a mass vaccination campaign.[2]

Between 28 April and 22 May 2001, stool specimens were obtained from 117 children at high risk of exposure, who had been admitted to hospital in 9 different districts. Children who had been on the same ward as one of the cases and the household contacts of both cases were also screened. Wild type human poliovirus 1 was found in two of the children screened — the sibling of one of these had shared the hospital ward with the first case — but neither child showed symptoms of poliomyelitis. Further stool specimens were collected from 244 children country-wide after an outbreak of viral meningitis due to Echovirus 30 between June and August 2001. Although 7 children carried a vaccine-derived virus, none was found with a wild virus. A final survey of 155 high-risk children aged <3 years in 10 district hospitals was conducted between 23 August and 11 September 2001. No wild polio viruses were detected.

To control the outbreak, a regional mass vaccination campaign was launched on 19 April 2001. A national vaccination campaign of 2 rounds with the goal of vaccinating all 468,720 children aged 0–6 years was conducted during the periods 28 May to 1 June and 25–29 June 2001. Administrative estimates of coverage suggest that 94 per cent of all children in the country were vaccinated during the first round and 95 per cent during the second.

This outbreak illustrates the occurrence of transmission over several months of a wild poliovirus imported into a country that had been free of poliomyelitis for almost 10 years. The outbreak occurred because a virus was imported into Bulgaria from an unknown source and infected population subgroups with low immunity. High coverage reported for the campaign nationwide, improved performance of surveillance for acute flaccid paralysis, and the absence of wild polio viruses in subsequent stool surveys of children at high risk suggest that circulation of the wild virus has been interrupted.

References

1. Noah N, Ramsay M, Twisselmann B. Imported cases of polio in Bulgaria. Eurosurveillance Weekly 2001;5:010524. <http://www.eurosurv.org/2001/010524.html>
2. World Health Organization. Imported wild poliovirus causing poliomyelitis, Bulgaria, 2001. Wkly Epidemiol Rec 2001;76: 332-5. <http://www.who.int/wer/pdf/2001/wer7643.pdf>

Acute flaccid paralysis in the Philippines

Three cases of acute flaccid paralysis (AFP) associated with circulating vaccine-derived poliovirus (cVDPV) isolates were reported in the Philippines between 15 March and 26 July, 2001. The first case-patient, a child aged 8 years from northern Mindanao Island (500 miles south of Manila) who had received 3 doses of oral polio vaccine (OPV), had onset of paralysis on 15 March. A second child aged 3 years from Laguna province on Luzon Island (60 miles south of Manila) who had received 3 OPV doses, presented with signs of meningitis but no paralysis on 23 July. A third child aged 14 months from Cavite province (25 miles from Manila and 45 miles north of Laguna province) who had received 2 OPV doses, had onset of paralysis on 26 July. None of the patients had travelled outside of their province of residence since birth. Characterisation of isolates from the 3 patients revealed type 1 polio viruses derived from Sabin vaccine strain type 1, with a 3 per cent genetic sequence difference between Sabin 1 vaccine and vaccine-derived poliovirus (VDPV) isolates. The 3 polio viruses were not identical but were closely related (99% sequence homology); they also appeared to share an identical recombination site with a non-polio enterovirus in the non-capsid region of the genome.

Wild poliovirus was last reported in the Philippines in 1993, and national vaccination rounds were last conducted in the Philippines in 1997 followed by regional immunisation days in 1998 and 1999. Among the areas covered were Cebu, Davao, Manila, and parts of Mindanao; however, coverage did not extend to the 3 provinces now reporting cVDPV cases. Routine coverage with 3 OPV doses has been approximately 80 per cent nationwide since the early 1990s; however, coverage gaps are likely, particularly in slum areas.

A combination of 2 concurrent events within the virus is necessary for cVDPV emergence: reversion of attenuating mutations to increase neurovirulence, and a presumed increase in transmission characteristics that might be related to recombination with a non-polio enterovirus. The molecular basis for the second property is not understood. This is now the third documented episode of poliomyelitis-like illness (acute flaccid paralysis - AFP) due to circulating vaccine-derived poliovirus (cVDPV) with reversion to neurovirulence. The two prior episodes were on the island of Hispaniola (Dominican Republic and Haiti) and in Egypt. In addition, there were reports of circulating vaccine-derived poliovirus in Israel identified in sewage sampling but not associated with clinical illness.

As wild poliovirus circulation continues, there is still a need to keep up intensified vaccination efforts, as the risk of disease is still present. The occurrence of cVDPV in association with clinical disease is very disturbing as it adds another factor into the risk benefit equation of vaccination recommendations.

Variant CJD, incidence and trends — UK

Source: Eurosurveillance Weekly*, Issue 46, 15 November 2001 (edited)*

By the beginning of November 2001, the total number of cases of variant Creutzfeldt-Jakob disease (vCJD) reported in the United Kingdom had reached 111.

To monitor the underlying trend in vCJD incidence and identify any differences in this trend by factors such as birth cohort and sex, quarterly analyses are performed that estimate the underlying trend. Models fitted to the data also enable short-term predictions for the expected number of deaths in the next year and an estimate of the total number of people with onset of symptoms who are yet to be identified. These quarterly analyses are now available on the CJD surveillance unit Website at <www.cjd.ed.ac.uk>, with the first report covering the period up to September 2001. This report included 107 cases, of whom 101 had died. The distribution of these 107 cases by year of symptom onset, diagnosis, and death is shown in the Table. The median age at death for the cases in each year is also given; the overall median age was 28 (range 14-74 years). The median number of days from onset to diagnosis was 333 days and from onset to death was 401 days. Of the 107 cases, 56 (52%) were male.

The recent analyses showed that the underlying incidence is increasing by 22 per cent per year using date of symptom onset or 27 per cent per year using date of death.

The analysis using date of death included 101 cases. No adjustment for reporting delay was required for deaths because most cases are diagnosed before death. The estimated current quarterly incidence of deaths is 7.5, and, assuming the current trend continues, the total number of deaths in the next 12 months is predicted to be 35 (22–50).

So far the age at death has not increased with time as might be expected for an outbreak due to a point source in time. This stable age is generated by the fact that the underlying trend is increasing more quickly in those born after 1970 (42 per cent per year) compared to those born before 1970 (12 per cent per year). The difference between these trends is not quite significant (P=0.087) but, if real, may be generated by factors such as age dependent exposure or incubation periods.

These models allow short-term predictions and trend estimates but cannot be used for long-term predictions or predictions of the total size of the epidemic. This can only be attempted using techniques such as back calculation, as recently performed by Huillard et al.[1] The quarterly analyses will continue to be performed in order to update short-term predictions, assess differences in trend by sex and cohort, and detect any changes in the incidence trend that might indicate that the epidemic has reached a peak.

Reference

1. Huillard d'Aignaux JN, Cousens, SN, Smith PG. Predictability of the UK variant Creutzfeldt-Jakob disease epidemic. Published online 25 October 2001; <www.sciencexpress.org> (Science Express Reports).

Missing gene increases variant Creutzfeldt-Jakob disease risk

Source BBC News Online, 14 November 2001 (edited)

Recent research suggests that people who lack a particular version of a gene involved in immune responses may be three times more likely to suffer new-variant Creutzfeldt-Jakob disease (vCJD). If the finding is borne out in larger studies, it could provide scientists with an important clue in their bid to develop therapies for the incurable brain disease. It may also help doctors to identify those people at risk. The gene, called DQ7, is part of a complex called HLA that produces molecules responsible for presenting bits of proteins to the immune system. It does not seem to offer protection against the sporadic form of the disease, in which rogue particles called prions are thought to form spontaneously in the brain, but it does appear to play a role in people who develop vCJD

The researchers studied the genetic make up of 50 patients with vCJD, approximately half of the population known to have the disease. Only 12 per cent of the patients possessed the DQ7 gene, compared with 36 per cent of the normal population. Writing in the journal *Nature*, the investigators suggest that the presence of DQ7 protects against vCJD. However, they warn that although they studied about half of all the people known to have contracted vCJD, their sample is still too small to be conclusive. The strong likelihood is that other genes are also involved — if a combination of genetic markers could be identified, then it might be possible to introduce mass screening for susceptibility to vCJD.

Reference

Pathogenesis: HLA-DQ7 antigen and resistance to variant CJD. GS Jackson, JA Beck, C Navarrete, J Brown, PM Sutton, M Contreras & J Collinge.

Table. Cases of vCJD reported in the United Kingdom, diagnosed by September 2001 by year of onset, diagnosis, and death

Year	Onset	Diagnosis	Death	Median age at death
1994	8	0	0	-
1995	10	7	3	-
1996	11	8	10	30
1997	14	12	10	26
1998	17	17	18	25.5
1999	29	17	15	29
2000	18+	27	28	25.5
2001	0+	19	17	28

Dengue/DHF updates

14 November 2001

Nicaragua: Concern about the high rate of dengue fever

Nicaraguan Health Ministry officials expressed their concern about the high incidence of dengue fever this year that has already resulted in 11 deaths. Four people have died due to dengue fever since September alone, increasing dengue-related fatalities to 11 thus far this year and exceeding the number of deaths the disease caused over the same period last year. Confirmed cases of the more serious haemorrhagic dengue fever have risen to 222, much higher than the 91 cases reported over the first 11 months of last year. Some 1,390 cases of dengue fever have been reported in Nicaragua so far this year, up considerably from the 741 cases reported during the same time period in 2000.

Panama: Sixth dengue haemorrhagic fever case causes alarm

The sixth case of dengue haemorrhagic fever in Panama was confirmed on 13 November 2001. The Panamanian Health Minister said that: 'This definitely indicates a dengue fever epidemic in Panama; so, both community and society have to collaborate in the elimination of the (breeding) grounds of the *Aedes aegypti* mosquito.' According to figures issued by the Health Ministry, there were 317 classic dengue cases in Panama in 2000, including one haemorrhagic type and no deaths were reported, compared to the 6 haemorrhagic-type and 698 classic cases registered this year.

Dengue fever — Hawaii

The number of confirmed cases in Hawaii has reached 74, with the Centers for Disease Control and Prevention (CDC) in Atlanta confirming the first case of the mosquito-borne illness on the 'big island' of Hawaii. So far 56 of the cases have been on Maui, where the first locally transmitted cases of dengue appeared in early September. The CDC said that it was the first outbreak of dengue in Hawaii in more than 56 years.

Epidemiology of hepatitis E in Spain

Source: Diario Medico, 19 September 2001 (edited)

According to research at the Hospital Valle de Hebron in Barcelona, the epidemiology of the hepatitis E virus (HEV) in non-endemic countries must be reviewed, given that there are sporadic cases of HEV hepatitis in which pigs may be the animal reservoir. In industrialised countries, HEV may be associated with sporadic imported cases, although some studies show that 1-5 per cent of the population of those countries have antibodies.

The research project describes the detection and genetic characterisation of HEV strains in Spain, in a region not considered endemic, and the identification of pigs as animal reservoirs of HEV. In order to identify the viral strains, researchers used nested RT-PCR followed by sequencing of the resulting amplified fragments. ELISA was used for antibody detection in human and swine serum samples. Tests showed that 20 per cent of the analysed samples of serum from pigs of various ages and locations had antibodies against HEV. In addition, some strains of HEV have been detected in residual waters in Barcelona and in the sera of patients from the same area with acute hepatitis. Over the past year, HEV RNA has been detected in 8 samples of residual waters. Of the 67 samples from hepatitis patients tested, 10 per cent had anti-HEV antibodies.[1]

The initial report identifying the relationship between HEV and a swine virus was published in 1997.[2] Since then, HEV has been under consideration as a zoonotic disease. In addition there have been concerns with respect to HEV and xenotransplantation of pig organs for human transplants. Clearly, it is an emerging disease in terms of our knowledge and recognition of the organism.

References

1. Pina S, Buti M, Cotrina M, Piella J, Girones R. HEV identified in serum from humans with acute hepatitis and in sewage of animal origin in Spain. *J Hepatol* 2000;33:826-833.
2. Meng XJ, Purcell RH, Halbur PG, Lehman JR, Webb DM, Tsareva TS, et al. A novel virus in swine is closely related to the human hepatitis E virus. *Proc Natl Acad Sci U S A.* 1997;94: 9860-9865. summary of findings can be found at: <http://www.niaid.nih.gov/publications/dateline/0398/hep.htm>

Cryptococcus Neoformans — *Canada*

Source: CBC News 3 September 2001(edited)

There have been about 25 cases of *Cryptococcus neoformans* in the province of British Columbia (BC) during the past 30 months: 5 times the normal rate. Four people have died from complications caused by the infection. The province's Centre for Disease Control is now concerned that otherwise healthy people are showing signs of *Cryptococcus neoformans* infection. Symptoms include chest pains, a stubborn cough, severe headaches, neck stiffness, and difficulty breathing.

There are several variations/strains of *Cryptococcus neoformans of which the two most common are C. neoformans* var. *neoformans* and *C. neoformans* var. *gattii.* The var. *gattii* is seen more frequently in tropical or subtropical climates (including parts of northern Australia). The organism is found in soil and bird droppings (including pigeons); the var. *gattii* has also been isolated from foliage and bark of certain species of eucalyptus trees. Sporadic cases of cryptococcosis occur in all parts of the world. Cell-mediated immunodeficiency and immunosuppression are predisposing conditions for clinical disease. Clinical disease includes a subacute or chronic meningitis, infection of lungs, kidneys, prostate and bone; skin involvement such as acneiform lesions, ulcers or subcutaneous nodules have also been described. Eye infections have resulted in blindness.

Mortality due to cryptococcosis increased from 0.09 per 100,000 in 1980, to 0.12 per 100,000 in 1994. Eighty per cent to 90 per cent of infections were AIDS-associated.

Yaws re-emergence — Papua New Guinea

Source: World News from Radio Australia 5 September 2001 (edited)

Health authorities in Papua New Guinea have reported an outbreak of yaws, a skin and bone disease, in the Bitapaka area on New Britain Island. Scientifically known as Frambesia, yaws mainly affects children living in humid

tropical regions. Its symptoms include lesions or skin eruptions that appear and disappear during the course of the disease. According to the acting district health officer in the Kokopo District, 3,000 cases of yaws have been confirmed over the past 4 weeks. Medical officers have carried out an intervention program, which includes awareness and prevention lessons.

According to the World Health Organization, yaws was supposed to have been fully eradicated. It affects people living in unhygienic conditions and can be spread by unsafe drinking water and lack of proper sanitation. Yaws has been known for several years to be re-emerging in Papua New Guinea. Once thought eradicated, it is apparent that the disease continues to be endemic there. It will probably remain so until problems of poverty and poor hygiene are solved.

Pacific Public Health Surveillance Network

The Pacific Public Health Surveillance Network serves to disseminate information about communicable diseases in the Pacific region through Pacnet. Pacnet may be accessed on registration, through the South Pacific Commission Website (http://www.spc.org.nc).

'Southern Hemisphere' influenza vaccine 2002

Source: Press release WHO/41 18 September 2001

The recommendation for the composition of the vaccine for the 2002 Southern Hemisphere influenza season has been decided and communicated to vaccine manufacturers, by the World Health Organization (WHO). About 200 million influenza vaccine doses are produced and given globally every year. The annual decision about the vaccine composition is made possible by the co-ordinated work of more than 110 influenza laboratories and four WHO Collaborating Centres.

WHO experts recommended that the influenza vaccine for 2002 in the Southern Hemisphere contain the following three components:

- A/Moscow/10/99(H3N2)-like virus
- A/New Caledonia/20/99(H1N1)-like virus
- B/Sichuan/379/99-like virus

This vaccine is intended for use from May to October 2002, the Southern Hemisphere influenza season. The timing of this WHO recommendation is critical to allow sufficient time for companies to produce a novel vaccine before the next influenza season starts. Based on the WHO recommendation, national authorities should approve the specific vaccine viruses and national public health authorities are responsible for recommendations regarding the use of vaccines.

The influenza season in the Northern Hemisphere will soon start and it is time for vaccination. Many countries have already begun advertising for vaccination campaigns focusing on high-risk groups (people 65 years or older; adults and children aged 6 months or older with chronic illnesses or who are immunocompromised).

The 'flu' has been estimated to infect as many as 100 million people each year in the Northern Hemisphere. While most healthy people fully recover from the flu, the disease can result in hospitalisation or even death. WHO, therefore, strongly recommends vaccination against influenza: the most important measure against the disease, particularly among those at high risk of developing complications.

Recommendations for influenza vaccine composition Northern Hemisphere: 2001-2002

- A/New Caledonia/20/99(H1N1)-like virus
- A/Moscow/10/99(H3N2)-like virus*
- B/Sichuan/379/99-like virus†

* The widely used vaccine strain A/Panama/2007/99 is an A/Moscow/10/99-like virus.

† B/Johannesburg/5/99 and B/Victoria/504/2000 are B/Sichuan/379/99-like viruses, which have been used for vaccine production.)

Molecular basis for influenza virulence

Molecular basis for high virulence of Hong Kong H5N1 influenza A viruses. Masato Hatta, Peng Gao, Peter Halfmann, and Yoshihiro Kawaoka *Science* 2001 293:1840-1842 (Abstract).

In 1997, an H5N1 influenza A virus was transmitted from birds to humans in Hong Kong, killing 6 of the 18 people infected. When mice were infected with the human isolates, 2 virulence groups became apparent. Using reverse genetics, we showed that a mutation at position 627 in the PB2 protein influenced the outcome of infection in mice. Moreover, high cleavability of the haemagglutinin glycoprotein was an essential requirement for lethal infection.

Recombination in the haemagglutinin gene of the 1918 'Spanish flu'. Mark J Gibbs, John S Armstrong, and Adrian J Gibbs *Science* 2001 293:1842-1845 (Abstract).

When gene sequences from the influenza virus that caused the 1918 pandemic were first compared with those of related viruses, they yielded few clues about its origins and virulence. Our reanalysis indicates that the haemagglutinin gene, a key virulence determinant, originated by recombination. The 'globular domain' of the 1918 haemagglutinin protein was encoded by a part of a gene derived from a swine-lineage influenza, whereas the 'stalk' was encoded by parts derived from a human-lineage influenza. Phylogenetic analyses showed that this recombination, which probably changed the virulence of the virus, occurred at the start of, or immediately before, the pandemic and thus may have triggered it.