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Editorial: Review of the Laboratory Virology and Serology Reporting Scheme

Tim JJ Inglis, Division of Microbiology and Infectious Diseases, PathCentre, Western Australia

The Laboratory Virology and Serology Reporting Scheme (LabVISE), the national surveillance scheme for virology and serology laboratory results has just undergone a detailed review. The data included in the LabVISE appraisal covers reports on a large number of viral and other pathogens over the decade 1991 to 2000. The total dataset available on LabVISE indicators stands at over 500,000 items since its inception in 1982. During the period of the current review there were significant changes in the scope and objectives of the surveillance program. Most notable of these were the establishment of the National Notifiable Disease Surveillance Scheme (NNDSS) in 1991, a simplification of LabVISE in 1995 and transfer of responsibility for the scheme to the National Public Health Laboratory Network in 1998. This report is therefore the first formal evaluation of LabVISE data since 1996. The dataset reported under the LabVISE program was also subject to change during this period, most notably the exclusion of hepatitis B and C, herpesvirus and *Neisseria gonorrhoea*, and a reduction in the number of data fields collected. Throughout this period denominator data were not provided and there have been significant inconsistencies in the completeness of reporting. There are particular deficiencies in some jurisdictions where major reference laboratories have not contributed data regularly. The private sector laboratories have been notable in their under-representation and overall, total test numbers have not been available.

Considering the difficulties of working within these limitations, the authors of the current report are to be congratulated for completing an undertaking of this size. If the project achieves nothing else, it provides a useful insight into the national perception of what infectious diseases matter and to whom. In the absence of accurate denominator data, specific populations, procedures or pathogens all suffer from bias created by their advocates and detractors. The LabVISE system has been useful in showing seasonal and epidemic activity, for example with influenza. In 1995 surveillance of most of the diseases that were not seasonal (such as herpes simplex, hepatitis B) was discontinued. Surveillance was continued for other diseases in order to get some national measure of seasonal/epidemic disease. However, it was recognised at this time that a better structured scheme was needed with improved representation of the Australian population and better denominator data.

It is a cruel irony with which most users of LabVISE data will be familiar that perception is often as important as accurate measurement of disease burden. For this reason, readers of the report will need to be cautious about changes in reporting that might be explained by fluctuating fashions in infectious disease practice, or the epidemiological self-fulfilling prophecies on which many pseudo-outbreaks are founded. In summary, the well-recognised limitations of the current LabVISE dataset render it difficult to use as a field epidemiology resource.

Undoubtedly the most practically useful material in this report is its demonstration of the nationwide presence of a range of non-notifable pathogens. The NNDSS may have taken the lead role in collating data on notifiable pathogens, but even on-line, laboratory-based notification has not overcome the problem of reliable denominator data or addressed the problem of slow turnaround of analysed data to local public health jurisdictions. LabVISE is therefore a useful reality check on NNDSS data, as is clear from measles virus results over the review period in which correlation between the two surveillance schemes is good despite higher notification rates to NNDSS. LabVISE has been one of the few sources of national data on laboratory-confirmed influenza. Isolates are analysed by the WHO Collaborating Centre for Research on Influenza to determine the virus strains for the annual review of vaccine composition. The persistent presence of other vaccine preventable viral infections in the Australian population is documented in the current review of LabVISE data and provides a valuable perspective on the efficacy of vaccination that supplements more targeted seroepidemiological studies. The presence of a range of respiratory, neurotrophic, enteric and other pathogens in the

LabVISE dataset is a timely reminder of the limited range of agents against which an effective vaccine exists.

It is a pity, then, that data on other bacterial pathogens such as *Leptospira* spp. are not presented in this review. Some of the criticisms levelled at LabVISE might have been more easily dispelled if the surveillance program had more input from bacteriologists. This is perhaps an appropriate point at which to admit to having taken the LabVISE program for granted at an important stage in its development. If this is a case of use-it-or-lose-it, then the nation's microbiologists really need to take LabVISE under their wing and advocate a more pro-active reporting process along the lines used in some European jurisdictions where all validated data is transmitted on-line. In return, I look forward to a more rapid completion of the epidemiological loop in which the information providers (the laboratories) are provided with equally rapid feedback of aggregate data. Presentation of laboratory data in a geographical context with time and space cluster analysis has become an urgent priority, particularly in view of events on 11 September last year and following. A preview of what a simple geographical representation of disease events might look like can be found on the EIDIOR website (http://www.e-tiology.com/).

It has become fashionable in some circles to criticise surveillance of infectious diseases as data collection for its own sake. The falling frequency of *Communicable Diseases Intelligence* (*CDI*) publication has reduced the immediacy of LabVISE aggregate data reports. Fortnightly posting of NNDSS data on an open-access website goes some way to compensating for the loss of periodic overview via the *CDI* route. Both reporting schemes act as a form of advocacy for continuing laboratory work on diseases for which there is no suitable chemotherapy and no available vaccine. Expert bodies such as the National Health and Medical Research Council do well to take note of these gaps in national defences against public health threats. It is one more reflection of the great Australian tyranny of distance that much of the number crunching is far removed from the public health front line. While we can argue exactly what constitutes that front line, it is clear that accurate, timely information is one of most useful weapons.

Is it not time that the various stakeholders established some common ground on the ownership of aggregated surveillance data and concentrated their efforts on finding more imaginative ways of launching their results into the public domain? There is an opportunity through ProMED or the various regional surveillance networks to take a regional lead in infectious disease surveillance. However, ProMED posts surprisingly little data from Australia given the wealth of data returned from laboratories to data-collecting centres. Indeed, regional surveillance networks such as the small Indian Ocean Rim network (EIDIOR) provide ready-made opportunities to openly share surveillance data in a region-wide capacity building exercise.

The authors of the present report on LabVISE are to be commended for their energy and enthusiasm. It would be a great shame if this valuable public health resource were not developed further.

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The Laboratory Virology and Serology Reporting Scheme, 1991 to 2000

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Abstract

Between 1991 and 2000, the Laboratory Virology and Serology Surveillance Scheme (LabVISE) received 340,730 laboratory reports of viral and non-viral pathogen identifications. In this report, data on 136 viruses and 31 non-viral pathogens is analysed. The age and sex distribution and seasonal fluctuations in infections are described. The major clinical diseases associated with LabVISE pathogens are reviewed together with a survey of recent activity reported in Australia. The contribution of LabVISE over the 10-year period to surveillance of poliomyelitis in Australia, up to and beyond the eradication goal, is described. The contribution of LabVISE to influenza surveillance and control in Australia is also described. Prospects for the continued role of LabVISE in the surveillance and control of viral meningitis, viral gastroenteritis and viral respiratory diseases are reviewed. *Commun Dis Intell* 2002;26:323-374.

Keywords: laboratory reports, viruses, surveillance, disease control

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Abbreviations

CMV	Cytomegalovirus
CNS	Central Nervous System
DAD	Disseminated adenovirus disease
DNA	Deoxyribonucleic acid
EBV	Epstein-Barr virus
HEV	Hepatitis E virus
HPIV	Human parainfluenza viruses
HPV	Human papillomaviruses
HSV-2	Herpes simplex virus type 2
LabDOSS	Laboratory Database of Organisms from Sterile Sites
LabVISE	Laboratory Virology and Serology Reporting Scheme
MMR	Measles-mumps-rubella vaccine
NLV	Norwalk-like virus
NNDSS	National Notifiable Diseases Surveillance System
PHLN	Public Health Laboratory Network
RRV	Ross River virus
RSV	Respiratory syncytial virus
USA	United States of America
WHO	World Health Organization

Introduction

The Laboratory Virology and Serology (LabVISE) Reporting Scheme is a passive surveillance scheme based on voluntary reports of infectious agents contributed by virology and serology laboratories around Australia. LabVISE provides information on a number of viruses and other infectious agents and basic demographic information of persons they infect.

In 1959 a group of virologists met to exchange information on viruses circulating in Victoria. Between 1962 and 1968 the group, which expanded to include virologists from other states, reported their findings quarterly in the *Medical Journal of Australia*. In 1975, the Commonwealth Department of Health in collaboration with virology laboratories, established the 'Virus Reporting Scheme', which was replaced in 1977 by the 'National Pathogen Reporting Scheme'. This scheme, consisting of 6 laboratories, sent data to the Commonwealth, which published the data in a fortnightly bulletin called the *National Microbiological Laboratory Reporting Scheme*. This scheme was replaced in 1992 by two parallel reporting schemes: Laboratory Database of Organisms from Sterile Sites (LabDOSS) and LabVISE. While LabDOSS collected data on bacterial and fungal infections, LabVISE collected data on pathogens diagnosed by virology and serology laboratories.

Meanwhile, a national database of communicable diseases was established in 1991 in the form of National Notifiable Diseases Surveillance System (NNDSS). The NNDSS, operating under the auspices of the Communicable Diseases Network Australia, reported on 49 diseases in the period 1991 to 2000. Several of these diseases were also reported by sentinel laboratories in LabVISE in the same period. In 1995, the laboratory schemes were reviewed and LabDOSS was discontinued, while LabVISE was simplified. There was a reduction in the number of pathogens under surveillance by LabVISE, primarily by removal of reports of hepatitis B and C isolations, which were being reported through the NNDSS. In addition, there was a reduction in the details collected on each isolate to a minimal dataset.

The National Communicable Diseases Surveillance Strategy (1996) recommended the strengthening of laboratory networks and collaborations between laboratories and epidemiologists. LabVISE was evaluated in 1999 and three options were presented to the Public Health Laboratory Network (PHLN). Of the three options, PHLN endorsed the retention and development of LabVISE as a broad based surveillance scheme with clear objectives and a feasibility study to assess additional uses of laboratory generated data and the possibility of real-time data transfer to state public health units and the commonwealth.

This report is the first assessment of data collected by LabVISE since 1996. LabVISE data for 1999 and 2000 were reported as part of the Australia's Notifiable Diseases Status reports for those years.[1] In this report, we review data collected since 1991 using the current list of organisms and data fields and compare LabVISE data with other data sources such as NNDSS (where applicable).

Materials and methods

Data collection and reporting laboratories

The LabVISE database contains over 645,000 records of infectious disease collected since 1982. Records include those previously collected by the 'National Pathogens Reporting Scheme' (1982–1992).

Data are reported to LabVISE as paper reports or electronic format. Electronic reporting by diskettes has been replaced more recently by e-mail. Over time, almost all laboratories have changed to report by e-mail although a few still rely on paper reports.

In 1999, the database was changed from a 'Focus mainframe' database to a 'MS Access' format to comply with year 2000 requirements. The Department of Health and Ageing had previously developed an Epi Info based data entry system to allow laboratories to enter and send data to LabVISE. This data extraction package was provided free of charge to contributing laboratories on request. In 1999, this system was replaced by the MS Access based system, 'LabData'.

In 1997, the LabVISE database was simplified by the removal of 12 fields (containing information on risk factors, clinical outcome, sources of clinical sample, methodology details and serogroup results). The data fields collected in LabVISE throughout the study period are shown in Table 1. Four fields are designated as mandatory and must be completed for a record to be accepted into the database.

In 1997 the number of organisms under surveillance in LabVISE was reduced by the exclusion of organisms such as hepatitis B and C, *Neisseria gonorrhoea* and herpesvirus. The organisms currently under surveillance and the totals reported between 1991 and 2000 are shown in Appendix 2. Only reports of viral pathogens, *Chlamydia*, *Mycoplasma*, and *Rickettsia* are analysed in this report.

The Surveillance and Epidemiology Section of the Commonwealth Department of Health and Ageing publishes reports of data from LabVISE in *Communicable Diseases Intelligence* (*CDI*). This bulletin was produced fortnightly between 1978 and September 1997, four weekly between October 1997 and March 2000, monthly between April and December 2000, and quarterly from 2001. LabVISE annual reports were published in *CDI* for the years 1992 to 1995,[2,3,4,5] the last two of these reports are also available on the Communicable Diseases Australia Website at http://www.health.gov.au/pubhlth/cdi/labannrep.htm.

Notes on interpretation

LabVISE data are difficult to interpret for a number of reasons. The representativeness of the data is uncertain, since there are no denominator data available and the reporting by pathogen has not been consistent. Although some major reference laboratories have been reporting to LabVISE, not all are represented. Laboratories from the Northern Territory have not been contributing regularly, although data from the Northern Territory are available in LabVISE via reference laboratories in other states. While public laboratories are well represented in LabVISE, larger private laboratories are not. As more pathology testing is being done in the latter in recent years, the representativeness of LabVISE becomes more uncertain. Although LabVISE data are reported by state and territory of the patient, disease rates have not been calculated. Alternative measurements such as rates of positive tests in each laboratory may be possible in the future, however, total test figures have not been available to date.

Since the number of reporting laboratories and total reports have varied over the 10-year period, we have not been able to draw conclusions about rates or outbreaks, except where independently confirmed. The quality of LabVISE data has

Table 1. Data field names and descriptions used in LabVISE, 1991 to 2000

Field name	Field data	Status*
Lab code	Unique 3-digit identification code for the sending laboratory	Mandatory
Lab ID	Unique patient identifier	Mandatory
Collection date	Date specimen was collected	Mandatory
2x2 identifier	Identifier composed of first 2 letters of first and first two letters of family name	Not mandatory
Sex	Gender of patient [Male (M), Female (F) or Unknown (U)]	Not mandatory
Date of birth	Patient's date of birth	Not mandatory
Age	Patient's age at date of specimen collection	Not mandatory
Postcode	Postcode of patients residence	Not mandatory
Diagnosis	Primary diagnosis, coded according to Table (Appendix 1)	Not mandatory
Organism	Primary organism isolated or identified in specimen (codes, Appendix 2)	Mandatory

* Mandatory fields must be complete for acceptance of a record into the LabVISE database

declined over time with details of viral serotypes for example, being less complete in more recent years. This limits the ability to comment on changes in viral serotypes circulating in Australia.

Further limitations on the interpretation of LabVISE data are the lack of agreed reporting protocols for contributing laboratories and the absence of diagnostic definitions, which would standardise reporting between laboratories. Although duplicate reports are removed from LabVISE, repeat testing of the same individual for the same pathogen on different occasions are not excluded, nor are the testing of one patient for the same condition by more than one laboratory. The mix of laboratories reporting to LabVISE is heavily biased toward the reference laboratories or laboratories of major hospitals, which may bias toward the reporting of rare infections. Finally, the decision to remove data on diagnostic method data from LabVISE reporting in 1996 was regrettable as the impact of new rapid screening technologies on infections reported can not be measured. The analysis of diagnostic methods used in LabVISE reported here are representative only of the period 1991 to 1996.

Results

Part A: General results

LabVISE reports 1991 to 2000

Between 1991 and 2000, LabVISE received 340,730 reports from between 13 and 26 laboratories. The numbers of reports and reporting laboratories are shown in Table 2. There has been a decline in both the number of reports and the number of contributing laboratories to LabVISE over the period 1991 to 2000 (Figure 1).

Figure 1. Total annual number of reports and number of laboratories reporting to LabVISE, 1991 to 2000

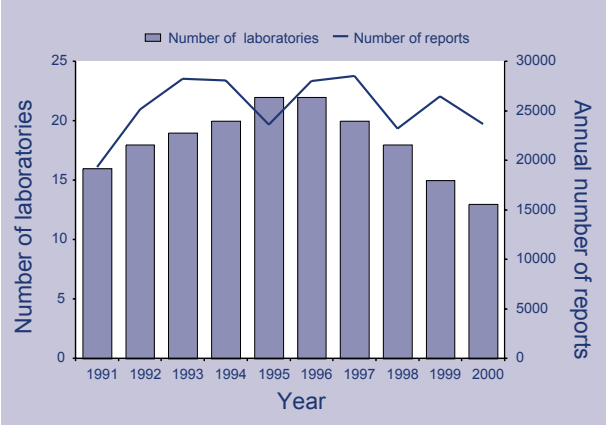


Table 2. Laboratory reports to LabVISE, 1991 to 2000, by laboratory

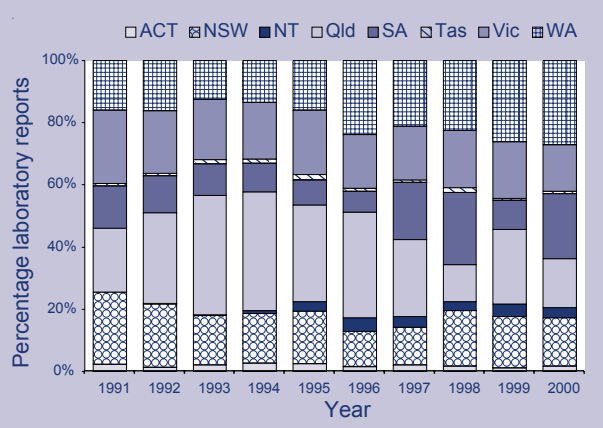
Jurisdiction	Laboratory name	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Australian Capital Territory	The Canberra Hospital	690	613	808	1,020	800	395	621	212	49	241
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	4,319	4,132	3,757	3,349	1,407	1,356	786	1,430	1,527	1,741
	Prince Henry/Prince of Wales Hospitals, Sydney	1,428	1,327	126	2,151	1,477	40	-	-	-	-
	New Children's Hospital, Westmead	892	845	779	764	677	499	975	1,101	1,253	924
	Royal Prince Alfred Hospital, Camperdown	-	-	-	136	371	288	259	483	573	542
	South West Area Pathology Service, Liverpool	-	792	1,268	1,695	1,713	1,203	997	1,206	1,001	346
	New England Pathology	126	151	160	-	-	-	-	-	-	-
	Royal North Shore Hospital	-	-	-	18	296	159	291	-	-	-
Northern Territory	Alice Springs Hospital	-	-	-	-	-	113	-	-	-	-
Queensland	Lynch Laboratory, Rockhampton	1,085	1,540	803	-	-	-	-	-	-	-
	Queensland Medical Laboratory, West End	-	3,750	8,751	10,012	10,793	7,953	5,219	2,944	7,669	4,403
	Townsville General Hospital	-	-	-	-	-	-	-	98	207	110
	State Health Laboratory, Brisbane	4,619	5,581	6,935	6,011	3,630	4,224	2,249	-	-	-
	Toowoomba Pathology Laboratory	129	17	-	-	-	-	-	-	-	-
	Nambour Hospital	-	-	4	99	52	-	-	-	-	-
South Australia	Institute of Medical and Veterinary Science, Adelaide	3,890	5,195	5,335	5,061	3,776	2,009	5,258	5,481	2,486	4,938

Table 2 (continued). Laboratory reports to LabVISE, 1991 to 2000, by laboratory

Jurisdiction	Laboratory name	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Tasmania	Northern Tasmanian Pathology Service, Launceston	-	76	222	161	184	95	110	156	105	128
	Royal Hobart Hospital, Hobart	231	273	306	472	596	293	109	234	24	-
	Hobart Pathology	19	-	-	-	-	-	-	-	-	-
Victoria	Monash Medical Centre, Melbourne	-	-	222	569	798	772	765	421	728	259
	Royal Children's Hospital, Melbourne	1,985	2,107	2,558	2,983	2,451	2,506	2,455	2,341	2,154	1,404
	Victorian Infectious Diseases Reference Laboratory, Fairfield	4,791	4,991	5,061	4,024	4,102	2,367	1,623	1,463	1,954	1,936
	Microbiological Diagnostic Unit, University of Melbourne	247	172	166	152	126	111	123	3	-	-
	Commonwealth Serum Laboratories, Melbourne	-	-	-	59	36	-	9	-	-	-
	Unipath Laboratories	-	-	-	-	436	251	-	-	-	-
Western Australia	PathCentre Virology, Perth	5,132	5,890	6,219	6,521	6,603	4,813	3,746	4,030	5,571	5,187
	Princess Margaret Hospital, Perth	990	1,386	1,266	1,785	1,252	1,687	1,387	885	1,314	1,398
	Western Diagnostic Pathology	-	-	-	-	900	2,951	1,691	1,086	173	107
	Royal Perth Hospital	-	-	-	-	-	185	85	-	-	-
Total*	**30,573 38,838 44,746 47,042 42,476 34,270**	**28,758**	**23,575**	**26,788**	**23,664**						

The number of organisms reported to LabVISE was reduced in 1995. This report will focus on the current list of organisms, most of which have been reported for the full study period (1991–2000). The number of reports for these organisms by year of collection and State or Territory of patient's residence is shown in Table 3. The proportion of reports from each Australian State and Territory has varied over the study period (Figure 2).

Figure 2. Laboratory reports to LabVISE, 1991 to 2000, by State or Territory



Completeness of data

Table 4 shows the completeness of data in four key (but not mandatory) fields common to LabVISE for the whole period from 1991 to 2000. While sex and diagnosis data were reported consistently over the years for the majority of LabVISE reports, in recent years important data such as age has been incomplete in more than half of all records. The completeness of data varied by laboratory, pathogen, and year.

Diagnosis

Data on diagnosis were available for 335,001 (98%) records from the study period. Of these 96,253 were coded as 'no clinical information available' and another 20,592 were coded as 'other diagnosis'. The 10 most common diagnoses (of 62 diagnostic categories accepted in LabVISE), which make up 201,959 or 90 per cent of the remaining 223,205 records are shown in Table 5.

Although samples submitted to LabVISE for investigations of genital (STI) disease make up the largest single category, the combined number of investigations of respiratory disease total 80,691 or 36 per cent of the total. It should be noted that the diagnostic descriptions are very broadly defined and the analysis that follows should be interpreted with caution.

The most frequently reported organisms in samples from patients with the 10 most common diagnoses are shown in Table 6.

Table 3. Laboratory reports to LabVISE, 1991 to 2000, by State or Territory

Year	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
1991	436	4,498	0	3,969	2,596	145	4,568	3,076	**19,288**
1992	345	5,101	65	7,309	2,984	233	5,025	4,074	**25,136**
1993	621	4,475	57	10,851	2,833	380	5,473	3,529	**28,219**
1994	766	4,484	259	10,731	2,583	351	5,137	3,757	**28,068**
1995	596	3,958	749	7,318	1,897	436	4,857	3,777	**23,588**
1996	444	3,141	1,256	9,508	1,843	294	4,820	6,671	**27,977**
1997	592	3,437	1,030	7,032	5,249	229	4,939	6,011	**28,519**
1998	419	4,110	670	2,758	5,415	366	4,270	5,208	**23,216**
1999	283	4,386	1,092	6,337	2,505	158	4,822	6,894	**26,477**
2000	397	3,688	776	3,729	4,964	179	3,527	6,402	**23,662**
Total	**4,899**	**41,278**	**5,954**	**69,542**	**32,869**	**2,771**	**47,438**	**49,399**	**254,150**

Table 4. Completeness of data in key fields in LabVISE, 1991 to 2000

Year	Sex* % complete	Age % complete	Postcode % complete	Diagnosis % complete
1991	97.6	77.0	2.9	100.0
1992	98.6	80.7	13.5	100.0
1993	98.9	83.1	51.6	99.9
1994	98.6	84.6	66.8	100.0
1995	98.9	91.5	74.8	99.9
1996	99.1	81.6	62.9	100.0
1997	99.1	80.9	60.9	99.9
1998	99.2	79.3	50.6	99.2
1999	99.3	54.7	74.9	94.2
2000	99.2	31.2	70.0	87.0

* Proportion of records identified as male or female, all the remainder were marked 'U' for unknown.

Table 5. The 10 most common diagnoses for which samples were submitted for testing to LabVISE laboratories, 1991 to 2000

Code	Diagnosis description	n	%
59	Genital disease (including sexually transmitted infections)	39,860	17.9
06	Superficial skin or mucous membrane diseases	36,677	16.4
02	Lower respiratory tract infection	36,320	16.3
11	Respiratory tract infection — unspecified	23,590	10.6
07	Gastrointestinal disease	23,177	10.4
01	Upper respiratory tract infection	20,781	9.3
17	Hepatitis	7,045	3.6
29	Bone/joint disease	5,541	2.5
G8	Malaise — general and/or mild fever	4,537	2.0
08	High fever	4,431	2.0

Table 6. The 10 most frequently isolated organisms in the 10 most frequently reported clinical diagnoses, LabVISE, 1991 to 2000

Rank	Genital diseases (n= 39,860) Virus/organism	Reports	Rank	Skin/mucous membrane disease (n=36,677) Virus/organism	Reports
1	*Chlamydia trachomatis*	18,208	1	Herpes simplex type 1†	12,029
2	Herpes simplex type 2†	14,257	2	Herpes simplex type 2†	8,596
3	Herpes simplex type 1†	5,539	3	Varicella–zoster virus	7,702
4	Herpes simplex (not typed)†	743	4	Rubella	1,768
5	*Treponema pallidum*	517	5	Herpes (not typed)	1,338
6	*Neisseria gonorrhoeae*†	178	6	Ross River virus	1,324
7	Cytomegalovirus	89	7	Measles virus	1,253
8	Varicella–zoster virus	67	8	Parvovirus	601
9	*Chlamydia trachomatis* A–K	35	9	Enterovirus (untyped)	322
10	*Chlamydia* spp.	35	10	Epstein-Barr virus	173
Rank	**Lower respiratory tract infection (n=36,320) Virus/organism**	**Reports**	**Rank**	**Respiratory tract infection — unspecified (n=23,590) Virus/organism**	**Reports**
1	Respiratory syncytial virus	16,349	1	Respiratory syncytial virus	9,202
2	*Mycoplasma pneumoniae*	3,750	2	Influenza A	2,259
3	Influenza A	3,367	3	Adenovirus (untyped)	1,629
4	Parainfluenza virus type 3	2,255	4	Parainfluenza virus type 3	1,455
5	Rhinovirus	1,753	5	*Mycoplasma pneumoniae*	1,441
6	Cytomegalovirus	1,699	6	Rhinovirus	1,303
7	Adenovirus (untyped)	1,163	7	Enterovirus (untyped)	1,013
8	Influenza B	1,093	8	Cytomegalovirus	880
9	*Bordetella pertussis*	690	9	Influenza B	615
10	Parainfluenza virus type 1	478	10	*Bordetella pertussis*	563
Rank	**Gastrointestinal disease (n=23,177) Virus/organism**	**Reports**	**Rank**	**Upper respiratory tract infection (n=20,781) Virus/organism**	**Reports**
1	Rotavirus	15,887	1	Respiratory syncytial virus	5,358
2	Adenovirus (untyped)	3,033	2	*Bordetella pertussis*	2,913
3	Enterovirus (untyped)	1,865	3	Rhinovirus	1,898
4	Human calcivirus*	683	4	Epstein-Barr virus	1,720
5	Adenovirus type 40	188	5	Parainfluenza virus type 3	1,336
6	Cytomegalovirus	161	6	Influenza A	1,225
7	Adenovirus type 2	111	7	Cytomegalovirus	988
8	Poliovirus (untyped)	87	8	Adenovirus (untyped)	919
9	Adenovirus type 1	68	9	Enterovirus (untyped)	618
10	Coronavirus	59	10	Parainfluenza virus type 1	587

Table 6 (continued). The 10 most frequently isolated organisms in the 10 most frequently reported clinical diagnoses, LabVISE, 1991 to 2000

Rank	Hepatitis (n=7,045) Virus/organism	Reports	Rank	Bone/joint disease (n=5,541) Virus/organism	Reports
1	Hepatitis C†	2,805	1	Ross River virus	4,296
2	Hepatitis A	1,890	2	Barmah Forest virus	308
3	Hepatitis B antibody†	1,832	3	Parvovirus	176
4	Epstein-Barr virus	124	4	Rubella	123
5	Cytomegalovirus	124	5	*Streptococcus* A	119
6	Hepatitis D	103	6	Epstein-Barr virus	72
7	*Coxiella burnetii*	29	7	Influenza A	55
8	Hepatitis B antigen†	23	8	Cytomegalovirus	55
9	*Chlamydia trachomatis*	19	9	*Coxiella burnetii*	49
10	Hepatitis E	17	10	Dengue type 2	40
Rank	**Malaise (n=4,537) Virus/organism**	**Reports**	**Rank**	**High fever (n=4,431) Virus/organism**	**Reports**
1	Epstein-Barr virus	710	1	Ross River virus	1,270
2	Cytomegalovirus	699	2	Epstein-Barr virus	458
3	Ross River virus	602	3	Cytomegalovirus	380
4	Coxiella burnetii	236	4	Influenza A	280
5	Influenza A	192	5	Respiratory syncytial virus	2
6	Hepatitis C†	152	6	*Coxiella burnetii*	185
7	*Mycoplasma pneumoniae*	145	7	Adenovirus (untyped)	138
8	Rubella	143	8	*Mycoplasma pneumoniae*	138
9	Influenza B	141	9	Barmah Forest virus	127
10	Barmah Forest virus	125	10	Enterovirus (untyped)	99

* Combines Norwalk like virus, small round virus and human calcivirus.

† Data collected up to 1996 only.

The ranking of organisms identified in different diagnoses over a 10-year period includes a number of pathogens no longer under surveillance through LabVISE. Among samples submitted with a primary diagnosis of genital disease, herpes simplex viral identifications combined comprise 20,539 (51% of the total), although data on herpesvirus were not included in LabVISE after 1996. Since the late 1970s the prevalence of herpes simplex type 2 (HSV–2) infections increased by 30 per cent in the United States of America (USA) and HSV–2 infects an estimated 86 million people worldwide.[6]

Among respiratory infections, respiratory synctial virus was most frequently identified in cases of all kinds of respiratory infection, while rotavirus was the predominant organism identified in cases of gastroenteritis. Cases of hepatitis tested in LabVISE were predominantly hepatitis C, although reporting of this pathogen to the system ceased in 1996.

Specimens processed and methods used

Although data fields for type of specimen and methods used for the diagnosis were no longer required in LabVISE after 1996, many laboratories continued to send these data fields. The 10 most common specimens received by LabVISE and the 10 most common methods used in LabVISE laboratories for the detection of antigens and antibodies are shown in Tables 7, 8 and 9.

Table 7. The 10 most common specimens received by LabVISE, 1991 to 2000

Rank	Specimen	n	%
1	Blood	80,019	25.0
2	Nasopharyngeal swab or aspirate	54,657	17.1
3	Other	42,937	13.4
4	Genital swab	42,782	13.4
5	Serum	34,039	10.6
6	Skin	25,462	8.0
7	Faeces/rectal swab	23,572	7.4
8	Urine	6,367	2.0
9	Eye	3,457	1.1
10	Cerebrospinal fluid	2,196	0.7

Table 8. The 10 most common methods used to detect microbial antigens in LabVISE laboratories, 1991 to 2000

Rank	Antigen description	n	%
1	Immunofluorescence	69,646	29.5
2	Enzyme-linked immunosorbent assay	38,221	16.2
3	Light microscopy	36,955	15.7
4	Immunoenzymatic techniques	33,138	14.0
5	Nucleic acid detection	18,354	7.8
6	Radio-immunoassay	17,547	7.4
7	Electronic microscopy	8,487	3.6
8	Other	3,862	1.6
9	Growth characteristics	2,935	1.2
10	Latex agglutination	2,899	1.2

Table 9. The 10 most common methods used to detect antibodies in LabVISE, 1991 to 2000

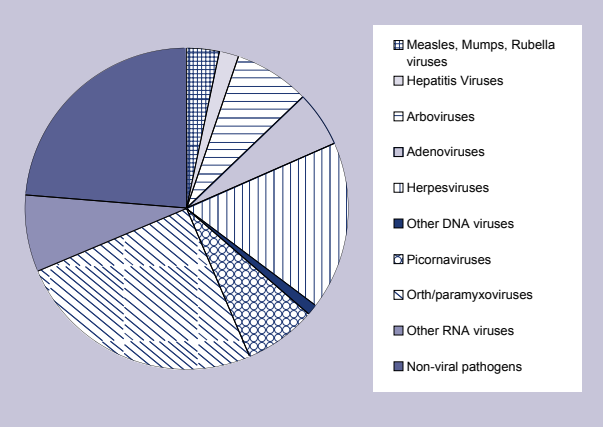
Rank	Antibody description	n	%
1	Enzyme-linked immunosorbent assay	71,611	42.3
2	Complement fixation	31,598	18.7
3	Immunofluorescence	26,001	15.3
4	Immunoenzymatic techniques	15,591	9.2
5	Other	9,165	5.4
6	Haemagglutination	3,230	1.9
7	Haemagglutination inhibition	3,169	1.9
8	Slide/tube agglutination	2,473	1.5
9	Radioimmunoassay	2,308	1.4
10	Latex agglutination	2,156	1.3

Pathogens under surveillance in LabVISE, 1991 to 2000

A complete list of pathogens under surveillance for this report is shown in Appendix 3 and is summarised in Table 10.

The totals for each year for each pathogen group are shown in Table 11. Figure 3 shows the relative proportions of each pathogen group in the total dataset.

Figure 3. Laboratory reports to LabVISE, 1991 to 2000, by pathogen group



In the study period, reports of non-viral pathogens increased from 17.9 per cent to 27 per cent of the annual reports to LabVISE. Among viral pathogens, ortho/paramyxoviruses made up on average 25 per cent of the annual reports, herpesviruses 17 per cent and other RNA viruses 8 per cent.

Part B: Analysis of data by pathogen

Measles, mumps and rubella viruses

LabVISE reports of measles, mumps and rubella during the study period have declined since 1994, when the last epidemic of measles occurred in Australia. As part of Australia's measles elimination strategy, the Measles Control Campaign in 1998 vaccinated 1.7 million Australian children regardless of their vaccination status, with the measles-mumps-rubella vaccine (MMR) . As a result of this campaign and continuing high vaccination coverage, measles activity in Australia is at an historic low.[7]

Measles, mumps and rubella are notifiable diseases that have been collected since 1991 from all Australian states and territories in the National Notifiable Diseases Surveillance System. A comparison of notifications for these diseases recorded in the NNDSS with LabVISE laboratory reports is shown in Table 12.

Table 10. Summary of pathogens under surveillance in LabVISE, 1991 to 2000

Pathogen group	Specific pathogens
Measles, mumps, rubella	Measles, mumps and rubella viruses
Hepatitis viruses	Hepatitis A, D and E
Arboviruses	Ross River virus, Barmah Forest virus, dengue (type 1 to 4), Murray Valley encephalitis virus, Kunjin virus, Japanese encephalitis virus, Kokabera virus, Stratford virus and flavivirus (unspecified)
Adenovirus	Types 1–17, 19, 21–22, 24, 26–32, 34, 35, 37, 40–47
Herpesviruses	Herpesvirus type 6, cytomegalovirus, varicella-zoster virus, Epstein-Barr virus
Other DNA viruses	Parvovirus, papovavirus, molluscum contagiosum, orf virus, poxvirus
Picornavirus	Coxsackievirus, echovirus, poliovirus, rhinovirus, enterovirus
Ortho/paramyxovirus	Influenza, parainfluenza, respiratory synctial virus
Other RNA virus	Rotavirus, astrovirus, reovirus (unspecified), calcivirus/Norwalk agent, coronavirus, small virus-like particles
Other non-viral pathogens	*Chlamydia, Mycoplasma, Rickettsia, Streptococcus, Yersinia, Brucella, Bordetella, Legionella, Leptospira, Cryptococcus, Treponema, Entamoeba, Toxoplasma, Echinococcus*

Table 11. Total number of reports to LabVISE, 1991 to 2000, by pathogen group and year and percentage of each year's total

Pathogen group		1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	Total
Measles, mumps, rubella	n	534	1,005	1,856	2,464	957	810	469	196	375	144	**8,810**
	%	*2.7*	*4.0*	*6.6*	*8.8*	*4.1*	*2.9*	*1.6*	*0.8*	*1.4*	*0.6*	***3***
Hepatitis viruses	n	482	417	510	393	455	426	643	392	384	159	**4,261**
	%	*2.5*	*1.6*	*1.8*	*1.4*	*1.9*	*1.5*	*2.3*	*1.7*	*1.5*	*0.6*	***2***
Arboviruses	n	955	2,013	2,744	2,577	1,271	3,570	2,311	872	1,726	1,682	**19,721**
	%	*5.0*	*8.0*	*9.7*	*9.2*	*5.4*	*12.8*	*8.1*	*3.7*	*6.5*	*7.1*	***8***
Adenovirus	n	1,518	1,722	1,885	1,542	1,182	1,382	1,017	1,162	1,309	1,205	**13,924**
	%	*7.9*	*6.9*	*6.7*	*5.5*	*5.0*	*4.9*	*3.6*	*5.0*	*5.0*	*5.1*	***5***
Herpesviruses	n	3,706	4,039	4,059	4,311	4,367	4,589	4,426	3,923	5,090	4,738	**43,248**
	%	*19.2*	*16.1*	*14.4*	*15.4*	*18.5*	*16.4*	*15.5*	*16.9*	*19.2*	*20.0*	***17***
Other DNA viruses	n	67	206	109	121	121	282	310	272	474	414	**2,376**
	%	*0.3*	*0.8*	*0.4*	*0.4*	*0.5*	*1.0*	*1.1*	*1.2*	*1.8*	*1.7*	***1***
Picornavirus	n	1,902	2,341	2,630	2,678	1,885	1,559	1,119	1,190	1,631	1,527	**18,462**
	%	*9.9*	*9.3*	*9.3*	*9.5*	*8.0*	*5.6*	*3.9*	*5.1*	*6.2*	*6.4*	***7***
Ortho/paramyxovirus	n	3,833	5,981	5,432	6,244	6,129	6,992	8,782	8,261	6,238	5,604	**63,496**
	%	*19.9*	*23.8*	*19.2*	*22.2*	*26.0*	*25.0*	*30.8*	*35.5*	*23.6*	*23.6*	***25***
Other RNA virus	n	2,832	2,277	2,090	2,332	1,693	1,582	1,522	1,444	2,322	1,864	**19,958**
	%	*14.7*	*9.1*	*7.4*	*8.3*	*7.2*	*5.7*	*5.3*	*6.2*	*8.8*	*7.9*	***8***
Other non-viral pathogens	n	3,458	5,135	6,904	5,406	5,528	6,798	7,919	5,547	6,928	6,374	***59,997***
	%	*17.9*	*20.4*	*19.3*	*24.5*	*23.4*	*24.2*	*27.8*	*23.1*	*26.2*	*27.0*	***24***

Table 12. Laboratory reports to LabVISE and notifications to NNDSS of measles, mumps and rubella, 1991 to 2000

Virus	Surveillance system	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Measles	LabVISE	256	204	853	1,199	153	57	67	49	172	44
	NNDSS	1,380	1,425	4,536	4,825	1,194	481	858	290	230	107
Mumps	LabVISE	32	48	77	87	69	37	40	44	58	49
	NNDSS	NN	23	28	90	157	126	191	182	184	212
Rubella	LabVISE	246	753	926	1,178	735	716	362	103	145	51
	NNDSS	620	3,810	3,812	3,374	4,590	2,556	1,389	745	376	321

NN Not notifiable

Measles reports in LabVISE included both viral isolations and seroconversions, whereas the NNDSS included cases of measles diagnosed on the basis of clinical findings or epidemiological links to another case. NNDSS notifications were 4–8 times higher than LabVISE reports. Laboratory confirmation of measles infection becomes increasingly important as Australia plans the elimination of the disease over the next few years. LabVISE laboratories could provide important information to supplement that of the NNDSS if a representative sample of all diagnostic laboratories was included. Despite these limitations, laboratories can provide important information on circulating measles virus genotypes in Australia. Genotyping data suggests that in some jurisdictions there are now no endemic measles strains circulating and that small outbreaks are frequently linked to imported cases (Lambert, Communicable Disease Conference 2001, abstract 60).

Surveillance of mumps in LabVISE preceded that in the NNDSS. Mumps notifications to the NNDSS began in 1992 and for some years, mumps was not reported from all jurisdictions. Thus from 1991 to 1993, LabVISE recorded more cases of mumps than the NNDSS. Notifications to the NNDSS include clinically diagnosed cases without laboratory confirmation, although some jurisdictions such as New South Wales require laboratory confirmation. Mumps is a rare disease in Australia. Although vaccination of Australian children with the MMR vaccine is expected to reduce mumps incidence in Australia, the impact has been less evident on the rate of mumps compared with the rate of measles (Gidding, Communicable Disease Conference, 2001, abstract 57). In a similar manner to measles, the laboratory diagnosis of mumps increases in importance, as the disease becomes rarer.

The incidence of rubella in Australia has been dramatically reduced as a result of widespread vaccination with MMR. Both NNDSS and LabVISE data show the impact of increased vaccination in Australia during the 1990s. Up to 2000, notifications of rubella to the NNDSS were clinically defined and did not require laboratory confirmation.

Hepatitis viruses

Up to 1996, hepatitis B and C were the predominant reports of hepatitis in LabVISE (Table 5). These reports were excluded from LabVISE from 1997 and were not analysed in this report. Hepatitis A and D were reported for the whole study period and hepatitis E has been reported since 1992. LabVISE reporting of hepatitis D and E predated reporting through the NNDSS by some years (Table 13) and reports from all states and territories to the NNDSS was not achieved for hepatitis D and hepatitis E until 2000.

Since the peak of notifications to NNDSS in 1997, the number of cases of hepatitis A has fallen significantly as a result of vaccination of high-risk groups. The trends in the NNDSS data have been reflected in the trends in LabVISE data. Hepatitis notifications to NNDSS include clinical cases of hepatitis and cases epidemiologically linked to a serologically confirmed case, and thus are expected to outnumber the laboratory-confirmed cases reported by LabVISE.

Since hepatitis D is diagnosed only by laboratory methods, LabVISE data should be close to that in NNDSS, subject to the limited number of laboratories contributing to LabVISE. In some jurisdictions (New South Wales and the Northern Territory), cases of hepatitis E (HEV) may be clinically defined as a hepatitis-like illness in the absence of other causes of hepatitis with a history of travel to HEV-endemic areas.

Table 13. Laboratory reports to LabVISE and notifications to NNDSS of hepatitis A, D and E, 1991 to 2000

Virus	Surveillance system	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Hepatitis A virus	LabVISE	445	371	451	363	424	407	624	384	375	146
	NNDSS	2,195	2,109	2,006	1,919	1,645	2,112	3,069	2,443	1,557	807
Hepatitis D virus	LabVISE	37	45	47	24	23	17	15	7	8	9
	NNDSS	NN	NN	NN	5	37	14	17	10	21	27
Hepatitis E virus	LabVISE	-	1	12	6	8	2	4	1	1	4
	NNDSS	NN	NN	NN	1	5	4	7	1	2	10

NN Not notifiable

Arboviruses

The numbers of arthropod-borne ('arboviruses') diseases reported in LabVISE between 1991 and 2000 are shown in Table 14.

Of the 8 arboviruses that were reported to LabVISE, three (Ross River virus, Barmah Forest virus and dengue) were also notifiable diseases reported to the NNDSS during the same period. Another 3 viruses (Murray Valley encephalitis virus, Kunjin virus and Japanese encephalitis virus) had been notifiable to NNDSS under the collective group Australian encephalitis but from 2001 became notifiable separately. The NNDSS also recorded 'Arbovirus unspecified' as a disease category in the period 1991 to 2000, which would have captured data on all other viruses listed here.

A month by month comparison of notifications of Ross River virus (RRV) to NNDSS and laboratory reports of RRV to LabVISE is shown in Figure 4. LabVISE reports show a seasonal variation matching that seen in the NNDSS notifications, with annual peaks in the first and second quarters (i.e. the summer months) of the year. RRV infection is diagnosed by virological or serological methods. The smaller numbers reported to LabVISE in the same time period, reflects the small number of laboratories contributing to LabVISE. The number of LabVISE reports were similar to the number of NNDSS notifications out of epidemic seasons (Figure 4).

Table 14. Laboratory reports to LabVISE of arboviruses, 1991 to 2000

Virus	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	Total
Ross River virus	833	1,319	1,895	2,240	988	3,249	2,016	676	1,423	1,268	**15,907**
Barmah Forest virus	36	251	208	273	202	232	201	44	180	169	**1,796**
Dengue total	29	385	528	35	25	62	64	70	88	181	**1,467**
Flavivirus (unspecified)	29	47	104	23	45	21	21	73	27	40	**430**
Kunjin virus	14	10	-	2	5	5	6	5	5	4	**57**
Murray Valley encephalitis virus	10	1	9	3	-	-	3	2	2	20	**50**
Japanese encephalitis virus	-	-	-	1	6	-	-	1	1	-	**9**
Stratford virus	3	-	-	-	-	1	-	1	-	-	**5**
Kokobera virus	1	-	-	-	-	-	-	-	-	-	**1**

Figure 4. Laboratory reports to LabVISE and notifications to NNDSS of Ross River virus infection, 1991 to 2000, by month of specimen collection

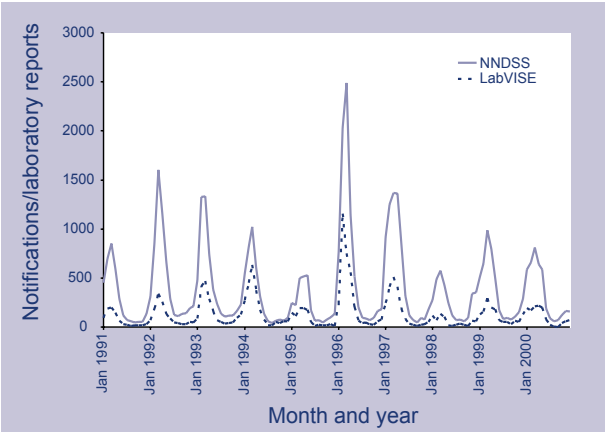
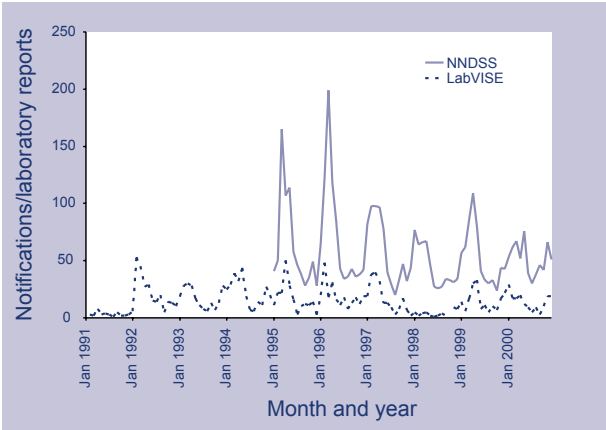


Figure 5. Laboratory reports to LabVISE and notifications to NNDSS of Barmah Forest virus infection, 1991 to 2000, by month of specimen collection



Barmah Forest virus laboratory reports have been recorded in LabVISE since 1991, 4 years before notifications were included in the NNDSS. A comparison of LabVISE reports and NNDSS notifications by month between 1991 and 2000 is shown in Figure 5. As for RRV, LabVISE laboratory reports and NNDSS notifications show an annual peak in the summer months. LabVISE reports were a larger proportion of NNDSS notifications out of epidemic seasons (Figure 5).

LabVISE reports of dengue virus include some data on the serotypes of dengue virus isolated (Table 5). However, the proportion of dengue virus among each year's reports that were serotyped has declined, particularly in recent years (Table 5) and there is no way to tell from LabVISE data whether the dengue infection was acquired overseas. The movement of new serotypes of dengue virus into Australia has important implications. The frequency of dengue haemorrhagic fever, a serious complication of dengue virus infection, increases when previously infected populations are exposed to different serotypes of the dengue virus. Since dengue is a major public health problem in areas to the north of Australia, and is occasionally a significant problem in Far North Queensland, surveillance of dengue and circulating dengue viral serotypes is essential.

Table 15. Laboratory reports to LabVISE of arboviruses, 1991 to 2000

Virus	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Dengue type 1	13	9	1	-	3	-	-	-	-	-
Dengue type 2	3	297	422	4	1	29	20	-	-	2
Dengue type 3	-	5	2	4	2	2	1	27	3	4
Dengue type 4	1	-	-	1	-	1	-	-	-	-
Dengue not typed	12	74	103	26	19	30	43	43	85	175
Dengue total	**29**	**385**	**528**	**35**	**25**	**62**	**64**	**70**	**88**	**181**

LabVISE reports of Murray Valley encephalitis virus infections are an important but partial record of this significant pathogen. Murray Valley encephalitis became a separately nationally notifiable disease from January 2001 along with Kunjin. Other arboviruses are also reported to the NNDSS as Arbovirus (not elsewhere classified).

Adenoviruses

Adenoviruses are DNA viruses which are clinically important because of their ability to cause acute respiratory infections and infections of the conjunctiva in humans. There are more than 47 serotypes of human adenoviruses. While human adenoviruses are ubiquitous with primary infection in the first year of life, there are geographical variations in the distributions of serotypes and associations of serotypes with different age groups. Broadly speaking, serotypes 1, 2, 5 and 6 are found in tonsils of young children, serotypes 3, 4 and 7 are found in young adults with upper respiratory tract infections, serotypes 8 and 19 are associated with adult eye infections and serotypes 11 and 21 are found in children with urinary tract infections.[8] The adenovirus serotypes associated with clinical syndromes in different age groups are shown in Table 16.

LabVISE laboratory reports of adenoviruses by year and serotype are shown in Table 17. Of the 13,924 reports, 10,826 were not further typed. Of the 2,468 serotypes identified, the most frequent serotypes identified in the period 1991 to 2000 were serotype 3 (687 reports, 28% of total), serotype 2 (591, 24%) and serotype 1 (513, 21%). In general, the proportion of untyped adenovirus reports increased from 63 per cent in 1991 to 86 per cent in 2000. The proportion of untyped adenovirus may reflect laboratory practices of batching samples for serotyping and the inability for LabVISE records to be updated with later serotyping information.

The age and sex distribution of adenovirus reports for the period 1991 to 2000 is shown in Figure 6. The male to female ratio was 1.3:1 and 58 per cent of the reports were from children aged less than 5 years.

Figure 6. Laboratory reports to LabVISE of adenovirus infection, 1991 to 2000, by age and sex

Table 16. Adenovirus serotypes associated with clinical syndromes in different age groups*

Group affected	Syndromes	Adenovirus serotypes
Neonates	Fatal disseminated infection	3,7,21,30
Infants	Coryza, pharyngitis (most asymptomatic)	1,2,5
Children	Upper respiratory disease	1,2,4–6
	Pharyngoconjunctival fever	3,7
	Haemorrhagic cystitis	11,21
	Diarrhoea	2,3,5,40,41
	Intussuception	1,2,4,5
	Meningoencephalitis	2,6,7,12
Young adults	Acute respiratory disease and pneumonia	3,4,7
Adults	Epidemic keritoconjunctivitis	8,19,37
Immunocompromised	Pneumonia with dissemination, urinary tract infection	5,31,34,35,39,42–47
	CNS disease including encephalitis	7,12,32

*Adapted from reference 8

Table 17. Laboratory reports to LabVISE of adenovirus, 1991 to 2000, by year of report and serotype

Viruses	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	Total
Adenovirus type 3	88	96	203	57	66	45	22	57	35	18	**687**
Adenovirus type 2	142	129	128	45	37	29	39	22	13	7	**591**
Adenovirus type 1	91	111	85	48	32	21	29	74	14	8	**513**
Adenovirus type 40	4	6	9	-	-	34	12	21	74	86	**246**
Adenovirus type 8	38	33	55	55	22	13	3	3	1	3	**226**
Adenovirus type 4	23	103	40	2	2	-	7	4	15	5	**201**
Adenovirus type 5	31	38	28	12	14	9	8	1	6	8	**155**
Adenovirus type 7	8	4	11	16	26	17	8	17	7	8	**122**
Adenovirus type 11	29	12	4	1	3	1	-	1	-	-	**51**
Adenovirus type 37	7	3	1	2	2	5	3	5	11	11	**50**
Adenovirus type 19	6	20	3	-	3	7	-	2	1	7	**49**
Adenovirus type 6	9	7	3	1	2	3	-	20	-	3	**48**
Adenovirus type 9	11	7	5	3	2	1	-	-	-	-	**29**
Adenovirus type 26	11	-	2	2	2	1	-	-	-	-	**18**
Adenovirus type 28	12	1	1	-	-	1	-	-	-	-	**15**
Adenovirus type 30	7	2	-	1	2	-	-	-	-	-	**12**
Adenovirus type 35	4	1	1	1	1	3	-	-	-	-	**11**
Adenovirus type 10	4	2	1	-	1	-	1	1	-	-	**10**
Adenovirus type 22	3	1	1	3	-	-	-	2	-	-	**10**
Adenovirus type 46	1	3	1	2	2	-	-	-	-	-	**9**
Adenovirus type 41	-	-	-	-	-	4	3	-	-	1	**8**
Adenovirus type 12	-	3	3	-	-	-	-	-	-	-	**6**
Adenovirus type 29	5	-	-	-	-	-	-	-	-	-	**5**
Adenovirus type 24	3	-	-	-	-	-	-	-	-	-	**3**
Adenovirus type 31	3	-	-	-	-	-	-	-	-	-	**3**
Adenovirus type 47	1	2	-	-	-	-	-	-	-	-	**3**
Adenovirus type 16	2	-	-	-	-	-	-	-	-	-	**2**
Adenovirus type 34	-	2	-	-	-	-	-	-	-	-	**2**
Adenovirus type 42	-	-	-	-	1	1	-	-	-	-	**2**
Adenovirus type 13	1	-	-	-	-	-	-	-	-	-	**1**
Adenovirus type 14	1	-	-	-	-	-	-	-	-	-	**1**
Adenovirus type 15	-	-	-	-	-	-	-	-	-	1	**1**
Adenovirus type 18	1	-	-	-	-	-	-	-	-	-	**1**
Adenovirus type 21	1	-	-	-	-	-	-	-	-	-	**1**
Adenovirus type 27	1	-	-	-	-	-	-	-	-	-	**1**
Adenovirus type 32	1	-	-	-	-	-	-	-	-	-	**1**
Adenovirus type 43	-	-	-	-	-	1	-	-	-	-	**1**
Adenovirus type 44	1	-	-	-	-	-	-	-	-	-	**1**
Adenovirus type 45	2	-	-	-	-	-	-	-	-	-	**2**
Adenovirus not typed/pending	966	1,136	1,300	1,291	962	1,186	882	932	1,132	1,039	**10,826**
Total	**1,518**	**1,722**	**1,885**	**1,542**	**1,182**	**1,382**	**1,017**	**1,162**	**1,309**	**1,205**	**13,924**

LabVISE reports of adenoviruses in which diagnosis details were available were analysed. The majority of adenovirus reports came from patients with respiratory, gastrointestinal or eye disease (45% respiratory, 33% gastrointestinal and 11% eye disease).

It is estimated that adenoviruses account for between 2–4 per cent of acute respiratory infections, which cause 4.5 million deaths annually in children, mostly in the developing world.[9] Adenoviruses also cause diarrhoea in children in developed countries. A prospective study in Canada has estimated that adenoviruses are responsible for around 4 per cent of community-acquired paediatric diarrhoea.[10] Adenoviruses were identified in between 3.4 and 4.9 per cent of stools from children hospitalised with acute gastroenteritis in Melbourne between 1995 and 1998.[11]

Adenovirus types vary in their geographic distribution and over time. Adenovirus type 41 infections increased in the Netherlands from 30 per cent to 95 per cent of all adenovirus infections between 1981 and 1986.[12] Adenovirus type 7 has been recorded as causing community and hospital outbreaks as well as sporadic cases in Australia.[13] Seven genome types of adenovirus 7 have been identified and a shift from Ad7c to Ad7c genome types was observed to occur in the late 1960s in Europe and in the mid-1970s in Australia.[14]

Adenovirus infections are significant in the immunocompromised. Disseminated adenovirus disease (DAD) in neonates has been reported in recent years. A review of 11 DAD cases in Texas (6 of whom were immunocompromised and 5 who were immunocompetent) showed a high mortality rate (83%). Mortality was reduced by treatment with antiviral agents and immunoglobulin.[15]

In HIV-positive patients, adenovirus infection risk was estimated at 28 per cent per year and increased with declining CD4+ T-cell counts. Infection was most commonly gastrointestinal or urinary and prolonged viral shedding in severely immunocompromised has been noted.[16]

Respiratory infections with cytomegalovirus (CMV) and community respiratory viruses including adenoviruses are important causes of infection and morbidity and mortality among lung transplant recipients.[17]

Herpesviruses

Viruses from the Herpesviridae family of DNA viruses, under surveillance through LabVISE, are herpesvirus type 6, cytomegalovirus (human herpesvirus 5), varicella-zoster virus (human herpesvirus 3) and Epstein-Barr virus (human herpesvirus 4). Total reports for these viruses to LabVISE between 1991 and 2000 are shown in Table 18. The major clinical syndromes associated with herpesviruses are summarised in Table 19.

Table 18. Laboratory reports to LabVISE of herpesviruses, 1991 to 2000

Virus	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	Total
Cytomegalovirus	1,820	1,728	1,561	1,727	1,404	1,373	1,017	766	1,220	1,312	**13,928**
Varicella-zoster virus	522	684	924	1,062	1,073	1,132	1,252	1,252	1,658	1,494	**11,053**
Epstein-Barr virus	1,361	1,625	1,570	1,516	1,887	2,084	2,151	1,903	2,196	1,926	**18,219**
Herpesvirus type 6	3	2	4	6	3	-	6	2	16	6	**48**

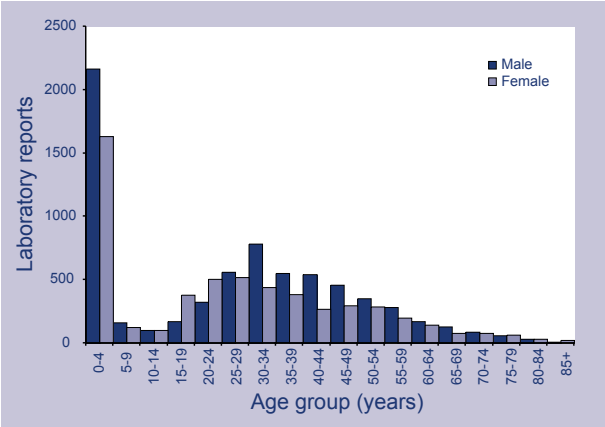
Table 19. Major clinical syndromes associated with herpesviruses under surveillance in LabVISE, 1991 to 2000

Virus	Clinical syndrome(s)
Cytomegalovirus	Cytomegalic inclusion disease, CMV mononucleosis, significant infection in AIDS patients
Varicella-zoster virus	Chickenpox, shingles
Epstein-Barr virus	Infectious mononucleosis
Herpesvirus type 6	Roseola infantum, fever, otitis media, encephalitis

Cytomegalovirus

Cytomegalovirus laboratory reports were reported more frequently from males than females (male to female ratio 1.3:1). The largest numbers of notifications were found in children aged less than 5 years (27% of total reports, Figure 7).

Figure 7. Laboratory reports to LabVISE of cytomegalovirus infection, 1991 to 2000, by age and sex



Cytomegalovirus was identified in patients presenting with a wide range of diagnoses during the study period. Respiratory infections accounted for 48 per cent of diagnoses in which CMV were found. CMV was also isolated from blood (32%), nasopharyngeal swabs (25%), urine (14%) or diagnosed from serum (12%).

CMV infection in humans is common and life-long although disease is rare. Secondary activation of latent infections is common although disease caused by primary infection is more serious. There are two periods of increased transmission — during the perinatal period and in the reproductive years. Transmission may occur during birth, during breast-feeding and between infants in nurseries. Sexual transmission is also common. Congenital CMV infections occur in 0.5–2.2 per cent of all live births, mainly in primiparous mothers who were infected for the first time with CMV during pregnancy.[18] Demographic and occupational factors also influence the risk of giving birth to an infant with congenital CMV infection.[19] Symptoms occur in less than a quarter of infected children; however, those that are infected demonstrate cytomegalic inclusion disease, characterised by jaundice and multiple organ involvement. Congenital CMV infections are the leading cause of congenital malformations in the developed world. Clinical trials of treatment regimens for congenital CMV infections are under way.[20]

There have been some profound changes in the epidemiology of CMV infections in children as a result of changes in breast-feeding and child rearing practices in Western countries over the past 15 years. In the USA, this has changed the prevalence of CMV among mothers and children in different socioeconomic classes. In middle and upper class households, which utilise child-care facilities, the exposure of mothers to CMV infection via their children will increase. In lower socioeconomic classes the relative decline in breast-feeding and the lower use of child-care facilities may increase the proportion of uninfected mothers.[21]

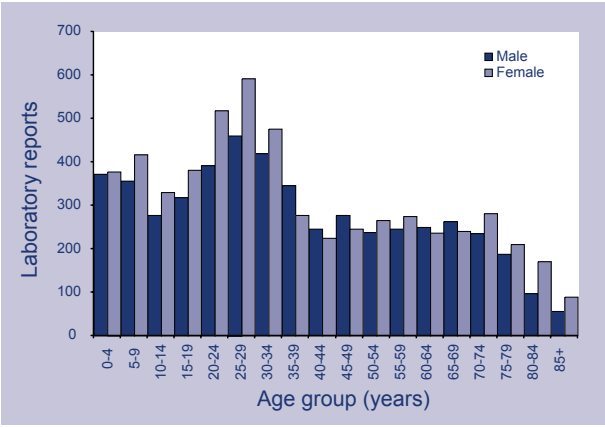
CMV infection by blood transfusion is common causing approximately 2.4 seroconversions per 100 units transfused.[18] Organ transplant recipients are also susceptible to infection with CMV.

CMV infections are significant in HIV/AIDS particularly as a cause of retinitis. In advanced AIDS, CMV infection may cause mononeuropathy multiplex.[22] Karposi's sarcoma in HIV positive patients was initially associated with CMV infection.[23] However, more recent work has identified a herpesvirus (human herpesvirus 8, Karposi's sarcoma associated herpesvirus, KSHV) as the etiologic agent of Karposi's sarcoma.[24]

Varicella-zoster virus

Reports of varicella-zoster virus (VZV) to LabVISE increased over the study period and comprised 4 per cent of the total reports. In 2000, LabVISE reports of VZV totalled 1,494. There were slightly more reports from females than males (male to female ratio 0.9:1). Laboratory reports were from all age groups with the largest numbers of notifications found in the 25–29 year age group (9% of total, Figure 8).

Figure 8. Laboratory reports to LabVISE of varicella zoster-virus infection, 1991 to 2000, by age and sex



The great majority of diagnoses associated with identification of VZV were skin/mucous membrane disease (89.5%) and the virus was most commonly isolated from skin (69%) or blood (19%).

VZV causes an acute generalised viral disease in children commonly termed 'chickenpox', while reactivation of the virus in adults and the elderly causes shingles. More than 90 per cent of people are infected with VZV by adolescence. While most VZV infections cause mild disease in children, disease severity is greater in adults and case fatality rates can be 20 times higher (in the 5–9 year age group 1 death per 100,000 population compared with adults 1 death per 5,000 population).[24]

While immunity is long lived, reactivation of the latent varicella infection is common in the elderly and up to 30 per cent of patients with shingles may suffer a post-shingles neuralgia.[24]

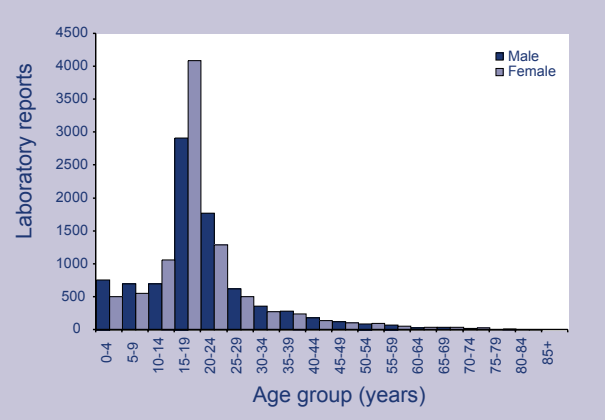
A varicella vaccine has been available in the USA since 1995. This vaccine has an efficacy among children of between 70 and 90 per cent. In 2002, the Australian Technical Advisory Group on Immunisation (ATAGI) is considering this vaccine for inclusion in the childhood immunisation schedule.

Epstein-Barr virus

Reports of Epstein-Barr virus (EBV) infection to LabVISE averaged 1,800 reports per year over the study period, about 7 per cent of total LabVISE reports between 1991 and 2000.

There were slightly more reports in females than males (male to female ratio 0.9:1). The largest numbers of notifications were in the 15 to 19 year age group (39% of the total, Figure 9).

Figure 9. Laboratory notifications of Epstein-Barr virus infection, 1991 to 2000, by age and sex



Over the study period, EBV was most often identified in patients presenting with reticuloendothelial disease (35%), and was most commonly isolated from blood (67%).

EBV causes an acute viral syndrome with fever, sore throat and lymphadenopathy accompanied by characteristic increases in the percentages of monocytes and lymphocytes (mononucleosis and lymphocytosis). The virus infects and transforms human B cells, although only 50 per cent of people infected will develop clinical infectious mononucleosis. While EBV infection is distributed worldwide, the peak of infection occurs in different age groups. Infection is more common among children in developing countries and more common among adolescents in developed countries. EBV is associated with the pathogenesis of Burkitt's lymphoma and nasopharyngeal carcinomas. Infection with EBV is possibly associated with Hodgkin's disease and non-Hodgkin's lymphomas particularly in HIV positive patients. Reactivation of latent EBV infection in HIV positive patients may cause interstitial pneumonia in infants and hairy leucoplakia and B-cell tumours in adults.[24]

Herpesvirus type 6

Only 48 reports of herpesvirus type 6 (HHV–6) were made to LabVISE during the study period. HHV–6 is the cause of 'sixth disease', Roseola infantum, an acute febrile rash occurring in children aged under 4 years. HHV–6 can be an opportunistic infection in transplant recipients. A meta-analysis of studies between 1986 and 1996[25] concluded that between 38 and 60 per cent of bone marrow transplant recipients and 31–55 per cent of solid organ transplant recipients were infected with HHV–6 two to four weeks after transplantation. Bone marrow suppression, interstitial pneumonia and encephalitis were the most commonly reported clinical diseases associated with HHV–6 infection.

Other DNA viruses

A number of other DNA viruses, reported to LabVISE in small numbers during the period 1991 to 2000, are shown in Table 20.

Table 20. Laboratory reports to LabVISE of other DNA viruses, 1991 to 2000

Virus	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	Total
Parvovirus	29	178	86	109	102	268	291	261	437	389	**2,150**
Papovavirus group	9	11	1	4	11	1	2	3	12	7	**61**
Molluscipox virus	21	10	8	4	3	7	5	2	15	11	**86**
Orf virus	5	7	4	2	1	1	9	6	8	7	**50**
Poxvirus group (not typed)	3	-	10	2	4	5	3	-	2	-	**28**

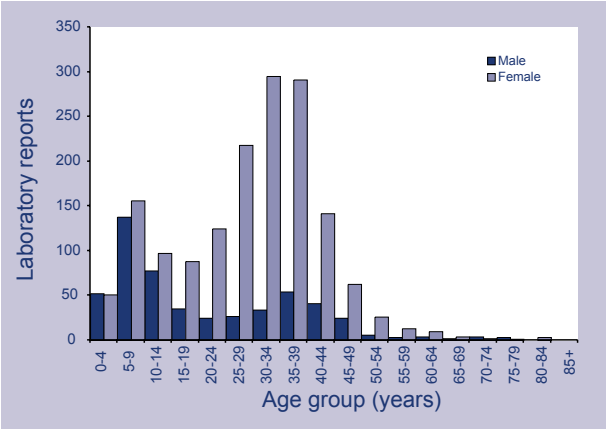
Among the parvoviruses, only parvovirus B19 is a known human pathogen.[26] Parvovirus causes prolonged epidemics of erythema infectiosum ('Fifth disease') among primary school aged children.[27] Fifth disease is a mild non-febrile illness characterised by a biphasic rash ('slapped cheek syndrome').[24] In Australia approximately 40 per cent of women are susceptible and around half of these are exposed to infection at home, typically via their school-aged children. Infection with parvovirus B19 in first half of pregnancy is associated with a 10 per cent excess foetal loss, anaemia and hydrops fetalis in 3 per cent. The overall risks of adverse events after occupational exposure to parvovirus B19 during pregnancy is low (excess foetal loss 2–6 per 1,000 pregnancies and foetal death from hydrops in 2–5/10,000 pregnancies.[27] It is recommended that susceptible pregnant women should be excluded from working with children during epidemics of parvovirus, which occur every 2 years and last for up to 2 years.[27]

Parvovirus infections may cause severe chronic anaemia in the immunosuppressed.[24]

Among reports of parvovirus to LabVISE, parvovirus was more commonly identified in women of childbearing age, possibly because of antenatal screening (Figure 10). The male to female ratio was 0.3:1, and 53 per cent of reports were in women aged between 15 and 45 years.

The papovavirus group includes human papillomaviruses and polyomaviruses. Human papillomaviruses (HPV) are the causative organism of human warts. At least three types of cutaneous HPV are recognised — cutaneous, plantar and anogenital. Diagnoses of the latter have been increasing since the 1980s.[28] Polyomaviruses JC viruses cause a rare demylinating disease in the immunocompromised (progressive multifocal leukoencephalopathy). Polyomaviruses JC and BK virus infections are common in childhood and the virus persists in the kidney.

Figure 10. Laboratory reports to LabVISE of parvovirus infections, 1991 to 2000, by age and sex



The poxvirus, molluscipoxvirus is the causative organism of the skin disease, molluscum contagiosum. Molluscum contagiosum skin papules occur on the abdomen, pubis, genitalia and inner thighs and persist without treatment for between 6 months and 2 years. The virus has not been isolated and serology is poorly defined. Transmission is by direct contact, including sexual, fomites and autoinoculation. Lesions may disseminate in HIV-infected persons.[29]

The orf virus is another poxvirus and the cause of contagious pustular dermatitis, a proliferative cutaneous disease. The virus is transmitted to humans by contact with infected sheep and goats. The disease is worldwide in distribution, especially among farm workers, and has been reported as an important occupational disease in New Zealand.[24]

Picornaviruses

The family *Picornaviridae* consists of two groups: the rhinoviruses and enteroviruses. Enteroviruses consist of five subgroups and these comprise a total of 67 serotypes: 31 echoviruses, 23 coxsackie A viruses, 6 coxsackie B viruses, 3 polioviruses and 4 'new' enteroviruses 68–71 (identified since 1970).[31] Reports of *Picornaviridae* to LabVISE are shown in Table 21.

Enteroviruses (general)

Picornavirus reports averaged 7.5 per cent of the annual reports to LabVISE throughout the study period. Enteroviruses made up nearly two-thirds of the total picornavirus reports (12,148, 65%) and 4.7 per cent of the total LabVISE reports.

Of enteroviruses with typing information, echovirus comprised 56 per cent, poliovirus 19.7 per cent, coxsackie B virus 14.9 per cent and coxsackie A virus 7.2 per cent.

Coxsackie A viruses

Coxsackie A viruses reported to LabVISE between 1991 and 2000 are shown in Table 22.

Table 21. Laboratory reports to LabVISE of Picornaviridae, 1991 to 2000

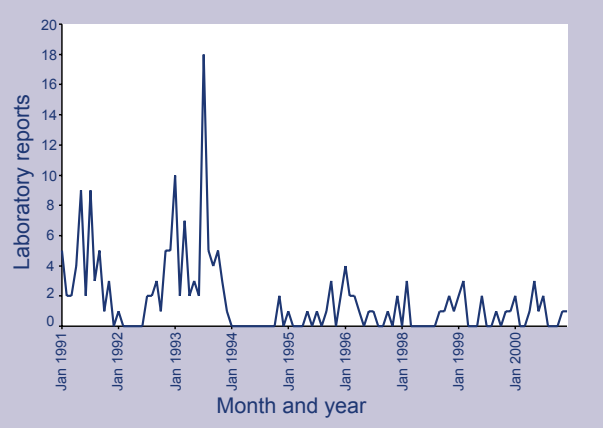
Virus	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	Total
Enterovirus not typed	673	781	943	1,101	891	742	484	538	753	815	**7,721**
Rhinovirus (all types)	653	683	868	905	650	662	549	420	501	420	**6,311**
Echovirus	155	497	502	443	206	49	25	149	264	193	**2,483**
Poliovirus	194	186	121	106	71	35	24	44	52	40	**873**
Coxsackie B virus	155	136	113	87	21	49	27	18	21	32	**659**
Coxsackie A virus	58	43	82	36	12	22	10	12	25	19	**319**
Enterovirus 71	13	15	1	-	34	-	-	9	15	6	**93**

Table 22. Laboratory reports to LabVISE of coxsackie A virus, 1991 to 2000, by serotype

Virus	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	Total
Coxsackie virus A9	45	19	62	2	9	9	5	8	10	11	**180**
Coxsackie virus A16	9	21	18	34	1	12	5	3	15	8	**126**
Coxsackie virus A2	2	-	-	-	-	-	-	-	-	-	**2**
Coxsackie virus A10	-	-	-	-	1	-	-	1	-	-	**2**
Coxsackie virus A7	-	-	-	-	-	1	-	-	-	-	**1**
Coxsackie virus A21	-	-	1	-	-	-	-	-	-	-	**1**
Coxsackie virus A (untyped)	2	3	1	-	1	-	-	-	-	-	**7**
Total	**58**	**43**	**82**	**36**	**12**	**22**	**10**	**12**	**25**	**19**	**319**

Diagnoses in which coxsackie A viruses were identified were most commonly in patients with skin or mucous membrane disease (38%) and meningitis (21%), and most often identified in specimens of nasopharyngeal swabs (25%). Most coxsackie A viruses are not readily isolated by cell culture except serotypes A9 and A16. As a result these were the most commonly isolated serotypes reported by LabVISE. Coxsackie A9 infections have occurred in outbreaks in 1985, 1988 and 1993 (Figure 11).[30]

Figure 11. Laboratory reports to LabVISE of coxsackie A9 infections, 1991 to 2000, by month of specimen collection



This virus is associated with aseptic meningitis in adults and children. Coxsackie virus A16 is the etiologic agent of hand, foot and mouth disease. In Western countries, cases of this disease among children in the same family are often seen. The infection is typified by fever followed by the appearance of oral vesicles and peripheral exantham on the skin of the hands and feet.[31] The clinical syndromes associated with coxsackie A viral infections are summarised in Table 23.

Coxsackie B viruses

Coxsackie B viruses were most often identified in patients presenting with meningitis (31%), lower respiratory tract illness (18%) and gastroenteritis (11%). Specimens of nasopharyngeal swabs (32%), faeces (26%) or cerebrospinal fluid (23%) were the most common sources.

Coxsackie B4 and B5 were the most common serotypes of coxsackie B viruses identified in LabVISE. Coxsackie B4 is associated with respiratory disease (summer gripe) mostly in children under 5 years of age. Coxsackie B5 is associated with meningitis and occurs in children and adults.[31]

Coxsackie B serotypes identified in LabVISE reports between 1991 and 2000 are shown in Table 24.

There were a number of outbreaks of coxsackie B4 viruses in 1991, 1993–1994, 1996 and 2000. Coxsackie B5 outbreaks occurred in 1991–1992, 1993 and 1996 (Figure 12).

The clinical syndromes associated with coxsackie B viral infection are summarised in Table 25.

Figure 12. Laboratory reports to LabVISE of coxsackie B4 and B5 viruses, 1991 to 2000, by month of specimen collection

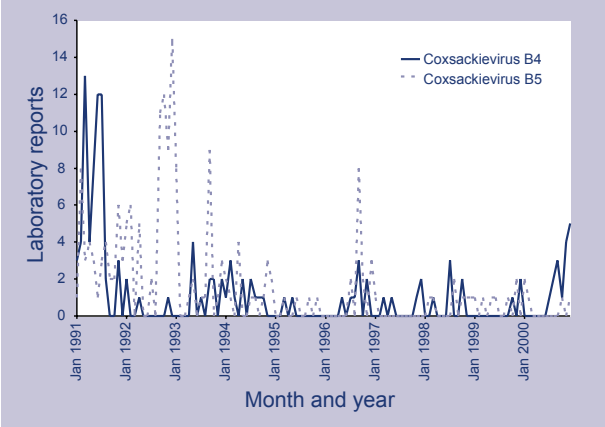


Table 23. Clinical syndromes associated with coxsackie A viral infection*

	Clinical syndrome	Coxsackie A serotypes
Illnesses associated with many enteroviruses in addition to coxsackie A	Aseptic meningitis	1–11,14, 16–18, 22, 24
	Encephalitis	2,5,6,7,9
	Paralysis	4,6,7,9,11,14,21
Illnesses more characteristic of particular groups or serotypes of coxsackie A	Herpangina	2–6, 8, 10, 22
	Hand, foot and mouth syndrome	5,7,9,10,16
	Exanthem	2,4,5,9,16
	Epidemic conjunctivitis	24
Undefined/uncertain etiologic role of coxsackie A viruses	Haemolytic uraemic syndrome	4
	Myositis	9
	Guillain-Barré syndrome	2,5,9
	Mononucleosis	5,6

* Modified from reference 31

Table 24. Laboratory reports to LabVISE of coxsackie B viruses, 1991 to 2000, by year and serotype

Virus	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	Total
Coxsackie virus B5	40	65	26	15	4	15	1	7	7	5	**185**
Coxsackie virus B4	61	4	11	12	2	8	5	6	3	16	**128**
Coxsackie virus B1	6	57	51	4	-	-	1	-	1	4	**124**
Coxsackie virus B2	34	2	8	23	4	20	6	1	10	6	**114**
Coxsackie virus B3	13	6	15	31	11	3	14	2	-	-	**95**
Coxsackie virus B6	-	1	1	2	-	-	-	1	-	-	**5**
Coxsackie virus B untyped	1	1	1	-	-	3	-	1	-	1	**8**
Total	**155**	**136**	**113**	**87**	**21**	**49**	**27**	**18**	**21**	**32**	**659**

Table 25. Clinical syndromes associated with coxsackie B viral infection*

	Clinical syndrome	Coxsackie B serotypes
Illnesses associated with many enteroviruses in addition to coxsackie B viruses	Aseptic meningitis	1–6
	Encephalitis	1–3,5,6
	Paralysis	1–6
Illnesses more characteristic of particular groups or serotypes of coxsackie B viruses	Exanthem	1,3,4,5
	Pleurodynia	1–5
	Pericarditis	1–5
	Myocarditis	1–5
	Generalised disease of the newborn	1–5
Undefined/uncertain etiologic role of coxsackie B viruses	Haemolytic uraemic syndrome	2,4
	Mononucleosis-like syndrome	5

* Modified from reference 31

Echoviruses

Echoviruses identified in LabVISE reports between 1991 and 2000 are shown in Table 26.

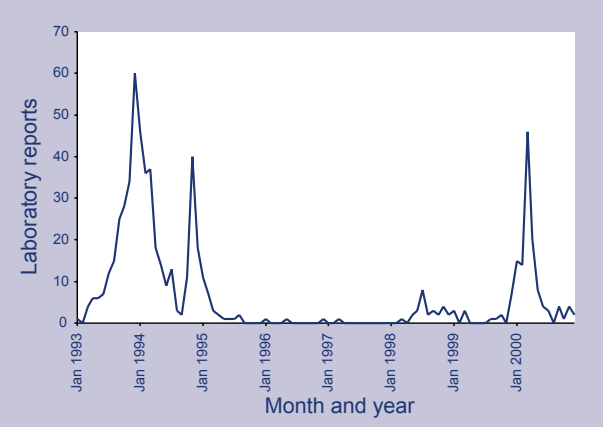
Table 26. Laboratory reports to LabVISE of echovirus, 1991 to 2000, by serotype

Virus	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	Total
Echovirus type 30	1	3	198	247	28	3	1	26	17	121	**645**
Echovirus type 11	10	13	141	26	4	1	5	50	166	7	**423**
Echovirus type 9	5	216	22	7	41	4	1	22	32	5	**355**
Echovirus type 6	9	85	10	107	12	1	1	5	16	1	**247**
Echovirus type 7	4	39	74	-	2	24	4	-	1	33	**181**
Echovirus type 17	59	38	4	-	-	-	-	2	-	-	**103**
Echovirus type 22	19	13	9	9	13	1	1	14	12	8	**99**
Echovirus type 14	7	15	23	12	35	4	-	1	-	1	**98**
Echovirus type 3	-	-	-	27	16	-	-	-	5	2	**50**
Echovirus type 25	2	31	5	1	3	-	-	-	3	-	**45**
Echovirus type 18	3	3	1	2	2	1	-	15	-	1	**28**
Echovirus type 4	2	14	-	-	1	1	1	5	1	-	**25**
Echovirus type 5	3	2	5	1	-	4	4	5	1	-	**25**
Echovirus type 16	14	9	-	-	-	-	-	-	-	-	**23**
Echovirus type 1	4	1	-	-	3	-	-	1	-	3	**12**
Echovirus type 33	1	-	-	-	1	1	1	-	4	4	**12**
Echovirus type 21	2	4	4	-	-	-	-	-	-	-	**10**
Echovirus type 31	1	1	-	-	1	-	1	-	1	2	**7**
Echovirus type 15	1	-	3	-	1	1	-	-	-	-	**6**
Echovirus type 24	2	1	-	-	3	-	-	-	-	-	**6**
Echovirus type 19	-	2	1	-	-	-	-	-	1	-	**4**
Echovirus type 2	-	1	-	-	-	-	1	1	1	-	**4**
Echovirus type 20	-	3	-	-	-	-	-	-	-	-	**3**
Echovirus type 23	-	-	-	1	1	-	-	-	-	-	**2**
Echovirus type 32	2	-	-	-	-	-	-	-	-	-	**2**
Echovirus type 8	-	2	-	-	-	-	-	-	-	-	**2**
Echovirus type 34	1	-	-	-	-	-	1	-	-	-	**2**
Echovirus type 12	-	-	1	-	-	-	-	-	-	-	**1**
Echovirus type 13	-	-	-	-	1	-	-	-	-	-	**1**
Echovirus not typed/ pending	3	1	1	3	38	3	3	2	3	5	**62**
Total	**155**	**497**	**502**	**443**	**206**	**49**	**25**	**149**	**264**	**193**	**2,483**

The most commonly identified echovirus serotypes during the study period were echovirus types 30, 11 and 9. Diagnoses in which echoviruses were identified were predominantly in cases of meningitis (41%) and were often isolated from cerebrospinal fluid.

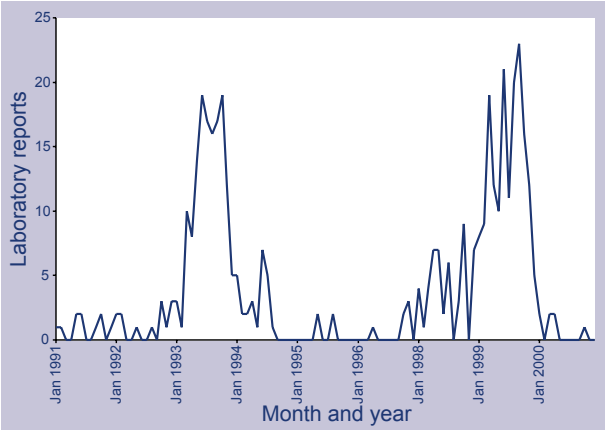
Echovirus 30 was the most common echovirus serotype identified in LabVISE during the study period. Echovirus 30 has caused large outbreaks of aseptic meningitis in many regions of the world during the last 40 years. Periods of increased reporting of echovirus 30 were evident in 1993 to 1994 and again in 2000 (Figure 13).

Figure 13. Laboratory reports to LabVISE of echovirus 30, 1991 to 2000, by month of specimen collection



Periods of increased activity of echovirus 11 associated with aseptic meningitis occurred at intervals of 2–4 years with major peaks in 1993 and 1999. More than half the cases were in children under 5 years (Figure 14).

Figure 14. Laboratory reports to LabVISE of echovirus 11, 1991 to 2000, by month of specimen collection



The clinical syndromes associated with infection with echovirus are shown in Table 27.

Table 27. Clinical syndromes associated with echoviruses*

	Clinical syndrome	Associated echovirus serotypes
Illnesses associated with many enteroviruses in addition to echoviruses	Aseptic meningitis	All except 24,26,29,32
	Encephalitis	2–4,6,7,9,11,14,17–19,25
	Paralysis	1–4,6,7,9,11,14,16,18,19,30
Illnesses more characteristic of particular groups or serotypes of echovirus	Exanthem	9,16 also 1–8,11,14,18,19,25,30,32,33
	Generalised disease of the newborn	4,6,7,9,11,12,14,19,21,51
	Neonatal diarrhoea	11,14,18
	Chronic meningoencephalitis in agammaglobulinemics	2,3,5,9,11,19,24,25,30,33
Undefined/uncertain etiologic role for echovirus	Myositis	9,11
	Haemolytic uraemic syndrome	22
	Guillan-Barré syndrome	6,22
	Infectious lymphocytosis	25

* Modified from reference 31

Polioviruses

Polioviruses are the cause of poliomyelitis, an infection of the central nervous system, which may result in acute flaccid paralysis. Poliovirus infection occurs via the gastrointestinal tract and in more than 90 per cent of cases, causes an inapparent infection.[24] Acute flaccid paralysis occurs in less than one per cent of infections. Three serotypes are recognised and immunity is serotype specific.[31] The oral polio vaccine, which has been in widespread use in Australia and throughout the world for the last 50 years, is a mixture of the three serotypes. Naturally occurring ('wild type') or live attenuated vaccine polioviruses circulate to a varying extent, depending on the impact of polio vaccine on transmission.[31]

Oral polio vaccination has placed the global eradication of poliomyelitis within reach. Numerous regions of the world, including the Western Pacific Region (which includes Australia) have been declared polio-free. The last case of poliomyelitis in Australia occurred in 1972 and the Western Pacific Region was declared polio-free in October 2000. However, continued surveillance is required since there is a continuing possibility of importation of cases of poliovirus from endemic areas.

Surveillance for poliovirus in Australia comprises reporting of poliomyelitis as a notifiable disease to the NNDSS, surveillance of all cases of acute flaccid paralysis, surveillance of vaccine associated paralytic polio cases, surveillance of enteroviruses and intratypic differentiation of all polioviruses isolated in Australia.

Poliovirus laboratory reports to LabVISE between 1991 and 2000 are shown in Table 28.

Thirty-seven per cent of poliovirus diagnoses were associated with a diagnosis of gastrointestinal disease and 50 per cent of the specimens in which poliovirus was identified were stool samples. No wild-type poliovirus has been isolated from any case of acute flaccid paralysis.[32] Continued surveillance of poliovirus will be required until the circulation of wild-type polio can be shown to have ceased. There is concern over the potential for live attenuated virus from the oral polio vaccine persisting in water supplies and possibly reverting toward wild-type neurovirulent phenotypes. This is one reason some countries such as the USA have changed to using an inactivated polio vaccine.[33]

Table 28. Laboratory reports to LabVISE of poliovirus, 1991 to 2000, by type

Virus	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	Total
Poliovirus type 1 (uncharacterised)	40	71	43	41	27	15	9	12	26	22	**306**
Poliovirus type 2 (uncharacterised)	54	45	35	41	29	14	9	25	16	8	**276**
Poliovirus type 3 (uncharacterised)	26	32	31	14	13	1	4	6	8	8	**143**
Poliovirus type 1 (vaccine strain)	-	-	-	-	-	1	-	1	-	-	**2**
Poliovirus type 2 (vaccine strain)	-	-	-	-	-	2	2	-	1	-	**5**
Poliovirus type 3 (vaccine strain)	-	-	-	-	-	-	-	-	-	1	**1**
Poliovirus not typed/ pending	74	38	12	10	2	2	-	-	1	1	**144**

Enteroviruses 71 and enterovirus (not typed)

Since the revision of the classification system of the taxonomic scheme for picornaviruses in 1970, four new serotypes have been discovered. These are enteroviruses 68–71. These 'new' enteroviruses are associated with distinct clinical symptoms and show a defined geographical distribution. In Australia, only enteroviruses 70 and 71 have been reported. Infections with enterovirus 70 occurred in a cluster in New South Wales in 1990, but since then, there have been no more reports of this virus to LabVISE.[30]

Enterovirus 71, the most recently discovered enterovirus has been recognised as a cause of cutaneous and central nervous system disease since 1969. Enterovirus 71 is the only non-poliovirus enterovirus known to have the potential to cause epidemic paralytic disease.

The relatively large number of untyped enteroviruses may reflect laboratory practices whereby reports to LabVISE are made only on initial identification or after exclusion of polioviruses, and further identification data are not sent.

Report to LabVISE of enterovirus 71 and enterovirus (untyped) are shown in Table 29.

Enterovirus 71 was first reported in Australia in 1972. A major outbreak of enterovirus 71 occurred in south-east Australia in 1986. Clinical diagnosis during this outbreak was largely skin and mucous membrane disease, meningitis (23%) and respiratory disease (20%).[30] An outbreak of hand, foot, and mouth disease caused by an enterovirus was reported in Western Australia in 1999.[34] In this outbreak, nine of 14 (64%) children developed neurological disease and four of these had long-term sequelae.

Rhinovirus

Rhinoviruses are a cause of the common cold with a worldwide distribution.[35] Rhinoviruses infect humans from early childhood with recurrent infection throughout life. In temperate regions, there are annual seasonal peaks in incidence.

Rhinovirus laboratory reports to LabVISE between 1991 and 2000 are shown in Table 30.

LabVISE reports of rhinovirus show an annual peak in late winter and early spring (Figure 15).

Figure 15. Laboratory reports to LabVISE of rhinovirus infections, 1991 to 2000, by month of specimen collection

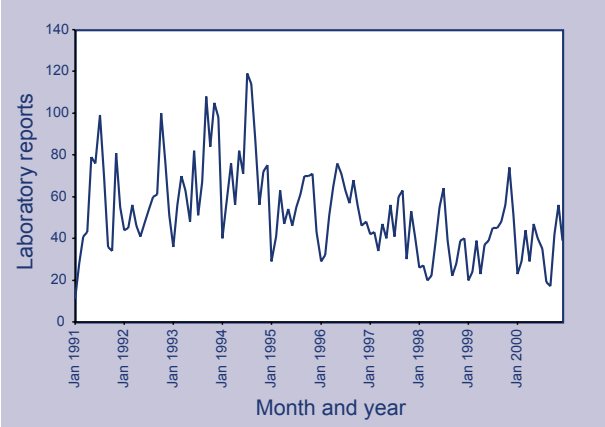


Table 29. Laboratory reports to LabVISE of enterovirus 71 and enterovirus (untyped), 1991 to 2000

Virus	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	Total
Enterovirus type 71	13	15	1	-	34	-	-	9	15	6	**93**
Enterovirus not typed/pending	673	781	943	1,101	891	742	484	538	753	815	**7,721**

Table 30. Laboratory reports to LabVISE of rhinovirus, 1991 to 2000

Virus	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	Total
Rhinovirus (all types)	653	683	868	905	650	662	549	420	501	420	**6,311**

Ortho/paramyxoviruses

Influenza virus

Influenza is a highly contagious acute respiratory disease which has caused epidemics and pandemics throughout the world for centuries. While most influenza infections are self-limiting, lower respiratory tract and cardiac complications, particularly in the elderly can lead to increased hospitalisations and deaths, particularly during the epidemic months.[36]

Up until 2001, LabVISE has been the only source of laboratory-confirmed influenza data for national influenza surveillance. Viral isolates are forwarded to the World Health Organization Centre for Reference and Research on Influenza for subtype and antigenic analysis. These data have been used to monitor circulating influenza viral strains and to determine the composition of the annual influenza vaccine for Australia.

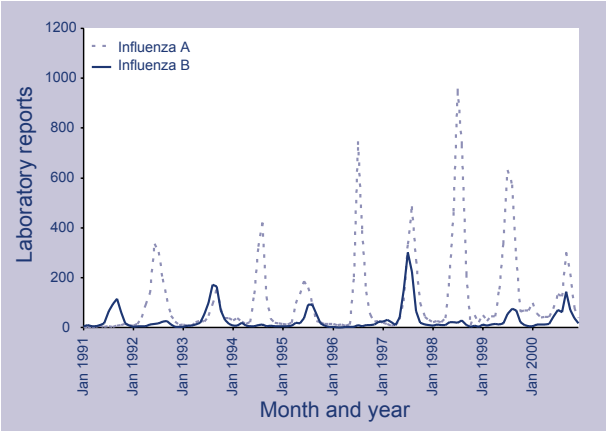
Influenza reports to LabVISE for 1991 to 2000 are shown in Table 31.

The monthly reports to LabVISE of influenza A and B, between 1991 and 2000 are shown in Figure 16. Typically, influenza A is predominant with outbreaks of influenza B every alternate year. Laboratory reports of influenza are largely from young children aged under 5 years.

Parainfluenza virus

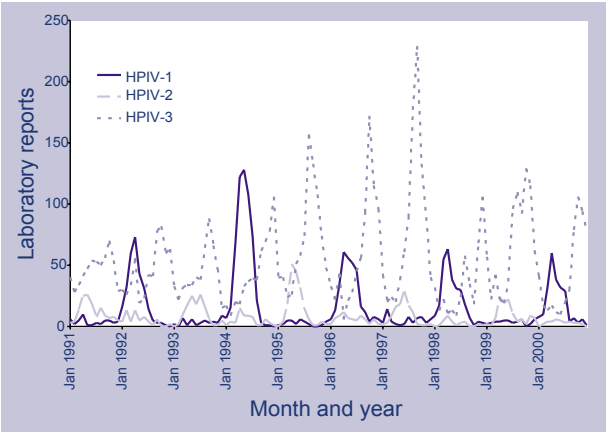
Human parainfluenza viruses (HPIV) are an important cause of acute respiratory infection in infants and children and are especially associated with laryngotracheobronchitis (croup). In the USA, the parainfluenza viruses are responsible for one-third of the estimated 5 million cases of lower respiratory infections occurring annually in children under 5 years of age.[37] Infections also occur in older age groups. Four serotypes are recognised. Biennial epidemics of HPIV–1 and HPIV–2 occur in autumn, while HPIV–3 causes annual epidemics, particularly among young infants aged less than 6 months. LabVISE reports of parainfluenza virus are shown in Table 32.

Figure 16. Laboratory reports to LabVISE of influenza A and influenza B infections, 1991 to 2000, by month of specimen collection



Laboratory reports of parainfluenza by serotype and month between 1991 and 2000 are shown in Figure 17. There are annual epidemics of parainfluenza type 3, while parainfluenza types 1 and 2 occur in biennial epidemics in alternate years in Australia. Laboratory reports to LabVISE for parainfluenza were predominantly for children aged 0–4 years. In 2000, 68 per cent of HPIV–1, 53 per cent of HPIV–2 and 68 per cent of HPIV–3 occurred in children aged 0–4 years.

Figure 17. Laboratory reports to LabVISE of human parainfluenza serotypes 1, 2 and 3, 1991 to 2000, by month of specimen collection



HPIV-1 human parainfluenza serotype 1
HPIV-2 human parainfluenza serotype 2
HPIV-3 human parainfluenza serotype 3

Table 31: Laboratory reports to LabVISE of influenza, 1991 to 2000, by strain type and annual influenza A:B ratio

Virus	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	Total
Influenza A	60	1,322	544	1,196	797	1,641	1,447	2,746	1,932	1,506	**13,191**
Influenza B	408	126	648	87	355	79	903	149	279	580	**3,614**
Ratio influenza A:B	0.1	10.5	0.8	13.7	2.2	20.8	1.6	18.4	6.9	2.6	

Table 32. Laboratory reports to LabVISE of parainfluenza virus, 1991 to 2000, by serotype

Virus	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	Total
Parainfluenza virus type 1	47	281	44	548	32	315	61	276	44	230	**1,878**
Parainfluenza virus type 2	143	60	127	61	178	73	112	30	114	36	**934**
Parainfluenza virus type 3	556	554	513	526	833	730	962	409	803	516	**6,402**
Parainfluenza virus type 4	-	-	-	-	2	7	-	3	2	-	**14**
Parainfluenza virus typing pending	59	80	46	68	36	32	239	5	1	1	**567**
Total	**805**	**975**	**730**	**1,203**	**1,081**	**1,157**	**1,374**	**723**	**964**	**783**	**9,795**

Table 33. Laboratory reports to LabVISE of respiratory syncytial virus, 1991 to 2000

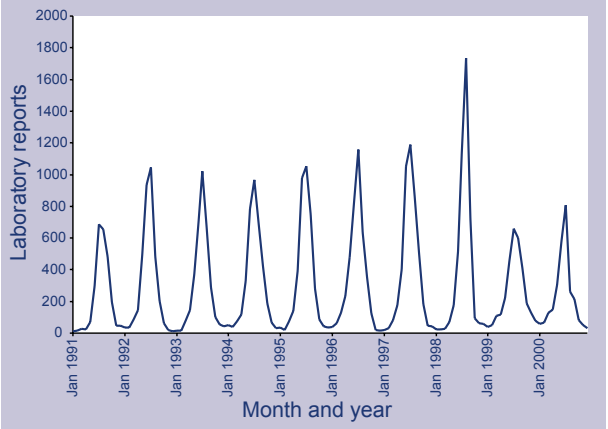
Virus	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	Total
Respiratory syncytial virus	2,555	3,556	3,506	3,749	3,889	4,068	4,588	4,641	3,059	2,735	**36,346**

Respiratory syncytial virus

Respiratory syncytial virus (RSV) infects almost all people in all regions of the world within the first years of life and is the major cause of lower respiratory illness in young children. RSV is an important cause of community-acquired pneumonia.[38] A recent study from the United Kingdom suggests that RSV infection may be confused with influenza-like illness.[39] RSV identifications were the single most common virus reported in LabVISE (14.3% of total between 1991 and 2000). LabVISE reports of RSV are shown in Table 33.

RSV epidemics occur annually in the winter months (Figure 18) and most patients are aged between 0–4 years. Thus of 2,735 reports in 2000, 2,446 (89.4%) were in children aged less than 5 years.

Figure 18. Laboratory reports to LabVISE of respiratory syncytial virus infection, 1991 to 2000, by month of specimen collection



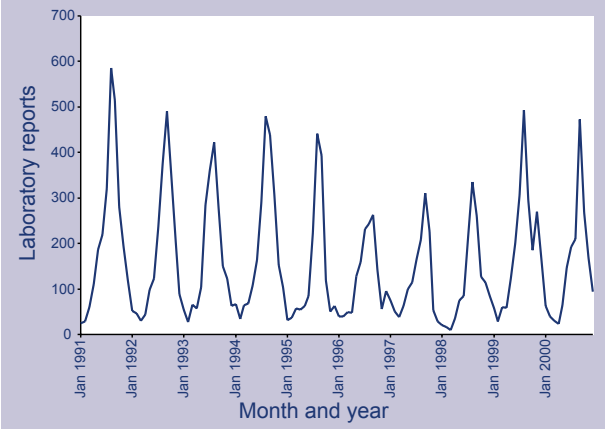
Other respiratory RNA viruses

Reports grouped under the LabVISE category 'other RNA viruses' are largely agents of viral gastrointestinal illness. LabVISE reports of these viruses are shown in Table 34.

Rotavirus

Reports of rotavirus infection were among the largest for any single virus in LabVISE (7.5% of total reports) between 1991 and 2000. Rotavirus infections are a common cause of diarrhoea in children and in Australia cause annual epidemics in the winter months (Figure 19).

Figure 19. Laboratory reports to LabVISE of rotavirus infection, 1991 to 2000, by month of specimen collection



Of the 1,771 reports of rotavirus in 2000, 1,556 (88%) were in children aged less than 5 years. From June 1999, a rotavirus surveillance program has been undertaken by the National Rotavirus Reference Centre, Royal Children's Hospital, Parkville, Victoria. Samples that test positive for rotaviruses by enzyme immunoassay or latex agglutination in collaborating laboratories are sent to the centre for serotyping. The Centre reports annually on circulating serotypes and outbreaks of rotavirus.[40]

Norwalk-like virus

For the purposes of this analysis, Norwalk-like virus includes reports of 'Norwalk agent', 'calicivirus' and 'small virus-like particles. Norwalk-like virus (NLV) is the leading cause of outbreaks of diarrhoea and vomiting in the United Kingdom.[41] These viruses are spread through contaminated food, aerosols, direct contact and environmental contamination. A recent report from the USA of a multi-state outbreak of NLV gastroenteritis associated with a common caterer, underlines the importance of this pathogen as food preparation becomes more centralised and distribution of products become more widespread.[42]

Astrovirus, and reovirus (unspecified)

Small numbers of astroviruses and reoviruses were reported to LabVISE during the study period (Table 34).

Astroviruses are a common cause of infantile gastroenteritis worldwide both as sporadic cases and as outbreaks. A study of children aged less than 5 years in Melbourne between 1995 and 1998, confirmed astrovirus as the cause of acute gastroenteritis in 3 per cent of cases.[11]

Reoviruses (unspecified) may include rotavirus. While reoviruses may cause human disease, infection is uncommon. Enteritis in infants and children and upper respiratory tract infections caused by reoviruses have been reported.[43]

Other non-viral pathogens

Chlamydial infections

Chlamydiae are a unique class of bacteria that are obligate intracellular parasites. Three *Chlamydia* species are recognised, all of which are human pathogens: *C. trachomatis, C. pneumoniae and C. psittaci*. The associated diseases, strains, mode of infection and host species are shown in Table 35.

Reports of chlamydial infections to LabVISE are shown in Table 36.

Table 34. Laboratory reports to LabVISE of 'other RNA viruses', 1991 to 2000

Virus	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	Total
Rotavirus	2,642	2,134	1,989	2,275	1,617	1,492	1,431	1,372	2,245	1,771	**18,968**
Norwalk-like virus	117	89	66	50	65	77	70	47	60	82	**723**
Astrovirus	21	14	4	1	6	3	4	10	3	-	**66**
Reovirus (unspecified)	8	12	7	3	-	-	-	-	2	2	**34**

Table 35. Characteristics of *Chlamydia* spp. and strains, modes of transmission and associated human diseases*

Species	Strains	Mode of transmission	Host species	Associated human diseases
C. trachomatis	LGV (L1, L2, L3)	Sexual	Humans	Lymphogranuloma venereum
	Trachoma (A,B,Ba,C)	Hand to eye, fomites, flies	Humans	Ocular trachoma
	Trachoma (B,Ba,D–K)	Sexual, hand to eye, neonatal	Humans	Ocular and genital disease, infant pneumonia
C. psittaci	Many	Aerosol	Birds, sheep, cats etc	Ornithosis (psittacosis)
C. pneumoniae	'TWAR'	Not defined aerosol?	Humans	Bronchitis, pneumonia

* Adapted from reference 44

Table 36. Laboratory reports of to LabVISE chlamydial infections, 1991 to 2000

Organism	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	Total
Chlamydia trachomatis A–K	59	11	-	1	1	1	1	-	1	-	**75**
Chlamydia trachomatis L1–L3	18	-	-	-	-	-	1	-	-	-	**19**
Chlamydia trachomatis not typed	2,615	2,563	2,835	2,178	2,579	3,803	3,980	3,158	3,295	3,154	**30,160**
Chlamydia pneumoniae	2	14	1	-	2	1	3	-	2	36	**61**
Chlamydia psittaci	139	97	74	114	176	62	51	70	78	102	**963**
Chlamydia spp. typing pending	1	10	9	10	6	1	7	-	1	-	**45**
Chlamydia species not typed	-	6	18	62	75	54	28	57	21	8	**329**
Total	**2,834**	**2,701**	**2,937**	**2,365**	**2,839**	**3,922**	**4,071**	**3,285**	**3,398**	**3,300**	**31,652**

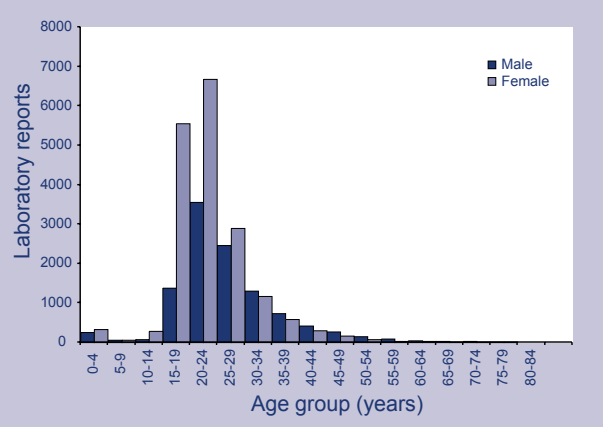
Chlamydia trachomatis

Notifications of *Chlamydial trachomatis* infection between 1991 and 2000 to the NNDSS and laboratory reports to LabVISE are shown in Table 37.

The NNDSS case definition for chlamydial infections requires the isolation of *Chlamydia trachomatis* or demonstration of chlamydial antigens in clinical specimens. For most of the study period and from most jurisdictions, notifications to the NNDSS of chlamydial infections were restricted to genital chlamydial infections. New South Wales did not start reporting chlamydial infections to the NNDSS until 1998. LabVISE laboratory reports have fallen from representing two-thirds of the NNDSS reports in 1991 to just 19 per cent in 2000. Whether laboratory reports in LabVISE were from clinical samples only from genital sites or whether they include samples from other infected sites cannot be determined.

The distribution of chlamydial infections by age and sex, reported to LabVISE are shown in Figure 20. The distribution is very similar to that seen in NNDSS notifications, with a female predominance (male to female ratio of 1:1.6) and the largest number of reports (57%) from young adults aged 15–24 years.

Figure 20. Laboratory reports to LabVISE of *Chlamydia trachomatis* infection, 1991 to 2000, by age and sex



Chlamydia psittaci

Notifications of *Chlamydia psittaci* infection between 1991 and 2000 to the NNDSS and laboratory reports to LabVISE are shown in Table 38.

Ornithosis has not been a notifiable disease in all Australian jurisdictions during the period 1991 to 2000 and consequently cases reported to the NNDSS do not represent national figures. While no agreed national NNDSS definition for ornithosis was used in this period, probable cases diagnosed based on an acute clinical illness compatible with ornithosis, were included. Laboratory diagnosis is based on increases in specific antibody titres, or more recently, detection of *C. psitttaci* by nucleic acid tests.

For a number of years, LabVISE reported more cases of ornithosis than the NNDSS. LabVISE reports showed a male predominance (male to female ratio 1.7:1) and a peak of reports from adult men aged 50–54 years. Figure 21 shows the age and sex distribution of laboratory reports of ornithosis to LabVISE. This age and sex distribution is similar to that found in notifications of ornithosis to NNDSS.

Figure 21. Laboratory reports to LabVISE of *Chlamydia psittaci* infections, 1991 to 2000, by age and sex

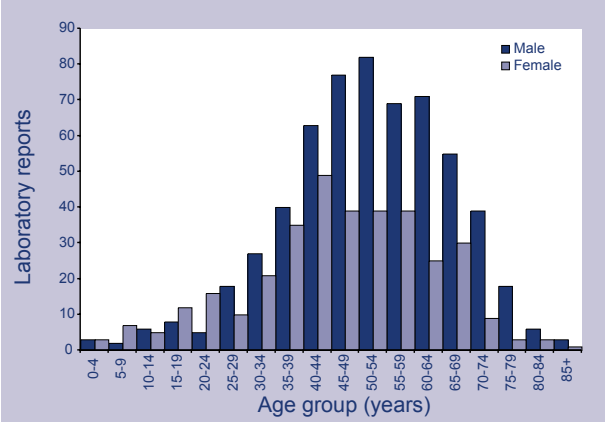


Table 37. Laboratory reports to LabVISE and notifications to NNDSS of *Chlamydia trachomatis* infections, 1991 to 2000

Surveillance system	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
LabVISE	2,692	2,574	2,835	2,179	2,580	3,804	3,982	3,158	3,296	3,154
NNDSS	4,044	6,293	6,500	6,450	6,398	8,445	9,242	11,339	14,082	16,853

Table 38. Laboratory reports to LabVISE and notifications to NNDSS of *Chlamydia psittaci* infections (ornithosis), 1991 to 2000

Surveillance system	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
LabVISE	139	97	74	114	176	62	51	70	78	102
NNDSS	136	98	94	86	186	86	35	64	84	100

Table 39. Laboratory reports to LabVISE, of *Mycoplasma* infections, 1991 to 2000

Organism	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	Total
Mycoplasma pneumoniae	381	1,580	1,760	820	334	1,009	1,640	1,285	1,125	686	**10,620**
Mycoplasma hominis	2	4	-	2	-	2	-	2	5	8	**25**
Total	**383**	**1,584**	**1,760**	**822**	**334**	**1,011**	**1,640**	**1,287**	**1,130**	**694**	**10,645**

Chlamydia pneumoniae

Chlamydia pneumoniae causes an acute respiratory disease similar to that caused by *Mycoplasma*. Infection is widespread and antibody prevalence is low in children, around 50 per cent in young adults and continues to be high into old age. Clinical disease is seen in all ages but most frequently in young adults.[24]

Mycoplasma pneumoniae

Mycoplasma pneumoniae is the cause of mycoplasma pneumonia (or primary atypical pneumonia), that presents predominantly as a febrile lower respiratory infection or occasionally as a pharyngitis, bronchitis or pneumonia.[24] The disease is worldwide in distribution and may occur in all age groups with occasional epidemics in institutions and military recruits.

Laboratory reports to LabVISE of *Mycoplasma* between 1991 and 2000 are shown in Table 39.

Mycoplasma pneumoniae infections reported to LabVISE were most commonly reported in female children aged 5–9 years (male to female ratio 0.9:1, Figure 22).

Mycoplasma pneumoniae reports show variation from year to year without a distinct seasonal peak (Figure 23).

Mycoplasma hominis is commonly isolated from the genitourinary tract (more commonly from women than men), the neonatal conjunctiva and peripartum blood. The organism is associated with cervicitis, vaginitis, conjunctivitis and peripartum sepsis.[45]

Figure 22. Laboratory reports to LabVISE of *Mycoplasma pneumoniae* infections, 1991 to 2000, by age and sex

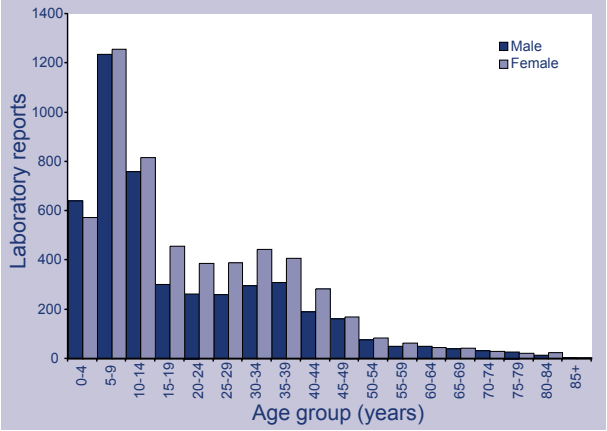


Figure 23. Laboratory reports to LabVISE of *Mycoplasma pneumoniae* infections, 1991 to 2000, by month of report

Rickettsia

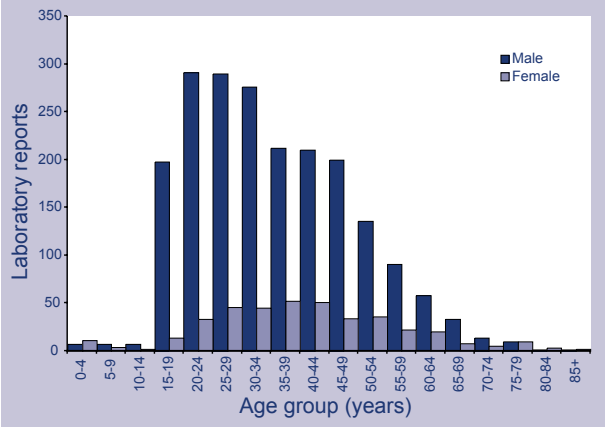
Coxiella burnetii

The rickettsial pathogen *Coxiella burnetii* is the cause of an acute febrile disease with a variety of clinical presentations of variable severity and duration (Q fever). The disease is particularly associated with livestock workers. Notifications of Q fever to the NNDSS and laboratory reports of *Coxiella burnetii* to LabVISE between 1991 and 2000 are shown in Table 40.

The NNDSS case definition for Q fever[46] is based on serology or isolation of the organism from a clinical sample. The trends in laboratory reports to LabVISE are similar to trends in NNDSS data, with a peak in reports in 1993 and lower reports in more recent years as a result of Q fever vaccine initiatives among abattoir workers, who are at high risk of contracting the disease.

An age, sex analysis of *Coxiella burnetii* infections shows a large male predominance (male:female ratio 5.2:1), similar to that shown in NNDSS data. The largest numbers of laboratory reports to LabVISE were from men aged 25–29 years (Figure 24).

Figure 24. Laboratory reports to LabVISE of *Coxiella burnetii* infections, 1991 to 2000, by age and sex



Small numbers of laboratory reports of other *Rickettsia* were sent to LabVISE during the period as shown in Appendix 3.

Other pathogens

From 1992 to 1996 and in 1999 and 2000, several hundred reports were made to LabVISE of group A streptococci. While streptococcal group A infections have declined dramatically in Australia, Indigenous Australians in northern Australia continue to suffer endemic infection with corresponding high rates of rheumatic fever,[47] acute post-streptococcal glomerulonephritis,[48] streptococcal pyoderma[49] and resulting chronic renal disease and rheumatic heart disease.

A significant number of reports to LabVISE during the period 1991 to 2000 were received for other pathogens under surveillance in the NNDSS (Appendix 2). These include isolations of *Yersinia, Brucella, Bordetella, Legionella, Leptospira, Treponema* and *Echinococcus*. Since data on these pathogens was collected sporadically over the period and represent only a small fraction of notified cases reported to NNDSS, no analysis has been attempted here. Similarly for small numbers of reports of *Cryptococcus, Entamoeba histolytica* and *Toxoplasmosis gondii* no meaningful comments can be offered.

The reader is referred to NNDSS annual reports published in *Communicable Diseases Intelligence* for detailed analysis of the epidemiology of these and other pathogens in Australia.

Table 40. Laboratory reports to LabVISE and notifications to NNDSS of *Coxiella burnetii* infection (Q fever), 1991 to 2000

Surveillance system	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
LabVISE	240	270	552	345	167	208	259	137	221	101
NNDSS	595	543	889	664	466	554	580	560	518	573

Discussion

This review of LabVISE data for the last 10 years has shown that this reporting scheme contains valuable data potentially important to public health in Australia. LabVISE collects important data on the viral diseases, particularly those of children that are not reported in other surveillance systems. These data supplement diseases under surveillance through the National Notifiable Diseases Surveillance System.

LabVISE has had a uniquely important role in the control of two diseases in the last 10 years, influenza and poliomyelitis.

Influenza epidemics occur in Australia annually in winter. Admissions to hospitals in Australia for influenza and pneumonia exceed 250,000 annually.[50] Pneumonia secondary to influenza infection in the elderly is an important cause of mortality.[36,50] For this reason, the Australian Government has provided influenza vaccination free of charge to Australians aged 65 years and above since 1999. Surveys of elderly Australians in 2000 and 2001 show that 77 per cent of this age group are taking annual influenza vaccination.[51] Each year, the composition of the Australian influenza vaccine is reviewed and altered to reflect the virus strains circulating in the previous influenza season. This information is derived in a large part from isolates collected through LabVISE laboratories.

Laboratory reports of influenza are reported throughout the year and allow surveillance of the annual epidemics. These data, combined with reports of influenza-like illness in sentinel general practices, are reported annually in the report of the National Influenza Surveillance Scheme.[52]

Since June 2001, influenza data are published fortnightly throughout the year on the Communicable Diseases Australia Website at http://www.health.gov.au/pubhlth/cdi/ozflu/flucurr.htm. It is essential for influenza surveillance and for vaccine production that LabVISE laboratories continue to isolate and characterise influenza viruses. It is only a matter of time until genetic mutations in the influenza virus trigger an influenza pandemic. Laboratory surveillance will be critical in the early stages of a pandemic to characterise the new virus and to monitor the geographical spread.

The last case of poliomyelitis in Australia was reported in 1972 and the Western Pacific Region was declared polio-free in October 2000. The WHO recommends continued surveillance of poliovirus in Australia, including laboratory investigations of all cases of acute flaccid paralysis and vaccine-associated paralytic polio and the monitoring of circulating enteroviruses in the community and intratypic differentiation of all polioviruses.[32] LabVISE contributes significantly to these aspects of polio surveillance. Such surveillance is still not reaching the targets set by WHO[32] and surveillance needs to continue until the global eradication of poliomyelitis is achieved. Recent outbreaks of polio in Bulgaria,[53] show that countries that have eliminated endemic polio are at risk from imported polio cases.

LabVISE has accumulated important information on viral pathogens of public health importance, particularly causative organisms of viral meningitis, viral gastroenteritis and viral respiratory diseases.

Viral meningitis surveillance is recommended by the WHO.[54] The rationale for this surveillance is that viral meningitis can occur in epidemics as well as sporadically and that although mortality is generally low, the associated morbidity and risk of long term sequelae in children are high. Laboratory identification of the causative virus and measurement of incidence by time, geographical area and age group are important to the identification and control of epidemics. There is no other national surveillance system in Australia, other than LabVISE, in a position to undertake this surveillance.

Outbreaks of viral meningitis associated with echovirus 11 and 30, enterovirus 71, and coxsackie A9 and B5, have been detected in Australia in recent years. However, there has been no nationally coordinated approach to these important public health problems.

Viral agents are responsible for the largest proportion of gastroenteritis in developed countries.[55] Rotavirus is a major cause of diarrhoea and may be an important cause of gastroenteritis in the elderly. In Australia, there are annual epidemics in the winter months and large outbreaks have occurred biennially in the Northern Territory.[56] During these epidemics, hospital consultations and admissions are greatly increased. Although deaths are rare in industrialised countries, rotavirus causes 800,000 childhood deaths annually in the developing countries.[57] The introduction of a rotavirus vaccine in 1998 was to be an important milestone in the control of this disease. The vaccine was subsequently withdrawn after cases of intestinal intussusception. Despite this, vaccination for rotavirus, particularly as part of the childhood immunisation program in developing

countries, remains a priority. Since 1999, the National Rotavirus Surveillance Program has serotyped rotaviruses isolated in laboratories, measured the emergence of new serotypes, and tracked their geographical spread in Australia.[40] Continued laboratory surveillance of rotavirus is important to monitor epidemics, to monitor circulating serotypes and to measure the impact of future vaccines.

Norwalk-like virus has emerged as a major cause of gastroenteritis in adults. The control of this agent is difficult, as transmission has been shown to be by aerosols and environmental contamination[58] as well as by contaminated food.[42] The epidemiology of the virus continues to be elucidated and surveillance of this agent is important for the control of community gastroenteritis.

Viral respiratory diseases include influenza, parainfluenza, respiratory syncytial virus and various adenoviruses. These viruses are important childhood pathogens. The importance of LabVISE to surveillance of influenza in Australia has been noted above. More common childhood infections with parainfluenza and RSV have been reported consistently to LabVISE over the years. Both viruses cause annual epidemics, with parainfluenza serotype 1 showing annual epidemics while serotypes 2 and 3 show biennial epidemics.

Since HPIV–3 causes more severe disease, monitoring of parainfluenza serotypes is important. Adenoviruses play a role in acute respiratory infections and monitoring of circulating serotypes will be an important activity to predict disease patterns in the Australian community.

The LabVISE scheme has been evaluated elsewhere[30] and a full discussion of the schemes strengths and weaknesses belong to another paper. However, it is obvious from the analysis performed here that there are limitations to the value of LabVISE data. As has been noted earlier the representativeness of the data in LabVISE is uncertain since there has been no measure from what population these results are drawn. The large number of reports from children in LabVISE reflects both the nature of the pathogens reported but also the inclusion of reports from major children's hospitals in Australia. By contrast, other age groups may be under-represented. The large proportion of tertiary hospital laboratories in LabVISE also biases the system to report less common pathogens or those more difficult to diagnose, seen perhaps more often in sicker individuals and not typical of the community at large. It is unclear what populations these large reference laboratories serve and laboratories have only reported positive results without reporting on the total number of tests performed. Thus, there are no denominators in the data to calculate rates. The National Respiratory and Enteric Virus Surveillance System of the Centers for Disease Control and Prevention in the USA, collect data as the percentage of positive isolates from laboratories, which allows monitoring of virus activity throughout the year. (www.cdc.gov/ncidod/dvrd/revb/nrcvss/index. Accessed, January 2002).

Another criticism of LabVISE has been the lack of focus, in that the surveillance system covers a large range of pathogens of varying public health significance. A focus on viral agents of meningitis, gastroenteritis and respiratory disease prioritised by public health importance may be an important option for the future of LabVISE. Changes to the pathogens under surveillance and the data collected on each notification in NNDSS impact on the usefulness of data collected by LabVISE. Notifications of measles, mumps, rubella, hepatitis, arboviral infections and chlamydial infection to NNDSS greatly exceed reports to LabVISE. Now that more microbiological data are collected on each NNDSS notification, the data collected by LabVISE has lost its value. Inclusion of these organisms in LabVISE may therefore be redundant and this supports the option that LabVISE should focus on a supplementary set of pathogens.

Reporting of LabVISE data has been poor for many years. This is the first full analysis of LabVISE data since 1996. The timeliness of reports through *Communicable Diseases Intelligence* is also poor as the publication schedule has changed to quarterly. Publication of reports on the Internet would be important in giving timely information.

The quality of data has declined over time with less complete identification of pathogens. A data collection system for NNDSS, which allows the updating of reports with additional detail, is being implemented. This allows the timely notification of a case and the subsequent completion of details including laboratory results. A similar system for LabVISE would allow timely reporting and complete characterisation of the pathogen.

This review of the last 10 years of LabVISE data shows that much valuable data has been accumulated and lessons learnt. With a new focus and commitment, LabVISE will continue to have an important role in public health in Australia.

Appendices

Appendix 1. Classifications of diagnoses for specimens reported to LabVISE, 1991 to 2000

Code	Diagnosis
00	Healthy — no illness
01	Respiratory tract infection — upper
02	Respiratory tract infection — lower
04	Central nervous system (CNS) — paralytic disease
05	CNS diseases — other (e.g. convulsions)
06	Superficial skin/mucous membrane diseases (rash, ulcer etc)
07	Gastrointestinal disease
08	High fever
09	Other than this list
10	Eye disease (e.g. conjunctivitis, keratitis, endophthalmitis)
11	Respiratory tract infection unspecified
12	Otitis media
13	Nervous system disease (unspecified)
14	Endocarditis
15	Trauma
16	Deep skin infection (wound, abscess, cellulitis, cyst)
17	Hepatitis
18	Septicaemia
19	Cardiovascular disease, unspecified
20	Endocarditis (native valve)
21	Endocarditis (prosthetic valve)
22	Otitis externa
23	Septic shock
24	Intra-abdominal infections (e.g. peritonitis, cholecystitis)
29	Bone and/or joint disease (including Bornholm disease)
30	Septic arthritis
31	Osteomyelitis
32	Otitis, unspecified
34	Myocarditis
35	Pericarditis
38	Reticulo-endothelial system disease
39	Glandular disease (salivary, endocrinous glands)
40	Epiglottitis
47	Hepatic disease — other (including jaundice)
48	Colitis
49	Intussusception
50	HIV/AIDS
51	Neutropenia

Appendix 1 (continued). Classifications of diagnoses for specimens reported to LabVISE, 1991 to 2000

Code	Diagnosis
52	Diabetes
53	Injecting drug use
54	Renal failure/haemodialysis
55	Transplant
56	Transplant
57	Immunosuppressed
58	Malignancy
59	Genital disease (including sexually transmitted infections)
60	Infection of female pelvis
61	Pregnant
62	Postnatal
63	Hospitalisation
64	Travel overseas
65	Animal exposure
66	Occupational
68	Pre-term neonate
69	Congenital disease
70	Recent surgery — gastrointestinal
71	Recent surgery — orthopaedic
72	Recent surgery — urinary tract
73	Recent surgery — thoracic
74	Recent surgery — vascular
75	Recent surgery — neurology
79	Recent surgery — other
80	Intravascular device — peripheral IV line
81	Intravascular device — central IV line
83	Intravascular device — artificial heart valve
84	Other prosthetic device
85	Phlebitis
88	Urinary tract infection (instrumentation/catheterisation)
89	Urinary tract disease
90	Blood transfusion
95	Probable contaminant
96	Hospital acquired
97	No data available
99	No clinical information available
A1	Sudden infant death syndrome
AA	Concomitant, but unrelated disease
E3	Encephalitis
G8	Malaise — general and/or mild fever
M3	Meningitis
P8	Pyrexia of unknown origin or severe prolonged fever

Appendix 2. Laboratory reports to LabVISE, 1991 to 2000, by year and organism type

Organism	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Measles, mumps, rubella										
Measles virus	256	204	853	1,199	153	57	67	49	172	44
Mumps virus	32	48	77	87	69	37	40	44	58	49
Rubella virus	246	753	926	1,178	735	716	362	103	145	51
Hepatitis										
Hepatitis A virus	445	371	451	363	424	407	624	384	375	146
Hepatitis D virus	37	45	47	24	23	17	15	7	8	9
Hepatitis E virus	-	1	12	6	8	2	4	1	1	4
Arboviruses										
Ross River virus	833	1,319	1,895	2,240	988	3,249	2,016	676	1,423	1,268
Barmah Forest virus	36	251	208	273	202	232	201	44	180	169
Dengue type 1	13	9	1	-	3	-	-	-	-	-
Dengue type 2	3	297	422	4	1	29	20	-	-	2
Dengue type 3	-	5	2	4	2	2	1	27	3	4
Dengue type 4	1	-	-	1	-	1	-	-	-	-
Dengue not typed	12	74	103	26	19	30	43	43	85	175
Murray Valley encephalitis virus	10	1	9	3	-	-	3	2	2	20
Kunjin virus	14	10	-	2	5	5	6	5	5	4
Japanese encephalitis virus	-	-	-	1	6	-	-	1	1	-
Kokobera virus	1	-	-	-	-	-	-	-	-	-
Stratford virus	3	-	-	-	-	1	-	1	-	-
Flavivirus (unspecified)	29	47	104	23	45	21	21	73	27	40
Adenoviruses										
Adenovirus type 1	91	111	85	48	32	21	29	74	14	8
Adenovirus type 2	142	129	128	45	37	29	39	22	13	7
Adenovirus type 3	88	96	203	57	66	45	22	57	35	18
Adenovirus type 4	23	103	40	2	2	-	7	4	15	5
Adenovirus type 5	31	38	28	12	14	9	8	1	6	8
Adenovirus type 6	9	7	3	1	2	3	-	20	-	3
Adenovirus type 7	8	4	11	16	26	17	8	17	7	8
Adenovirus type 8	38	33	55	55	22	13	3	3	1	3
Adenovirus type 9	11	7	5	3	2	1	-	-	-	-
Adenovirus type 10	4	2	1	-	1	-	1	1	-	-
Adenovirus type 11	29	12	4	1	3	1	-	1	-	-
Adenovirus type 12	-	3	3	-	-	-	-	-	-	-
Adenovirus type 13	1	-	-	-	-	-	-	-	-	-
Adenovirus type 14	1	-	-	-	-	-	-	-	-	-
Adenovirus type 15	-	-	-	-	-	-	-	-	-	1
Adenovirus type 16	2	-	-	-	-	-	-	-	-	-
Adenovirus type 18	1	-	-	-	-	-	-	-	-	-
Adenovirus type 19	6	20	3	-	3	7	-	2	1	7
Adenovirus type 21	1	-	-	-	-	-	-	-	-	-

Appendix 2 (continued). Laboratory reports to LabVISE, 1991 to 2000, by year and organism type

Organism	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Adenoviruses, cont.										
Adenovirus type 22	3	1	1	3	-	-	-	2	-	-
Adenovirus type 24	3	-	-	-	-	-	-	-	-	-
Adenovirus type 26	11	-	2	2	2	1	-	-	-	-
Adenovirus type 27	1	-	-	-	-	-	-	-	-	
Adenovirus type 28	12	1	1	-	-	1	-	-	-	-
Adenovirus type 29	5	-	-	-	-	-	-	-	-	-
Adenovirus type 30	7	2	-	1	2	-	-	-	-	-
Adenovirus type 31	3	-	-	-	-	-	-	-	-	-
Adenovirus type 32	1	-	-	-	-	-	-	-	-	-
Adenovirus type 34	-	2	-	-	-	-	-	-	-	-
Adenovirus type 35	4	1	1	1	1	3	-	-	-	-
Adenovirus type 37	7	3	1	2	2	5	3	5	11	11
Adenovirus type 40	4	6	9	-	-	34	12	21	74	86
Adenovirus type 41	-	-	-	-	-	4	3	-	-	1
Adenovirus type 42	-	-	-	-	1	1	-	-	-	-
Adenovirus type 43	-	-	-	-	-	1	-	-	-	-
Adenovirus type 44	1	-	-	-	-	-	-	-	-	-
Adenovirus type 45	2	-	-	-	-	-	-	-	-	-
Adenovirus type 46	1	3	1	2	2	-	-	-	-	-
Adenovirus type 47	1	2	-	-	-	-	-	-	-	-
Adenovirus not typed/ pending	966	1,136	1,300	1,291	962	1,186	882	932	1,132	1,039
Herpesviruses										
Herpesvirus type 6	3	2	4	6	3	-	6	2	16	6
Cytomegalovirus	1,820	1,728	1,561	1,727	1,404	1,373	1,017	766	1,220	1,312
Varicella-zoster virus	522	684	924	1,062	1,073	1,132	1,252	1,252	1,658	1,494
Epstein-Barr virus	1,361	1,625	1,570	1,516	1,887	2,084	2,151	1,903	2,196	1,926
Other DNA viruses										
Papovavirus group	9	11	1	4	11	1	2	3	12	7
Cowpox virus	1	-	-	-	-	-	-	-	-	-
Molluscum contagiosum	21	10	8	4	3	7	5	2	15	11
Contagious pustular dermatitis (Orf virus)	5	7	4	2	1	1	9	6	8	7
Poxvirus group not typed	2	-	10	2	4	5	3	-	2	-
Parvovirus	29	178	86	109	102	268	291	261	437	389
Picornaviruses										
Coxsackie A										
Coxsackievirus A2	2	-	-	-	-	-	-	-	-	-
Coxsackievirus A7	-	-	-	-	-	1	-	-	-	-
Coxsackievirus A9	45	19	62	2	9	9	5	8	10	11
Coxsackievirus A10	-	-	-	-	1	-	-	1	-	-
Coxsackievirus A16	9	21	18	34	1	12	5	3	15	8
Coxsackievirus A21	-	-	1	-	-	-	-	-	-	-
Coxsackievirus A untyped/pending	2	3	1	-	1	-	-	-	-	-

Appendix 2 (continued). Laboratory reports to LabVISE, 1991 to 2000, by year and organism type

Organism	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Picornaviruses, cont.										
Coxsackie B										
Coxsackievirus B1	6	57	51	4	-	-	1		1	4
Coxsackievirus B2	34	2	8	23	4	20	6	1	10	6
Coxsackievirus B3	13	6	15	31	11	3	14	2	-	-
Coxsackievirus B4	61	4	11	12	2	8	5	6	3	16
Coxsackievirus B5	40	65	26	15	4	15	1	7	7	5
Coxsackievirus B6	-	1	1	2	-	-	-	1	-	-
Coxsackievirus B untyped/pending	1	1	1	-	-	3	-	1	-	1
Echoviruses										
Echovirus type 1	4	1	-	-	3	-	-	1	-	3
Echovirus type 2	-	1	-	-	-	-	1	1	1	-
Echovirus type 3	-	-	-	27	16	-	-	-	5	2
Echovirus type 4	2	14	-	-	1	1	1	5	1	-
Echovirus type 5	3	2	5	1	-	4	4	5	1	-
Echovirus type 6	9	85	10	107	12	1	1	5	16	1
Echovirus type 7	4	39	74	-	2	24	4	-	1	33
Echovirus type 8	-	2	-	-	-	-	-	-	-	-
Echovirus type 9	5	216	22	7	41	4	1	22	32	5
Echovirus type 11	10	13	141	26	4	1	5	50	166	7
Echovirus type 12	-	-	1	-	-	-	-	-	-	-
Echovirus type 13	-	-	-	-	1	-	-	-	-	-
Echovirus type 14	7	15	23	12	35	4	-	1	-	1
Echovirus type 15	1	-	3	-	1	1	-	-	-	-
Echovirus type 16	14	9	-	-	-	-	-	-	-	-
Echovirus type 17	59	38	4	-	-	-	-	2	-	-
Echovirus type 18	3	3	1	2	2	1	-	15	-	1
Echovirus type 19	-	2	1	-	-	-	-	-	1	-
Echovirus type 20	-	3	-	-	-	-	-	-	-	-
Echovirus type 21	2	4	4	-	-	-	-	-	-	-
Echovirus type 22	19	13	9	9	13	1	1	14	12	8
Echovirus type 23	-	-	-	1	1	-	-	-	-	-
Echovirus type 24	2	1	-	-	3	-	-	-	-	-
Echovirus type 25	2	31	5	1	3	-	-	-	3	-
Echovirus type 30	1	3	198	247	28	3	1	26	17	121
Echovirus type 31	1	1	-	-	1	-	1	-	1	2
Echovirus type 32	2	-	-	-	-	-	-	-	-	-
Echovirus type 33	1	-	-	-	1	1	1	-	4	4
Echovirus type 34	1	-	-	-	-	-	1	-	-	-
Echovirus not typed/ pending	3	1	1	3	38	3	3	2	3	5

Appendix 2 (continued). Laboratory reports to LabVISE, 1991 to 2000, by year and organism type

Organism	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Picornaviruses, cont.										
Polioviruses										
Poliovirus type 1 (uncharacterised)	40	71	43	41	27	15	9	12	26	22
Poliovirus type 2 (uncharacterised)	54	45	35	41	29	14	9	25	16	8
Poliovirus type 3 (uncharacterised)	26	32	31	14	13	1	4	6	8	8
Poliovirus type 1 (vaccine strain)	-	-	-	-	-	1	-	1	-	-
Poliovirus type 2 (vaccine strain)	-	-	-	-	-	2	2	-	1	-
Poliovirus type 3 (vaccine strain)	-	-	-	-	-	-	-	-	-	1
Poliovirus not typed/ pending	74	38	12	10	2	2	-	-	1	1
Rhinovirus (all types)	653	683	868	905	650	662	549	420	501	420
Other enteroviruses										
Enterovirus type 71 (BCR)	13	15	1	-	34	-	-	9	15	6
Enterovirus not typed/ pending	673	781	943	1,101	891	742	484	538	753	815
Picorna virus not typed	1	-	-	-	-	-	-	-	-	2
Ortho/paramyxoviruses										
Influenza										
Influenza A virus	54	1,144	512	1,122	696	1,571	1,350	2,744	1,898	1,499
Influenza A virus H1N1	5	8	-	-	92	-	1	-	1	-
Influenza A virus H3N2	1	170	32	74	9	70	96	2	33	7
Influenza B virus	408	126	648	87	355	79	903	149	279	580
Influenza C virus	1	-	-	-	-	-	-	-	-	-
Influenza virus — typing pending	2	1	4	8	2	19	448	2	-	-
Parainfluenza										
Parainfluenza virus type 1	47	281	44	548	32	315	61	276	44	230
Parainfluenza virus type 2	143	60	127	61	178	73	112	30	114	36
Parainfluenza virus type 3	556	554	513	526	833	730	962	409	803	516
Parainfluenza virus type 4	-	-	-	-	2	7	-	3	2	-
Parainfluenza virus typing pending	59	80	46	68	36	32	239	5	1	1
Respiratory syncytial virus	2,555	3,556	3,506	3,749	3,889	4,068	4,588	4,641	3,059	2,735
Other RNA viruses										
Paramyxovirus (unspecified)	3	1		1	5	28	22	-	4	-
HTLV–1	8	2	13	1	4	10	17	15	12	9
Rotavirus	2,642	2,134	1,989	2,275	1,617	1,492	1,431	1,372	2,245	1,771
Astrovirus	21	14	4	1	6	3	4	10	3	-
Reovirus (unspecified)	8	12	7	3	-	-	-	-	2	2
Calicivirus	37	19	12	6	1	6	-	1	1	-
Norwalk agent	21	6	21	11	48	59	67	44	59	82
Coronavirus	36	26	11	2	1	-	-	-	-	-
Small virus (like) particle	59	64	33	33	16	12	3	2	-	-

Appendix 2 (continued). Laboratory reports to LabVISE, 1991 to 2000, by year and organism type

Organism	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Non-viral pathogens										
Chlamydia										
Chlamydia trachomatis – A–K	59	11	-	1	1	1	1	-	1	-
Chlamydia trachomatis — L1–L3	18	-	-	-	-	-	1	-	-	-
Chlamydia trachomatis (not typed)	2,615	2,563	2,835	2,178	2,579	3,803	3,980	3,158	3,295	3,154
Chlamydia pneumoniae	2	14	1	-	2	1	3	-	2	36
Chlamydia psittaci	139	97	74	114	176	62	51	70	78	102
Chlamydia spp. (typing pending)	1	10	9	10	6	1	7	-	1	-
Chlamydia species	-	6	18	62	75	54	28	57	21	8
Mycoplasma										
Mycoplasma pneumoniae	381	1,580	1,760	820	334	1,009	1,640	1,285	1,125	686
Mycoplasma hominis	2	4	-	2	-	2	-	2	5	8
Rickettsia										
Coxiella burnetii (Q fever)	240	270	552	345	167	208	259	137	221	101
Rickettsia prowazeki	-	-	1	-	-	-	-	-	-	2
Rickettsia australis	-	8	3	3	24	18	10	2	2	2
Rickettsia tsutsugamushi	-	-	-	-	6	13	26	-	2	11
Rickettsia — spotted fever group	-	21	-	-	2	-	-	-	1	44
Rickettsia species — other	1	2	11	6	7	5	7	8	13	12
Gram positive bacteria										
Streptococcus group A	-	73	292	340	553	154	-	-	368	348
Streptococcus group B	-	5	-	-	-	-	-	-	-	-
Gram negative bacteria										
Yersinia enterocolitica	-	5	5	34	31	-	-	1	10	15
Brucella abortus	-	-	-	13	1	-	-	-	-	1
Brucella species	-	15	3	8	7	3	-	-	11	5
Bordetella pertussis	-	20	348	620	608	943	1,801	770	845	689
Bordetella parapertussis	-	-	1	3	-	-	-	-	-	1
Bordetella species	-	73	264	159	261	244	2	-	-	-
Legionella pneumophila	-	1	-	3	5	5	18	8	17	44
Legionella longbeachae	-	1	3	5	22	20	31	37	51	59
Legionella species	-	3	8	27	11	14	10	-	-	5
Cryptococcus										
Cryptococcus species	-	13	30	22	18	13	20	7	9	18
Spirochetes										
Leptospira interrogans	-	-	-	-	-	2	-	-	-	-
Leptospira canicola	-	1	2	1	-	2	1	-	-	-
Leptospira icterohaemorrhagiae	-	2	4	2	-	-	-	-	-	-
Leptospira pomona	-	5	7	6	4	10	7	-	-	-

Appendix 2 (continued). Laboratory reports to LabVISE, 1991 to 2000, by year and organism type

Organism	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Spirochetes, continued										
Leptospira autumnalis	-	-	-	1	-	-	-	-	-	-
Leptospira grippotyphosa	-	-	-	1	-	2	-	-	-	-
Leptospira hardjo	-	8	20	25	9	25	8	3	1	-
Leptospira australis	-	2	4	5	3	7	5	-	-	-
Leptospira species	-	10	22	45	26	58	1	-	55	63
Treponema pallidum	-	267	547	431	452	102	1	1	774	909
Protozoa										
Entamoeba histolytica	-	3	9	7	20	8	-	-	7	17
Toxoplasma gondii	-	35	48	84	107	7	1	1	9	16
Helminths										
Echinococcus granulosus	-	7	23	23	11	2	-	-	4	18

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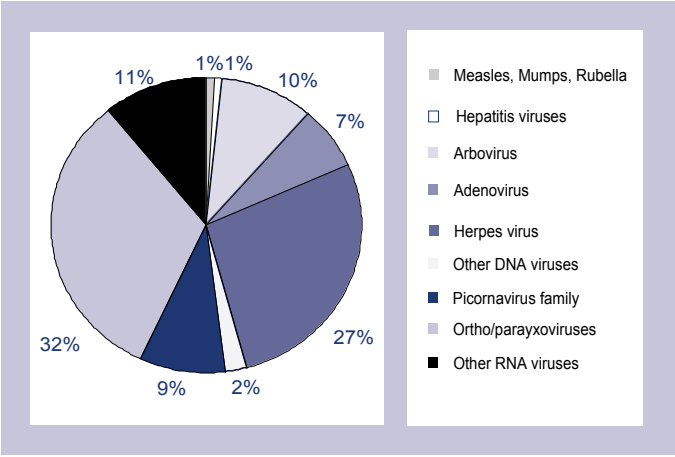
Errata

The following corrections to *Communicable Diseases Intelligence* Vol 26 No 2 should be noted.

Page 187: Figure 55. Labvise reports, Australia, 2000.
The last point in the legend should read 'Other RNA viruses' instead of 'Ortho/paramyxoviruses'. The correct figure is reproduced below.

Figure 55. Labvise reports, Australia, 2000



Page 265. 'Evaluation of the Australian CJD Surveillance System' by Monica Robotin.
Under the title heading should read 'Monica Robotin[1,2]' instead of 'Monica Robotin[1]'

Page 303: Table 4. Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 January to 31 March 2002, and total reports for the year[2].
The last four column headings are incorrect. The column headings should read as follows:

'This period 2002'; 'This period 2001'; 'Year to date 2002' and 'Year to date 2001'

Enhancing foodborne disease surveillance across Australia in 2001: the OzFoodNet Working Group

In alphabetical order: Rosie Ashbolt,[1] Rod Givney,[2] Joy E Gregory,[3] Gillian Hall,[4] Rebecca Hundy,[2] Martyn Kirk,[5] Ian McKay,[6] Lynn Meuleners,[7] Geoff Millard,[8] Jane Raupach,[2] Paul Roche,[6] Nittita Prasopa-Plaizier,[3] Mohinder K Sarna,[7] Russell Stafford,[9] Nola Tomaska,[4] Leanne Unicomb,[10] Craig Williams,[5] the OzFoodNet Working Group

Abstract

In 2000, the OzFoodNet network was established to enhance surveillance of foodborne diseases across Australia. OzFoodNet consists of 7 sites and covers 68 per cent of Australia's population. During 2001, sites reported 15,815 cases of campylobacteriosis, 6,607 cases of salmonellosis, 326 cases of shigellosis, 71 cases of yersiniosis, 61 cases of listeriosis, 47 cases of shiga-toxin producing *E. coli* and 5 cases of haemolytic uraemic syndrome. Sites reported 86 foodborne outbreaks affecting 1,768 people, of whom 4.0 per cent (70/1,768) were hospitalised and one person died. There was a wide range of foods implicated in these outbreaks and the most common agent was *S.* Typhimurium. Sites reported two international outbreaks; one of multi-drug resistant *S.* Typhimurium Definitive Type 104 due to helva imported from Turkey, and one of *S.* Stanley associated with dried peanuts from China. The National Centre for Epidemiology and Population Health conducted a national survey of gastroenteritis. Preliminary data from interviews of 2,417 people suggests that the incidence of foodborne illness is significantly higher than previously thought. OzFoodNet initiated case control studies into risk factors for *Campylobacter*, *Salmonella*, *Listeria*, and shiga-toxin producing *E. coli*. OzFoodNet developed a foodborne disease outbreak register for Australia; established a network of laboratories to type *Campylobacter*; prepared a survey of pathology laboratories; reviewed Australian data on listeriosis; and assessed the usefulness of sentinel surveillance for gastroenteritis. This program of enhanced surveillance has demonstrated its capacity to nationally investigate and determine the causes of foodborne disease. *Commun Dis Intell* 2002;26:375-406.

Keywords: foodborne disease, surveillance, Salmonella *Typhimurium*, Listeria, Campylobacter, *gastroenteritis*

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Introduction

In 2000, the Commonwealth Department of Health and Ageing (DoHA) established a collaborative network, coined OzFoodNet, to enhance the existing surveillance mechanisms for foodborne disease. This was one of a number of projects that the department established to build a strong base for national policy development in the area of food safety.[1]

The OzFoodNet initiative built upon the experience of an 18-month trial of active foodborne disease surveillance in the Hunter region of New South Wales.[2] This pilot was modelled on the FoodNet system of active surveillance in the United States of Amercia (USA), and provided much insight into establishing OzFoodNet (see http://www.cdc.gov/foodnet/).[3]

Mission and aims

The mission of OzFoodNet is to apply a concentrated effort at a national level to investigate and understand foodborne disease; to describe more effectively its epidemiology and to provide better evidence on how to minimise foodborne illness in Australia.

OzFoodNet aims to:

- estimate the incidence and cost of foodborne illness in Australia;
- improve our understanding of the epidemiology of foodborne disease, by enhancing surveillance and conducting special studies on foodborne pathogens;
- identify inappropriate practices in domestic and commercial settings which lead to food contamination and foodborne illness;
- assess the efficacy of current and proposed food hygiene standards and their enforcement by jurisdictions;
- provide data essential for future risk assessments and policy interventions; and
- train people to investigate foodborne illness.

The work of OzFoodNet will improve surveillance of foodborne disease across Australia, but many of these goals may only be realised in years to come.

Organisation

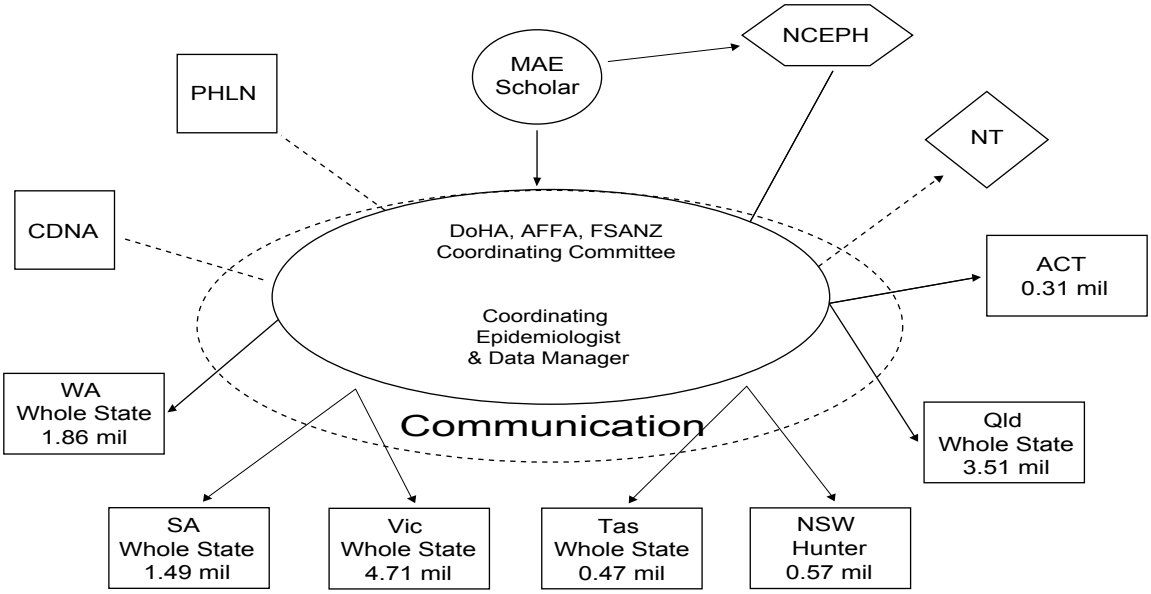
OzFoodNet involves many different agencies and has required a major collaborative effort to establish (Figure 1). The Commonwealth Department of Health and Ageing provides funding and strategic management for the OzFoodNet program of work. The department convenes a regular management group that includes senior managers from Food Standards Australia New Zealand (FSANZ) (formerly the Australian New Zealand Food Authority) and the Commonwealth Department of Agriculture, Fisheries and Forestry — Australia. Australia's peak body for communicable disease control, the Communicable Diseases Network Australia (CDNA), oversees the work of OzFoodNet.

In 2000, DoHA provided funding for the six Australian States and the Australian Capital Territory to participate in OzFoodNet. Each of the seven funded jurisdictions has employed one or more epidemiologists to participate in OzFoodNet. These epidemiologists report to the jurisdiction's manager of communicable disease surveillance. Each epidemiologist conducts work that is locally or nationally important for prevention of foodborne diseases. The work program includes a mixture of surveillance, outbreak investigation, and applied research.

A coordinating epidemiologist and a data manager were employed to ensure that the work is conducted efficiently and consistently. Site epidemiologists provide regular reports of foodborne disease incidence to the coordinating epidemiologist, who is the OzFoodNet representative on CDNA.

Every 3 months, the OzFoodNet epidemiologists and a wider group meet to discuss surveillance and control of foodborne diseases and the progress of applied research studies (Figure 2). This wider group forms the basis of the OzFoodNet Working Group, which includes partners from state and territory health departments, the National Centre for Epidemiology and Population Health (NCEPH), the Public Health Laboratory Network (PHLN), and federal government agencies. OzFoodNet also communicates regularly through monthly teleconferences, and a list server.

Figure 1. Outline of OzFoodNet sites showing population covered (in millions) and relationship to other bodies



NCEPH – National Centre for Epidemiology and Population Health
MAE – Master of Applied Epidemiology (Field Epidemiology Training Program)
PHLN – Public Health Laboratory Network
CDNA – Communicable Diseases Network Australia
DoHA – Department of Health and Ageing
FSANZ – Food Standards Australia New Zealand
AFFA – Department of Agriculture, Fisheries and Forestry — Australia

Figure 2. Participants at the OzFoodNet face-to-face meeting in Hobart, September 2001

Pictured from left: Gill Hall (NCEPH), Jane Raupach (SA), Donna Cassoni (DoHA), Martyn Kirk (FSANZ), Nittita Prasopa-Plaizier (Vic), Rebecca Hundy (SA), Leanne Unicomb (Hunter Health Area, NSW), Vanessa Madden (Tasmania), Luba Tomaska (FSANZ), Lynne Meuleners (WA), Russell Stafford (Qld), Geoff Millard (ACT), Joy Gregory (Vic)

OzFoodNet currently covers a population of 12.9 million people, or 68 per cent of Australia's population. The states of Queensland, Tasmania, South Australia, Victoria and Western Australia enhanced their surveillance for foodborne disease across the whole state. In New South Wales, the health department enhanced foodborne disease surveillance in the Newcastle region which is covered by the Hunter Health Area. The Australian Capital Territory joined the Network in July 2001 and the Northern Territory participated in OzFoodNet as an observer during 2001.

OzFoodNet reports to the management group on a quarterly basis. The Department of Health and Ageing uses the data and findings to feed into national committees formulating policy, such as the Food Regulation Standing Committee, the Development and Implementation Sub-Committee and the Technical Advisory Group.

Scope of this report

This first annual report for OzFoodNet synthesises the work and reports of all site epidemiologists for 2001. The report details:

- the incidence of foodborne disease across Australia;
- information on risk factors for foodborne illness;
- ways of improving surveillance for foodborne disease;
- the status of the OzFoodNet projects across Australia;
- outcomes from OzFoodNet activities during 2001; and
- recommendations arising from the work of OzFoodNet.

Incidence of foodborne disease

National foodborne disease incidence

This section documents trends in the incidence of enteric diseases in OzFoodNet sites. OzFoodNet epidemiologists provide regular summaries of foodborne disease incidence from notifiable disease datasets. The OzFoodNet data on sporadic disease are a subset of the information reported to the National Notifiable Diseases Surveillance System (NNDSS), but are more detailed and allow interpretation at the state, territory or public health unit level. NNDSS annual reports should be consulted for national notification rates of foodborne diseases. OzFoodNet provides a national picture by recording details of outbreaks and clusters occurring across jurisdictional boundaries. Improved communication and cross-jurisdictional investigations provide important information about the food handling practices that have led to food contamination and the causes of foodborne disease.

Interpreting the data

It is important to recognise that subtle differences between the three sources of data used in this report, OzFoodnet, NNDSS and the National Enteric Pathogens Surveillance System (NEPSS) can make interpretation difficult. Some of the inherent limitations of the data include:

- Data in the surveillance systems may come from different information sources, e.g. the proportion of notifications received from medical practitioners varies from jurisdiction to jurisdiction.
- Each surveillance system will have different delays in receipt and processing of reports, which can affect the total number reported in any time period.
- Where the surveillance data are reported, the reporting date is often different, e.g. sometimes the 'date of onset of symptoms' is used, while at other times reports will relate to the 'date of specimen collection', or the 'date of receipt of notification'. In this report, the 'date of receipt of notification' is also used, except for historical comparisons where we use the 'date of onset'.

Managers of the various surveillance schemes may still be cleaning data at the time of reporting. This cleaning will involve checking for accuracy of information on the database, and removing duplicate entries.

The data reported usually reflect a complex mix of biases that are inherent in public health surveillance. One bias that particularly affects surveillance data is ascertainment bias, i.e. some groups of the population are more likely to be detected as cases by the surveillance system. In notifiable disease datasets it is common to have an over representation of younger children, people who are elderly or immunocompromised, and people who are severely affected by the illness. This is usually because these patients are more likely to seek medical attention, and doctors are more likely to conduct tests on these patients. The data are rarely representative of the true burden of infection in the community, or the gradient of symptoms associated with infection.

The states and territories have differing approaches to surveillance, which may be reflected in the data. This also impacts the way that different jurisdictions choose to report data, such as outbreaks or clusters.

Some diseases are not notifiable in certain jurisdictions, e.g. individual cases of *Campylobacter* infection are not notifiable in New South Wales.

OzFoodNet data

OzFoodNet reports surveillance data for several bacterial pathogens and summary information from outbreaks potentially related to food and water. In this report, data are reported by the date of receipt of notifications at the health agency, unless specified. Historical comparisons use date of onset of symptoms for comparative purposes. Summary data for OzFoodNet sites on notified cases are shown in Appendix 1.

The Hunter OzFoodNet site supplied data for all of New South Wales. These data were used where possible for reporting total figures. Data for the Northern Territory are not reported unless specified.

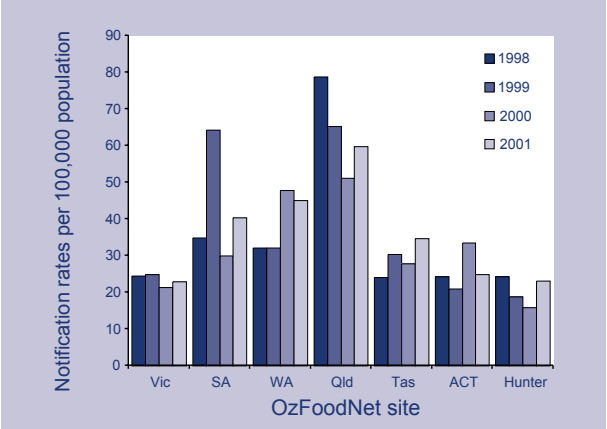
Rates were calculated using the Australian Bureau of Statistics estimated resident populations for 2001. Where appropriate, we directly standardised regional rates of disease within jurisdictions by age to estimated resident population for Australia, 2000.

Salmonella

In 2001, OzFoodNet sites reported 6,607 cases of *Salmonella* infection, which represented an increase of 2.1 per cent over the mean of the previous 3 years.* The overall rate of *Salmonella* notification in OzFoodNet sites was 34.1 cases per 100,000 population, and ranged from 23.1 cases per 100,000 population in the Hunter region to 59.8 cases per 100,000 population in Queensland (Figure 3).

Overall, notification rates of salmonellosis for 2001 were increased in the states of Tasmania (26.2%), Western Australia (20.7%) and the Hunter Health Area, New South Wales (17.1%) when compared with the 3-year mean rates for 1998–2000. There were moderate declines in the number of notifications of *Salmonella* in Queensland (-12.4%), South Australia (-6.3%), the Australian Capital Territory (-5.5%), and in Victoria (-2.8%) from the 3-year mean values.

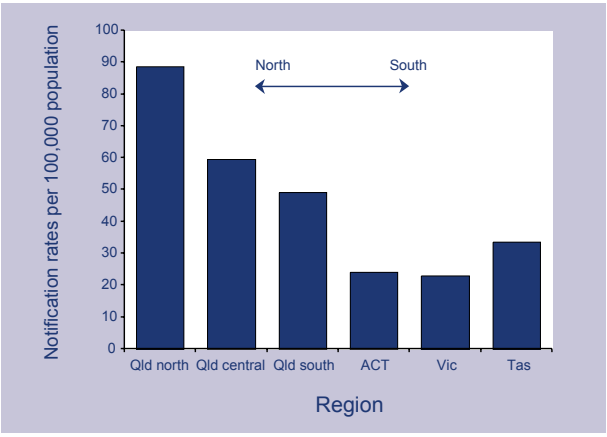
Figure 3. Crude notification rates of salmonellosis, 1998 to 2001, by site and year



OzFoodNet sites reported that the ratio of males to females was approximately 1:1, and ranged from 1.2:1 in Victoria to 0.9:1 in Tasmania. The median age of cases ranged between 18–21 years at all OzFoodNet sites, except for Queensland where the median age was 9 years. There were no major changes in the median ages of salmonellosis cases from 2001 to 2000.

Rates of salmonellosis are highest in northern areas of Australia, with the highest rates in the Kimberley region.[4] Western Australia reported that the Kimberly region had a rate of 559 cases per 100,000 population. OzFoodNet sites reported that notification rates increased from south to north along eastern Australia (Figure 4).

Figure 4. Standardised rates of *Salmonella* notifications in OzFoodNet regions in eastern Australia, 2001, by date of notification†



† Notifications were analysed by date of receipt at the health department. Rates were directly standardised to the Australian Bureau of Statistics estimated resident population for Australia in 2000.

* In this report, historical comparisons use date of onset of patient's symtoms or nearest equivalent for analysis.

Table 1. Numbers, rates and proportions of top five *Salmonella* infections, 2000 to 2001, by site

OzFoodNet site	Top five *Salmonella* infections					
	***Salmonella* type (serovar & phage type)**	**2001**	**Rate 2001***	**Proportion (%)†**	**2000**	**Ratio‡**
ACT	Typhimurium 9	10	3.2	12.8	31	0.3
	Stanley	5	1.6	6.4	1	5.0
	Bovismorbificans 14	4	1.3	5.1	0	–
	Paratyphi B bv Java Dundee	2	0.6	2.6	1	2.0
	Enteritidis RDNC 11	2	0.6	2.6	0	
Hunter	Typhimurium 135	15	2.8	12.2	10	1.5
	Typhimurium 126	9	1.7	7.3	3	3.0
	Typhimurium 64	9	1.7	7.3	14	0.6
	Birkenhead	5	0.9	4.1	9	0.6
	Typhimurium U290	3	0.6	2.4	0	–
New South Wales	Typhimurium 135	202	3.1	11.9	115	1.8
	Typhimurium 9	133	2.0	7.8	138	1.0
	Typhimurium 126	98	1.5	5.8	56	1.8
	Birkenhead	87	1.3	5.1	73	1.2
	Infantis	41	0.6	2.4	25	1.6
Queensland	Virchow 8	177	4.9	8.2	189	0.9
	Saintpaul	164	4.5	7.6	184	0.9
	Typhimurium 135	137	3.8	6.3	118	1.2
	Birkenhead	130	3.6	6.0	102	1.3
	Aberdeen	81	2.2	3.7	52	1.6
South Australia	Typhimurium 126	110	7.3	18.0	5	22.0
	Typhimurium 9	49	3.3	8.0	26	1.9
	Typhimurium 108	31	2.1	5.1	11	2.8
	Typhimurium 64 var	21	1.4	3.4	0	–
	Infantis	19	1.3	3.1	8	2.4
Tasmania	Mississippi	98	20.8	59.0	73	1.3
	Typhimurium 9	11	2.3	6.6	22	0.5
	Typhimurium 135	5	1.0	3.0	5	1.0
	Infantis	3	0.6	1.8	4	0.8
	Saintpaul	2	0.4	1.2	2	1.0
Victoria	Typhimurium 9	127	2.6	11.4	186	0.7
	Typhimurium 135	96	2.0	8.6	70	1.4
	Typhimurium 4	79	1.6	7.1	37	2.1
	Typhimurium 170	73	1.5	6.5	36	2.0
	Virchow 34	35	0.7	3.1	60	0.6
Western Australia	Typhimurium 135	80	4.2	9.0	68	1.2
	Saintpaul	45	2.4	5.1	42	1.1
	Chester	31	1.6	3.5	12	2.6
	Muenchen	23	1.2	2.6	29	0.8
	Stanley	21	1.1	2.4	5	4.2

* Rate per 100,000 population

† Proportion of total *Salmonella* notified for this jurisdiction.

‡ Ratio of the number of reported cases in 2001 compared with 2000.

During 2001, there were 520 notifications of *Salmonella* Typhimurium phage type 135 to OzFoodNet sites (including New South Wales) making it the most common infection (Table 1). There were 330 notifications of *Salmonella* Typhimurium phage type 9, which has been a common phage type for many years. South Australia recorded the emergence of *Salmonella* Typhimurium phage type 126, which had previously been rare in this state. The incidence of this phage type also increased in other Australian jurisdictions during 2001, particularly New South Wales and Queensland.

Certain *Salmonella* serovars were localised to specific geographical areas in Australia. During 2001, *Salmonella* Birkenhead was the fourth most common serovar for both New South Wales and Queensland. This elevated notification rate relates to an endemic focus of *Salmonella* Birkenhead in northern New South Wales and south-eastern Queensland. In Tasmania, 59 per cent (98/166) of *Salmonella* reports were the Mississippi serovar, which is rarely reported anywhere else in Australia. The notification rate for *Salmonella* Mississippi in Tasmania was 20.8 cases per 100,000 population, which was the highest specific rate of any serovar in OzFoodNet sites.

During 2001, NEPSS recorded 6,912 cases of *Salmonella* and documented specific epidemiological changes. The most notable of these changes was the emergence of *Salmonella* Typhimurium 126 across Australia. NEPSS also detected increases in *Salmonella* Stanley, *Salmonella* Typhimurium 170, *Salmonella* Typhimurium DT 104, and other serovars. NEPSS collaborated with state and territory health departments and OzFoodNet on a regular basis and participated in several joint investigations and routine teleconferences. NEPSS is a valuable Australian resource due to the data they collect on human and non-human sources of *Salmonella* and other enteric pathogens.

The rates of salmonellosis in Australia are higher than in the United States of America, but lower than in New Zealand (personal communication, Michael Baker, ESR, New Zealand, 1 August 2002).[3] In 2001, the FoodNet active surveillance system in the United States of America reported an incidence of 15.1 cases per 100,000 population compared to 64.7 cases per 100,000 population in New Zealand. It is difficult to compare the true incidence between countries due to the different healthcare systems and cultural settings.

Campylobacter

In 2001, OzFoodNet sites reported 15,815 cases of *Campylobacter* infection, which equated to a rate of 125 cases per 100,000 population however, data was not available for New South Wales, and the Hunter Health Area, New South Wales. This notification rate represented a 20.6 per cent increase over the mean for the previous 3 years. The increase was consistently observed in each quarter of 2001, with the highest rates in spring.

Rates of campylobacteriosis increased in all sites, ranging from 3.4 per cent in Queensland to 63.2 per cent in Western Australia (Figure 5). The increased rate in Western Australia is partly attributable to the introduction of voluntary laboratory notifications in 2000 for the first time. Geographically, the rates of *Campylobacter* infection were higher in southern parts of Australia in contrast to the rates observed for *Salmonella* infections (Figure 6). The north south trend was less marked for *Campylobacter* infections, but this phenomenon has been observed in other countries.[5] The highest rate of *Campylobacter* infection was 174 notifications per 100,000 population in South Australia.

Figure 5. Crude notification rates of *Campylobacter* infection, 1998 to 2001, by site and year

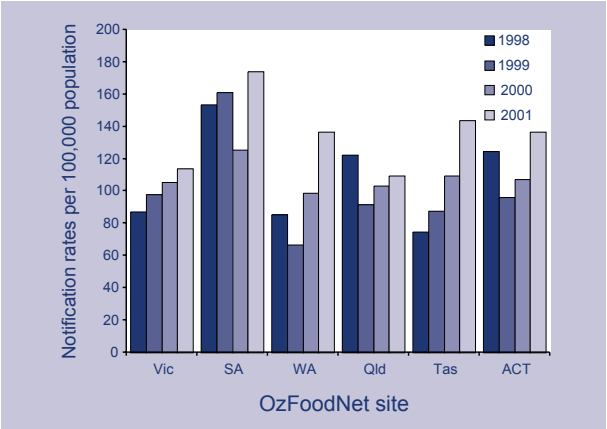
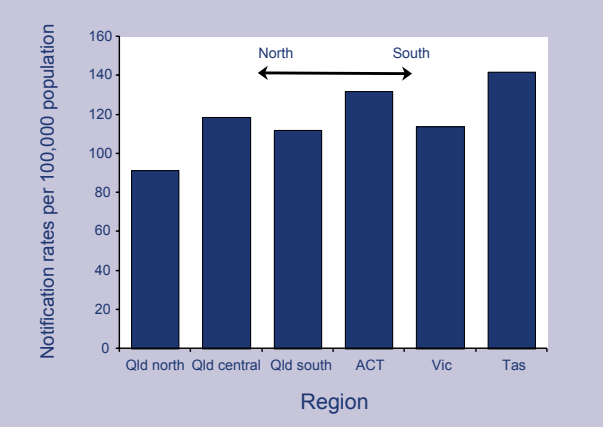


Figure 6. Standardised rates of *Campylobacter* notifications in OzFoodNet regions in eastern Australia, 2001, by date of notification*



* Notifications were analysed by date of receipt at the health department. Rates were directly standardised to the Australian Bureau of Statistics estimated resident population for Australia in 2000.

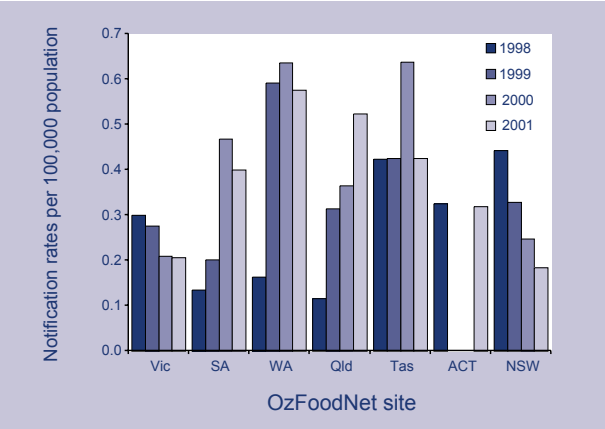
Sites reported a slight predominance of males (range of male to female ratio: 1:2–1.3:1) amongst notified cases. The median age of cases ranged from 26 to 32 years. Six outbreaks were reported due to *Campylobacter* in 2001. Apart from an overall increase in rates, OzFoodNet sites did not record any significant changes in the epidemiology of *Campylobacter* infections from 2001 to 2000.

There are substantial differences in rates of campylobacteriosis between countries. The rate in USA FoodNet sites is 13.8 cases per 100,000 population and 271.5 cases per 100,000 population in New Zealand (personal communication, Michael Baker, ESR, New Zealand, 1 August 2002).[3] It is difficult to determine whether these represent true differences in community incidence of the disease, or relate more to healthcare access, laboratory testing procedures and surveillance modalities.

Listeria

OzFoodNet sites reported 61 cases of listeriosis in 2001, which represents a notification rate of 0.3 cases per 100,000 population. This was an increase of 4 per cent compared to the mean of the previous 3 years. Western Australia had the highest notification rate amongst OzFoodNet sites and the incidence increased in Queensland over the last 3 years (Figure 7).

Figure 7. Crude notification rates of *Listeria* infections in OzFoodNet sites, 1998 to 2001, by site and year



The majority of cases during 2001 were reported in elderly people who were immunocompromised. OzFoodNet sites reported that the median age of non-pregnancy associated cases ranged from 60 to 86 years. Thirteen per cent (7/54) of non-pregnancy associated cases died. In Queensland, the outcome of 47 per cent (8/17) cases was unknown. Sites reported six maternal foetal infections during 2001, which equated to a rate of 2.3 cases per 100,000 births. (Births data from AIHW National Perinatal Statistics Unit for 1999 and includes live births and foetal deaths.[6]) The foetus or neonate died in three of these cases, giving a neonatal mortality rate of 50 per cent.

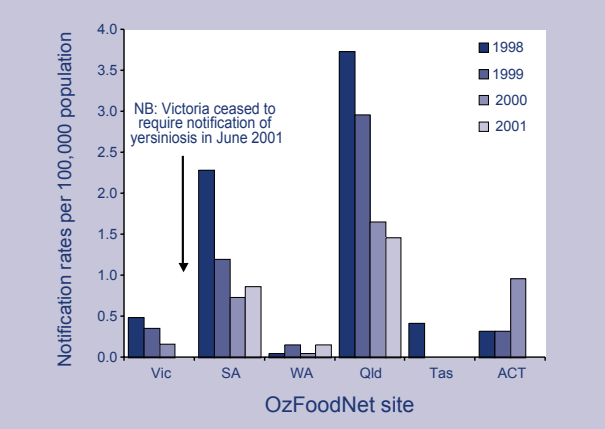
Yersinia

The CDNA agreed to stop reporting notifications of *Yersinia* infections to the NNDSS as of January 2001. The main reasons for this were the apparent decline in incidence and the absence of identified outbreaks. In May 2001, the Victorian Government revised regulations governing reporting of infectious diseases, at which time they removed yersiniosis from the list of reportable conditions. Currently, no other Australian jurisdiction has amended legislation to remove yersiniosis from lists of reportable conditions.

In 2001, OzFoodNet sites reported 71 cases of yersiniosis, which equated to a rate of 0.6 cases per 100,000 population. The overall rate was 50 per cent of the mean of the previous 3 years. The reasons for this decline in yersiniosis are unclear, but follow similar trends in other countries. Queensland reported 75 per cent (53/71) of all cases and had the highest rate of 1.5 cases per

100,000 population (Figure 8). The rates of yersiniosis were similar in all 3 Queensland Health zones, and ranged from 1.1 cases per 100,000 population in the Central zone to 1.9 cases per 100,000 population in the Northern zone.

Figure 8. Crude notification rates of yersiniosis, 1998 to 2001, by site and year



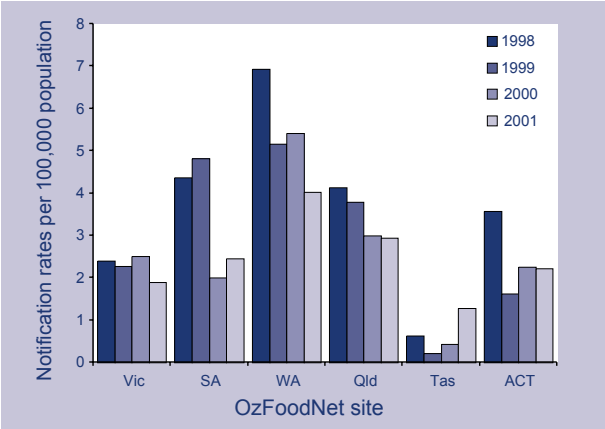
The median age of cases of yersiniosis ranged from 6 to 26 years in different sites. In the two jurisdictions with the majority of cases, South Australia and Queensland, males were more common than females with a male to female ratio of 1.8:1 and 1.5:1 respectively.

The decrease in *Yersinia* notifications may be due to changes in laboratory testing practices rather than a true decline in incidence. Despite the declining rates of this disease, it is important for health agencies to continue surveillance for yersiniosis. The rates of yersiniosis in neighbouring New Zealand are 11.5 per 100,000 population, which is significantly higher than Australia (personal communication, Michael Baker, ESR, New Zealand, 1 August 2002).

Shigella

OzFoodNet sites reported 326 cases of shigellosis during 2001, which equated to a notification rate of 2.6 cases per 100,000 population. The rate of notification decreased by 23 per cent from the mean of the previous 3 years and only Tasmania observed an increase in the 3-year period (Figure 9). The median ages ranged from 20–43 years. Males were more commonly reported from all sites, except for Tasmania and Western Australia. There were no reported outbreaks of shigellosis or confirmed links with food. In Australia, the majority of shigellosis infections are thought to be due to person-to-person transmission, or are acquired overseas.

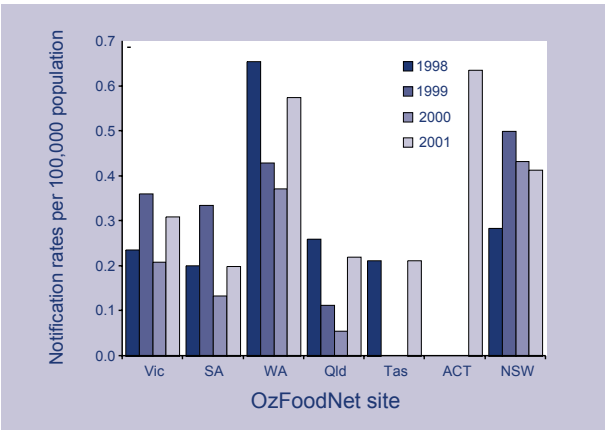
Figure 9. Crude notification rates of shigellosis, 1998 to 2001, by site and year



Typhoid

OzFoodNet sites reported 67 cases of typhoid infection during 2001. This represents an overall notification rate of 0.3 cases per 100,000 population, which was similar to previous years. The highest rates were reported in Western Australia (Figure 10). Where travel status was known, sites reported that 92.5 per cent (37/40) of cases of typhoid had recently travelled overseas. Fifty-five per cent (22/40) of these cases had recently returned from Indonesia.

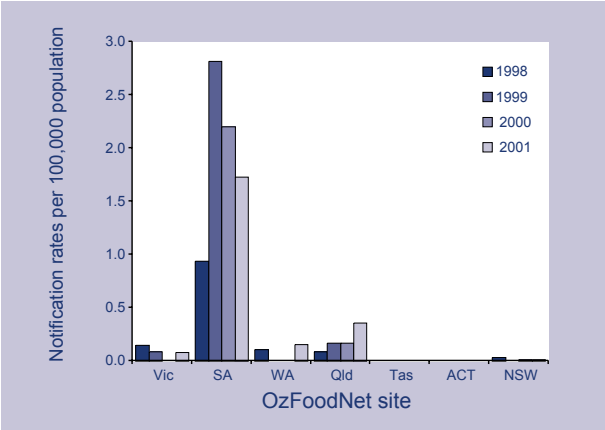
Figure 10. Crude notification rates of typhoid, 1998 to 2001, by site and year



Shiga-toxin producing *E. Coli*

OzFoodNet sites reported 47 cases of shiga-toxin producing *E. coli* (STEC) infection during 2001. The notification rate of 0.2 cases per 100,000 population was a 15 per cent increase over the mean rate for the previous 3 years (Figure 11). South Australia (26 cases) and Queensland (13 cases) reported the majority of cases. The median age of cases ranged from 10–28 years and females were more commonly infected than males in Queensland, South Australia and Victoria. All of the cases appeared to be sporadic.

Figure 11. Crude notification rates of shiga-toxin *E. coli*, 1998 to 2001, by site and year



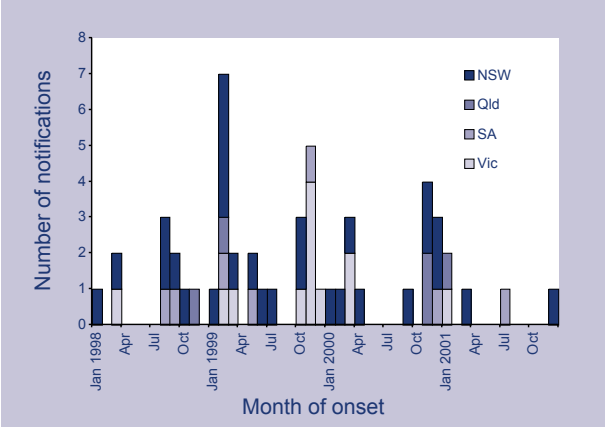
The highest rate of STEC infections was in South Australia, due to the specific testing of bloody stool (both microscopic and macroscopic) for the presence of shiga-toxin or the gene coding for production of the toxin. The majority of reports in South Australia were detected by polymerase chain reaction. Only 12 per cent (3/26) of cases in South Australia were reported to be due to *E. coli* O157. Victoria reported that two out of 4 cases were due to *E. coli* O157, while Queensland reported that four out of 10 cases were due to this serovar.

Haemolytic uraemic syndrome

There were 5 cases of haemolytic uraemic syndrome (HUS) reported during 2001, corresponding to an overall rate of 0.02 cases per 100,000 population. There were 2 cases reported in New South Wales, and one case reported in each of Victoria, South Australia and Queensland (Figure 12). The median age of cases was 16 years (range 2–53 years) and the male to female ratio was 1:1. One case, an 83-year-old male, died giving a case fatality rate of 20 per cent.

It is likely that there is substantial under-reporting of this serious disease. The Queensland site reported that there were 21 patients recorded in hospitalisation statistics for the financial year 2000/01, compared to only 3 cases for the same time period on the notification dataset. There is very little known about the notification fraction for diseases potentially due to food, and this is an area of future work for OzFoodNet.

Figure 12. Numbers of notifications of haemolytic uraemic syndrome, 1998 to 2001, by month of onset and site



Foodborne disease outbreaks

During 2001, several significant outbreaks occurred and some important themes emerged. This section discusses some of these outbreaks and summarises preliminary data. Common themes from these outbreaks are discussed in the section on 'Information on risk factors'. For a summary list of individual outbreaks associated with food or water reported by OzFoodNet sites see Appendix 2.

In 2001, OzFoodNet sites reported 86 outbreaks that were potentially related to food (Table 2). The 86 outbreaks affected approximately 1,768 people, of whom 4.0 per cent (70/1,768) were hospitalised and one person died. The majority of outbreaks occurred in summer and autumn, although there was a peak in December relating to pre-Christmas functions (Figure 13).

Figure 13. Foodborne disease outbreaks, 2001, by month and agent

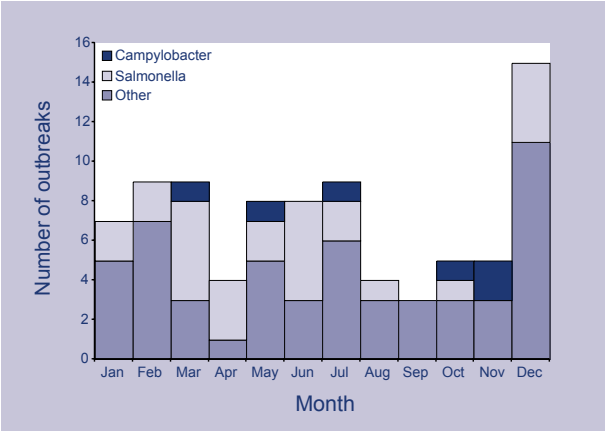


Table 2. Number of outbreaks and clusters, 2001, reported by OzFoodNet site and pathogen

OzFoodNet	Number of outbreaks, by pathogen type							Vehicles identified		Median number of cases per outbreak (range)	Analytical studies		Clusters investigated[||]
	Salmonella	*Campylobacter*	Parasites	Toxin	Viral	Unknown*	Total	Conf[†]	Susp[‡]		CCS[§]	Cohort	
ACT	0	0	0	0	0	6	6	0	6	46 (19–110)	0	0	3
Vic	7	2	0	3	4	7	23	9	5	22 (3–269)	2	11	26
Hunter	1	1	0	1	0	8	11	5	5	4 (2–20)	0	1	3
Qld	5	2	1	9	1	5	23	15	1	8 (2–87)	2	4	5
WA	3	0	0	0	2	5	10	3	3	20 (4–56)	0	7	1
SA	8	1	0	0	0	0	9	6	0	13 (2–90)	2	4	7
Tas	2	0	0	0	0	1	3	0	1	7 (6–9)	0	2	3
Aust	1	0	0	0	0	0	1	1	0	27	0	0	–
Total	**27**	**6**	**1**	**13**	**9**	**34**	**86**	**39**	**20**	**10 (2–269)**	**6**	**29**	**48m**

* Outbreaks where the aetiology was suspected, but not confirmed have been categorised as 'Unknown'.

† Confirmed vehicle, either microbiologically and/or epidemiologically.

‡ Suspected vehicle, from descriptive epidemiology and biological plausibility, and/or non-specific microbiological indicators.

§ Case control study.

|| The Australian Capital Territory and Tasmania reported investigating only clusters of non-salmonellosis, where Victoria, the Hunter and Queensland reported clusters of *Salmonella*, and South Australia reported additional investigation of suspected person-to-person transmission.

Thirty-one per cent (27/86) of outbreaks were due to *Salmonella*, while 37 per cent (32/86) were of unknown aetiology. OzFoodNet sites reported that a food vehicle was confirmed for 45 per cent (39/86) of outbreaks and suspected for a further 23 per cent (20/86) of outbreaks. To investigate these outbreaks, health departments conducted 29 cohort studies and 6 case control studies. In addition, sites reported details of 48 investigations into temporal or geographical increases of enteric pathogens, although this number is an underestimate as this information is poorly recorded.

Significant outbreaks

Australia participated in two international outbreak investigations during 2001. In the first, the Victorian Department of Human Services (DHS) investigated an outbreak of *Salmonella* Typhimurium Definitive Type 104 associated with helva imported from Turkey.[7] The Victorian DHS investigated this in conjunction with Sweden, Norway and other European countries. Eighty-seven per cent (20/23) of Australian cases occurred in Victoria, with 2 cases occurring in New South Wales and one in Queensland.

The second international outbreak was due to *Salmonella* Stanley associated with dried peanuts imported from China.[8] OzFoodNet coordinated the investigation into this outbreak at the request of CDNA. The investigation was unusual in that it involved small numbers of cases in several Australian jurisdictions. OzFoodNet epidemiologists and health department staff conducted hypothesis-generating interviews, which were collated centrally. The source of the outbreak became clear when the Victorian DHS and the Microbiological Diagnostic Unit, Public Health Laboratory tested dried peanuts nominated by 2 cases. The peanuts subsequently tested positive for three *Salmonella* serovars: Stanley, Newport and Lexington. This finding triggered an international product recall and assisted health agencies in Canada and the United Kingdom who were investigating similar outbreaks.

The largest community-wide outbreak in 2001 occurred in South Australia and was due to *Salmonella* Typhimurium phage type 126.[9] The outbreak lasted for several months, with cases emerging in other jurisdictions later in the epidemic. South Australian investigators conducted a case-control study showing that illness was associated with consumption of chicken. The department also identified corroborating evidence for this link, including descriptive epidemiology and microbiological evidence from samples of raw chicken. The South Australian DHS observed a decrease in human cases of this infection following interventions in the local chicken industry, at the breeder farm, hatchery and processing plant levels.

This outbreak raised again the question of the role that contaminated chicken products play in the epidemiology of *Salmonella* and *Campylobacter* in humans in Australia. Following the outbreak, a submission to the Food Regulation Standing Committee prompted an examination of this issue by a Development and Implementation Sub-Committee working group.

In June 2001, Queensland investigated a statewide increase in *Salmonella* Bovismorbificans phage type 32. Investigators suspected that the outbreak was linked to a food product purchased from a fast food restaurant, and conducted a case control study. The study implicated a product containing iceberg lettuce. Environmental investigations identified a mechanical slicer at the processing facility that was positive for *Salmonella* Bovismorbificans phage type 32.

Agents and vehicles

Thirty-one per cent (27/86) of outbreaks during 2001 were due to *Salmonella*, with S. Typhimurium causing 16 outbreaks (Table 3). The proportion of *Salmonella* outbreaks with good quality evidence for an implicated source was very high, with 52 per cent (14/27) having analytical and/or microbiological evidence. Despite *Campylobacter* being the most commonly notified pathogen to health authorities, only 6 outbreaks were recorded. Queensland recorded 83 per cent (5/6) of outbreaks of ciguatera poisoning due to fish that were locally caught and consumed. Consumption of fish caused 2 outbreaks of oily diarrhoea due to escolar wax esters and one of histamine poisoning. Norwalk-like viruses were responsible for 8 per cent (7/86) of outbreaks, although it is likely that many outbreaks of unknown aetiology could be caused by these viruses.

Outbreak settings

A summary of outbreaks by settings reveals that 29 per cent (25/86) of outbreaks were associated with restaurants, which affected an estimated 381 people (Table 4). Outbreaks at conferences and functions affected the most people (765 cases) and had the largest median outbreak size of 40.5 persons. The hospitalisation rate was very low in this outbreak setting. There were 5 outbreaks in aged care settings affecting 51 people, 10 of whom were hospitalised. Fast food and takeaway businesses were implicated in a smaller number of outbreaks. These outbreaks had a smaller median size of 3 persons.

Table 3. Number of outbreaks reported, 2001, by aetiological agent, and level of evidence

Agent category	A	A+M	D	D+M	M	Total
C. perfringens	1	1	2	-	-	**4**
Norwalk virus	3	-	4	-	-	**7**
Campylobacteriosis	1	-	4	1	-	**6**
Ciguatera	-	-	6	-	-	**6**
Cryptosporidiosis	-	1	-	-	-	**1**
Escolar wax esters	-	-	-	2	-	**2**
Salmonella other	-	1	7	1	-	**9**
Salmonella Typhimurium	3	6	6	1	-	**16**
Salmonella Virchow	-	-	1	-	1	**2**
Scombrotoxicosis	-	-	1	-	-	**1**
Suspected Norwalk virus	-	-	2	-	-	**2**
Suspected campylobacteriosis	-	-	1	-	-	**1**
Suspected salmonellosis	-	-	1	-	-	**1**
Suspected toxin	-	-	8	-	-	**8**
Unknown	2	-	18	-	-	**20**
Total	**10**	**9**	**61**	**5**	**1**	**86**

D Descriptive evidence implicating the suspected vehicle or suggesting foodborne transmission.

M Microbiological confirmation of agent in the suspect vehicle and cases.

A Analytical association between illness and one or more foods.

Table 4. Number of foodborne disease outbreaks, 2001, by settings

Setting	Outbreaks (n)	Affected (n)	Hospitalised (n)	Hospitalisation rate (%)	Median number affected (Range)
Aged care	5	51	10	19.6	14.5 (3–49)
Camp	6	207	2	1.0	30 (11–87)
Community	5	161	16	9.9	23 (6–88)
Conference/function	15	765	2	0.3	40.5 (2–269)
Home	13	81	24	29.6	7 (2–16)
Hotel	5	36	3	8.3	8 (6–22)
Nationwide	1	27	-	-	-
Restaurant	25	382	8	2.1	8.5 (2–95)
Takeaway	11	50	5	10.0	3 (2–10)
Total	**86**	**1,759**	**70**	**4.0**	**9 (2–269)**

It is important to consider when reviewing these data that the setting plays an important role in the recognition and investigation of an outbreak. An outbreak in a conference setting where many people eat common food is easily recognised because many people become ill. Outbreaks associated with takeaway food are difficult to detect. While the volume of food prepared might be very large, it is difficult to identify consumers of contaminated takeaway food as they may be widely dispersed in the community. Some of these outbreaks reported here may have resulted from food safety problems in settings other than those mentioned, as contributing factors have not been taken into account.

No food vehicle was identified in 31 per cent (27/86) of outbreaks in 2001 (Table 5). The most common categories were for meat and poultry, which were responsible for 14 per cent (12/86) and 13 per cent (11/86) of outbreaks respectively. There were 3 outbreaks that were associated with eggs. Fish or shellfish were responsible for, or suspected to have caused 11 outbreaks. The majority of seafood-associated investigations were descriptive, as they were small toxin-related outbreaks where diagnosis was made on clinical grounds. Desserts were responsible for 7 per cent (6/86) of outbreaks, while salads, vegetables or fruits were responsible for 3 per cent (4/86) of outbreaks.

There were 2 outbreaks associated with contaminated drinking water, although investigators only obtained descriptive epidemiological data. One of these was a camp water supply with a high coliform count, and the other was a remote mine site where bore water was suspected as the cause. During 2001, there were 2 outbreaks due to unpasteurised milk. One of these was a small outbreak of cryptosporidiosis associated with milk intended for animal consumption. The other outbreak was suspected to be caused by unpasteurised milk consumed while on a school camp.

Table 5. Outbreaks reported to OzFoodNet sites, 2001, by vehicle category and level of evidence

Vehicle category	Level of evidence					Total
	A	A+M	D	D+M	M	
Dessert*	2	4	-	-	-	**6**
Drinking water†	-	-	2	-	-	**2**
Eggs	-	1	-	-	-	**1**
Suspected eggs	-	-	2	-	-	**2**
Fish/shellfish/seafood	-	-	7	2	-	**9**
Suspected fish/shellfish	-	-	2	-	-	**2**
Milk‡	-	1	1	-	-	**2**
Miscellaneous	-	-	-	1	-	**1**
Mixed vehicles	2	-	-	-	1	**3**
Pizza	-	-	5	-	-	**5**
Poultry	-	1	2	1	-	**4**
Suspected poultry	1	-	6	-	-	**7**
Red meat/meat products	3	1	-	1	-	**5**
Suspected red meat/meat products	1	-	6	-	-	**7**
Salad/vegetable/fruit§	1	1	1	-	-	**3**
Unknown	-	-	27	-	-	**27**
Total	**10**	**9**	**61**	**5**	**1**	**86**

* One outbreak was suspected to be caused by the dessert based on mildly elevated relative risks.

† One outbreak was suspected to be due to drinking water contamination, based on circumstantial evidence.

‡ One outbreak was suspected to be due to unpasteurised milk based on circumstantial evidence, and descriptive epidemiology.

§ One outbreak was suspected to be caused by salads consumed at a barbecue.

D Descriptive evidence implicating the suspected vehicle or suggesting foodborne transmission.

M Microbiological confirmation of agent in the suspect vehicle and cases.

A Analytical association between illness and one or more foods.

The settings in which outbreaks occurred varied with the agent implicated (Table 6). Outbreaks due to salmonellosis occurred in many settings, compared to outbreaks due to *C. perfringens* where 75 per cent (3/4) of outbreaks occurred in restaurants. Conference or functions, or restaurants were the setting for all of the suspected toxin outbreaks, which would be explained by poor handling of foods. All ciguatera poisoning outbreaks reported in 2001 occurred in homes.

It is important to interpret these data cautiously, as we have only reported food vehicles and not sources of infection or cause of contamination (Box 1).

Box 1. Attributing source of *Salmonella* Typhimurium 64 outbreak

An outbreak in Western Australia of *Salmonella* Typhimurium 64 was epidemiologically linked to fried ice cream. Fried ice cream has been categorised as a dessert. The cause of this outbreak was related to several potential breaches in food safety, including:

- using raw eggs to make the batter;
- using bread crumbs that were also used for crumbing chicken and other meats; and
- inadequate cooking.

The original cause of contamination in this outbreak could have been either raw eggs or cross-contaminated bread crumbs.

Table 6. Agents responsible for foodborne disease outbreaks associated with different settings, OzFoodNet sites, 2001

	Aged care	Camp	Community wide	Conference/function	Home	Hotel	Nationwide	Restaurant	Takeaway	Total
C. perfringens	-	-	-	-	-	1	-	3	-	**4**
Norwalk virus	-	1	-	2	-	-	-	4	-	**7**
Campylobacteriosis	1	-	-	1	-	-	-	3	1	**6**
Ciguatera	-	-	-	-	6	-	-	-	-	**6**
Cryptosporidiosis	-	-	1	-	-	-	-	-	-	**1**
Escolar	-	-	-	1	-	-	-	1	-	**2**
Salmonella other	2	1	2	-	1	1	1	1	-	**9**
Salmonella Typhimurium	1	2	2	-	4	1	-	4	2	**16**
Salmonella Virchow	-	-	-	1	1	-	-	-	-	**2**
Scombrotoxicosis	-	-	-	-	-	-	-	1	-	**1**
Suspected Norwalk virus	-	-	-	2	-	-	-	-	-	**2**
Suspected campylobacteriosis	-	1	-	-	-	-	-	-	-	**1**
Suspected salmonellosis	-	-	-	-	-	-	-	-	1	**1**
Suspected toxin	-	-	-	5	-	-	-	3	-	**8**
Unknown	1	1	-	3	1	2	-	5	7	**20**
Total	**5**	**6**	**5**	**15**	**13**	**5**	**1**	**25**	**11**	**86**

Risk factors for foodborne illness

OzFoodNet sites identified some important risk factors for foodborne infections during 2001. Epidemiologists identified these by reviewing data on foodborne outbreaks and discussing the results of investigations. During 2001, OzFoodNet started a series of case control studies for common infections, which will further characterise risk factors for foodborne illness. The major risk factors for infection that OzFoodNet identified during 2001 are grouped in the following categories: imported foods; takeaway foods; seafood; and red meat and poultry.

Imported foods and *Salmonella* contamination

Like many other countries, Australia is importing increasing amounts of foods from overseas countries. In 2001, there were two major outbreaks in Australia associated with imported foods. The first of these was the outbreak of antibiotic resistant *Salmonella* Typhimurium Definitive Type 104 due to helva imported from Turkey.[7] The second outbreak was an outbreak of *Salmonella* Stanley due to dried peanuts imported from China.[8]

While both of these outbreaks were small in terms of numbers of cases (50 overall) they have important implications for Australia and the food industry. When foods contaminated by microorganisms are imported they can pose a serious risk for primary industry and the processed food sector. *Salmonella* Typhimurium DT 104 has the potential to be a serious threat to primary industry due to its virulence and antibiotic resistant characteristics.[10] *Salmonella* Enteriditis phage type 4 is another agent that could prove devastating to the egg producing industry if it becomes established in Australia. It is vital that health and agriculture agencies are able to rapidly recognise outbreaks and identify the source.

Outbreaks due to imported foods have important resource implications for health and other regulatory authorities. Identifying the source of the food vehicle is difficult, as these foods often have a wide distribution and cases may be widely and thinly spread. Small numbers of cases of *Salmonella* Stanley were identified in every Australian jurisdiction except Tasmania and the Northern Territory. This type of investigation requires a coordinated response from all jurisdictions. Although a food vehicle may be identified, it may be difficult to control future product importation. For example, testing all food products containing peanuts coming into Australia is virtually impossible due to the huge range of products containing these nuts.

These outbreaks have shown that there is an obvious need to strengthen networks between Australian and international investigators. Health Canada was trying to identify a source for a similar outbreak of *Salmonella* Stanley in British Columbia during September 2001. The OzFoodNet posting to international electronic mailing lists about contaminated peanuts assisted them to identify the source of their outbreak.

The Victorian DHS was only able to confirm the source for the outbreak of *Salmonella* Definitive Type 104 in Victoria after Turkish helva was confirmed as the source of a similar outbreak in Sweden. These two international investigations involved intensive liaison with health authorities in Canada, China, Turkey, the United Kingdom, Sweden, Norway and other European countries.

While investigators find it difficult to identify imported food vehicles, it is even more difficult to identify the original source of contamination in the source country. Both of these investigations tracked a specific product back to a country of origin, but were unable to identify how the product became contaminated. This is a cause for concern, as it makes prevention effort almost impossible. The concept of product traceability is currently under discussion in international forums, such as the Codex Alimentarius Commission.

Health agencies are increasingly identifying outbreaks associated with foods that are distributed internationally.[11,12,13] *Salmonella* is frequently recognised as causing international outbreaks, but other agents have also been implicated.[11] Imported foods are possibly responsible for many more cases of illness that currently go unrecognised by Australia's surveillance systems.

Takeaway foods

The increasing consumption and volume of takeaway food served in Australia means that we are recognising more outbreaks associated with this sector. In 2001, there were 10 outbreaks associated with fast foods and one community-wide outbreak associated with products served by fast food restaurants. Many of these outbreaks were relatively small, but occurred repetitively.

In 2001, there were 3 small clusters of *Salmonella* and *Campylobacter* infections associated with takeaway kebabs. The vertical spits used to cook these products may not allow adequate internal cooking during busy periods. A recent survey of kebabs in Victoria showed that in 41.1 per cent of instances meat did not reach a surface temperature of 75˚C, and 23 per cent of proprietors were cutting under-cooked meat off kebab spits.[14] There were several small outbreaks associated with takeaway chickens, where the cause of contamination could not be determined.

Pizza was suspected as the vehicle for 5 outbreaks, one of which was due to *S.* Typhimurium 126 and the remainder of unknown aetiology. These outbreaks were small, due to the nature of consumption of these products, i.e. generally in small groups, making outbreak recognition difficult. OzFoodNet sites reported that pizzas have historically been the cause of toxin related outbreaks, particularly due to *S. aureus*. These bacterial toxins have been due to poor storage of raw ingredients immediately prior to pizza preparation. Pizza is a food that may also be undercooked, particularly during busy periods when cooking times are reduced.

Knowledge of safe times and temperatures for cooking food is essential for food businesses to ensure safe food. Although a validated food safety program can greatly assist businesses ensure that their food is safe, one of the major *Salmonella* outbreaks in 2001 was associated with a supplier with a certified safety program.

Seafood related illness

During 2001, there were 10 outbreaks associated with seafood that indicate potential risks for consumers. These included:

- six outbreaks of ciguatera poisoning following reef fish consumption;
- two outbreaks of oily diarrhoea associated with escolar consumption;
- one outbreak of histamine poisoning after eating Mahi Mahi; and
- one outbreak of *Salmonella* Missisippi suspected to be associated with oysters.

Ciguatera poisoning is a commonly reported illness, particularly in Queensland, where the majority of outbreaks occurred in 2001. Ciguatera poisoning may cause serious illness. In one outbreak, 11 out of 14 people were hospitalised as a result of their illness. In another outbreak, all 3 people consuming fish were affected and one person died. All outbreaks occurred in a home setting. The fish species implicated in these outbreaks included coral trout (n=2), Spanish mackerel (n=2), spotted mackerel (n=1), and barracuda (n=1). These species are recognised as a high risk for ciguatera poisoning. There is an obvious need to increase the education of amateur fishermen about species likely to cause ciguatera poisoning and the location of high-risk reefs and fishing locations.

During 2001, there were 2 outbreaks of diarrhoea associated with consumption of escolar (*Lepidocybium flavobrunneum*) or oilfish (*Ruvettus pretiosus*). There have been several outbreaks of this diarrhoeal syndrome around Australia in recent years, particularly in South Australia, Victoria and New South Wales. The outbreaks in 2001 affected 42 per cent (20/47) of people attending a conference in Newcastle and 33 per cent (5/15) of people attending a restaurant in Melbourne.

The marketing names used for these species are confusing, as they may be called butterfish, rudderfish, oilfish or escolar. Escolar and oilfish are the only two species that have the potential to cause illness. These fish have a very high content of indigestible wax ester, which causes oily diarrhoea, nausea and vomiting. The two other outbreaks associated with seafood in 2001, were a small outbreak of histamine poisoning (4 cases) and one of salmonellosis associated with oysters (6 cases). Histamine poisoning is not commonly reported in Australia, compared to other countries.[15] The symptoms are short-lived and often affect small numbers of people. *Salmonella* outbreaks are not commonly associated with seafood, although oysters may be contaminated with human pathogens when grown in contaminated water.[16]

Red meat and poultry

Twenty-seven per cent (23/86) of outbreaks reported by OzFoodNet sites were attributed to poultry or red meat products (Table 7). Many of these outbreaks were related to contamination post-cooking. There were 2 outbreaks of *C. perfringens* associated with cooked red meats, and four suspected toxin-related outbreaks associated with spit roast meats. The outbreak of Norwalk-like virus occurred at a large function where it was suspected that dishes or platters containing chicken became contaminated.

Table 7. Outbreaks associated with poultry or red meat/meat products, 2001, by agent and vehicle

Agent category	Poultry	Red meat/ meat products	Suspected poultry	Suspected red red meat/ meat products	Total
C. perfringens	-	2	-	-	**2**
Norwalk virus	-	-	1	1	**2**
Campylobacteriosis	2	-	-	-	**2**
Salmonella Typhimurium	1	3	-	1	**5**
Salmonella Virchow	1	-	-	-	**1**
Suspected salmonellosis	-	-	-	1	**1**
Suspected toxin	-	-	-	4	**4**
Unknown	-	-	6	-	**6**
Total	**4**	**5**	**7**	**7**	**23**

Salmonella was responsible for 7 outbreaks associated with these foods, five of which were due to *Salmonella* Typhimurium serovar. The South Australian Department of Human Services investigated a large outbreak of *Salmonella* Typhimurium phage type 126. The department investigation demonstrated a strong association between illness and consuming locally produced chicken meat. They also identified concurrent epidemics of this *Salmonella* in local chicken flocks. The chicken industry instituted a range of interventions, which was likely to have resulted in a subsequent decrease in the number of human cases.

During 2001, OzFoodNet sites investigated 38 clusters of *Salmonella* infections affecting 235 people. These included serovars commonly isolated from animal sources, such as Typhimurium, Virchow, and Bovismorbificans. Eleven of these clusters were various phage types of S. Typhimurium and accounted for 158 notified cases. Many of these clusters appeared to have links to red meat and/or poultry, either through human-animal contact or contaminated food.

Some of the reasons that investigators suspected that these clusters were related to these sources were:

- reports of isolation of these organisms from non-human sources in the NEPSS database;
- sporadic cases where the source of infection was known, e.g. a farmer infected with a certain type of *Salmonella* coincident with an outbreak in an animal herd;
- mixed infections with other organisms, such as *Campylobacter*, that are commonly associated with the suspected source;
- previous experience with outbreaks and sporadic cases of the specific *Salmonella* infection; and
- surveys of foods.

Identifying the source of these human infections is very difficult since poultry and red meats are very commonly consumed. While it is very difficult to identify sources, it is vital that public health agencies can compare data on *Salmonella* isolates from different sources to generate hypotheses.

State and territory health departments routinely consult NEPSS data on isolates from non-human sources to assist with investigations, although the underlying sampling distribution is unknown. It is often very difficult to obtain data from industry that are relevant to the investigation. To overcome these problems, jurisdictions could consider developing a long-term survey of *Salmonella* and *Campylobacter* in red meat and poultry at the retail level to monitor trends. If the sampling plan is well devised and the survey is conducted over a long period of time, investigators may be able to correlate these data with human infections. It is also vital for health agencies to improve liaison with industry and departments of agriculture.

Burden of disease

Foodborne disease imposes a substantial burden on the community and healthcare system.[17] One of the primary aims of OzFoodNet is to determine the incidence of foodborne disease in Australia. In 1999, the Australia New Zealand Food Authority estimated that there were approximately 4.2 million cases of foodborne disease each year, costing in excess of A$2.6 billion.[18]

NCEPH is conducting a National Gastroenteritis Survey on behalf of OzFoodNet to determine the incidence of gastroenteritis, which will be used to estimate the burden of foodborne disease. Two sites, Queensland and Victoria, also collected data about gastroenteritis through their state-based computer-assisted telephone interview (CATI) systems during 2001. This section reports on the progress of the National Gastroenteritis Survey and the preliminary results from the two state-based surveys.

Early estimates from the data collected in these three surveys indicate that the incidence of gastroenteritis is approximately one episode per person per year.[19] If we consider that roughly 35 per cent of gastrointestinal disease may be due to food, then there may be as many as 7 million cases of foodborne disease in Australia each year.[17] This is considerably higher than previous estimates.[18]

The National Gastroenteritis Survey

NCEPH started the OzFoodNet National Gastroenteritis Survey in September 2001. The main aim of this cross-sectional survey is to determine the incidence of gastroenteritis in Australia and to contribute to more reliable estimates of foodborne disease. The survey will also allow OzFoodNet to:

- identify regional or seasonal trends in gastroenteritis;
- determine the health seeking behaviours of persons with gastroenteritis; and
- determine the faecal testing patterns of medical practitioners who treat patients with gastroenteritis.

The National Gastroenteritis Survey uses the CATI technique to record people's experience of gastroenteritis in the previous month. The survey will run from September 2001 to August 2002 and will enrol approximately 6,000 people from all Australian states and territories. The results will be analysed by varying case definitions of gastroenteritis. This will range from the broadest possible, such as any acute episode of vomiting or diarrhoea in the last 4 weeks through to more stringent criteria, such as three or more loose stools or two episodes of vomiting in any 24-hour period. To ensure that the data are relevant to foodborne disease, OzFoodNet will exclude people attributing symptoms to non-infectious causes.

The preliminary data available in December 2001 covered the 4 months between September and December 2001 from 2,417 interviews of people across Australia. The unweighted results showed that approximately 12 per cent of respondents experienced symptoms of gastroenteritis in the previous 4 weeks. Preliminary analysis of the data suggests that there is variation by region, age and a medical history of chronic illness.[20] In the 4-month period there was modest variation across the jurisdictions with the highest level being recorded in the Northern Territory (Table 8). The Northern Territory recorded nearly twice the incidence of gastroenteritis of most other jurisdictions.

Table 8. Proportion of respondents with symptoms of gastroenteritis,* September to December 2001, by State and Territory

State	Proportion with gastroenteritis (%)
New South Wales†	10
Northern Territory	21
Queensland	11
South Australia	12
Tasmania	11
Victoria	11
Western Australia	10
Total	**12**

* Unweighted and all-inclusive definition of Gastroenteritis

† Includes the Australian Capital Territory, and an over sample in the Hunter Area Health Service

There was considerable difference in incidence by age, with younger children in the 0–4 year age group having the highest level of gastroenteritis. Approximately 20 per cent of this age group experienced gastroenteritis in the past 4 weeks compared with 5 per cent of older adults.

About 20 per cent of people with gastroenteritis visited their doctor or casualty department for treatment, but only about 3 per cent had a stool sample taken for testing. About a third took some form of medication, mostly painkillers. About a third of working people missed a day or more of work when they had gastroenteritis.

Victorian Population Health Survey

The Victorian Department of Human Services surveyed 7,494 persons aged 18 years or older as part of the Victorian Population Health Survey conducted between August and November 2001.

The survey used a CATI methodology to collect data about a range of health topics and demographic information. In the survey there were seven questions relating to gastroenteritis. The case definition for an episode of gastroenteritis was three or more loose stools, or two or more episodes of vomiting in a 24-hour period. Survey respondents were asked if they had experienced gastroenteritis in the previous 4 weeks. Persons with chronic conditions in which diarrhoea or vomiting were predominant symptoms were excluded from analysis.

The survey found that 10.1 per cent of adults had either diarrhoea or vomiting in the past 4 weeks when people with chronic gastrointestinal symptoms were excluded (Table 9). Twenty-one per cent of these people sought medical assistance for their illness, and 3.4 per cent had a faecal specimen tested.

Queensland Health 2001 Omnibus Survey

The Queensland Department of Health surveyed a total of 3,081 persons aged 18 years or older as part of the Queensland Health 2001 Omnibus Survey conducted between March and May in 2001. The Survey also collected data on children aged 7 months to 4 years from a nested survey of 386 parents or caregivers.

The survey used a CATI methodology to collect data about a range of health topics and demographic information. In the survey, there were 17 questions relating to gastroenteritis in adults and 13 addressed to carers of young children. The case definition for an episode of diarrhoea was three or more loose stools in a 24-hour period. Respondents were asked about episodes of diarrhoea during the preceding month. Persons with chronic conditions in which diarrhoea is a symptom were excluded from analysis.

The survey found that 13.6 per cent of adults and 18.9 per cent of children had acute diarrhoea in the preceding month (Table 10). Persons aged 18–39 years were almost twice as likely as those aged 40 years and older to report acute diarrhoea in the preceding month.

There was no significant difference for incidence of acute diarrhoea between persons living in a capital city or other major urban areas and persons living in rural and remote areas. There was no significant difference in the incidence of acute diarrhoea between lower and higher socio-economic groups as measured by the Australian Bureau of Statistics Socio-economic Indices for Areas, which is different to reports in the literature.[21]

Parents of young children with diarrhoea were more than twice as likely to seek medical care compared with adults (RR 2.5; 95% CI 1.8–3.5), although doctors requested stool specimens from similar proportions of presenting adults and young children.

Table 9. Self-reported gastroenteritis reported in the previous 4 weeks for adults over 18 years, Victorian Population Health Survey, August to December 2001

	Adults (n=7,494)	
	n	%
Gastroenteritis	760	10.1
Days off work/school/study/home duties	172	22.6*
Consulted doctor/nurse/medical person	157	20.7*
Stool tested	26	3.4*
Hospitalised	20	2.6*

* The denominator for proportions reporting days off work, consultation to doctor, stool testing and hospitalisation is the number of survey respondents reporting gastroenteritis (*n* = 760).

Table 10. Acute diarrhoea reported in the previous month, comparing adults and children aged between 7 months and 4 years, Queensland Health Omnibus Survey, March to May 2001

	Adults (n=3,081)		Children (n=386)	
	n	%	n	%
Acute diarrhoea	418	13.6	73	18.9
Consulted doctor	77	18.4*	34	46.6*
Stool collected	11	2.6*	6	8.2*

* The denominator for proportions reporting consultation to doctor and stool testing is the number of survey respondents reporting gastroenteritis (n = 418 for adults and n = 73 for children).

Improving surveillance

OzFoodNet aims to improve the investigation and reporting of foodborne disease throughout Australia. During 2001, OzFoodNet reviewed and evaluated surveillance of foodborne disease in different jurisdictions. These discussions highlighted that surveillance in different jurisdictions varies in sensitivity to detect and investigate outbreaks. OzFoodNet aims to ensure that each jurisdiction enhances the sensitivity of their surveillance system in a way that is sustainable in the longer term.

Communicating nationally

OzFoodNet has developed into the major forum vehicle for discussing foodborne disease incidence at the national level in Australia. OzFoodNet contributes to CDNA, which is Australia's peak body for surveillance and response to communicable diseases. CDNA meets each fortnight by teleconference to discuss issues about communicable diseases that are of national importance.[22] OzFoodNet is able to investigate clusters of foodborne disease that occur in more than one Australian jurisdiction.

During 2001, OzFoodNet started circulating a short summary report of outbreaks and clusters occurring at each site. These reports are circulated each fortnight and detail:

- the occurrence of point source outbreaks occurring in the site;
- results from current and previous investigations;
- any increases in enteric pathogens; and
- the current incidence of important foodborne diseases, such as: listeriosis, STEC and *Salmonella* Enteritidis infections.

OzFoodNet holds monthly teleconferences to update members about the occurrence of clusters of disease and discuss the progress of joint projects. If cluster investigations involve more than one jurisdiction more frequent teleconferences are conducted.

National outbreak coordination

In July 2001, CDNA requested that OzFoodNet coordinate the investigation into an outbreak of S. Stanley that was occurring in people with Asian surnames in several Australian jurisdictions. OzFoodNet convened teleconferences to discuss state and territory investigations of cases. All jurisdictions agreed to pool de-identified data into a spreadsheet for descriptive analysis and hypothesis generation.

This outbreak investigation was unusual in that very few cases were notified in each jurisdiction. Some jurisdictions only had one or 2 cases notified. It demonstrated the need for centralising data and coordinating investigations nationally. OzFoodNet also coordinated summaries of several smaller clusters of *Salmonella* infections occurring across different jurisdictions.

National case definitions

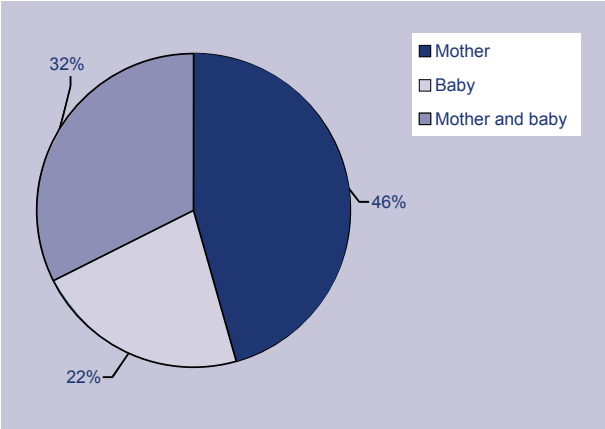
All contributors using the same case definitions and applying them consistently improve public health surveillance. During 2001, the CDNA revised the case definitions for national surveillance of communicable diseases. This review, which included input from OzFoodNet included several diseases potentially tranmitted via food.

Case series of listeriosis

The FSANZ requested that OzFoodNet compile data on human listeriosis for a risk assessment on *Listeria* in seafood. OzFoodNet obtained data from all states and territories on cases of listeriosis reported between 1998 and 2000.

The data required considerable checking and interpretation, but yielded important insights into surveillance for listeriosis. An example of this was the inconsistencies in recording materno-foetal infections between states and territories. States and territories reported 49 listeriosis cases in pregnant women that corresponded to 37 distinct infections. For each pregnancy-associated infection, jurisdictions recorded either the mother or the baby as a single case, or they recorded both the mother and the baby on the dataset (Figure 14). This means that the numbers of listeriosis cases occurring in each jurisdiction are not comparable. The review also highlighted many information gaps on routine surveillance databases, such as information on risk factors and Indigenous status.

Figure 14. Notifications of listeriosis in pregnant women, 1998 to 2000, by method of State and Territory dataset entry (*n*=49 cases)



Timeliness and completeness of Salmonella reporting

Effective surveillance of *Salmonella* relies on data that are transmitted in a timely fashion and recorded systematically.[23] In 2001, OzFoodNet epidemiologists evaluated surveillance for foodborne diseases. These evaluations highlighted some deficiencies inherent in the system, which became obvious during multi-jurisdictional investigations.

OzFoodNet epidemiologists worked with local data providers and reference laboratories to improve the timeliness of surveillance data. Some examples of improvements are listed below.

- By changing the way data were reported from the reference laboratory, the OzFoodNet-Hunter site was able to decrease the median time delay between specimen collection to receipt of a serovar result from 21 days to 17 days.
- The OzFoodNet-Tasmania site was able to improve the timeliness of *Salmonella* reports by recording sero-groupings, as the predominant serovar. Mississippi is the only one belonging to the E/G group. S. Mississippi accounted for 59 per cent (96/166) of notifications in Tasmania during 2001. While not providing definitive results, this change will allow the Tasmanian Department of Health and Community Services to identify potential outbreaks of *Salmonella* Mississippi, and non-Mississippi serovars.
- The OzFoodNet-Western Australia site was able to liaise with the local reference laboratory to increase the frequency of sending *Salmonella* isolates requiring phage typing to reference laboratories in South Australia and Victoria. Minimising the time taken for batching isolates is vital for outbreak detection and control.

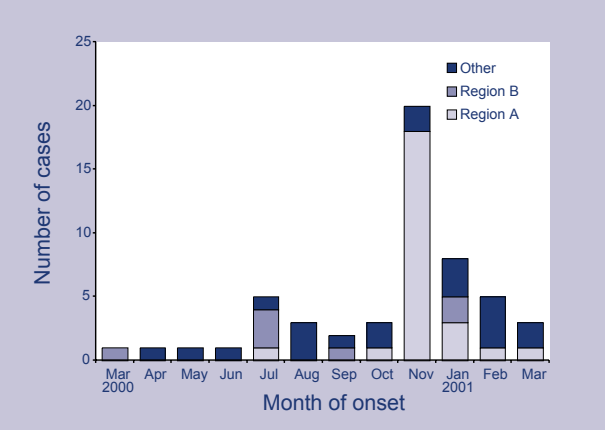
Despite these examples of improvements to *Salmonella* timeliness, there are still many gains yet to be made in this area. Timeliness should improve considerably with the introduction of electronic reporting from laboratories to health departments.

It is equally important for health agencies to accurately record reports of *Salmonella* on surveillance databases. The quality of datasets around the country can influence detection of clusters for investigation (Box 2).

Box 2. A psuedo outbreak due to data entry error

At a routine teleconference, an epidemiologist identified a recent increase in *Salmonella* Typhimurium phage type 4 in a neighbouring geographic region (Region A) of their state (Figure 15). An epidemiologist in another state reported a concurrent increase of *S.* Typhimurium 4 at the same time. Upon further investigation, the increase in the first state was found to be entirely due to a data entry error. This national discussion about this pseudo-outbreak again highlighted the importance of rigorous quality assurance in surveillance data collection.

Figure 15. Pseudo outbreak of *Salmonella* Typhimurium 4 due to data entry error in the neighbouring region to an OzFoodNet site



There was a marked improvement in completeness of *Salmonella* typing information on surveillance databases in jurisdiction between 2000 and 2001 (Table 11). There was a 5.3 per cent increase in salmonellosis cases on notification databases with appropriate typing data, up from 88.0 per cent in 2000 to 93.3 per cent in 2001. Importantly, the rates of completeness particularly improved in the states of Western Australia and New South Wales in these 2 years. Western Australia reported the largest improvement of 23.1 per cent from 2000 to 2001, which was due to the health department receiving voluntary laboratory notifications of communicable diseases at this time. South Australia had the highest rate of completeness with appropriate information for 99.8 per cent of all cases in 2001.

It is likely that the majority of salmonellas isolated at primary laboratories are typed due to a well-developed system of referral. The overall improvements observed in 2001 can be partly attributed to the interaction of OzFoodNet epidemiologists with surveillance systems. While there was an improvement in this area from 2000 to 2001, it is an area that OzFoodNet epidemiologists need to monitor and improve in the future.

Increasing OzFoodNet coverage

During 2001, the Northern Territory participated in OzFoodNet as observers. The Food Branch of the New South Wales Health Department also participated in several teleconferences and attended face-to-face meetings.

The Australian Capital Territory joined OzFoodNet as a fully funded member in August 2001. The OzFoodNet epidemiologist in the Australian Capital Territory is also assisting NCEPH with the estimation of the burden of foodborne disease in Australia. At the time of writing, contracts had recently been finalised which will see OzFoodNet coverage to include all of New South Wales and the Northern Territory.

Efficient surveillance of infectious diseases relies upon good liaison between health agencies and public health laboratories. OzFoodNet has continued to work collaboratively with laboratories in each jurisdiction and the PHLN and is undertaking several studies with strong laboratory involvement, which has associated benefits for surveillance.

International developments

In 2001, OzFoodNet established collaborative links with international agencies conducting surveillance and research into foodborne diseases. Several countries have conducted similar studies to OzFoodNet, which will yield important insights into the incidence and control of foodborne disease. These collaborations have included agencies, such as the USA Centers for Disease Control and Prevention, the Food Safety Authority of Ireland, Health Canada, the Institute of Environmental Science and Research New Zealand, the United Kingdom Public Health Laboratory Service, and the World Health Organization.

Table 11. Completeness of *Salmonella* typing data on State and Territory surveillance databases, 2000 and 2001

Information required	Per cent of notifications with appropriate typing information, by notification date															
	Tas		NSW		WA		ACT		Vic		Qld		SA		OzFoodNet sites	
	2000 (n=127)	2001 (n=166)	2000 (n=1,341)	2001 (n=1,670)	2000 (n=936)	2001 (n=898)	2000 (n=100)	2001 (n=76)	2000 (n=1,009)	2001 (n=1,091)	2000 (n=1,818)	2001 (n=2,169)	2000 (n=452)	2001 (n=613)	2000 (n=5,783)	2001 (n=6,683)
Salmonella serotype	96.9	98.2	92.8	94.1	92.2	95.2	96.0	98.7	97.7	97.7	97.2	97.0	99.6	99.8	95.5	96.5
S. Bovismorbificans phage type	100	100	50.0	39.4	62.5	57.1	–	100	96.3	96.7	100	100	100	100	76.4	83.2
S. Enteritidis phage type	100	100	78.2	80.8	34.5	85.5	100	100	100	100	94.8	90.2	100	100	78.3	89.4
S. Hadar phage type	–	–	52.9	38.9	80.0	85.7	0.0	100	81.8	100	100	73.3	100	100	75.8	77.8
S. Heidelberg phage type	0.0	–	18.2	84.6	0.0	0.0	100	100	75.0	100	90.6	91.8	–	–	68.6	88.6
S. Typhimurium phage type	97.0	96.4	88.1	95.7	55.2	87.8	100	100	99.8	99.7	93.1	95.8	100	100	87.7	96.4
S. Virchow phage type	100	–	38.2	67.2	80.0	66.7	100	100	99.1	100	97.4	95.0	100	100	90.7	92.5
Salmonella with information	**95.3**	**97.6**	**80.5**	**87.8**	**68.1**	**89.4**	**95.0**	**98.7**	**97.1**	**97.4**	**95.0**	**94.7**	**99.6**	**99.8**	**88.0**	**93.3**

OzFoodNet projects

During 2001, OzFoodNet collaborators initiated several projects to investigate and understand foodborne disease, some of which were national in scope. This section briefly details the nature of these projects and the current status of this work.

National projects

During September, NCEPH collected the first month's data for the national gastroenteritis survey. Starting this study was a major achievement and required considerable collaboration. NCEPH also prepared a report into future directions for OzFoodNet, which outlined research gaps in Australia for foodborne disease.

OzFoodNet developed national case control studies for *Campylobacter* and *Salmonella* Enteritidis to identify risk factors for infection. During 2001, sites in Tasmania, Victoria and Western Australia started the *Campylobacter* study and the remaining sites made preparations. In 2001, OzFoodNet developed a proposal for a listeriosis case control study and piloted the methodology. At the December face-to-face meeting, this was changed to a case series in all but two sites. OzFoodNet sites in the Hunter and Queensland will run the original protocol as a case control study. The results of this case series will provide important information nationally on the underlying risk factors for infection and high-risk foods.

OzFoodNet will conduct a case control study of STEC/HUS in South Australia, which has the highest rates of STEC notification in Australia due to intensive screening. Investigators continued to revise the protocol for the national laboratory survey. This survey will determine the faecal testing practices of laboratories around Australia, and will provide important information that will assist interpretation of notification data.

An outbreak register for Australia

Australia's lack of a systematic system of recording data on outbreaks of enteric disease has hampered our understanding of foodborne disease.[24] Summary data from outbreaks can provide useful information for the development of policy.[25]

Before OzFoodNet commenced, the Hunter Health Area, New South Wales initiated a retrospective survey of outbreak information from all states and territories between the years 1995 to 2000. This data collection, coined OzBreaks, contains detailed information on 208 outbreaks. OzBreaks is currently being analysed in collaboration with OzFoodNet epidemiologists.

To improve the quality of this information, OzFoodNet developed a register to provide a prospective record of Australian disease outbreaks associated with food and water. The OzFoodNet working group agreed to collect outbreak information from 1 January 2001 onwards. The OzFoodNet data manager developed a database and form, based on those used by the World Health Organization European regional office and the USA Centers for Disease Control and Prevention.

OzFoodNet epidemiologists have conducted a trial of the new register and made recommendations for improvement. The CDNA has requested that OzFoodNet expand the register to include outbreaks of intestinal illness not related to food. To ensure that the system for surveillance of outbreaks works properly, OzFoodNet is communicating with international investigators and formally evaluating the register in July 2002.

Development of a national *Campylobacter* typing network

The Hunter Health Area, New South Wales site conducted a case control study of *Campylobacter* infections that commenced prior to OzFoodNet. One hundred and eighty isolates from this study have been typed by several phenotypic and genotypic methods. The OzFoodNet-Hunter epidemiologist along with microbiologists will evaluate the testing methods for their epidemiological usefulness, cost, speed, simplicity and concordance. The outcome of this evaluation will assist the identification of suitable testing methods for *Campylobacter* isolates collected as part of the national case-control study.

This evaluation is unique in that the assessment of the different typing schemes is epidemiological in nature. Comparison of typing is quite common in microbiological research, but often lacks epidemiological input. In this instance, the case control study data for the most common subtypes from a range of typing schemes will be analysed.[26]

Another benefit of this typing network is that it may provide an opportunity to develop into a network for typing organisms associated with other disease outbreaks. This method of sharing microbiological data has provided many countries with an increased capacity to control foodborne disease.[27] Sharing pulsed field gel electrophoresis patterns using BioNumerics software is the basis of the successful PulseNet system.[28]

Projects in single sites

OzFoodNet epidemiologists or collaborators have developed several other studies within their jurisdictions. These include:

- a molecular typing project in Western Australia looking at automated ribotyping of bacterial foodborne pathogens, and development of a typing library using BioNumerics software;
- a pilot study looking at enhancing Environmental Health Officer reports of foodborne disease outbreaks in Victoria;
- case control studies for locally important *Salmonella* infections, including the following serovars and phage types:
  - S. Birkenhead in Queensland and northern New South Wales;
  - S. Mississippi in Tasmania;
  - S. Typhimurium 126 in South Australia; and
  - S. Typhimurium 135 in New South Wales, Victoria and Western Australia.

Two sites, Queensland and Victoria, attempted to establish sentinel surveillance for gastroenteritis in general practice. Despite intensive liaison with divisions of general practice, both sites found it difficult to recruit recorders for the scheme. OzFoodNet has decided not to proceed with sentinel GP surveillance at this stage, particularly when other groups such as the Royal Australian College of General Practice already collect such data.

Conclusion and recommendations

OzFoodNet is much more than a data gathering exercise. OzFoodNet has demonstrated its capacity to investigate and respond to outbreaks at the national level and can potentially provide an early-warning capacity for bioterrorism events associated with food.

In time, OzFoodNet will be able to assess the efficacy of current and proposed food hygiene standards and their enforcement by jurisdictions. OzFoodNet represents a significant investment in applied research into foodborne disease. It is important for the results of this work to become incorporated into policy formulation. The results of analytical studies initiated in 2001 will provide a useful insight into the occurrences of foodborne disease in Australia.

Recommendations regarding common risk factors

As a result of recurring outbreaks associated with commonly eaten foods, OzFoodNet recommends that Australian regulatory authorities:

1. consider developing guidelines for the safe preparation of takeaway kebabs and pizza;
2. educate amateur fishermen about the dangers of eating reef fish from areas affected by ciguatera poisoning;
3. provide effective guidelines to aged care facilities aimed at preventing foodborne disease outbreaks; and
4. monitor, with OzFoodNet, the incidence of escolar-associated outbreaks, following national efforts to prevent these outbreaks.

Recommendations regarding improving foodborne disease surveillance

To improve national surveillance of foodborne disease, OzFoodNet recommends that:

5. Health and food safety agencies should continue to improve international liaison regarding food safety alerts and disease outbreaks about widely distributed foods.
6. Health, food safety agencies and agricultural agencies should consider developing a long-term survey of retail meats across Australia to determine the prevalence of specific *Salmonella* types and *Campylobacter* to aid communicable disease investigations.
7. Health, food safety, industry and agricultural agencies should develop closer links to share information about the occurrence of foodborne pathogens.
8. OzFoodNet should develop short guidelines on investigating national clusters to outline responsibilities and expectations of all parties.
10. The Commonwealth Department of Health and Ageing in conjunction with CDNA should consider building on the *Campylobacter* typing network coordinated by OzFoodNet-Hunter to enable rapid sharing of molecular typing data on bacterial pathogens.
11. OzFoodNet epidemiologists should develop standard reporting practices for pregnancy-associated listeriosis.

12. State and territory health departments should continue to conduct rigorous checks on the quality of surveillance data maintained on surveillance databases.
13. State and territory health departments should consider using completeness and timeliness of *Salmonella* reporting as a potential performance indicator of surveillance and capacity to control disease.
15. OzFoodNet should review the under-reporting of haemolytic uraemic syndrome to state and territory health departments.

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Appendices

Appendix 1. Notification summary of infections potentially due to food for OzFoodNet sites, 2001, by date of onset

		ACT	NSW	Hunter	Qld	SA	Tas	Vic	WA	Total
Campylobacter	n	429	NN	NN	3,969	2,617	676	5,515	2,609	**1,5815**
	rate	136.5	NN	NN	109.4	174.2	143.7	114.2	136.6	**125.0**
Salmonella	n	78	1,619	125	2,171	607	163	1,107	862	**6,607**
	rate	24.8	24.8	23.1	59.8	40.4	34.7	22.9	45.1	**34.4**
Yersinia	n	0	NN	NN	53	13	0	2	3	**71**
	rate	0.0	NN	NN	1.5	0.9	0.0	0.0	0.2	**0.6**
STEC	n	0	1	0	13	26	0	4	3	**47**
	rate	0.0	0.0	0.0	0.4	1.7	0.0	0.1	0.2	**0.2**
HUS	n	0	2	0	1	1	0	1	0	**5**
	rate	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	**0.03**
Typhoid	n	2	27	3	8	3	1	15	11	**67**
	rate	0.6	0.4	0.6	0.2	0.2	0.2	0.3	0.6	**0.3**
Shigella	n	7	NN	NN	107	37	6	92	77	**326**
	rate	2.2	NN	NN	2.9	2.5	1.3	1.9	4.0	**2.6**
Listeria	n	1	12	2	19	6	2	10	11	**61**
	rate	0.3	0.2	0.4	0.5	0.4	0.4	0.2	0.6	**0.3**

NN Not notifiable.

Rate = Rate per 100,000 population

Appendix 2. Outbreak summary for OzFoodNet sites, 2001

State	Month of outbreak	Setting category	Agent responsible	Number affected	Hospitalised	Evidence study	Responsible vehicles
Australia	July	Nationwide	S. Stanley	27		D+M	Imported dried peanuts
ACT	December	Conference/ function	Suspected toxin	22	0	D	Suspected spit roast meal
	December	Conference/ function	Suspected toxin	110	0	D	Suspected spit roast meal
	December	Conference/ function	Suspected toxin	68	0	D	Suspected spit roast meal
	December	Conference/ function	Suspected toxin	31	0	D	Suspected spit roast meal
	September	Conference/ function	Suspected viral	61	0	D	Suspected salad at barbecue
	December	Restaurant	Suspected toxin	19	0	D	Suspected Turkish banquet
Hunter	October	Conference/ function	Escolar wax esters	20	0	D+M	Escolar
	June	Takeaway	Unknown	4	0	D	Pizza
	May	Takeaway	Unknown	8	0	D	Pizza
	May	Takeaway	Unknown	4	0	D	Pizza
	October	Takeaway	S. Typhimurium 126	2	1	D	Chicken pizza
	April	Restaurant	Unknown	6	0	D	Suspected seafood sauce
	July	Restaurant	Unknown	10	0	D	Suspected honey chicken
	July	Takeaway	Unknown	2	0	D	Suspected takeaway chicken
	May	Takeaway	Unknown	2	0	D	Suspected chicken kebab
	May	Takeaway	Unknown	3	0	D	Suspected BBQ chicken
	November	Restaurant	*Campylobacter*	2	0	D	Unknown
Qld	January	Camp	Unknown	87	0	D	Drinking water
	January	Home	Ciguatera poisoning	14	11	D	Spanish mackerel
	January	Home	Ciguatera poisoning	2	0	D	Spotted mackerel
	June	Home	Ciguatera poisoning	3	3	D	Barracuda (Sphyraena jello)
	November	Home	Ciguatera poisoning	4	0	D	Coral trout
	November	Home	Ciguatera poisoning	9	0	D	Spanish mackerel
	February	Restaurant	Histamine fish poisoning	4	0	D	Mahi Mahi
	August	Community	Cryptosporidiosis	8	3	A+M	Unpasteurised pets milk (cow)
	July	Conference/ function	Norwalk virus	56	0	A	Salads, steak sandwiches
	March	Conference/ function	S. Virchow PT 8	2	0	D	Chicken
	July	Restaurant	*Campylobacter*	2	0	D+M	Duck liver
	March	Takeaway	*C. jejuni*	3	0	D	Chicken kebabs
	January	Restaurant	*C. perfringens*	9	0	A+M	Reef & beef meal

Appendix 2 (continued). Outbreak summary for OzFoodNet sites, 2001

State	Month of outbreak	Setting category	Agent responsible	Number affected	Hospitalised	Evidence study	Responsible vehicles
Qld	July	Restaurant	*C. perfringens*	8	0	A	Beef curry
cont	May	Community	S. Bovismorbificans 32	36	6	A+M	Chicken salad in pita bread
	February	Aged care	S. Heidelberg PT 1	12	6	D	Suspected eggs
	February	Aged care	Unknown	19	0	D	Unknown
	March	Aged care	S. Muenchen	3	0	D	Unknown
	February	Conference/ function	Unknown	6	0	D	Unknown
	June	Conference/ function	Suspected viral	10	1	D	Unknown
	December	Hotel	Unknown	6	0	D	Unknown
	June	Hotel	S. Montevideo	8	1	D	Unknown
	July	Restaurant	*C. perfringens*	7	0	D	Unknown
SA	December	Home	S. Typhimurium 135a	11	4	A+M	Tiramisu dessert
	December	Restaurant	S. Typhimurium 64var	28	0	A+M	Mango pudding
	March	Takeaway	S. Typhimurium 126	9	3	A	Custard tart with strawberries and a jelly glaze
	March	Aged care	S. Typhimurium 135	17	3	A+M	Raw egg (mince & potato pie & rice pudding)
	May	Community	S. Typhimurium 126	88		A+M	Chicken
	June	Home	S. Typhimurium 135a	2	0	D+M	Homemade italian sausage
	January	Restaurant	S. Typhimurium 29	8	1	D	Unknown
	June	Restaurant	S. Zanzibar	2	0	D	Unknown
	May	Restaurant	*C. jejuni*	10	1	D	Unknown
Tas	April	Home	S. Typhimurium 9	6	1	D	Suspected duck egg whites
	April	Home	S. Mississippi	7	0	D	Unknown
	February	Home	Unknown	9	0	D	Unknown
Vic	June	Community	S. Typhimurium 104	23	7	A+M	Turkish Helva
	March	Home	Ciguatera poisoning	16	0	D	Coral trout
	August	Restaurant	Wax ester (butterfish diarrhoea)	5	0	D+M	Butterfish
	December	Home	S. Virchow 34	11	2	M	Barbequed chicken or beef
	March	Hotel	Unknown	15	0	A	Combination cheese platter, mushroom risotto, Thai prawns
	February	Restaurant	Unknown	5	0	D	Suspected pizza
	July	Hotel	S. Typhimurium 99	22	2	A	Lamb's fry
	August	Restaurant	S. Typhimurium 99	95	1	A	Eye fillet meal
	October	Conference/ function	*Campylobacter*	50	0	A	Tomato and cucumber salad

Appendix 2 (continued). Outbreak summary for OzFoodNet sites, 2001

State	Month of outbreak	Setting category	Agent responsible	Number affected	Hospitalised	Evidence study	Responsible vehicles
Vic	December	Community	S. Mississippi	6	0	D	Suspected oysters
cont	August	Camp	Suspected *Campylobacter* (1 +ve)	12	0	D	Suspected unpasteurised milk
	January	Home	S. Typhimurium 170	14	3	D	Unknown
	February	Restaurant	Norwalk virus	65	0	A	Suspected sausages
	May	Takeaway	Unknown (1 positive *Salmonella*)	3	1	D	Suspected kebabs
	November	Aged care	*Campylobacter*	49	1	D	Unknown
	April	Camp	S. Typhimurium 9	30	1	D	Unknown
	December	Conference/ function	Unknown (suspected toxin)	269	0	D	Suspected soup or roast beef
	December	Hotel	*C. perfringens*	9	0	D	Suspected potato and bacon soup
	December	Restaurant	Unknown (suspected toxin)	33	1	D	Unknown
	February	Restaurant	Norwalk virus	31	0	D	Unknown
	January	Restaurant	Norwalk virus	9	0	D	Unknown
	March	Restaurant	Norwalk virus	16	0	D	Unknown
	September	Restaurant	Unknown (suspected toxin)	7	0	D	Unknown
WA	October	Conference/ function	Unknown	50	1	A	Cranachan (dessert)
	June	Restaurant	S. Typhimurium 64	36	4	A+M	Fried ice cream
	March	Camp	S. Typhimurium 135 var	29	0	D	Suspected bore water supply
	December	Conference/ function	Norwalk virus	56	0	A	Suspected chicken
	July	Restaurant	Unknown	6	0	D	Possible undercooked turkey
	November	Takeaway	Unknown	10	0	D	Suspected chicken
	February	Camp	S. Wandsworth	50	0	D	Unknown
	October	Camp	Norwalk virus	11	1	D	Unknown
	December	Conference/ function	Unknown	4	0	D	Unknown
	September	Restaurant	Unknown	7	0	D	Unknown

D Descriptive evidence implicating the suspected vehicle or suggesting food or waterborne transmission.

A Statistical association between illness and one or more foods determined from a formal epidemiological study.

M Microbiological confirmation of agent in the suspect vehicle and cases.

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Annual report of the Australian Meningococcal Surveillance Programme, 2001

The Australian Meningococcal Surveillance Programme

Abstract

Since 1994, The National Neisseria Network has examined and analysed isolates of *Neisseria meningitidis* from cases of invasive meningococcal disease in Australia by means of a collaborative laboratory program. The phenotypes (serogroup, serotype and serosubtype) and antibiotic susceptibility of 338 isolates of *N. meningitidis* from invasive cases of meningococcal disease were determined in 2001. Most disease was caused by serogroup B (206 isolates, 61%) or serogroup C (122 isolates, 36%) meningococci. However, there was considerable diversity in the phenotypes circulating in the different states and territories. Serogroup B strains predominated in all jurisdictions except Victoria and Tasmania and were isolated from sporadic cases of invasive disease. Serogroup B phenotypes were generally disparate with phenotype B:4:P1.4 being the most common and phenotype B:15:P1.7 was also widely distributed. Infections with a novel phenotype that was first noted in 1999, C:2a:P1.4(7), were again common in Victoria, especially in adolescents and adults, but were infrequently seen elsewhere in Australia. In Tasmania, a different phenotype, C:2a:P1.5,2 accounted for 11 of 16 isolates, again predominantly in infections of young adults. The number of isolates in Queensland increased to 78 from 43 in 2000 and was due to more strains of both serogroup B and serogroup C meningococci. About two-thirds of all isolates showed decreased susceptibility to the penicillin group of antibiotics (MIC 0.06 to 0.5 mg/L). All isolates tested were susceptible to third generation cephalosporins. From 1999, reports have also included diagnoses made by non-culture based methods in these analyses. Data relating to 135 laboratory-confirmed but culture negative cases supplemented information on culture-confirmed cases in this report. *Commun Dis Intell* 2002;26:407–418.

Keywords: antibiotic resistance, Neisseria meningitidis, *meningococcal disease*

Introduction

A national laboratory-based program for the examination of isolates of *Neisseria meningitidis* from cases of invasive meningococcal disease (IMD) commenced in 1994 through the collaboration of reference laboratories in each jurisdiction and is designed to supplement data from clinical notification schemes. Information on the phenotype (the serogroup, the serotype and subserotype), on occasion the genotype, and the antibiotic susceptibility of invasive isolates are obtained from examination of isolates. In addition, data from non-culture based laboratory testing, derived from nucleic acid amplification assays and serological examination, are included in the analyses. The characteristics of the meningococci responsible for IMD are important both for individual patient management and to tailor the public health response. The recent availability of a conjugate serogroup C vaccine and the prospect of porin-based vaccines for serogroup B meningococcal disease increase the need for precise data on circulating meningococcal subtypes.

Annual reports summarising data gathered since the inception of the program were published in *Communicable Diseases Intelligence*.[1-7] The following report analyses the characteristics of meningococci isolated in the calendar year 2001.

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Methods

The National Neisseria Network (NNN) is a collaborative program for the laboratory surveillance of the pathogenic *Neisseria*, *N. meningitidis* and *N. gonorrhoeae*.[1-8] A network of reference laboratories in each Australian State and Territory (see acknowledgments) undertakes meningococcal isolate surveillance throughout Australia.

Isolate based surveillance

Each case was based upon isolation of a meningococcus from a normally sterile site and defined as IMD according to the Public Health Laboratory Network definitions. Information on the site of infection, the age and sex of the patient and the outcome (survived/died) of the infection was sought. The isolate-based subset of the program categorises cases on the basis of site of isolation of the organism. Where an isolate is grown from both blood and cerebrospinal fluid (CSF) cultures in the same patient, the case is classified as one of meningitis. It is recognised that total number of cases and particularly the number of cases of meningitis, e.g. where there was no lumbar puncture or else where lumbar puncture was delayed and the culture sterile, is underestimated. However, the above approach has been used since the beginning of this program and is continued for comparative purposes.

Phenotyping of invasive isolates of meningococci by serotyping and serosubtyping was based on the detection of outer membrane protein antigens using a standard set of monoclonal antibodies obtained from the National Institute for Public Health, the Netherlands.

Antibiotic susceptibility was assessed by determining the minimal inhibitory concentration (MIC) to antibiotics used for therapeutic and prophylactic purposes. This program uses the following parameters to define the various levels of penicillin susceptibility/resistance when determined by a standardised agar plate dilution technique.[8]

sensitive, MIC ≤ 0.03 mg/L;

less sensitive, MIC 0.06 — 0.5 mg/L;

relatively resistant MIC ≥ 1 mg/L.

Strains with MICs which place them in the category of 'sensitive' or 'less sensitive' would be considered to be amenable to penicillin therapy when used in currently recommended doses. However, precise MIC/outcome correlations are difficult to obtain because of the nature of IMD.

Non-culture-based laboratory-confirmed cases

Additional laboratory confirmation of suspected cases of IMD is increasingly available by means of non-culture-based methods such as nucleic acid based amplification assays (NAA) and serological techniques. NAA testing is essentially by polymerase chain reaction (PCR) techniques[9] and has been progressively introduced in the different jurisdictions. Data from the results of these investigations were included for the first time in the 1999 report. The serological results are based on results of tests performed using the methods and test criteria of the Manchester PHLS reference laboratory, the United Kingdom as assessed for Australian conditions.[10,11] Where age, sex and outcome data for patients with non-culture-based diagnoses are available these were also recorded. The site of a sample of a positive PCR test is also used to define the clinical syndrome. This separation is not possible for cases diagnosed serologically.

Results

Numbers of isolates from culture-confirmed cases

A total of 338 invasive isolates of meningococci were examined in 2001, 50 less than the 388 examined in 2000. There were 100 isolates from patients whose infections were acquired in New South Wales (30% of all isolates), 78 (23%) from Queensland, 77 (23%) from Victoria, 32 (9%) from Western Australia, 22 (6%) from South Australia, 16 (5%) from Tasmania, 8 (2%) from the Northern Territory and 5 (1%) from the Australian Capital Territory (Table 1). The number of isolates increased in Queensland to 78 from 43 in 2000. In New South Wales there were 41 fewer isolates and in Victoria 31 isolates less than in 2000. Numbers also decreased in Western Australia to 32 from 50 in 2000 but in other jurisdictions the total numbers of isolates were little changed.

Table 1. *Neisseria meningitidis* isolates, Australia, 2001, by State or Territory and serogroup

State/ Territory	Serogroup										Total	
	B		C		A	Y		W135		NG*		
	n	%	n	%	n	n	%	n	%	n	n	%
ACT	5	100.0	0	0.0	0	0	0.0	0	0.0	0	**5**	**1.5**
NSW	65	65.0	33	33.0	0	1	1.0	1	1.0	0	**100**	**29.5**
NT	7	87.5	1	12.5	0	0	0.0	0	0.0	0	**8**	**2.5**
Qld	49	62.8	25	32.0	0	3	4.0	1	1.2	0	**78**	**23.0**
SA	14	63.6	7	31.8	0	0	0.0	1	4.6	0	**22**	**6.5**
Tas	3	18.8	13	81.2	0	0	0.0	0	0.0	0	**16**	**4.7**
Vic	37	48.0	38	49.4	0	1	1.3	1	1.3	0	**77**	**22.8**
WA	26	81.2	5	15.6	0	0	0.0	1	3.2	0	**32**	**9.5**
Total	**206**	**61.0**	**122**	**36.0**	**0**	**5**	**1.5**	**5**	**1.5**	**0**	**338**	**100.0**

* Not viable for serogrouping or not serogroupable.

Seasonality

Sixty-nine (21%) cases occurred between 1 January and 31 March, 62 (19%) between 1 April and 30 June, 127 (39%) between 1 July and 30 September and 70 (21%) between 1 October and 31 December. A winter peak of meningococcal disease is usual.

Age group

The age distribution of patients infected with invasive isolates in each State and Territory is shown in Table 2. Nationally, the peak incidence of meningococcal disease occurred in those aged 4 years and under. Those aged less than one year or in the 1–4 age group accounted for 47 (14%) and 62 (18%) cases respectively. A secondary peak was noted in the 15–19 year age group when 54 cases accounting for 16 per cent of the total were recorded. A further 35 cases (10%) occurred in those aged 20–24 years. The number (89) and proportion (26%) of culture-positive cases in the 15–24 year age range was less than the 126 (32%) recorded in 2000. This age range was particularly affected in Queensland and Tasmania in 2001.

Serogroup, serotype and serosubtype (phenotype) distribution

The distribution of the isolates by serogroup is shown in Table 1. Nationally, 206 serogroup B isolates represented 61 per cent of all strains. The 122 serogroup C strains (36%) was similar to the number (128) and proportion (33%) detected in 2000. The number of serogroup W135 and serogroup Y strains was lower than in recent years. No serogroup A isolates were encountered.

Some important differences in the distribution of serogroups were evident when data were disaggregated by region. Serogroup B predominated in national data (61%) and in all jurisdictions except Victoria and Tasmania. When examined regionally, Western Australia (81% of isolates), the Australian Capital Territory (100%), South Australia (63%), the Northern Territory (87%), Queensland (63%) and New South Wales (65%) had high proportions of serogroup B strains. However, in Victoria serogroup B isolates were 48 per cent of the total and in Tasmania only 18 per cent. Group B disease comprised mainly unlinked and apparently sporadic cases.

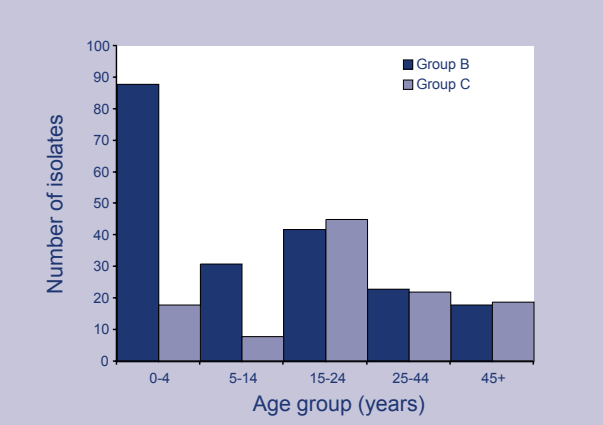
Serogroup C strains were most prominent in Tasmania where 13 of 16 isolates were serogroup C. The proportion of serogroup C infections in Victoria

decreased slightly to 49 per cent and their number decreased to 38 from the 58 isolated in 2000. In 1998, there were only 7 (17.5%) serogroup C strains in Victoria. Serogroup C isolates also declined in number in New South Wales in 2001 and the 33 strains represented 33 per cent of all isolates. In 2000, there were 55 (39%) serogroup C isolates and in 1999, 45 (37%) group C strains in New South Wales. There were 25 group C isolates (32%) in Queensland, 7 (32%) in South Australia, 5 (15%) in Western Australia, one in the Northern Territory but none in the Australian Capital Territory.

The increase in numbers of isolates in Queensland was in both serogroup B (18 more cases) and serogroup C (15 more cases). The decrease in culture-positive cases in Victoria was mainly in serogroup C isolates. Twenty fewer serogroup C meningococci were isolated in 2001. In Tasmania, there was a significant shift to serogroup C from serogroup B isolates.

Serogroup distribution has been typically age-associated, with serogroup B disease concentrated in younger age groups and serogroup C infections predominating in adolescents and young adults. In 2001, 89 (82%) of all isolates in those aged less than 4 years were serogroup B and the 18 serogroup C isolates comprised 16 per cent of cultures nationally in this age group (Table 2, Figure 1). In those aged 5–14 years, 31 serogroup B meningococcal cultures represented over 60 per cent of the 50 isolates and the 18 serogroup C strains represented 36 per cent. Serogroup B and C isolates were isolated in essentially equal proportions for all age groups over 14 years (Figure 1).

Figure 1. Number of serogroup B and C isolates, Australia, 2001, by age



However, jurisdictional differences in the distribution of serogroup B and C meningococcal isolates were again evident in 2001 (Table 2, Figures 1, 2, 3 and 4). In Western Australia and the Territories, serogroup B isolates predominated in all age groups, and in all centres, serogroup B was more commonly encountered in those aged 4 years and under. New South Wales, Queensland and South Australia closely followed the national pattern with regard to age-associated serogroup distribution. In Victoria and Tasmania serogroup C isolates were especially prominent in older, i.e. adolescent and young adult, age groups but were also seen more often in younger age groups than in other jurisdictions.

Figure 2. Number of serogroup B and C isolates, Victoria, 2001, by age

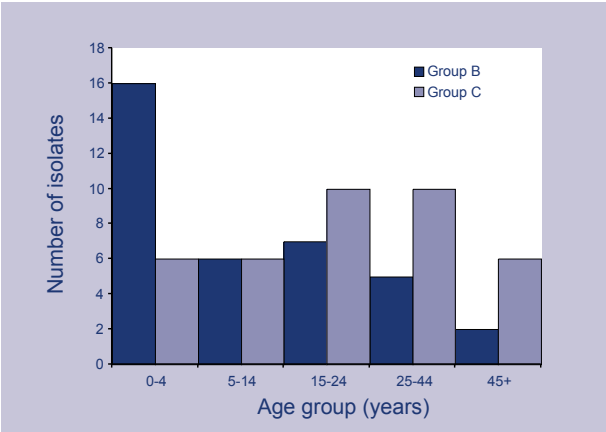


Figure 3. Number of serogroup B and C isolates, New South Wales, 2001, by age

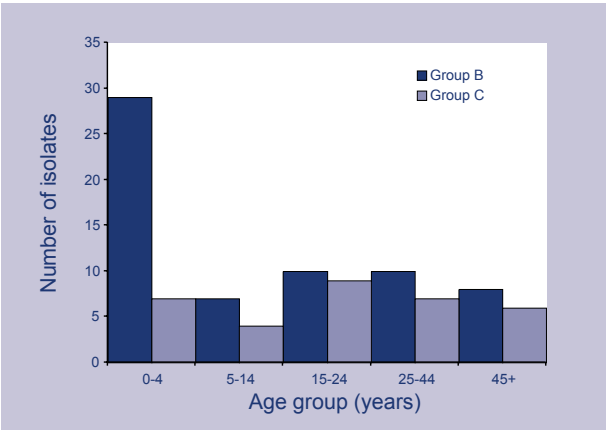


Figure 4. Number of serogroup B and C isolates, Queensland, 2001, by age

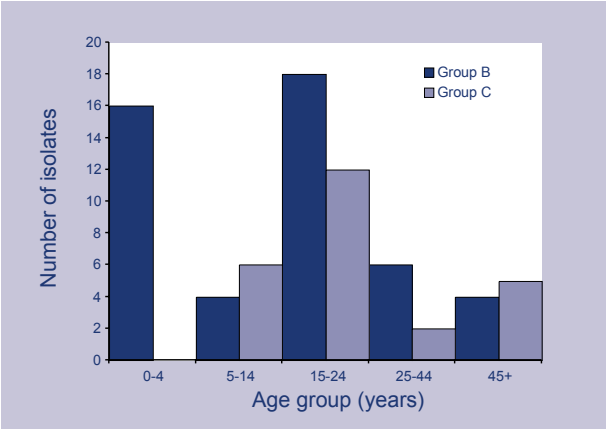


Table 2. *Neisseria meningitidis* isolates, Australia, 2001, by State or Territory, serogroup and age

		Age group (years)										
		<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	NS	Total
ACT	Total	1	0	0	0	2	0	1	1	0	0	**5**
	B	1	0	0	0	2	0	1	1	0	0	**5**
NSW	Total	20	16	5	6	11	8	17	9	7	1	**100**
	B	18	11	3	4	5	5	10	6	2	1	**65**
	C	2	5	2	2	6	3	7	2	4	0	**33**
NT	Total	2	2	1	1	0	0	0	0	1	1	**8**
	B	2	1	1	1	0	0	0	0	1	1	**7**
	C	0	1	0	0	0	0	0	0	0	0	**1**
Qld	Total	5	12	8	3	16	15	8	5	5	1	**78**
	B	5	11	4	0	10	8	6	3	1	1	**49**
	C	0	0	4	2	6	6	2	1	4	0	**25**
SA	Total	3	5	3	2	3	3	0	1	2	0	**22**
	B	2	5	2	2	1	1	0	0	1	0	**14**
	C	0	0	1	0	2	2	0	1	1	0	**7**
Tas	Total	2	2	1	0	6	3	2	0	0	0	**16**
	B	1	1	1	0	0	0	0	0	0	0	**3**
	C	1	1	0	0	6	3	2	0	0	0	**13**
Vic	Total	6	16	6	6	12	6	15	4	5	1	**77**
	B	5	11	3	3	5	2	5	1	1	1	**37**
	C	1	5	3	3	6	4	10	3	3	0	**38**
WA	Total	8	9	5	3	4	0	2	1	0	0	**32**
	B	7	7	4	3	3	0	1	1	0	0	**26**
	C	0	2	1	0	1	0	1	0	0	0	**5**
Australia	n	47	62	29	21	54	35	45	21	20	4	**338**
	%	13.9	18.3	8.5	6.2	15.9	10.5	13.3	6.2	6	1.2	
Serogroup B	n	41	47	18	13	26	16	23	12	6	4	**206**
Australia	%	20	22.8	8.7	6.3	12.6	7.8	11.1	5.9	3	1.5	**61**
Serogroup C	n	4	14	11	7	27	18	22	7	12	0	**122**
Australia	%	3.2	11.4	9	5.7	22.1	14.7	18	5.7	9.9	0	**36**
Other Australia	n	2	1	0	1	1	1	0	2	1	1	**10**

Table 3. Commonly isolated serotypes and serosubtypes and phenotypes of *N. meningitidis* of interest, Australia, 2001, by State or Territory

	Serogroup B				Serogroup C			
	Serotype	n	Serosubtype	n	Serotype	n	Serosubtype	n
ACT	4	2	1.4	2				
	14	1	1.4	1				
			1.6	1				
NSW	4	35	1.4	22	2a	28	1.5	16
			1.7	1			1.5,2	2
			1.14	4			1.2	2
			1.15	1			1.4	2
			1.13	1			nst	6
		nst†	4		2b	0		
	15	5	1.7	3	nt*	3	1.15	1
	1	3	1.5	1			1.16	1
			1.14	1	1	2	1.14	2
	nt*	21	1.4	6				
			1.5,2	1				
			nst	6				
NT	4	3	1.4	3	2a	1	1.5,2	1
	15	1	1.15	1				
	14	3	nst	3				
Qld	4	4	1.4	4	2a	19	1.5	8
	15	8	1.7	5			1.5,2	4
	1	5	1.14	3			1.4	4
	nt	32	1.4	12	2b	1	1.2	1
			1.14	3	nt	5	1.15	4
Vic	4	4	1.4	3	2a	37	1.5	3
	15	8	1.7(16)	5			1.2	2
			nst	2			1.4	19
	2b	2	1.16	1			nst	13
	nt	22	1.4	7	2b	1	nst	1
			1.15	4				
			nst	8				
SA	4	4	1.4	2	2a	2	1.4	2
	15	3	1.7	2	1	3	1.14	3
	14	1	nst	1				
	1	2	1.14	1	nt	2	1.16	1
	nt	2	1.4	1			nst	1
			1.15	1	2b	0		
Tas					2a	12	1.5,2	11
							1.4,7	1
WA	4	0			2a	4	1.5	4
	15	3	1.7(16)	3	2b	0		
	nt	23	1.4	7	nt	1	1.15	1
			1.15	4				
			nst	7				

* Not typeable.

† Not serosubtypeable.

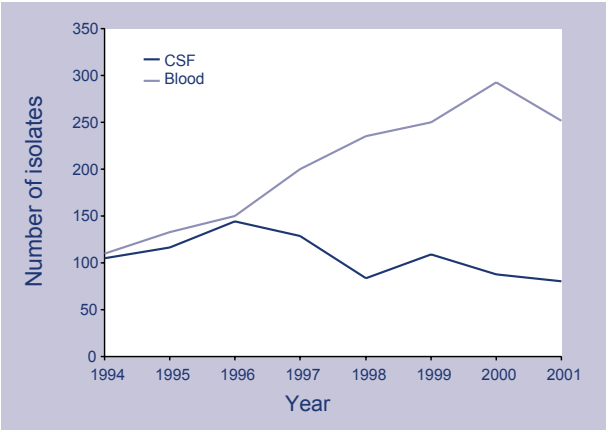
There was again considerable phenotypic heterogeneity amongst invasive isolates as determined by serotyping and serosubtyping. The predominant serotypes/serosubtypes in each State and Territory are shown in Table 3. Serogroup B meningococci are more difficult to characterise by serological methods and a number could not be phenotyped. B:4:P1.4(7) strains predominated in New South Wales and were also present in Queensland, South Australia and Victoria. B:15:P1.7 strains were present in New South Wales, Queensland, Victoria, and Western Australia.

There was less heterogeneity amongst serogroup C meningococci. Isolates were usually serotype 2a. Phenotype C:2a:P1.4(7), which appeared in Victoria in 1999, requires special comment. There were 10 such isolates in Victoria in 1999, 24 in 2000 and 19 in 2001. This phenotype remains uncommon elsewhere in Australia. Phenotype C:2a:P1.2 was also frequently isolated in Victoria in 2000 (10 isolates) but only 2 strains of this phenotype were seen in Victoria in 2001. This phenotype was also rarely identified in other centres. New South Wales was the other state where serogroup C strains were present in larger numbers. Phenotypes C:2a:P1.5 and C:2a:P1.5,2 accounted for 70 per cent of the 33 serogroup C strains isolated there. The C:2a:P1.5 phenotype was present in most jurisdictions and 11 of a total of 16 isolates in Tasmania were of this phenotype. Serotype 2b strains were rarely encountered.

Site of isolation

There were 80 isolates from CSF either alone or with a blood culture isolate and 252 from blood cultures alone. There were 5 isolates from synovial fluid and one from skin. Trends in the relative number of isolates from CSF and blood are shown in Figure 5. The ratio of CSF isolates to blood culture isolates was 0.31:1, similar to that recorded in 2000.

Figure 5. Numbers of meningococcal isolates from CSF and blood culture, Australia, 1994 to 2001



Outcome data for cases with sterile site isolates

Outcome data (survived or died) were available for 180 patients (53%). Twenty-three deaths were recorded (12.7%) (Table 4). Outcomes were available in 53 per cent of both serogroup B infections and serogroup C infections.

Table 4. Outcome of meningitic and septicaemic cases of meningococcal infection, culture positive cases, Australia, 2001, by serogroup

Disease type	Outcome	Serogroup					Total
		B	C	Y	W135	NG	
Meningitis	Survived	24	11	0	0	0	**35**
	Died	2	3	0	0	0	**5**
	Total	26	14	0	0	0	**40**
Septicaemia	Survived	77	35	3	2	0	**117**
	Died	5	13	0	0	0	**18**
	Total	82	48	3	2	0	**135**
All cases*	Total	110	65	3	2	0	**180**
	Died	7	16	0	0	0	**23**

* Includes 3 serogroup C and 2 serogroup B isolates from joint aspirates from patients who survived.

There were 7 (6.3%) deaths in patients with serogroup B infections and 16 (24%) in patients with serogroup C infections.

Where outcomes were known, there were 5 deaths in 40 patients (12%) with meningitis. Two of these patients were infected with serogroup B, and 3 with a serogroup C strain.

Eighteen deaths were recorded in 135 bacteraemic patients (13%). There were 82 cases of serogroup B meningococcal bacteraemia with 5 deaths (6%) and another 48 cases were caused by serogroup C strains among whom 13 fatalities were recorded (27%). No fatalities were recorded with serogroup Y or W135 bacteraemias.

Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillin

All of the 338 isolates were tested for their susceptibility to penicillin. Using defined criteria, 112 strains (33%) were fully sensitive to penicillin and 226 (67%) less sensitive (MIC 0.06 to 0.5 mg/L). These proportions differ only slightly from those recorded in recent years. The highest MIC was 0.5 mg/L, recorded for 2 isolates.

Other antibiotics

All isolates were susceptible to ceftriaxone (and by extrapolation to other third generation cephalosporins). Three isolates were resistant to the prophylactic antibiotic rifampicin at 1 mg/L but all isolates were susceptible to ciprofloxacin.

Numbers and sources of non-culture diagnoses of invasive meningococcal disease

One hundred and thirty-five additional cases of invasive meningococcal disease were diagnosed by non-culture methods in 2001. There were 137 diagnoses of invasive meningococcal disease where PCR and/or serology were positive in the absence of positive cultures (Table 5). In two instances where both serology and PCR testing were performed, both tests were positive. However, it was more usual to have available samples suitable for testing by only one of the above techniques. Thus there were 107 instances where PCR testing in isolation was positive and 28 cases where serology testing alone was positive.

With PCR testing it was also possible to categorise the disease type by source of specimen in a manner similar to that used for culture-positive cases (Table 5). Of the 109 cases positive by PCR, 56 were from CSF or CSF and blood, 52 from blood only and one from another site. This is a different distribution from that obtained with culture-based diagnosis. Culture-based diagnosis of blood yielded 2.5 times the number of positive cultures compared with cultures of CSF. With PCR based diagnosis equal numbers of positive results were obtained from blood and CSF.

Serogroup and age distribution of non-culture based invasive meningococcal disease

In addition to diagnostic PCR, this technique can also be used to ascertain the serogroup involved in the disease process. In most centres this is still restricted to serogroup B and C determinations. There were 109 cases where a PCR-based diagnosis was made and in 84 of these the serogroup was also determined (Table 6).

For those cases diagnosed by serology alone, age distribution was different with most diagnoses (25/28) occurring in those aged 10 years or more (Table 7). This reflects in part the difficulty in obtaining serum samples from young children. The categorisation of IMD by site of organism capture cannot be determined in those diagnosed by serological methods. Additionally, serogroup determination by serological testing was not possible in 2001, although this will be available for serogroup C cases from 2002.

Outcome data for invasive meningococcal disease based on non-culture-based diagnosis

For IMD diagnosed by PCR-based tests, the outcome was known in 47 instances, with 5 deaths (10.6%). There were 3 deaths where blood PCR alone was positive (2 of serogroup B and one serogroup C). There were two instances of deaths (one each of serogroup B and C) where PCR was positive on a CSF sample. Twenty-seven of 28 cases diagnosed serologically survived and the outcome was unknown in the remaining case.

Table 5. Source of non-culture-based diagnosis of invasive meningococcal disease, Australia, 2001

Diagnostic method	Number
All non-culture-based diagnoses	137
PCR positive*	109
CSF PCR positive	48
CSF and blood PCR both positive	8
Blood PCR positive	52
Other	1
Serology positive in the absence of positive PCR	28

* Including those with positive serology.

Table 6. Serogroup and age distribution of invasive meningococcal disease diagnosed by PCR, Australia, 2001

	Age group (years)										
Group	**<1**	**1–4**	**5–9**	**10–14**	**15–19**	**20–24**	**25–44**	**45–64**	**64+**	**Unknown**	**Total**
B	7	12	5	2	6	9	5	4	1	1	**52**
C	3	6	2	0	6	5	3	5	0	1	**31**
Y	0	0	0	0	1	0	0	0	0	0	**1**
U*	5	5	5	2	1	2	1	4	0	0	**25**
All	**15**	**23**	**12**	**4**	**14**	**16**	**9**	**13**	**1**	**2**	**109**

* Undetermined

Table 7. Age distribution of serologically diagnosed cases of invasive meningococcal disease, Australia, 2001

Age group (years)	<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	>65	Total
Cases	1	0	3	3	6	4	8	2	1	**28**

Discussion

The total of 338 isolates examined by the NNN laboratories in the Australian Meningococcal Surveillance Programme in 2001 was less than the 388 examined in 2000. The number of isolates examined by the NNN from 1994 until 2000 has ranged between 323 and 388. When data are disaggregated by jurisdiction however, differences become more apparent. The number of isolates available in Victoria increased from 41 in 1998 to 94 in 1999 and 108 in 2000, but declined to 77 in 2001. In contrast, the number of isolates from Queensland decreased from 81 in 1998 to 66 and 43 in 1999 and 2000 respectively, then increased to 78 in 2001. Isolate numbers in New South Wales and Western Australia decreased in 2001 but in other centres varied little from 2000 totals.

It is difficult to be certain of the reliability of these numbers of positive cultures if used as an index of trends in disease rates. The number of isolates available for examination will always be less than the number of clinically notified cases because clinical surveillance case definitions include culture-negative cases. The number of culture-negative cases will vary according to the implementation of and adherence to the 'early treatment' practices now advocated for management of IMD. These culture-negative cases are increasingly confirmed by other methods, but the introduction and use of non-culture-based diagnostic methods has varied in different jurisdictions over time. For these reasons, there should be some wariness in comparing trend data from different states and territories in recent years. Nevertheless in 2001, 137 cases were confirmed by non-culture-based methods and when these were added to the 338 positive cultures, a total of 475 laboratory-confirmed cases was recorded. In 2000, 147 clinical cases were confirmed only by non-culture-based laboratory examinations and there were a further 388 culture-positive cases giving a total of 535 laboratory-confirmed cases.

The ratio of cases of meningitis to bacteraemia in culture-confirmed cases (0.31:1) maintained an existing trend (Figure 5). Previous reports also noted differences in meningitis/septicaemia rates when these were derived from culture-based and non-culture-based methods. This trend was the subject of comment in earlier reports.[6,7] It was noted that the initial introduction of PCR-based diagnosis saw positive CSF samples representing 2.5 times the number of diagnoses from blood. It was suggested that a possible bias arises insofar as PCR was initially more often performed only on CSF samples and that the sensitivity of PCR techniques in blood samples is less than that for CSF. In the 2000 data, there was an increase in PCR based diagnoses from blood and a corresponding 'correction' in the proportions of diagnoses from CSF and blood by this technique. In 2001 the numbers diagnosed from each source by PCR were again almost identical.

The predominant disease pattern throughout the country remained sporadic infection with serogroup B meningococci. The proportion of serogroup C cases in aggregated data (36%) was essentially unchanged from 2000 (37%) after increasing from 25 per cent in 1998 and 32.5 per cent in 1999. Serogroup C were the majority of strains isolated in Tasmania and Victoria but serogroup C represented about one third of cases in New South Wales, Queensland and South Australia. In Western Australia and the Territories, serogroup C was uncommonly encountered. While serogroup C infections have been prominent in Victoria and New South Wales for a number of years, their numbers in Tasmania in 2001 represent a significant change and their proportions in both Queensland and South Australia also increased. No serogroup A meningococci were isolated and the proportion of serogroup Y and W135 strains declined.

In 2001, the age distribution of IMD was typical with children aged 4 years or less the most frequently infected. A secondary peak in incidence in young adults and adolescents was also observed. By contrast, in 2000, those in the 15–24 age group had more infections than those aged 4 years or less in aggregated data. The larger case numbers in young adults in New South Wales and Victoria influenced this aspect of meningococcal disease patterns in 2000. However, this pattern again varied by region in 2001 with Queensland and Tasmania having the highest proportion of young adult cases while New South Wales and Victoria returned to the usual pattern. Serogroup B infections were the most frequently seen in the infant age group whereas serogroup C isolates continued to be over-represented in the young adult age group (Figures 1, 2, 3 and 4).

Phenotyping data emphasise the considerable heterogeneities that exist in meningococcal subtypes causing IMD in different jurisdictions. The group B phenotype B:4:P1.4(7), associated with hyperendemic disease in New Zealand for many years, is of more than academic interest. A trial of a porin-based vaccine for infections with serogroup

B:4:P1.4 strains will commence shortly in New Zealand so the presence of this strain in Australia warrants particular attention. In New South Wales this phenotype represented at least 20 per cent of all isolates, although it was not as prominent in other jurisdictions. Because a proportion of serogroup B strains are normally not typable with monoclonal antibodies, use of por gene sequencing techniques may be required to establish the real incidence of infection due to this subtype. Phenotype B:15:P1.7 remained widely distributed.

In 1999 the group C phenotype C:2a:P1.4(7) was detected in Victoria, accounting for much of the increase in serogroup C disease in that State at that time. This phenotype persisted in 2000 and represented about 22 per cent of all isolates. In 2001, about 25 per cent of all isolates in Victoria were C:2a:P1.4(7), although their number declined. In contrast, in Tasmania phenotype C:2a:P1.5,2 predominated (11 of 16 isolates) and only a single strain of the 'Victorian' phenotype was identified. Elsewhere C:2a:P1.5 was more common. The 'Victorian' phenotype was found infrequently in other states. This considerable temporal and geographic variation in meningococcal subtypes and the volatility in the predominant phenotypes is well-recognised and is a result of genetic recombination through horizontal gene transfer.[12]

Mortality data was assessable in only 180 culture positive cases and the 23 deaths, giving a mortality of 12.7 per cent, may not accurately represent the true situation. Although a higher mortality was observed for serogroup C infections, other factors such as age, and time from onset to presentation and treatment, on which data were not available, may also explain the difference between outcomes due to different serogroups. The 5 fatal cases recorded from another 47 cases of PCR-based diagnoses was slightly less in percentage terms and the rate was not different between serogroups. Serologically diagnosed cases are by their nature milder and in patients who by definition survive long enough to mount a detectable antibody response. The combined mortality for all serogroups was 11 per cent.

No invasive isolates resistant to penicillin were detected in 2001. The highest MIC recorded was 0.5 mg/L in 2 isolates and the proportion of 'less susceptible' strains was unchanged. All isolates were susceptible to the third generation cephalosporins and the prophylactic agent ciprofloxacin. Occasionally isolates resistant to rifampicin are encountered and in 2001, 3 strains were resistant at 1 mg/L.

Since 1994, the NNN has examined over 2,500 strains from all states and territories. There has been a continuing evolution and development of laboratory techniques over this period so that it is not always possible to make full comparisons of data gathered in different years. Additionally, the NNN data are used to supplement information collected separately by clinically based surveillance of invasive meningococcal disease. Within this context however, the NNN data remain an essential component of IMD surveillance in Australia. Accurate data are needed to allow informed responses by those responsible for individual and public health management of IMD. The recent release of conjugate serogroup C vaccines reinforces this need. For further details the relevant NNN member in each jurisdiction should be contacted.

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Isolates were received in the reference centres from many laboratories throughout Australia. The considerable time and effort involved in forwarding these strains is recognised and these efforts are greatly appreciated. These data could not have been provided without this assistance and the help of clinical colleagues and public health personnel.

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Annual report of the Australian National Poliovirus Reference Laboratory and summary of acute flaccid paralysis surveillance, 2001

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Abstract

The National Poliovirus Reference Laboratory at the Victorian Infectious Diseases Reference Laboratory (VIDRL) is responsible for poliovirus testing for Australia and the Pacific Island countries. It is also a regional reference laboratory for the Western Pacific Region of the World Health Organization. Surveillance for acute flaccid paralysis, a clinical manifestation of poliomyelitis, is coordinated at VIDRL in collaboration with the Australian Paediatric Surveillance Unit. There were 60 unique notifications of acute flaccid paralysis (AFP) in 2001, of which 44 were classified by the polio expert committee as eligible non-polio AFP cases, that is, from patients resident in Australia and aged less than 15 years. Polioviruses were isolated from one AFP patient and characterised as Sabin oral poliovirus vaccine-like for all 3 serotypes. In the same period, the National Poliovirus Reference Laboratory identified 40 Sabin-like viruses from 74 referred isolates and specimens, and an additional five non-Sabin-like polioviruses as part of the laboratory containment of poliovirus. The Western Pacific Region, of which Australia is a member nation, was declared free of circulating wild poliovirus in October 2000. However, during 2001, viruses derived from the Sabin oral poliovirus vaccine caused 3 cases of poliomyelitis in the Philippines, also a member nation of the Western Pacific Region. The identification of circulating vaccine-derived poliovirus in the Philippines has emphasised the necessity of maintaining a high level of vaccination coverage within Australia and an effective surveillance system to detect cases of poliomyelitis. *Commun Dis Intell* 2002;26:419-427.

Keywords: poliovirus, vaccine-derived poliovirus, acute flaccid paralysis, surveillance

Introduction

The National Poliovirus Reference Laboratory at the Victorian Infectious Diseases Reference Laboratory (VIDRL) is responsible for the characterisation of all poliovirus isolates in Australia. This includes Sabin oral polio vaccine-like viruses, isolated incidentally from nasopharyngeal aspirates taken from recently immunised infants when investigating other illnesses such as bronchiolitis. The laboratory is also responsible for testing stool samples from all patients with acute flaccid paralysis (AFP) in Australia.

Acute flaccid paralysis is the major clinical manifestation of poliovirus infection, occurring in 0.1 per cent to one per cent of infections. Clinical surveillance for cases of AFP and the subsequent laboratory testing of faecal specimens for poliovirus will also detect cases of poliomyelitis caused by imported wild-type poliovirus, circulating vaccine-derived poliovirus (cVDPV) or vaccine-associated paralytic poliomyelitis (VAPP). In Australia, AFP surveillance with laboratory support was initiated in March 1995 to meet the certification standards of the World Health Organization (WHO) poliomyelitis eradication program[1] and since 2000, has been coordinated at VIDRL in collaboration with the Australian Paediatric Surveillance Unit (APSU).

In 1994, the World Health Organization declared the region of the Americas to be free of circulating wild poliovirus. This was followed by a similar declaration for the Western Pacific Region in October 2000 and represented a milestone in the WHO program for the global eradication of poliomyelitis.[2] Despite these achievements, outbreaks of poliomyelitis have recently occurred in both regions due to viruses derived from the Sabin oral polio vaccine.[3,4,5] This has focussed attention on the need for quality surveillance and extensive characterisation of all poliovirus isolates.

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Methods

AFP surveillance

The approach adopted in Australia for AFP surveillance has been presented in detail elsewhere[1] and is briefly outlined here. Any paediatrician seeing a patient aged less than 15 years and presenting with AFP is requested to notify the surveillance coordinator by telephone, complete a detailed questionnaire and arrange for the collection of 2 faecal samples 24 hours apart and within 14 days of the onset of paralysis. A follow-up questionnaire requesting clinical details 60 days after the onset of paralysis is required for cases where a definitive diagnosis cannot be made from the first questionnaire and available laboratory results. In addition, all paediatricians are asked to return a reply-paid survey card to the APSU each month, indicating the number of patients seen with a range of rare conditions, including AFP.

The Polio Expert Committee, appointed by the Australian Commonwealth Department of Health and Ageing, reviews all notifications of AFP. Cases may be classified as poliovirus due to wild poliovirus, VDPV or VAPP; non-polio AFP; or non-AFP. Australia's population aged under 15 years was estimated at approximately 3,922,000 in 2001. In order for Australia to fulfil the WHO surveillance target of one AFP case per 100,000 population in this age group,[6] 40 notifications of AFP cases would have been expected during 2001.

Laboratory investigations

The National Poliovirus Reference Laboratory at VIDRL characterises all viruses isolated from AFP cases and all incidental isolations of polioviruses, for instance, viruses isolated from the nasopharynx of infants following vaccination. Results of laboratory investigations in this report are for tests performed in the calendar year 2001, irrespective of the date of notification of an AFP case or receipt of a referred incidental poliovirus. The laboratory also tests untyped enteroviruses referred from other Australian laboratories.

All polioviruses isolated by cell culture are tested to determine whether they are Sabin or non-Sabin-like. The WHO global network of poliovirus reference laboratories uses standardised methods for enzyme-linked immunosorbent assay (ELISA), nucleic acid probe hybridisation and polymerase chain reaction (PCR) for the intratypic differentiation of the 3 serotypes of Sabin and wild-type polioviruses. To maintain standards, all laboratories within the WHO poliovirus global network undergo accreditation on an annual basis and participate in quality assurance programs.

Intratypic differentiation of poliovirus isolates

Nucleic acid probe hybridisation and PCR are directed to highly conserved regions of the poliovirus genome and differentiate the 3 poliovirus serotypes from one another and Sabin-like from non-Sabin-like viruses. Differentiation of viruses by the ELISA is based on polyclonal antisera to polioviruses. The ELISA test is capable of detecting minor amino acid changes to the antigenic regions within the poliovirus capsid protein. A Sabin virus that has accumulated mutations may result in a VDPV with a test result of non-Sabin-like by ELISA but Sabin-like by the nucleic acid probe hybridisation and PCR tests. WHO laboratory protocols require all polioviruses isolated on or after 1 January 2001 to be tested by two methods of intratypic differentiation, one of which must be the ELISA.

Viral isolation and neutralisation

Faecal specimens are transported cold to the poliovirus reference laboratory to ensure virus viability. Specimens are extracted in chloroform and cell culture media and added to a panel of continuous cell lines. The main cell line for isolation of poliovirus is L20B, a mouse fibroblast line with a stable genetic integration of the poliovirus receptor.[7] Other cell lines, including RD-A (human rhabdomyosarcoma) and HEp2C (human epithelium carcinoma) are used for the isolation of non-polio enteroviruses as well as polioviruses. Identification of both polioviruses and non-polio enteroviruses is confirmed by antisera neutralisation.

Nucleic acid probe hybridisation

Inactivated viral ribonucleic acid (RNA) is immobilised on nylon membranes and individually tested with two digoxigenin-labelled oligonucleotide probes.[8] One probe is specific for a highly conserved sequence within the 5' non-translated region of enteroviruses and the other a Sabin serotype specific probe directed to the VP1 gene. A positive result with both probes indicates the isolate is Sabin-like, while a positive result only with the enterovirus probe may indicate a wild-type poliovirus.

Polymerase chain reaction and nucleotide sequencing

An alternative to the nucleic acid probe hybridisation method is to amplify viral RNA by PCR. The oligonucleotide primers are directed to the same genomic regions and interpretation of results is comparable to the probe hybridisation method. Sequencing of poliovirus isolates is performed using primers directed to the 5' non-translated region, VP1 and 3D subgenomic regions. Sequencing of non-polio enteroviruses is based on the method of Oberste.[9]

Enzyme-linked immunosorbent assay

The poliovirus ELISA differentiates between Sabin and wild-type strains using serotype specific antisera to intact virus particles of one strain that have been cross-adsorbed against the heterologous strain.[10] Results may indicate a Sabin-like virus, a non-Sabin-like virus or one with properties common to both strains (double reactive), arising from antigenic drift due to mutation of the Sabin virus genome.

Results

AFP surveillance in Australia

During 2001, 81 AFP notifications from 60 cases were received and reviewed by the Polio Expert Committee. Forty-six (76%) cases were first notified to the National Poliovirus Reference Laboratory at VIDRL with the balance notified through the APSU monthly reporting system. There were no cases of poliomyelitis and 44 cases were classified as non-polio AFP occurring among patients aged less than 15 years and resident in Australia (Figure 1). Other notifications were duplicates; from cases aged 15 years or more; from non-Australian residents; with date of paralysis onset in 2000; or were classified as non-AFP. As in previous years, Guillain-Barré syndrome was the most common single diagnosis, accounting for almost one third of all AFP cases (Table 1).

The WHO has defined AFP surveillance targets and Australia's AFP surveillance for 2001 is compared with these targets in Table 2. In 2001, the target of one notified and confirmed case per 100,000 population aged less than 15 years was exceeded (1.1/100,000 population). However, the proportion of cases with adequate stool samples was still below target, with only 36 per cent of notified cases having adequate stool samples, compared with a target of at least 80 per cent. Summaries of Australia's AFP surveillance from 1995 to 2001 are given in Table 3 and Figure 2. Figure 2 demonstrates the increasing proportion of duplicate notifications in the 7 years of surveillance.

Table 1. Classification of all cases notified through AFP surveillance, Australia, 2001

Case classification by Polio Expert Committee	Number	Per cent of non-polio AFP cases	Per cent of all cases
Polio AFP	0		
Non-polio AFP diagnosed as:	44		73
Guillain-Barré syndrome	14	32	
Transverse myelitis	6	14	
Acute demyelinating encephalomyopathy	3	7	
Non-polio enterovirus*	9	20	
Infant botulism	2	4	
Other	10	23	
Non-AFP	2		3
Non Australian resident/age >15 years/onset in 2000	10		17
Insufficient information for classification	4		7
Total cases	**60**		**100**

* Non-polio enterovirus isolations at the National Poliovirus Reference Laboratory: Coxsackie A24 = 1; Echovirus 11 = 2; Enterovirus 71 = 1; Enterovirus 71 identified in other laboratory = 5.

Figure 1. AFP notifications and testing of stool samples from AFP cases at the National Poliovirus Reference Laboratory, 2001

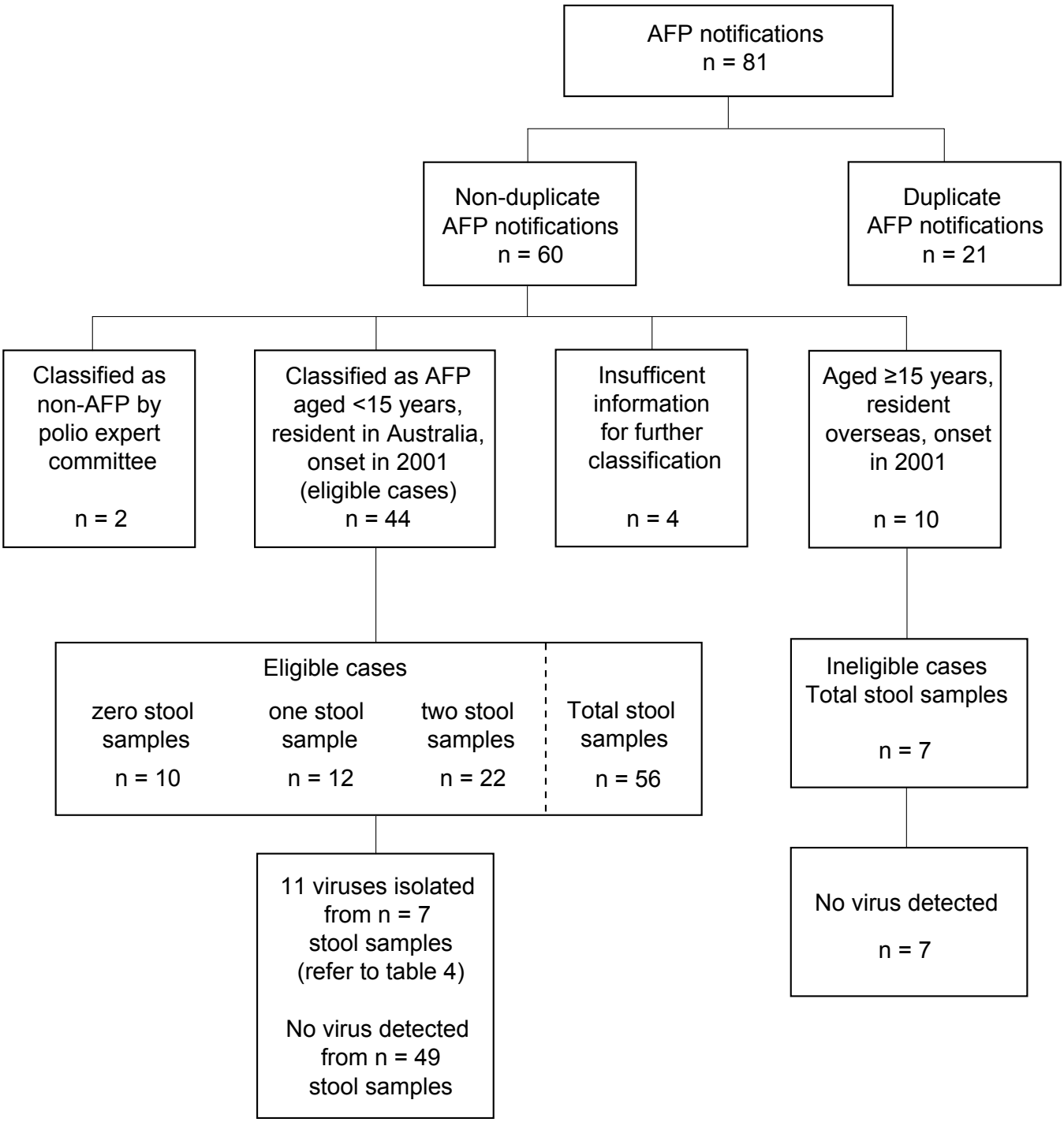


Table 2. AFP surveillance in Australia, 2001, compared with WHO indicator targets

WHO recommendation	Indicator	AFP surveillance 2001
Non-polio AFP cases per 100,000 population aged < 15 years	1/100,000	48 notified - 1.2/100,000 population 44 classified by the Polio Expert Committee 1.1/100,000 population
Percentage of routine surveillance sites that provide routine reports including (zero reports) on time	>80%	98% (completed monthly reports from the Australian Paediatric Surveillance Unit)
Percentage of AFP cases that are investigated	>80%	92%
Percentage of AFP cases that are investigated within 48 hours of notification	>80%	57% (investigated for questionnaire completion and stool collection within 48 hours of notification)
Percentage of AFP cases with a follow-up examination for residual paralysis at 60 days after the onset of paralysis	>80%	92% (only cases whose diagnosis and outcome could not be established by the initial questionnaire and laboratory tests required a 60 day follow-up)
Percentage of AFP cases with 2 adequate stool samples collected at least 24 hours apart within 14 days of onset of paralysis	>80%	36%

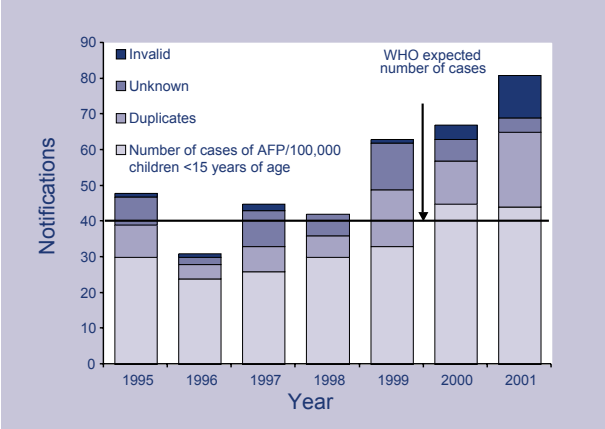
Table 3. Australia's AFP surveillance compared with WHO targets, 1995 to 2001

Year(s)	WHO target number of AFP cases detected per year in Australia and classified as eligible by the Polio Expert Committee*	Total number of Australian resident cases ascertained and classified by the Polio Expert Committee (per cent of target)	Proportion of cases with 2 stools, collected at least 24 hours apart and within 14 days of the onset of paralysis, tested for the exclusion of wild poliovirus at the National Poliovirus Reference Laboratory (WHO target > 80%)
1995-1999†	195 (average 39)	148 (76%)	25%
2000	39	45 (115%)	31%
2001	40	44 (110%)	36%

* An eligible case has acute flaccid paralysis, is aged <15 years and is resident in Australia. A minimum of 39 cases should be found in Australia per year, equivalent to 1/100,000 resident population aged <15 years.

† Data from the 7th Annual report of the Australian Paediatric Surveillance Unit.

Figure 2. Classification of notified acute flaccid paralysis cases, Australia, 1995 to 2001



Laboratory investigations

The origin of viruses isolated from AFP cases incident in 2001, is shown in Figure 1 and the origin of all isolates referred to the National Polio Reference Laboratory in 2001 is shown in Figure 3.

AFP cases

No wild poliovirus was detected in any AFP case tested in 2001. Sabin vaccine-like poliovirus serotypes 1, 2 and 3 were isolated from 2 faecal specimens of a single patient. No significant nucleotide sequence variation was detected between each of the poliovirus and corresponding Sabin vaccine serotypes. *Clostridium botulinum* type B organism and toxin were also detected from this patient at the Women's and Children's Hospital, Adelaide, and the case was classified as infant botulism. Enterovirus 71 (EV71) was isolated from 3 cases, echovirus 11 from 2 cases, Coxsackie A24 from one case and an untyped adenovirus from another case. Two of the EV71 viruses were isolated in 2001 from AFP cases with onset late in 2000. After 14 days of culture no virus was detected from the remaining 61 specimens, including stool specimens from cases incident in 2000 (Table 4).

Referred enterovirus isolates

Seventy-four viruses were identified from 79 referred isolates (Table 4). These included 40 polioviruses, generally recovered from recently immunised infants, all of which were Sabin-like. Thirteen of 27 non-polio enteroviruses referred to the National Poliovirus Reference Laboratory for identification, were characterised as EV71, Coxsackie A9 and echovirus serotypes 6, 9 and 13 (Table 4). Five uncharacterised polioviruses, referred to VIDRL for intratypic differentiation from an Australian laboratory following a review of stored untyped poliovirus isolates as part of poliovirus laboratory containment, were characterised as non-Sabin-like serotype 2. Table 5 summarises the laboratory activities from 1995 to 2001.

Figure 3. Isolates referred to the National Poliovirus Reference Laboratory, 2001

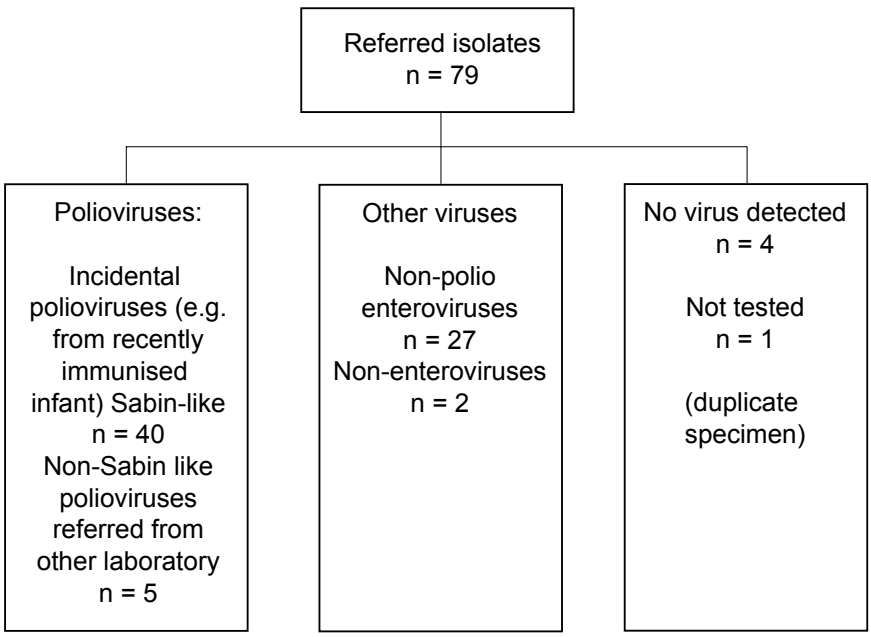


Table 4. Results of National Poliovirus Reference Laboratory testing performed, Australia, 2001

Isolation result	Isolates from AFP cases		Referred enterovirus isolates*	Total isolates
	< 15 years	≥ 15 years		
Poliovirus Sabin-like type 1†	2	0	14	**16**
Poliovirus Sabin-like type 2†	2	0	10	**12**
Poliovirus Sabin-like type 3†	2	0	16	**18**
Poliovirus non-Sabin-like type 3‡	0	0	5	**5**
Adenovirus type 5	0	0	1	**1**
Adenovirus (untyped)	1	0	0	**1**
Coxsackie A9	0	0	1	**1**
Coxsackie A24	1	0	0	**1**
Echovirus 6	0	0	6	**6**
Echovirus 9	0	0	2	**2**
Echovirus 11	2	0	0	**2**
Echovirus 13	0	0	2	**2**
Enterovirus 71§	3	0	2	**5**
Non-polio enterovirus	0	0	14	**14**
Rhinovirus	0	0	1	**1**
No virus detected after 14 days culture‖	61	7	4	**72**
Not tested¶	2	0	1	**3**
Total	**76**	**7**	**79**	**162**

* Includes polioviruses isolated incidentally from recently immunised infants.

† Three poliovirus serotypes in each of 2 specimens.

‡ Isolates referred as part of laboratory containment of poliovirus.

§ EV71 isolated from 2 cases with onset in late 2000 and one with onset in 2001.

‖ Includes specimens from patients with onset in 2000 but tested in 2001.

¶ Inappropriate specimens (e.g. urine).

Table 5. Summary of enterovirus testing at the National Poliovirus Reference Laboratory, Australia, 1995 to 2001

Year	Poliovirus		Non-polio enterovirus	Non-enterovirus detected or no virus detected	Total isolates tested
	Sabin-like	Non-Sabin-like			
1995	190	0	200	13	**403**
1996	224	0	198	9	**431**
1997	124	0	76	0	**200**
1998	52	0	15	4	**71**
1999	60	1	9	9	**79**
2000*	45	0	44	47	**136**
2001	46	5	33	75	**159**

* From 2000, Australian laboratories have been referring untyped enteroviruses and undifferentiated poliovirus as part of the laboratory containment of poliovirus.

Quality assurance, accreditation and training

The National Poliovirus Reference Laboratory at VIDRL retained its full accreditation status after a review by a representative of the WHO's Vaccine Assessment and Monitoring Team in August 2001. Proficiency panels for the techniques of poliovirus isolation and serotyping from faecal samples, nucleic acid probe hybridisation, ELISA and diagnostic PCR were also successfully completed in 2001. During November 2001, in collaboration with the polio reference laboratory from the Netherlands, VIDRL hosted an ELISA workshop with participants from the poliovirus laboratories of China, Hong Kong, Japan, New Zealand, the Philippines and Singapore.

Regional surveillance conducted by the National Poliovirus Reference Laboratory

More than 400 enteroviruses, including 285 polioviruses, were characterised in the laboratory's role as a regional reference laboratory. The laboratory was involved in the isolation and characterisation of 3 cVDPV isolates from the Philippines. All cVDPV isolates demonstrated a non-Sabin-like reaction in the poliovirus ELISA. Nucleotide sequencing of the isolates revealed they were derived from the Sabin oral polio vaccine, with more than 3 per cent nucleotide sequence variation in the VP1 gene and up to one per cent variation between the isolates. A recombination event had occurred within the non-capsid region with a non-polio enterovirus in all 3 viruses.

Discussion

AFP surveillance in Australia

Australia was certified free of circulating endemic wild poliovirus in 2000, although the last case of poliomyelitis in Australia due to an endemic infection was in the early 1970s.[11] Australia achieved the WHO target for notification of AFP cases (prospectively) for the first time in 2000.[12] Retrospective reviews of hospital records had to be undertaken in order for Australia to reach the target necessary for certification.[13,14] In 2001, Australia was again able to achieve the AFP surveillance target (prospectively). The AFP surveillance system has improved steadily since 1996.

Guillain-Barré syndrome remains the major cause of AFP in countries that are not poliovirus endemic. However, other viruses such as enterovirus 71 can cause AFP. Enterovirus 71 is a common cause of hand, foot and mouth disease but has also been associated with neurological disease including encephalomyelitis. Some cases of hand, foot and mouth disease with neurological complications have had a similar clinical presentation to poliomyelitis. Large outbreaks, which included fatal cases as a result of enterovirus 71 infection with severe neurological disease, have recently occurred in Taiwan[15] Malaysia,[16] Singapore[17] and Western Australia.[18,19]

AFP surveillance has also highlighted the occurrence of infant botulism in Australia, with 3 cases identified in 2000 and a further 2 cases in 2001. Poliovirus was isolated incidentally from the faeces of three of these 5 cases. Since infant botulism occurs during the first 12 months of life, this may be around the time of the administration of the oral polio vaccine. The similarity of presenting symptoms makes it imperative to differentiate infant botulism from VAPP.

Uncharacterised polioviruses identified as non-Sabin-like

Five non-Sabin-like poliovirus isolates were identified amongst a collection of uncharacterised polioviruses referred to the reference laboratory in 2001. This highlights the importance for all laboratories to undertake a thorough examination of the contents of their freezers to identify any material (biological or environmental) that potentially contain poliovirus. The WHO strategy for the post-eradication phase of poliomyelitis is for the global containment of all polioviruses in specified laboratories prior to cessation of poliovirus immunisation. The finding of previously unsuspected non-Sabin like polioviruses in one Australian laboratory highlights the importance of following WHO guidelines for the containment of poliovirus. Any laboratory that has material no longer required but potentially containing poliovirus or untyped enteroviruses should destroy the material by incineration or refer the samples to the National Poliovirus Reference Laboratory. If the material is still required, an aliquot should be referred to the reference laboratory for testing.

Implications for Australia of AFP surveillance in other Western Pacific Region countries

The circulating vaccine-derived serotype 1 polioviruses isolated from the Philippines were determined to have more than 3 per cent nucleotide substitutions within the VP1 gene compared to the parental Sabin sequence and to have undergone a recombination event with a non-polio enterovirus in the non-capsid region.[3] These genomic modifications were comparable to those of the cVDPVs isolated from the island of Hispaniola in 2000.[4,5] The two recent cVDPV outbreaks occurred in countries that are part of

WHO administrative regions, the Americas and the Western Pacific, that had been declared free of circulating wild poliovirus. Widespread vaccination programs have interrupted both outbreaks with ongoing surveillance to monitor potential VDPV circulation. However, the experience in these countries underlines the need for maintaining a high coverage of polio vaccination, even where the circulation of wild poliovirus has been eliminated. High quality clinical and laboratory surveillance is important to detect cVDPV, cases of VAPP, and cases of AFP due to imported wild poliovirus. It is for these reasons that Australia must continue surveillance for some years after the world has been certified as free of circulating wild poliovirus.

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We would like to acknowledge Margery Kennett, who established the poliovirus reference laboratory in 1990 and retired in 2001, Ann Turnbull and Aishah Ibrahim for excellent technical assistance, the Australian Paediatric Surveillance Unit for collaboration with AFP surveillance and the Polio Expert Committee for review of AFP cases. We would like to thank all notifying clinicians and all laboratories that have forwarded specimens and Dr Andrew Lawrence, Women's and Children's Hospital, Adelaide, for isolation of *Clostridium botulinum* type B organism and toxin.

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Sentinel Chicken Surveillance Programme in Australia, 1 July 2001 to 30 June 2002

Annette K Broom,[1] Peter I Whelan,[2] Joe Azuolas,[3] Dominic Dwyer,[4] Linda Hueston,[4] John S Mackenzie,[5] Lorna Melville,[6] Scott A Ritchie,[7] David W Smith[8]

Abstract

Detection of flavivirus seroconversions in sentinel chicken flocks located throughout Australia is used to provide an early warning of increased levels of Murray Valley encephalitis (MVE) and Kunjin virus activity in the region. During the 2001/2002 season low levels of flavivirus activity were detected in northern Australia compared to previous years. MVE and Kunjin virus activity was detected in the Kimberley and Pilbara regions of Western Australia and the Northern Territory but not in north Queensland, New South Wales or Victoria. This is in contrast to the previous season when MVE activity was detected both in northern Australia and, for the first time in over 20 years, in New South Wales. Two cases of Murray Valley encephalitis were reported from the north of Western Australia during the 2001/2002 wet season. *Commun Dis Intell* 2002;26:428-429.

Keywords: disease surveillance, Murray Valley encephalitis, kunjin, flavivirus

Introduction

The Sentinel Chicken Surveillance Programme is used to provide an early warning of increased flavivirus activity in Australia. The main viruses of concern are Murray Valley encephalitis (MVE) and Kunjin (KUN) viruses. MVE virus causes the disease Murray Valley encephalitis (formerly known as Australian encephalitis), a potentially fatal disease in humans. Encephalitis is less frequent in cases of Kunjin virus infection and these encephalitis cases have a lower rate of severe sequelae. These viruses are enzootic in the Kimberley region of Western Australia and in the Top End of the Northern Territory and possibly in far north Queensland (Western Cape and Gulf country). They are epizootic in the Pilbara, Gascoyne and Midwest regions of Western Australia, central Australia and in western and central Queensland. MVE virus is also responsible for occasional severe epidemics of encephalitis in south-eastern Australia, the most recent occurring in 1974.

In the northern areas of Australia, MVE and KUN virus activity varies depending on the extent and location of wet season rainfall and flooding in the region. Record rainfall was recorded in the north of Australia during the 1999/2000 wet season and cases of Murray Valley encephalitis were reported from both central Australia and Western Australia. In 2000/2001 MVE activity was detected in New South Wales for the first time since 1974 but no cases were reported. However, cases were reported from central Queensland and central Australia in 2000/2001. Kunjin virus activity was also detected in New South Wales and Victoria in 2000/2001.

MVE and KUN virus activity is monitored in Australia by detecting seroconversions in sentinel chicken flocks.[1] Since 1974, a number of sentinel chicken flocks have been established in 5 Australian states to provide an early warning of increased MVE virus activity. These programs are funded by individual state health departments and each state has a contingency plan, which will be implemented if one or more chickens in a flock seroconverts to MVE virus. From 1992 to 2001 the results of the state sentinel chicken programs were reported bimonthly in *Communicable Diseases Intelligence*. In 2002 important results were posted on the Communicable Diseases Australia website for each State and this report is a brief summary of the results obtained from the individual state programs.

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Currently, 30 flocks are maintained in the north of Western Australia, eight in the Northern Territory, 10 in New South Wales, 10 in Victoria and two in northern Queensland. The flocks in Western Australia and the Northern Territory are sampled and tested all year round but those in New South Wales and Victoria are tested only in the summer months, during the main MVE risk season. Two flocks were established in northern Queensland (Mount Isa and Normanton) early in 2002 after a fatal human case of Murray Valley encephalitis was reported from Mt Isa during the 2000/2001 wet season. These chickens were tested weekly from January to June 2002. All flock locations, except Queensland, were presented earlier in *Communicable Diseases Intelligence*.[1]

Western Australia

Sentinel flocks in Western Australia are tested for flaviviruses fortnightly during the 'high risk' period (November to May) and monthly at other times. Overall, low-level MVE and KUN virus activity was detected in the Kimberley and Pilbara regions in the north of Western Australia during the 2001/2002 wet season and the majority of seroconversions in both regions was due to MVE virus.

MVE activity was first detected in December 2001 at Kununurra in the north-east Kimberley and seroconversions to both MVE and KUN viruses were detected later in December at 3 sites in the West Kimberley. The first health warning was issued by the Department of Health, Western Australia (DoH, WA) in December and a human case of Murray Valley encephalitis was reported from Kununurra in late December 2001. MVE activity spread throughout the Kimberley region in January and February and a second, as yet unconfirmed case was reported from the West Kimberley in early March 2002.

MVE activity spread to the Pilbara in March and initially activity was restricted mainly to flocks located at 2 large dams (Harding Dam near Karratha and Ophthalmia Dam near Newman). Activity spread to other areas of the Pilbara in April and May 2002. This low-level activity continued throughout both northern regions from April to early June. In response to these results additional health warnings were issued to residents and visitors to the north of the State by DoH, WA in February, March and May 2002. To date activity has not spread to the coastal area of Exmouth in the Pilbara and no activity has been detected further south or east in the Gascoyne, Murchison, Midwest, Goldfields and Wheatbelt regions.

Northern Territory

MVE activity is usually initiated later in the Northern Territory than in Western Australia. The flocks are bled monthly by veterinary officers of the Department of Business Industry and Resource Development and volunteers and the serum samples tested by staff of the Arbovirus Surveillance and Research Laboratory in Perth. The first MVE seroconversions were detected in February 2002 at Tennant Creek. MVE activity spread to Alice Springs and Katherine in March. Kunjin virus activity was detected in the Darwin region (Howard Springs) and Katherine region in March 2002 and this activity continued at Howard Springs, Leanyer and Beatrice Hill to May. Media warnings were issued by the Northern Territory Department of Health and Community Services in December 2001 after the first heavy rains and in February, April, May and June 2002 after sentinel chicken seroconversions and rises in vector numbers in different areas of the Northern Territory.

There was one case of MVE in the Top End with onset in July 2001 in the Mudginberri/Oenpelli area, but this was part of the previous year virus activity. No other cases of MVE were recorded in the Northern Territory, in contrast to the previous year with 2 cases each of Murray Valley encephalitis and Kunjin encephalitis in the Alice Springs locality. The absence of cases and reduced MVE seroconversions in Alice Springs is thought to be due to an extensive insecticide application and drainage measures of Ilparpa swamp on the outskirts of Alice Springs. This resulted in a significant drop in vector numbers compared with the previous year.

North Queensland

The 2 sites in Queensland were monitored weekly for 6 months (January to June 2002) but no flavivirus antibodies were detected.

New South Wales and Victoria

Samples from sentinel chicken flocks were tested weekly for flavivirus antibodies in New South Wales from December 2001 to April 2002 and in Victoria from October 2001 to March 2002. In the 2001/2002 season no MVE or KUN virus activity was detected in these regions. This is in contrast to the previous season when both MVE and KUN virus antibodies were detected in sentinel flocks.

Reference

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OzFoodNet: enhancing foodborne disease surveillance across Australia: quarterly report, January to March 2002

The OzFoodNet Working Group

Introduction

OzFoodNet is a collaborative network of epidemiologists, microbiologists and food safety specialists conducting applied epidemiological research into foodborne disease and improving existing surveillance mechanisms for foodborne disease. The Commonwealth Department of Health and Ageing established OzFoodNet in 2000 and the network has had representation on the Communicable Diseases Network Australia (CDNA) since 2001.

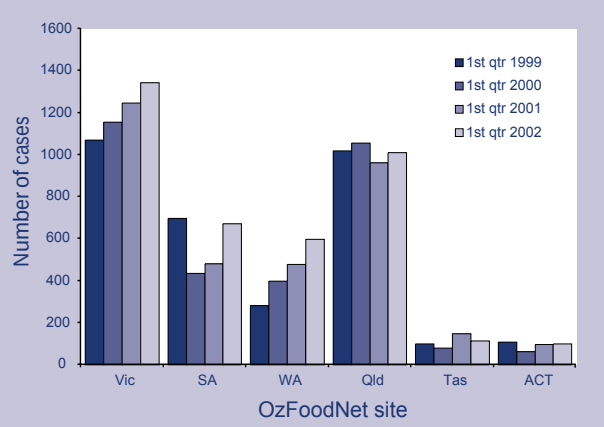
This first quarterly report of OzFoodNet for 2002 summarises the incidence of foodborne disease in the 6 States of Australia and the Australian Capital Territory between January and March 2002. During the first quarter of 2002, OzFoodNet continued to collect data on the incidence of gastroenteritis and its causes around Australia. All Australian jurisdictions collaborate in OzFoodNet. The New South Wales Health Department has enhanced surveillance in the Hunter Region, although data are reported for all of New South Wales where available. The Northern Territory participates as an observer, and data are only included where specified.

All data are reported using the date the report was received by the health agency.

Notifications in the first quarter

In the first 3 months of 2002, notifications of *Campylobacter* infection continued to be higher than historical means, except for Queensland and the Australian Capital Territory (Figure 1). During the first quarter 2002, OzFoodNet sites reported 3,842 notifications of campylobacteriosis, which represented a 19.6 per cent increase over the mean for the same quarter for the years 1998 to 2001. This does not include data for New South Wales or the Hunter Area Health Service, as *Campylobacter* is not notifiable in this State. The median age of cases ranged between 26 to 30 years. The male to female ratio of cases ranged from 1.1–1.5:1.0 across all jurisdictions. There were no reported outbreaks of *Campylobacter* infection during the quarter, although Tasmania investigated a localised increase in northern Tasmania.

Figure 1. Notifications of campylobacteriosis in OzFoodNet sites during the first quarter in the years 1998 to 2002



The incidence of salmonellosis was higher than previous years in all OzFoodNet sites, except for South Australia and Western Australia. Sites reported a total of 2,162 cases of salmonellosis during the first quarter of 2002, which represented a 20.6 per cent increase over the mean for the

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same quarter for the years 1998 to 2001. The median ages of cases ranged from 9 to 26 years in OzFoodNet sites. The Queensland OzFoodNet site reported the highest rate of salmonellosis and the lowest median age of cases (9 years). The male to female ratio differed appreciably in different sites, with the Australian Capital Territory recording 0.4 males for every female, through to Western Australia recording 1.4 males to every female. Sites reported six *Salmonella* outbreaks during the quarter.

During the quarter, there were 3 serovars that were common in three or more states: *Salmonella* Typhimurium (phage types 9, and 135), and *S*. Saintpaul (Table 1). The Victorian Department of Human Services reported the continued emergence of *S*. Typhimurium 170, which also increased in New South Wales and Queensland.[1] The Communicable Diseases Network Australia requested that OzFoodNet coordinate the multi-state investigation of this phage type. The investigation required intensive efforts from the three states, but did not identify any conclusive source for the increase (see section on outbreaks).

There was also a significant increase in the incidence of *S*. Typhimurium 9 in New South Wales and Western Australia. Queensland reported a major increase in cases of *S*. Hvittingfoss, although no obvious source of infection was identified. The OzFoodNet site in Tasmania reported that there were fewer cases of *S*. Mississippi compared to the same quarter in the previous year (ratio of cases in the first quarter 2002 to the mean number of cases in the first quarter for the last 4 years = 0.6).

During the first quarter of 2002, the National Enteric Pathogen Surveillance Scheme reported that the five most common *Salmonella* infections nationally were *S*. Typhimurium 9 (279 cases), *S*. Typhimurium 135 (262 cases), *S*. Saintpaul (145 cases), *S*. Virchow 8 (137 cases), and *S*. Typhimurium 170 (132 cases) (personal communication, Mark Veitch, The University of Melbourne, 13 April 2002).

State health departments received 15 notifications of listeriosis during the first quarter of 2002, which was 20 per cent lower than the number of notifications for the previous 4 years (20 cases). All of these cases were reported in older people with severe immunocompromising conditions. The median age of cases ranged from 74 to 81 years, and the overall male to female ratio was 1.5:1.0. There were no materno-foetal infections reported during the quarter.

OzFoodNet sites reported 19 cases of shiga-toxin producing *E. coli* infections during the quarter; with cases notified from South Australia (n=13), Western Australia (n=3), Queensland (n=2), and New South Wales (n=1). There were no common links identified between the cases. No serotype was recorded for 53 per cent (10/19) of infections. Seven were reported as *E. coli* O157 infections. The median ages of cases in different sites ranged from 34 to 76 years, with females predominating (1.0:1.5). There were 2 cases of haemolytic uraemic syndrome reported, one in Victoria and one in New South Wales. The case in Victoria was due to *E. coli* Ont:H-, and no serotype was reported for the New South Wales case.

OzFoodNet sites reported that during the quarter there were 35 cases of typhoid, which represented a 37 per cent increase on the mean of the previous 4 years. Sites also reported 99 cases of shigellosis and 23 cases of yersiniosis, which represented decreases of 18 per cent and 54 per cent from the mean of the previous 4 years, respectively.

Foodborne disease outbreaks

During the first quarter of 2002, OzFoodNet sites reported 26 outbreaks of gastrointestinal infections with a probable food source, compared to 25 outbreaks for the first quarter of 2001. The outbreaks affected an estimated 784 people, of whom 34 were hospitalised and one person died (Table 2). Sites conducted 12 retrospective cohort studies to investigate these outbreaks, and the remainder of investigations relied on descriptive information.

Table1. Number of notifications for the five most common *Salmonella* infections reported to OzFoodNet sites for the first quarter 2002 compared to the first quarter 2001

OzFoodNet site	Top 5 *Salmonella* infections	Number of cases				
		1st Qtr 2002	1st Qtr 2001	YTD 2002	Total 2001	Ratio*
ACT	S. Typhimurium 9	14	4	14	10	3.5
	S. Typhimurium 135	3	2	3	2	1.5
	S. Typhimurium 64	2	1	2	2	2.0
	S. Infantis	1	3	1	3	0.3
	S. Hvittingfoss	1	0	1	1	–
Hunter	S. Typhimurium 135	10	6	10	15	1.7
	S. Typhimurium 9	10	2	10	3	5.0
	S. Potsdam	8	2	8	2	4.0
	S. Montevideo	4	0	4	1	–
	S. Typhimurium U290	4	0	4	3	–
New South Wales	S. Typhimurium 9	145	58	145	132	2.5
	S. Typhimurium 135	78	75	78	201	1.0
	S. Typhimurium 170	56	2	56	33	28.0
	S. Birkenhead	42	33	42	88	1.3
	S. Typhimurium 4	17	20	17	41	0.9
Queensland	S. Virchow 8	137	63	137	177	2.2
	S. Saintpaul	112	66	112	169	1.7
	S. Birkenhead	57	62	57	134	0.9
	S. Aberdeen	56	29	56	81	1.9
	S. Hvittingfoss	51	12	51	52	4.3
South Australia	S. Typhimurium 126	17	14	17	110	1.2
	S. Typhimurium 108	11	1	11	31	11.0
	S. Typhimurium 9	10	19	10	49	0.5
	S. Agona	7	3	7	6	2.3
	S. Typhimurium 4	7	0	7	7	–
Tasmania	S. Mississippi	33	56	33	102	0.6
	S. Typhimurium 135	8	1	8	5	8.0
	S. Typhimurium 9	3	3	3	12	1.0
	S. Anatum	1	0	1	0	–
	S. Hadar 14	1	0	1	0	–
Victoria	S. Typhimurium 135	66	43	66	92	1.5
	S. Typhimurium 170	48	17	48	72	2.8
	S. Typhimurium 9	34	56	34	127	0.6
	S. Typhimurium 126	21	2	21	16	10.5
	S. Saintpaul	12	2	12	10	6.0
Western Australia	S. Typhimurium 135	35	35	35	51	1.0
	S. Typhimurium 9	22	4	22	15	5.5
	S. Saintpaul	13	20	13	47	0.7
	S. Typhimurium 135a	9	10	9	17	0.9
	S. Muenchen	7	11	7	26	0.6

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

Table 2. Outbreaks transmitted by food or water reported by OzFoodNet sites, January to March 2002

State	Month of outbreak	Setting	Agent responsible	Number affected*	Hospitalied	Evidence	Responsible vehicles
ACT	March	Miscellaneous	Unknown	131	0	D	Unknown
	March	Hotel	Unknown	82	0	D	Unknown
Hunter	February	Restaurant	S. Potsdam	17	2	M	Egg based dressings
	February	Restaurant	*V. parahaemolyticus*	2	0	D	Unknown
	February	Conference/ function	*C. perfringens*	16	0	M	Spit roasted beef and/or pork
Qld	January	Aged care/ healthcare setting	S. Typhimurium 102	12	2	D	Unknown
	January	Restaurant	*C. perfringens*	2	0	D	Unknown
	February	Restaurant	Unknown	6	0	D	Unknown
	February	Home	Ciguatera	2	1	D	Striped perch
	February	Takeaway	*S. aureus*	8	0	D	Pizza
	February	Restaurant	Unknown	6	0	D	Unknown
	February	Institution	S. Potsdam	2	0	D	Unknown
	March	Home	S. Typhimurium 135a	10	8	D	Salmon rice patties
	March	Home	Ciguatera	2	0	D	Spanish mackerel
SA	January	Community	S. Typhimurium 78	5	2	D	Unknown
Tas	March	Restaurant	S. Typhimurium 135	5	3	D	Suspected chicken stock
Vic	February	Conference/ function	Unknown	32	1	S	Chocolate mud cake
	February	Hotel	Human calicivirus	12	0	D	Unknown
	February	Hotel	Unknown	18	0	D	Unknown
	February	Aged care/ healthcare setting	Unknown	13	N/A	D	Suspect chemical poisoning
	March	Restaurant	Unknown	8	0	D	Unknown
	March	Home	S. Typhimurium 135	19	2	S	Roast chicken
	March	Conference/ function	Unknown	12	0	D	sandwiches suspected
	March	Conference/ function	Mixed aetiology	272	13	M	Meal of rice, meat, salad, bread and yoghurt
WA	February	Restaurant	Human calicivirus	60	0	M	Seafood salad
	February	Conference/ function	Unknown	30	NK	D	Unknown

D = Descriptive evidence implicating the suspected vehicle or suggesting foodborne transmission.

S = Statistical association between illness and one or more foods.

M = Microbiological confirmation of agent in the suspect vehicle and cases.

N/A = Not applicable

NK = Not known

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

Fifty-six per cent (14/25) of outbreaks occurred in February. Six outbreaks were due to *Salmonella* contamination and 10 were of unknown aetiology. Two outbreaks were due to *Salmonella* Typhimurium 135, and two due to *Salmonella* Potsdam. There were two outbreaks due to human caliciviruses and two outbreaks of ciguatera poisoning. There were four outbreaks due to bacterial toxins following poor handling in terms of temperature following cooking. The majority of outbreaks (48%) occurred in conjunction with meals at restaurants, conferences or functions.

The Hunter reported an outbreak of *C. perfringens* poisoning due to a function where meats were cooked on a spit roast. The meat was prepared by a company in Sydney and transported to the Hunter. The outbreak was very similar to outbreaks that the Australian Capital Territory reported immediately prior to Christmas.[1] Discussion amongst investigators identified that these separate events were supplied by the same company operating under different names. OzFoodNet sites identified several other outbreaks from previous years that were associated with this company in Western Australia, New South Wales and Queensland. Many of the outbreaks appeared to be caused by spit roast meats that remained at temperatures allowing bacterial toxin production for long periods of time. This outbreak highlighted the benefits of regular communication amongst investigators and the regulatory challenges for food safety in Australian jurisdictions.

During the quarter, Queensland, New South Wales and Victoria collaborated in an investigation of a large community-wide increase of *S.* Typhimurium 170 (not included in outbreak table). One hundred and nine cases were notified between October 2001 and March 2002. The majority of these cases occurred in Victoria, with this state also recording the first cases in this outbreak. Cases occurred in rural and urban areas and there was no obvious geographic clustering. Despite conducting many comprehensive hypothesis-generating interviews the investigation team was unable to definitively identify a vehicle or source for the outbreak. There were several anecdotal links to consumption of red meat and chicken, including:

- several human cases in Queensland purchasing meats from the same butcher, which was supplied by a Victorian distributor;
- concurrent animal isolates of the same organism at similar times and geographic locations (National Enteric Pathogen Surveillance Scheme);
- *S.* Typhimurium 170 commonly isolated from chicken meat (National Enteric Pathogen Surveillance Scheme);
- a subset of cases were infected with *S.* Typhimurium 170 that was resistant to either kanamycin and/or neomycin, which are aminoglycoside antibiotics used in veterinary applications; and
- infections in people in contact with cow herds that were also infected with *S.* Typhimurium 170.

These links were difficult to confirm, but highlighted the need for understanding the animal sources of *Salmonella*. The investigation team recommended improved collaboration between the health and agricultural portfolios for the purposes of disease investigation and data collection.

Applied research

In the first quarter of 2002, a further two OzFoodNet sites commenced recruiting patients and controls for the national *Campylobacter* case control study, bringing the total to five sites. Sites will continue to collect data for the *Campylobacter* case control study until September 2002. The South Australian site commenced a case control study into risk factors for acquiring shiga-toxin producing *E. coli*. This study is not expected to finish for two years, as there are so few cases reported. OzFoodNet sites started interviewing patients for the *Listeria* case control study, although only two sites (Queensland and Hunter) are recruiting controls. Sites continued to recruit patients and controls for the national *Salmonella* Enteritidis case control study and studies into locally endemic *Salmonella* serovars.

In the first quarter of 2002, 1,599 people were interviewed as part of the national OzFoodNet gastroenteritis survey. Overall 12.2 per cent of people experienced gastroenteritis compared with 11.8 per cent for the previous quarter. There were noticeable differences by season in different jurisdictions (Table 3). During January and February 2002, residents of New South Wales reported the highest crude proportion of people experiencing gastroenteritis in the previous month, and Tasmanian and Western Australian residents reported the lowest. Nationally, the prevalence of gastroenteritis was highest for respondents interviewed in the month of January (14.2%) (Figure 2). This survey records self-reported gastroenteritis and does not distinguish foodborne illness from other causes of gastroenteritis.

Figure 2. Unweighted results of the national OzFoodNet gastroenteritis survey showing the proportion of respondents reporting an episode of gastroenteritis in the previous month (n = 3,916), September 2001 to March 2002

The data collected in this survey will contribute to OzFoodNet's calculation of an estimate of the proportion of gastroenteritis due to food. During the quarter, OzFoodNet held discussions with international programs researching gastrointestinal disease and agreed to compare survey date and findings.

References

1. The OzFoodNet Working Party. OzFoodNet: enhancing foodborne disease surveillance across Australia: quarterly report, October to December 2001. *Commun Dis Intell* 2002;26:248–252.

Table 3. Unweighted results of the national OzFoodNet gastrointestinal survey between October and December 2001 and January and February 2002, showing the number and proportion of respondents reporting an episode of gastroenteritis in the previous month

State or Territory	October – December 2001			January – February 2002		
	No. with gastroenteritis	No. interviewed	%	No. with gastroenteritis	No. interviewed	%
NSW*	38	357	10.4	40	255	15.7
NT	40	202	19.8	28	204	13.7
Qld	27	286	9.4	22	203	10.8
SA	37	292	12.7	22	187	11.8
Tas	29	268	10.8	23	215	10.7
Vic	30	250	12.0	26	229	11.4
WA	22	229	9.6	22	206	10.7
Total	**223**	**1,884**	**11.8**	**183**	**1,499**	**12.2**

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

Editorial: Diarrhoea associated with consumption of escolar (rudderfish)

Craig Shadbolt,[1] Martyn Kirk,[2] Paul Roche[3]

This issue of *Communicable Diseases Intelligence* contains three reports of recent outbreaks of oily diarrhoea associated with consumption of ocean-fish. In each outbreak, the oily diarrhoea was caused by indigestible wax esters contained within the fish. Common names of fish associated with these outbreaks included rudderfish, butterfish, oilfish, ruddercod and escolar.

Information supplied by OzFoodNet epidemiologists[1] and the South Australian Department of Human Services[2] describe outbreaks in Victoria, New South Wales and South Australia. In November 1999, two restaurant outbreaks[3] associated with butterfish were reported in Victoria, the first involving 14 cases. Victoria's most recent incident involved a further 4 cases at a restaurant in August 2001 (Gregory, this issue).[4] The South Australian Department of Human Services' Public and Environmental Health Service conducted an investigation into reports of illness after consumption of rudderfish. Ninety-eight cases of illness reports were received (Givney, this issue).[5] Similarly, at a conference luncheon in New South Wales[6] in October 2001, 20 persons became ill after consuming escolar (Yohannes, this issue).[7]

Another recent outbreak occurred in New South Wales (Marianne Tegel, NSW Health, personal communication, April 2002) when a restaurant served patrons with what the owner believed was 'ruddercod'. Five people were adversely affected. The restaurant received complaints and the owner informed authorities that an alternate source of seafood would be used in future. Information supplied from Sydney Fishmarket Pty Ltd (Bryan Skepper, Sydney Fishmarket Pty Ltd, personal communication, April 2002) also indicates that outbreaks from consumption of escolar have been occurring in the restaurant and catering setting for a number of years.

Based on the size of the annual escolar catch (up to 400 tonnes, Hans Jusseit, East Coast Tuna Boat Owners Association, personal communication, March 2002), it has been assumed that many, if not the majority of people eating these fish species do not develop any illness. However, the attack rates described in these three short reports range from 20/44 (45%) to 10/15 (67%). There is probably a significant under-reporting of illness associated with consumption of these fish as the symptoms can be mild and short-lived. In South Australia, 60 additional cases of oily diarrhoea associated with consumption of escolar/rudderfish were identified by the Department of Human Services, following media reports of the issue.[5] There are little data available to identify people susceptible to oily diarrhoea induced by consumption of fish with a high wax ester content. Yohannes could find no association between the development of illness and body mass index, age or general health status. People with bowel problems, malabsorption or pregnancy may be at increased risk of diarrhoea because of eating escolar. It is also possible that seasonal and geographic differences may influence the level of indigestible wax ester content in fish.

In those who are susceptible, the onset of symptoms occurs with a median of 2.5 hours and a range of 1 to 90 hours after consumption.[7] The symptoms described in the three reports show wide variation. Symptoms range from mild and rapid passage of oily yellow or orange droplets, to severe diarrhoea with nausea and vomiting. The milder symptoms have been referred to as 'keriorrhoea' (literally 'flow of wax').[8]

Data from the CSIRO Marine Research[9] show that two species of fish, *Lepidocybium flavobrunneum* (escolar) and *Ruvettus pretiosus* (oilfish), contain approximately 20 per cent (by weight) of indigestible wax ester oil. Both species are by-catches from tuna longlines on the east and west coasts of Australia,[10] caught in quantities of sufficient size that banning them from sale is not considered an option by the fishing industry.

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Some fish, such as orange roughy, have very high oil content just below the surface of the skin. Removal of skin and superficial flesh ('deep skinning') may remove the offending oil portion, leaving a more palatable fish. At present, it is not known whether the wax ester is evenly distributed throughout the flesh of escolar, or lies just below the surface of the skin. Hence, it is uncertain whether the wax ester can be removed by 'deep skinning' of escolar and/or oilfish.

The above outbreaks were addressed at a meeting of the Technical Advisory Group (TAG), which comprises food and agriculture officers from all States and Territories, and Commonwealth agencies responsible for food safety. A TAG working group which included representatives from the seafood industry and CSIRO Marine Research was formed. The group determined that the main problem was one of species identification. Some fish are landed and marketed incorrectly. Whilst named in the Australian Seafood Handbook, a picture of *L. flavobrunneum* (escolar) was lacking. Many fishermen appeared to be landing and selling this particular fish as 'rudderfish'.

Rudderfish species also contain similar proportions of oil, but not the indigestible wax ester seen in escolar and oilfish. Rudderfish are also landed as a result of trawl operations. Illustrations of rudderfish, escolar and oilfish (provided by Don Nichols of the West Australian Seafood Quality Management Initiative) are shown in Figures 1 to 4. Butterfish (*Scatophagus* sp.) is mistakenly used as a marketing name for rudderfish, or escolar, particularly in Victoria. Unlike escolar and rudderfish, which are caught in deeper waters, butterfish are found in the shallows of northern Australia.

The problem of misidentification and mislabelling occurs throughout the entire supply chain, with businesses and consumers being unaware of the potential problems associated with consumption. The working group agreed that misidentification or mislabelling of fish was the most important aspect which needed to be addressed. The Fish Names Committee was alerted to the importance of this issue from a marketing and food safety perspective. To assist industry and consumers with identification and labelling, the common names of escolar and oilfish were endorsed for *L. flavobrunneum* and *R. pretiosus*, respectively. Industry representatives on the working group indicated that there were good national networks amongst fishermen and processors. These networks could be used as a vehicle for distribution of information, including pictures of rudderfish and those species responsible for wax ester diarrhoea. The Australian Seafood Handbook would also be updated to include a picture of *L. flavobrunneum.*

Figure 1. Oilfish, *Ruvettus pretiosus*

Figure 2. Escolar, *Lepidocybium flavobrunneum*

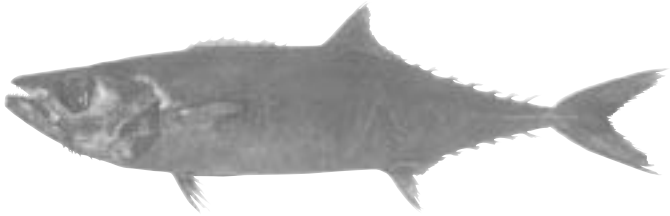


Figure 3. Rudderfish, *Centrolophus niger*

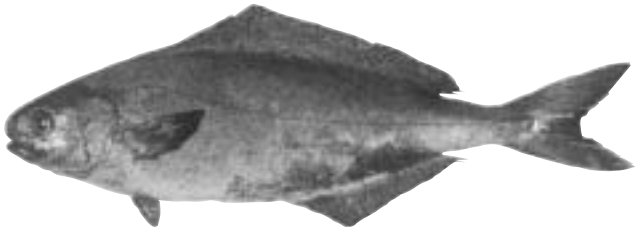
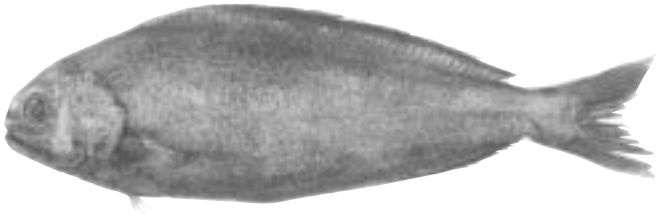


Figure 4. Rudderfish, *Tubbia* sp.



This action allows health authorities to release nationally consistent information on escolar and oilfish. Despite the majority of effects being mild, warnings have been released in some jurisdictions indicating that these fish are not suited to catering, and should be avoided by those with a bowel condition or pregnant women. People eating escolar for the first time are advised to initially consume small portions to determine their susceptibility.

Correct identification of species with a high wax ester content, proper labelling by fish vendors, and appropriate warnings for restaurateurs and the consumer will assist with the reduction of undesirable effects from escolar and oilfish consumption. This will protect the health and safety of consumers, and the image of the food service and seafood industry sectors. More research needs to be done within the public health and seafood industry sectors to identify contributing factors to oily diarrhoea susceptibility, the risks associated with consumption in vulnerable populations and preparation methods which may reduce the risk.

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Outbreaks of diarrhoea associated with butterfish in Victoria

Joy Gregory

Introduction

In the last 3 years the Department of Human Services in Victoria has recorded 3 outbreaks of gastroenteritis believed to be associated with consumption of 'butterfish'. In Victoria, escolar (*Lepidocybium flavobrunneum* and *Ruvettus pretiosus*) and rudderfish (*Centrolophus* sp.) are commonly marketed under the name 'butterfish'. True butterfish, (*Scatophagus* species) is caught as a by-catch in seine nets and traps in the shallows of northern Australia and is very unlikely to be available in any of the southern states of Australia. This short report summarises the three Victorian outbreaks.

Outbreak 1, November 1999

The first outbreak was reported in a group of approximately 80 people who attended a function at a restaurant in November 1999. The Communicable Diseases Section conducted a cohort study and interviewed 63 per cent (50/80) of guests who attended the function. Eleven attendees developed symptoms, predominantly of diarrhoea (92%), abdominal pain (92%) and nausea (50%). Vomiting was not a feature of this outbreak with only 8 per cent reporting this symptom. The diarrhoea was described as watery and there was a median incubation period of 2.5 hours after consumption of the meal. Most people recovered within 24 hours. The dinner was a set menu consisting of a choice of two entrees, two main meals and two desserts. Only one food item, crumbed and deep-fried fillets of butterfish served as a main course, had a statistically significant relative risk (RR=9.37; 95%CI 1.31–67.20). The alternate main meal grilled lamb, had a statistically significant protective association (RR 0.12; 95%CI 0.02–0.83). A sample of 'butterfish', taken from the wholesale suppliers to the restaurant, was analysed and found to be either escolar (*Ruvettus pretiosus*) or rudderfish (*Centrolophus* sp.).

Outbreak 2, November 1999

The second outbreak was reported in a group of 15 people who attended a restaurant also in November 1999. Interviews with this group were unable to be completed but it is known that 10 persons reported symptoms, predominantly of diarrhoea described by one case as yellow oily diarrhoea, after consumption of grilled 'butterfish' which was the common food consumed by all cases. A sample of left-over butterfish from the restaurant was obtained and was found to be either escolar (*Ruvettus pretiosus*) or rudderfish (*Centrolophus* sp.).

Outbreak 3, August 2001

A third outbreak reported in August 2001 affected five out of a group of 15 work colleagues who attended a restaurant for a lunch meal. Four cases consumed 'butterfish' and experienced symptoms of diarrhoea and nausea within 2 hours of consumption. Statistical analysis was not carried out as too few people were interviewed. Leftover fish sampled from the restaurant was analysed and found to be escolar (*Ruvettus pretiosus*).

Discussion

Prior to these outbreaks, there had been no gastrointestinal outbreaks associated with 'butterfish' recorded in Victoria. Investigation of the 3 Victorian outbreaks revealed that the chefs of the restaurants where the outbreaks occurred were unaware of the purgative properties of escolar and rudderfish. In addition, receipts retained by the restaurants indicated that 'butterfish' was purchased so the chefs were also not aware of the correct species that had been purchased on these occasions. The purgative properties of escolar and rudderfish have been documented in literature[1,2,3,4] but outbreaks may be poorly recorded.

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Illness associated with rudderfish/ escolar in South Australia

Rodney C Givney

Nineteen out of 41 people who attended a dinner on 10 March 1999 developed gastrointestinal symptoms, 18 of them within 2 days. The dinner was held at a restaurant associated with a tertiary educational institution in metropolitan Adelaide.

The associated educational institution had reported the growth of a faecal coliform on in-house testing from its potable reticulated water supply sampled on 9 March 1999 after the repair of a damaged water pipe. On the advice of the Environmental Health Branch of the Department of Human Services, South Australia (DHS SA) water from the suspected system was boiled before consumption until further microbiological testing by the relevant water authority indicated it was safe to drink.

The Communicable Disease Control Branch of DHS SA carried out a cohort study. The restaurant provided a menu and 40 of the 41 people who attended the dinner were questioned regarding items on the menu that were eaten. Only one food item, rudderfish, served as a main course, had a significant risk ratio: 2.53 (confidence interval 1.13–5.70). Fourteen of the 19 persons who ate the fish reported illness. By contrast, water served at the dinner posed no risk: risk ratio 1.00 (confidence interval 0.35–2.83).

After a more recent event in South Australia, in October 1999, an implicated so called rudderfish fillet was speciated as *Lepidocybium flavobrunneum* by protein fingerprinting.[1] According to industry sources this fish is imported into South Australia from Queensland and Western Australia. It is not clear if other species which cause the same symptoms are also sold under this or other names or if the *Lepidocybium flavobrunneum* is sold under names other than rudderfish. The recommended marketing name for *Lepidocybium flavobrunneum* is escolar.[1]

Usually people complain of diarrhoea, often oily and orange coloured, within hours of consumption. The diarrhoea may be urgent enough to cause repeated faecal incontinence. Abdominal discomfort, nausea and occasionally vomiting have also been reported.

The cause of illness seems to be the high oil content of the fish rather than a recognised toxin or bacterial contamination. Nevertheless, when any fish is identified as a possible cause of food poisoning it is recommended that advice be sought to arrange testing to exclude microbiological and toxic causes.

In South Australia, between 1997 and October 1999, there have been seven other reports of abdominal symptoms following consumption of rudderfish, involving at least 19 people. After media interest following the October cases, a further 60 people phoned DHS SA with complaints of illnesses which occurred from 1997 to 1999 following rudderfish consumption. The Food Unit of the Environmental Health Branch then advised seafood retailers to display a sign advising that rudderfish might cause these problems. Since then there have been no further complaints to date related to rudderfish consumption in South Australia.

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Postscript: 'Rudderfish' is now a recognised marketing name but for a different fish (*Centrolophus*, *Scedophilus* and *Tubia* species).

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An outbreak of gastrointestinal illness associated with the consumption of escolar fish

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Abstract

An outbreak of gastrointestinal illness occurred amongst attendees of a conference lunch in the Hunter area, New South Wales, in October 2001. A distinctive symptom reported by many ill persons was the presence of oily diarrhoea. The Hunter Public Health Unit investigated the outbreak by conducting a telephone interview of the cohort of conference attendees using a standard questionnaire. Twenty persons out of 44 attendees (46%) became ill following the conference. The median incubation period was 2.5 hours (range 1–90 hours). The most common symptoms reported were; diarrhoea (80%) — 38 per cent of these reported oily diarrhoea; abdominal cramps (50%); nausea (45%); headache (35%) and vomiting (25%). For analyses, a case was defined as a person who developed oily diarrhea, or diarrhoea within 48 hours, or had at least two other symptoms of gastroenteritis within 6 hours, of the conference lunch. Seventeen persons had symptoms that met the case definition. None of the foods or beverages consumed were significantly associated with illness, however, all cases had consumed fish and none of those who did not eat fish (4 persons) became ill. Moreover, only 'fish' or 'potato chips' could explain a significant proportion of the illness. Analysis of the oil composition of the fish consumed was consistent with the known profile of the species marketed as 'escolar'. Among those who consumed fish the following potential risk factors did not have a significant association with the illness: Body Mass Index, age, health status and the amount of fish consumed. We concluded that consumption of fish within the marketing group escolar can cause severe abdominal cramping, nausea and vomiting, in addition to incontinent diarrhoea. *Commun Dis Intell* 2002;26:441-445.

Keywords: fish, outbreak, diarrhoea, Australia, escolar, rudderfish, wax ester

Introduction

Purgative properties are reported for members of the escolar (*Lepidocybium flavobrunneum, Ruvettus pretiosus*) and rudderfish (*Centrolophus niger* and *Tubia* species) marketing groups.[1] Escolar are commonly sold in the domestic market mislabeled as 'rudderfish' or 'butterfish'. Their oil profiles have been found to be very distinctive from each other and other fish species.[2] Studies have found that both escolar and rudderfish have higher oil composition in proportion to their wet mass (2–25%[2]) than most seafood, but it is the high wax ester content in escolar oil (>90%[2]) that explains the purgative property.[2,3] In humans, wax esters accumulate in the rectum causing oily diarrhoea.[3] In October 2001, the Hunter Public Health Unit received a report of diarrhoea from a person who had attended a conference lunch at a local catering centre. Further investigation identified a number of conference attendees who had developed gastrointestinal illness after attending the conference lunch where the main meal was reported to be rudderfish. The Public Health Unit investigated the outbreak with the aim of preventing further cases of gastrointestinal illness and identifying the causative agent.

Methods

Epidemiologic investigation

The Public Health Unit conducted a cohort study of all conference attendees who attended the lunch. A list of conference attendees was obtained from the conference organisers and an effort was made to contact the entire cohort by telephone. A standard questionnaire was used to obtain information on the type and quantity of food and beverages consumed. Detailed information on clinical symptoms and duration of symptoms were also collected. In addition to the standard questions the study incorporated questions related to the use of medication, health status, height and weight and description of build that could be used to examine

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the impact of other factors on illness. Body mass index (BMI) was calculated for each interviewee (weight/height^2).

Relative risks (RR) with 95 per cent confidence intervals (95% CI) were calculated to estimate measures of association between exposure and illness. To further investigate factors associated with being a case, logistic regression analysis was performed, using BMI, age, health status and the amount of fish consumed as covariates. For the logistic regression analysis BMI and age were both categorised into 2 groups with the mean as the cut off. Statistical analysis was performed using Epi Info version 6.04c and SPSS version 11.0.

For the purpose of the analysis a case was defined as a person who developed oily diarrhoea, or diarrhoea within 48 hours, or suffered at least 2 symptoms that included; nausea, abdominal cramps, vomiting or headache within 6 hours of eating at the conference lunch. Diarrhoea was defined as three or more loose stools in 24 hours.

Environmental and laboratory investigations

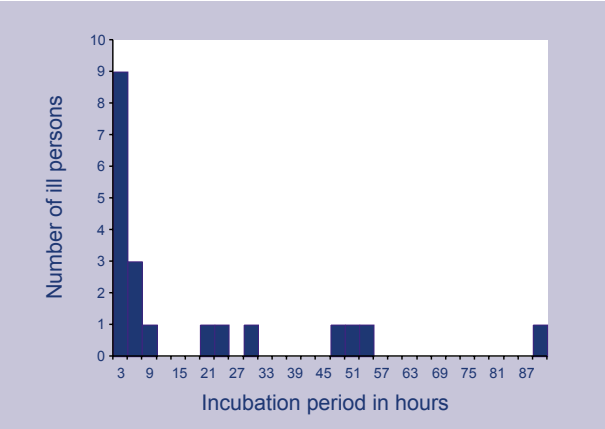
Hunter Public Health Unit Food Surveillance Officers inspected the fish market and lunch venue and reviewed food preparation and handling procedures. Two remaining pieces of fish from lunch and a sample of the oil in which it was cooked were sent to the CSIRO Marine Research Laboratory, Hobart, Tasmania, for oil content and composition analysis and possible identification of the species. The methods of analysis involved the extraction of oil with solvents and the determination of individual oil classes using an Iatroscan thin-layer chromatograpgy-flame-ionization detector (TLC-FID) analyzer, and is reported elsewhere.[2]

Results

Public Health Unit officers interviewed 94 per cent (44/47) of persons who ate at the conference lunch. Of these, 46 per cent (20/44) reported gastrointestinal symptoms (Table 1). The male to female ratio for persons reporting gastrointestinal symptoms was 1.2:1. The median duration of illness was 22 hours (range 5–78 hours). The most frequent symptom reported was diarrhoea (16/20), which was also reported as the most severe symptom by 89 per cent of ill persons. Thirty-eight per cent of persons with diarrhoea described the diarrhoea as oily.

The median time between lunch and onset of illness was 2.5 hours (range 1–90 hours) (Figure). Symptoms of abdominal cramping, vomiting, nausea and headache, generally proceeded diarrhoea (Table 1). Fifty-six per cent (9/16) of persons with diarrhoea reported additional symptoms, which included abdominal cramping (8/9), nausea (6/9), vomiting (3/9) or headache (4/9). Thirty-five per cent of ill persons could not perform their normal activities for a median of 2 days (range 0.5–5 days), however no one sought medical attention.

Figure. Incubation period of illness reported by persons who attended a conference lunch, Hunter, New South Wales, 2001



Seventeen ill persons (17/20) met the case definition and were included in the analysis. Of the 3 ill persons who did not meet the case definition, two had watery diarrhoea more than 48 hours after the lunch and one did not have diarrhoea but had other symptoms more than 6 hours after lunch. Food specific attack rates for cases showed that consumption of 'fish' or 'potato chips' could explain a significant proportion of the illness (Table 2). There were no cases who did not eat the fish and everyone consumed approximately the same amount of fish. No other foods or beverages showed a statistical association with illness.

Logistic regression analysis was performed on data from persons who ate fish. BMI was calculated for 39 interviewees. The mean BMI for cases was 25.8 (SD 3.2) and non-cases was 26.8 (SD 6.5). The results showed that cases and non-cases did not differ by BMI, age or health status.

Table 1. The prevalence of various symptoms among the persons who reported illness after attending a conference lunch, Hunter, New South Wales, 2001

Symptom	Prevalence (%) n=20	Median incubation period
Diarrhoea — watery only	50	4 hours (range 2–90 hours)
Diarrhoea — oily	30	4 hours (range 2–90 hours)
Abdominal cramps	50	2 hours (range 1–53 hours)
Nausea	45	2 hours (range 1–6 hours)
Headache	35	2 hours (range 1–27 hours)
Vomiting	25	2 hours (range 1–2 hours)

Table 2. Food-specific attack rates among persons who attended a conference lunch, Hunter, New South Wales, 2001

Food items	Persons who consumed item		Persons who did not consume item		Relative risk	95% CI
	Total ill	Attack rate (%)	Total ill	Attack rate (%)		
Fish	17/40	43	0/4	0.0	Undefined	Undefined
Potato chips	17/39	44	0/5	0.0	Undefined	Undefined
Apple slice	2/8	25	15/34	44	0.6	0.6–2.0
Coconut slice	2/3	67	15/40	38	1.8	0.7–4.4
Curried egg	3/7	43	13/33	39	1.1	0.4–2.8
Honeydew	1/10	10	16/32	50	0.2	0.0–1.3
Kiwi	2/9	22	15/34	44	0.5	0.1–1.8
Other foods	7/12	58	10/32	31	1.9	0.9–3.8
Pineapple	2/4	50	14/35	40	1.3	0.4–3.6
Rockmelon	6/16	38	11/27	41	0.9	0.4–2.0
Vanilla slice	3/7	43	14/37	38	1.1	0.4–2.9
Watermelon	4/17	24	13/26	50	0.5	0.2–1.2

Table 3. Oil content and composition of fish samples from the outbreak, Hunter, New South Wales 2001, compared with that of escolar and rudderfish reference specimens[2]

Oil content and composition		Fish samples from outbreak		Reference specimens[2]	
		Sample 1	Sample 2	Escolar species	Rudderfish species
Oil content (% of wet body mass)		21.7	22.4	17.8–21.2	1.7–24.8
Oil composition (% in oil)	Wax ester	96.4	97.6	90.1–96.9	n.d. – 1.5
	Triglyceride	1.9	0.3	n.d. – 1.5	0.3–14.9
	Free fatty acids	n.d.	n.d.	n.d. – 0.7	0.6–21.6
	Polar lipids	1.7	2.1	2.1–5.7	1.3–42.1
	Hydrocarbon	n.d.	n.d.	n.d. – 1.1	n.d. – 93.4
	Diacylglyceryl ether	n.d.	n.d.	n.d. – 0.5	2.2–92.5

n.d.= not detected

Environmental and laboratory investigations

No breach of food preparation and handling procedures was detected. The results of the analysis of oil content and composition of the fish showed that the fish samples had oil content of 22 per cent (percentage of weight), which is in excess of the average Australian marine fish oil content of 1 per cent[1,2] and 97 per cent of the oil content was wax ester (Table 3). These results are consistent with the oil content of members of the escolar marketing group (*Lepidocybium flavobrunneum, Ruvettus pretiosus*),[2,4] and suggest that the fish served at the conference meal were escolar, and not rudderfish, as shown on the sale invoice to the catering venue.

Discussion

The investigation identified an outbreak of gastrointestinal disease caused by the consumption of escolar. Escolar has been described as having a purgative effect due to the high wax ester content in the oil of the fish accumulating in the rectum causing oily diarrhoea. In this outbreak we identified the effects of escolar consumption to involve more severe symptoms of gastrointestinal illness, including diarrhoea, nausea, headaches, abdominal cramps and vomiting. It is unclear why some people who consumed fish became ill and some did not. In this investigation BMI, age, health status and the amount of fish consumed did not affect the outcome.

There is a paucity of information describing the symptoms associated with escolar consumption and as a result it may be an under-recognised cause of gastrointestinal disease. The health effects of the consumption of escolar are not well described in literature. Berman *et al* (1981)[3] distinguished the effects of wax ester from hydroxyoleic acid, which is the purgative element in castor oil. They claimed that consumption of wax ester resulted in a passing of accumulated oil in the rectum, while consumption of the hydroxyoleic acid caused diarrhoea by some irritant effect on the bowel. Therefore, they proposed to label the diarrhoea caused by escolar as keriorrhoea, a Greek word to mean flow of wax. However, this suggestion was based on symptoms described by only two cases. We found that cases suffered not only the inconvenience of incontinent diarrhoea, but also abdominal cramps, nausea, headache and vomiting.

While BMI, age and health status and amount of fish consumed were not associated with illness, there are other factors that could mediate the severity and occurrence of the gastrointestinal effects of reported escolar consumption. These factors include variability in wax ester content of different fillet cut depths and mixing of fillets from different fish species sold as ‘rudderfish’ at the wholesale or retail levels. These may result in differential exposure in a cohort of consumers.

A limitation of this study was that we did not collect stool samples for microbiological analysis from those who were ill because 4 days had lapsed before the event was reported. In the absence of any stool samples from ill persons, we could not rule out that the illness was caused by an infectious pathogen. Toxicity such as histamine poisoning from fish is not a likely explanation, as the onset is more rapid (45 minutes) than the incubation period observed in this outbreak and symptoms differ as histamine poisoning symptoms usually include fever, flushing and rapid pulse rate.[5] The lack of illness among those who did not consume the fish did not allow a relative risk to be calculated, however consumption of 'fish' or 'chips' explained the highest proportion of the illness reported. Potato chips are not a plausible cause of the illness. Although glycoalkaloids found in potatoes can cause illness, the oral dose required for such effects is higher than would be expected from a serve of potato chips[6] and neurological disorder was not reported by members of the cohort. In this study potato chips have a strong correlation with consumption of fish. Dose response could not be assessed from this study, as there was little variation in the amount of fish consumed by each person.

This investigation highlights the need for escolar hazard guidelines to protect both traders and the public. There may be a number of restaurateurs and caterers that are unaware of the potential health effect of escolar. Escolar, a deep-sea fish of the tropical and temperate oceans, is harvested by long line trawlers from southern Queensland, along the south of the continent and up the Northwest Shelf of Western Australia.[1] In New South Wales, more than 60 tonnes of fish is marketed annually under the label of 'rudderfish' at one auction house alone (Sydney Fish Market, Information Sheet, 17 August 2001). In Japan, the Ministry of Health prohibits the sale of the two species of escolar.[4] In its 1998 hazard guide, the United States of America Federal Drug Administration recommended that *Lepidocybium flavobrunneum* not to be marketed in interstate commerce.[7] There may be a need for greater education of fish wholesalers and retailers to prevent future outbreaks. Our investigation also highlighted that selling escolar as 'rudderfish' may indicate a breakdown in quality control in the fish industry. It is important to correctly identify species at the wholesale level to ensure that only species suitable for human consumption are sold. The Department of Agriculture, Fisheries and Forestry — Australia is currently addressing this issue. In April 2002 its committee for seafood marketing names made recommendations for public consultations aimed at resolving existing misidentification and mislabeling of escolar and rudderfish.

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Gastroenteritis outbreak in a sporting team linked to barbecued chicken

Paul Armstrong,[1,2] David Peacock,[1] Scott Cameron[2]

Background

On 25 May 2001, the Centre for Disease Control, Northern Territory Department of Health and Community Services in Darwin, was alerted by local media reports to an apparent outbreak of gastroenteritis that occurred in a visiting interstate sporting team 2 days before. The 16-member team was competing in the Arafura Games, a biennial, international sports competition conducted in Darwin. After corroborating the report by interviews with the team management and by reviewing hospital records, an outbreak investigation was initiated.

Methods

Hypothesis generating interviews were conducted with the team members. Information was collected regarding food consumption history, demographic details, symptomatology, and time of illness onset. From these interviews, a meal organised for team members only and consumed several hours prior to onset of symptoms by affected team members, was identified as the likely source of the outbreak. A retrospective cohort study was conducted to determine any link between illness and eating particular foodstuffs at this meal. The case definition was defined as: 'any member of the team who ate at this team meal (commencing 11pm 23 May) and who became ill with one or more symptoms of vomiting, abdominal pain or diarrhoea, from 11pm, 23 May to 11am, 24 May'. The information was entered into a database using Epi Info Version 6 software. Relative risks were calculated for each food item.

The then Territory Health Services Environmental Health team investigated the food handling practices of the supermarket delicatessen where the food items consumed at the common team meal were purchased. Their aim was to identify potential environmental source(s) of the foodborne illness, and enforce public health legislation where appropriate.

Results

Epidemiological investigations

Descriptive study

On 23 May 2001, after their sporting commitments were completed, the team and their management met at their hotel for a late evening meal consisting of food purchased from a supermarket 6 hours prior to the meal. The foods purchased were 3 hot barbecued chickens, potato salad, coleslaw, bread rolls, fruit juice in small cartons, and confectionary. Soon after they were purchased, one of the team unpacked and handled one of the chickens and placed it on the only plate available (denoted 'plate chicken' in analytical study below). The other chickens were left in their wrappers untouched ('wrapper chicken') and all the chickens, as well as the other food items, were refrigerated until the meal commenced, $5^1/_2$ hours later. Between $2^1/_2$ and 4 hours after the meal commenced, 6 members of the team (3 male, 3 female; age range 18-26 years) became unwell, initially with malaise (5/6), severe vomiting (5/6) and crampy abdominal pain (4/6), and diarrhoea some hours later (6/6). Five presented to an accident and emergency department and all were discharged after receiving supportive treatment. No samples were obtained for microbiological diagnosis. No other clusters of acute gastrointestinal disease were reported around the time of this outbreak, neither in the hotel where the team were residing nor elsewhere in Darwin.

Analytical study

All 16 team and staff members completed the questionnaire (100%). Two members, who did not participate in the evening team meal and remained well, were excluded from the analysis. Six members of the team had symptoms in keeping with the case definition, giving an overall attack rate of 6/14 (37.5%).

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The number of team members who ate the various food items, and the relative risks for becoming ill, are shown in the Table. Two team members who ate chicken could not remember which chicken they ate; one became ill and one did not. These 'unknowns' can be analysed in a number of ways in calculating relative risks for eating the two types of chicken. The most conservative approach, assuming the former ate wrapper chicken and the latter ate plate chicken, yielded a relative risk for eating plate chicken of 5.0 (Table).

Environmental investigation

At the time of the site inspection, acceptable standards of food safety practices were observed and there were no potential sources of the outbreak identified.

Discussion

This small outbreak of an acute gastrointestinal illness has all the hallmarks of food poisoning due to a pre-formed toxin produced by an enterotoxin-producing bacterium, although microbiological proof is lacking. The short incubation period with abrupt onset, the symptomatology, and the short, self-limiting nature of the illness, are all typical of disease caused by either of the 2 pathogens that are commonly implicated in such illnesses, *Staphylococcus aureus* and *Bacillus cereus*.[1] Illness caused by B. cereus is usually associated with eating boiled or fried rice that has been cooked and kept warm for an extended period.[2,3] In this outbreak, S. *aureus* was considered to be the more likely cause, being a commonly recognised aetiological agent for foodborne outbreaks associated with poultry,[1,4] the likely vehicle for enterotoxin in this outbreak.[5,6] High salt foods like commercial barbecued chickens favour the growth of S. *aureus* over other bacteria.

The most conservative estimate of relative risk for eating 'plate chicken' in our analysis was 5.0, making it the most likely food vehicle. The 'plate chicken' may have become contaminated whilst it was in the store, either prior to cooking or during handling by store employees after cooking, or during handling by the purchaser. However, there were no other reports of food poisoning in the region around the time of this outbreak and the conclusion of the environmental investigation was that food handling practices of the store were acceptable. It is more likely that the team member who handled the food was the source of contamination, especially considering the 2 chickens that were not handled by this team member were not associated with illness. The considerable heat load on the team's motel refrigerator when all of the food items were placed within it several hours prior to the meal, could have slowed the rate of cooling of the chickens, thereby allowing enterotoxin to be produced in sufficient quantities to cause disease.

There was a failure of the notification procedure at the beginning of this outbreak which delayed the initiation of the investigation. Gastroenteritis is a notifiable condition in the Northern Territory if it occurs in an institution, in a food handler, or if two or more cases that are apparently related are recognised. The 5 cases who presented to hospital were clearly related yet were not notified because

Table. Association between exposure to a particular food item eaten at the evening meal and symptoms of an acute gastrointestinal illness

	No. ill team members		No. well team members		
Food item	**Ate item**	**Did not eat item**	**Ate item**	**Did not eat item**	**RR**
'Plate' chicken	5	1	2	6	5.0
'Wrapper' chicken	3	3	4	2	0.8
Potato salad	4	2	5	3	1.1
Coleslaw	3	3	5	3	0.8
Fruit juice	4	2	6	2	0.8
Bread rolls	5	1	7	1	0.8
Confectionary	4	2	6	2	0.8

the treating team was unaware of the necessity to do so. Immediate remediable action included a presentation to Accident and Emergency staff regarding notification requirements pertaining to diseases likely to be seen in their setting. This information will be incorporated into the regular presentation given by the Centre for Disease Control to Accident and Emergency medical staff given at times of staff turnover.

In the setting of an apparent cluster of related cases, efforts should have been taken to collect specimens for microbiological analysis. In the Accident and Emergency Department, appropriate specimens would have included faeces for microscopy/culture of conventional enteric pathogens, and vomitus and faeces for microscopy, culture and enterotoxin testing for *S. aureus* and *B. cereus* (enterotoxin testing is normally only available at public health laboratories). In suspected foodborne outbreaks caused by *S. aureus* or *B. cereus*, further specimens should ideally be taken during the epidemiological and environmental investigation. These would include hand and nasal swabs from the food handler for culture of S. aureus, and samples of the implicated food (if it is still available) for culture and enterotoxin testing for both organisms.[1] With regard to *S. aureus*, valuable epidemiological evidence can potentially be gained from matching phage-types isolated from the food handler, the food items, and the case. Less important is obtaining samples from fomites associated with food preparation, such as the plate that the implicated chicken was stored and served upon, as these are unusual sources of contamination with enterotoxin producing organisms. Because the illness caused by these organisms is a short self-limiting one, and the organism and enterotoxin are cleared relatively quickly, effort should be made to collect the samples within 48 hours after onset of symptoms.

The public health consequence of foodborne outbreaks caused by enterotoxin-producing bacteria is mainly morbidity associated with a short term, often incapacitating illness, but one that rarely leads to death or long term health sequelae. Unlike foodborne outbreaks where the mechanism of spread is waterborne or by the faecal-oral route, food poisoning outbreaks due to preformed enterotoxin ingestion are not self-perpetuating. Apart from physical discomfort experienced by affected team members, and disruption to their sporting program, no other adverse public health consequences eventuated in the outbreak described here.

In summary, this small outbreak of an acute gastrointestinal illness linked to barbecued chicken has features that strongly suggest an enterotoxin-producing bacterium as the causative agent, although microbiological proof is lacking. It is not possible to be definitive about the cause of the contamination of the chicken but the most likely scenario is that the team food-handler was the source. Although mortality and longer-term morbidity are uncommon with food poisoning caused by enterotoxin-producing bacteria, this outbreak highlights its capacity to cause short term, moderately-severe illness in a young and healthy population. It underscores the need for proper food handling practices, both in-store and by the consumer, and reinforces the importance of appropriate microbiological specimen collection from cases of apparent gastroenteritis outbreaks, as well as the public health importance of timely notification of such outbreaks.

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Outbreak of *Cryptosporidium* linked to drinking unpasteurised milk

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In August and September 2001, the Sunshine Coast Public Health Unit received notifications that 8 children from the Sunshine Coast, Queensland, had laboratory-confirmed *Cryptosporidium* in faecal samples. Four children were hospitalised and all recovered. Dates of onset were consistent with a protracted common source dispersed in the community. An epidemiological and environmental investigation sought details of symptom history and exposure to potential sources of *Cryptosporidium*, including animal contact, commercial and non-commercially available unpasteurised milk, non-potable water and other persons with gastroenteritis. For the case control study, a case was defined as a child with laboratory-confirmed *Cryptosporidium*, with symptom onset in August 2001 and living on the Sunshine Coast. Considering the potential confounders of age and geographic location, substantial effort was made to obtain age-matched controls from the treating general practitioner. Controls had attended the general practitioner for conditions other than gastroenteritis. Controls for 3 cases were unable to be obtained, and thus only unmatched analysis was undertaken.

All 8 cases experienced vomiting and diarrhoea. Three controls (18%) had diarrhoea and two had vomiting (12%) in August 2001, with no laboratory confirmation of cause. The incubation period was available for 6 cases only, where a range of one to 9 days was recorded.

Unmatched analysis was performed using SPSS v10.07. Drinking of commercially obtained unpasteurised milk in the 2 weeks prior to the onset of illness was the only exposure significantly associated with a laboratory-confirmed diagnosis of *Cryptosporidium* (Table). For this exposure the Odds Ratio (OR) was 32.7, and 95 per cent confidence interval (CI) 2.9-374.

Table. Exposure history and odds ratios

Exposure in 2 weeks prior to onset	Cases exposed (n=8)	Controls exposed (n=17)	OR	95%CI
Unpasteurised milk	7	3	32.7	2.9-374
Contact with farm animals, zoo animals or animal sanctuaries	2	5	0.80	0.11-5.7
Contact with pets	8	15	2.74	0.1-64*
Contact with pets with diarrhoea	0	0	No exposure	
Swimming in lake, dam, or private or public pool	4	3	6.2	0.89-44
Pasteurised milk	6	15	1.25	0.05-35*
Milk direct from a farm	0	1	0.85	0.03-24*
Purchased water or water from a tank	4	10	0.7	0.13-3.8
Contact with people with gastrointestinal symptoms	5	8	2.2	0.3-15

* Estimated OR (95% CI). Estimation was required as all cases were exposed to pets and to pasteurised milk and none were exposed to milk directly from a farm. The OR and 95 per cent CI were estimated by adding 0.5 to each cell frequency.

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All 10 samples of the commercial product of unpasteurised milk were found to be of unacceptable quality for unpasteurised milk (Food Standards Code 1.6.1), due to a high plate, coliform and/or *E. coli* counts. Milk samples were mixed with equal volumes of distilled water and Tween-20, centrifuged at 1000 x G.[1] A cream layer (approx. 1mL) was present after centrifugation. Both the cream layer and the pellet were further concentrated using immunomagnetic separation and stained using an immunofluorescent reagent containing *Cryptosporidium*-specific monoclonal IgG1 antibodies. Concentrated samples were examined microscopically and analysed using the ELISA SYSTEMS™ *Cryptosporidium* Detection in Water Microwell ELISA kit.[2] Five samples returned positive results for *Cryptosporidium* antigen in the milk fat. In addition, the single available sample of unconsumed commercial unpasteurised milk obtained from a case tested positive for the antigen. The detection level for the ELISA kit, as stated by the manufacturer, is 10 oocysts per well and/or 30 nanograms per mL of concentrated *Cryptosporidium* antigen. Two negative control samples were also analysed, a pasteurised commercially available milk sample and an unpasteurised milk sample from a local dairy supplier.

This is the first reported outbreak of cryptosporidiosis associated with drinking unpasteurised cow milk in Australia. A single report from the United Kingdom describes an outbreak in children when the pasteurisation process was faulty[3] and a mother and child are believed to have been infected by unpasteurised goat milk in Australia.[4] It is illegal to sell unpasteurised cow milk for human consumption in Queensland. This milk was labelled as unpasteurised pet milk. This outbreak illustrates the dangers associated with drinking unpasteurised milk. Calves are frequently infected with *Cryptosporidium,*[5] oocysts can be recovered from adult cows and milk can be contaminated through mechanisms such as poor udder hygiene. Cryptosporidial oocysts will not survive pasteurisation.[6]

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Observational methods in epidemiologic assessment of vaccine effectiveness

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Abstract

Observational methods are important in the measurement of vaccine effectiveness (VE) as experimental designs cannot be used for measurement of vaccines already on the vaccination schedule. Furthermore, efficacy measured in clinical trials under ideal conditions may differ to effectiveness in the field under non-ideal conditions and in different populations. In addition to post-licensure surveillance, observational VE studies are particularly important when disease incidence does not predictably decrease with increased vaccine coverage, when high proportions of vaccine failure among reported cases suggest a problem with the vaccine or when issues arise that were not predicted in pre-licensure evaluations. Commonly used study types for evaluating VE include cohort studies, household contact studies, case-control studies, the screening method and case-cohort studies. There are many potential biases in all observational VE studies which should be considered in the study design and analysis stage. Of the five observational study types reviewed, cohort studies undertaken during an outbreak investigation offer the simplest means of VE estimation and is the preferred study design where the situation permits. Where this is not possible the screening method is the most economical and rapid method. It is essential that the effectiveness of all vaccination programs be evaluated. As new vaccines are introduced to the schedule, booster doses are added and the timing of doses changed, the role of observational methods in the evaluation of VE will become even more important. To date, few observational VE studies have been undertaken in Australia, suggesting the under-utilisation of these methods. *Commun Dis Intell* 2002;26:451-457.

Keywords: vaccine effectiveness

Introduction

Vaccine efficacy is the percentage reduction of disease incidence in a vaccinated group compared with an unvaccinated group, under ideal conditions. Vaccine efficacy studies are typically undertaken pre-licensure using double-blind randomised controlled trials, with all participants initially susceptible to the disease.[1] Once a vaccine has been shown to be efficacious and is licensed, the use of a placebo is unethical. Therefore an experimental design cannot be used for vaccines on the vaccination schedule and so observational methods must be employed. Furthermore, efficacy measured in clinical trials under ideal conditions may differ to effectiveness in the field under non-ideal conditions and in different populations.[2]

Vaccine effectiveness depends upon vaccine efficacy but is also affected by other factors such as transportation and storage at appropriate temperatures ('cold chain') and proper administration and timing of doses. The terms 'vaccine effectiveness' and 'vaccine efficacy' are often used interchangeably and the abbreviation VE is used for both vaccine efficacy and effectiveness.[3] In this review VE is used as an abbreviation for vaccine effectiveness.

The Australian vaccination schedule is constantly changing as new vaccines are introduced, booster doses are added and the timing of doses changed.[4] To maintain public and provider confidence in vaccination programs, it is essential that the effectiveness of new vaccines and changes to the schedule of existing vaccines be evaluated. The evaluation of current vaccines/schedules should also be monitored to enable detection of variations in effectiveness over time which may result from changes in the target population or in the epidemiology of the disease. In the case of new vaccines these effectiveness studies may be

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incorporated as a component of post-licensure surveillance. In addition to post-licensure surveillance, observational vaccine effectiveness studies are particularly important when disease incidence does not predictably decrease with increased vaccine coverage, when high proportions of vaccine failure among reported cases suggest a problem with the vaccine or when issues arise that were not predicted in pre-licensure evaluations.[2] However, it should be noted that when most of the population is vaccinated, most cases will be vaccine failures, so a high proportion of vaccine failures is not necessarily indicative of a declining vaccine effectiveness or efficacy.

A number of observational methodologies can be used in the assessment of vaccine effectiveness, some of which may be incorporated into routine surveillance of vaccine preventable diseases. This paper discusses the potential biases and limitations of observational VE studies, outlines five commonly used study types and provides examples from the literature of where these study types have been used.

Calculating VE

All VE studies involve comparison of the relative risks of disease in the vaccinated group(s) with the unvaccinated group(s), hence any study type from which relative risk can be estimated can be used to calculate VE.[2] The standard equation for calculating VE as a percentage is:[2]

$$VE\ (\%) = \left(\frac{ARU - ARV}{ARU} \right) \times 100$$

where ARU is the attack rate in the unvaccinated group and ARV is the attack rate in the vaccinated group. Rearranging the formula gives the following:[2]

$$VE\ (\%) = \left[1 - \left(\frac{ARV}{ARU} \right) \right] \times 100$$

where $\frac{ARV}{ARU}$ is equivalent to the relative risk.
In case-control studies the relative risk is approximated by the odds ratio.

Protection against what? Defining the study question

Immunisation may produce more than one effect, both at the individual and population level.[3] Individual effects include the production of an immunologic response, protection against infection or in some cases only against disease or severe disease, a reduction in the degree or duration of infectivity, or even behavioural effects such as changes in the rate of contact with potentially infectious sources.[3] Population effects include a reduction in transmission of disease and/or infection. When designing a study to estimate VE, it is important to clearly define the question of interest, in particular whether individual and/or population effects are of interest, as this determines the appropriate choice of unit of observation, comparison group, parameter of effect, and level of information required.[3]

The question of interest is dependent upon the objective of the control program. If the objective of a control program is to reduce morbidity then high coverage with a vaccine protecting only against disease, or even severe disease, may be satisfactory.[5] In contrast if herd immunity or eradication is the goal, then the vaccine must clearly protect against infection.[5]

Potential biases in all study types

Any factor which differentially raises or lowers the apparent attack rate in either the vaccinated or unvaccinated group will bias the VE estimate. There are many potential biases in observational VE studies that need to be minimised in either the design or analysis phase of a study. In addition, the results should be presented in such a way that enables the reader to judge the extent to which potential biases have operated and to estimate their impact on the estimation of VE.[2]

Case definition

Ideally, case definitions should be sensitive and specific. Whilst a high sensitivity gives a more precise estimate of VE, the point estimate is not unduly affected by a low sensitivity as long as the case definition has equal sensitivity in the vaccinated and unvaccinated groups.[2] This is a problem with pertussis, as vaccinated persons often experience a milder form of the disease which is less likely to fit a clinical case definition. In these situations, it is the effectiveness of the vaccine against more serious disease, rather than against all disease or infection, that is being estimated.

For VE estimation the specificity of a case definition is generally more crucial than its sensitivity, as the misclassification of other illness as cases would equalise the attack rates in the two groups resulting in a falsely low VE estimate.[2] The rarer the disease, and the greater the incidence of misclassified illness, then the greater the bias toward low VE.[2] This bias is even greater when the case definition has low sensitivity.[2]

Point estimates of pertussis VE increase with increasing specificity of clinical case definitions[2] or when based on clinically severe or bacteriologically positive cases.[5] The same phenomenon has been observed in pertussis vaccine trials, where the greater the clinical severity of cases accepted as pertussis, the higher the VE estimates.[6] However, although laboratory confirmation increases specificity, it may lead to other biases as a result of problems with case ascertainment.[2]

Case ascertainment

In a pre-licensure trial, bias in case ascertainment is minimised by randomisation and by blinding the observer to vaccination status, neither of which is generally possible in an observational study. In observational studies, vaccinated and unvaccinated persons are self selected groups who may not have equal access to health care services, hence equal case detection cannot be assured.[2]

Studies using passively notified cases are particularly prone to bias in case ascertainment, as individuals with disease may not all have an equal probability of being notified. If notifications correlate with good public health practice and easy access to medical services and hence are associated with high vaccine uptake, then vaccinated individuals may be preferentially notified, resulting in an underestimate of VE.[5] Or if, as is the case with pertussis,[6,7] the vaccine gives greater protection against more severe disease and there is a correlation between clinical severity and the probability of a physician recognising and then notifying a case, VE will be overestimated.[5] A more serious problem occurs if, independently of disease severity, unvaccinated cases are more (or less) likely to be recognised and/or notified than vaccinated cases. For example a physician's knowledge that a child is fully vaccinated against pertussis could reduce the index of suspicion that an illness is in fact pertussis, resulting in an overestimate of VE.[5] The extent of this bias is difficult to estimate.

A pertussis outbreak investigation in the United Kingdom in 1987 found that only 31 of 90 children with bouts of coughing lasting for two or more weeks followed by whooping, vomiting or choking/turning blue (probable cases), were notified.[8] Using notified cases only the VE estimate was 88 per cent. This fell to 75 per cent when probable cases were included and 68 per cent when the case definition included all children with bouts of coughing lasting at least 2 weeks.[8] The author found that notified children were younger and less likely to be vaccinated, suggesting that children were less likely to be diagnosed and notified as pertussis if they were known to have been vaccinated.

Ascertainment of vaccination status

Classification errors in vaccination status reduce VE estimates unless there is a bias towards misrepresenting vaccinated cases as unvaccinated.[5] Studies relying on parental recall of a child's vaccination status tend to overestimate vaccination coverage, whereas studies which require verification with written records may underestimate vaccination coverage.[9]

VE estimation for diseases against which more than one dose of vaccine is necessary for full protection require information on the number of doses of vaccine given. If partial vaccination affords some protection against disease, then the way partially vaccinated cases are handled in the analysis can affect the VE estimate. If partially vaccinated cases are classified as unvaccinated but still receive some protection, the attack rate in the unvaccinated is lowered, whereas classifying partially vaccinated cases as fully vaccinated will raise the attack rate in the vaccinated. If the effectiveness of the full course of vaccination is being measured, then cases who are partially vaccinated should be excluded from the analysis wherever possible.[2]

Comparability of vaccinated and unvaccinated groups — potential confounding

In randomised controlled trials potentially confounding variables are randomly distributed among the experimental and control groups. In observational studies the groups may differ in many ways, only some of which may be recognised by the investigator.[2] Unrecognised or unmeasurable differences between the experimental and control groups such as increased susceptibility due to poor nutrition in unvaccinated

marginalised groups may pose serious threats to validity. However, the most important potential confounder in VE studies is exposure to disease. VE calculations generally assume equal exposure to infection in vaccinated and unvaccinated individuals or groups. Exposure to infection may in turn be associated with variables such as age and place of residence.

For a variable to be considered as a confounder it must be independently related to both the risk of disease and to vaccination status. Not all variables which differ in frequency between vaccinated and unvaccinated groups fulfil this requirement. Orenstein *et al*[2] give the example of a case-control study whereby cases, by definition, will have been more exposed to disease than controls but this difference in exposure does not bias the VE estimate unless the probability of exposure is also related to the probability of being vaccinated. If groups who have a greater risk of exposure (e.g. children who attend day care) are more likely to be vaccinated, then VE will be underestimated.

The indirect effects of vaccination can affect the probability of exposure in both vaccinated and unvaccinated groups, but not necessarily to an equal extent. If VE is estimated at a population level, where the study group is comprised of exposed and unexposed individuals, then whether or not the vaccine has been administered randomly will have a significant effect on the estimate.[10] If groups with high vaccine coverage are at low risk of exposure to infection, for example due to herd immunity, and VE is viewed as the degree of protection afforded to an individual who has been exposed to the disease, then clustering of vaccine status in the population may produce falsely high VE estimates.[5,10] Similarly, groups with low vaccine coverage may have greater exposure to infection resulting in falsely low VE estimates.[2] However, if the overall effectiveness of the vaccination program is being studied, then it is appropriate to include the indirect protection of vaccination in the calculation.

Age

Age may be associated with both the probability of vaccination and the probability of having had prior exposure to the disease. Where immunity from disease and/or vaccination is acquired at a young age and diminishes with time, age may be a proxy measure for time since vaccination. Data should be analysed separately for narrow age groups or otherwise standardised for age.[5]

Prior disease

If prior disease is not associated with vaccination status then VE estimates will be unbiased.[2] However, vaccinated and unvaccinated groups may differ with respect to prior disease, in which case ignoring previous histories may bias the VE estimate.[2] However, the effect of this bias must be weighed against the problem of obtaining valid histories, which for some diseases may not be feasible.

Study types

A variety of observational methods may be used to estimate VE including the well established cohort and case-control design. Each methodology has its advantages and disadvantages and methodological problems have been identified for all study types.

Cohort studies

A cohort design is most appropriate when a discrete population at risk can be defined.[11] Most cohort studies of VE have been retrospective and have generally been undertaken as part of an outbreak investigation. The cohort, often based in a school, child care centre or geographically defined area, is defined and the vaccination status of all members of the cohort is ascertained. The relative risk of disease in the vaccinated compared with the unvaccinated group is then calculated thus enabling the calculation of VE. Examples of disease outbreaks in Australia where VE has been measured using a cohort study design include a pertussis outbreak in an Australian Capital Territory school[12] and measles outbreaks in Western Australia,[13] the Australian Capital Territory,[14] Central Australia[15] and Queensland.[16]

Orenstein *et al*[1] list five criteria which minimise bias in cohort studies which are part of an outbreak investigation:

- absence of substantial prior disease activity in the studied age group;
- both vaccinated and unvaccinated individuals are included in the study population;
- adequate numbers in the population in the age group to be studied;
- high overall attack rate; and
- good vaccination records available.

Household contact studies

Household contact studies are used to measure the secondary attack rate of disease in household contacts of index cases. VE is calculated by combining the total population of the households under study, excluding the primary and co-primary cases, to form vaccinated and unvaccinated cohorts.[2] The methodology corrects for potential differences in exposure between vaccinees and non-vaccinees, thus reducing the bias that may result from differential exposure.[1] Orenstein *et al*[1] comment that next to outbreak investigations, this technique has been evaluated most and is an acceptable alternative to outbreak investigations. However, Fine and Clarkson[5] point out that the relative simplicity of the household secondary attack method should not be taken as license for its uncritical application and interpretation. No Australian household contact studies were identified in the literature.

Pertussis VE estimates derived from household secondary attack rates are generally lower than those obtained by other methods, regardless of diagnostic criteria used for case ascertainment.[17] One possible bias in these study types, which relates to pertussis vaccines and level of exposure, is the assumption that these vaccines are less effective under conditions of heavy exposure such as that within households.[5] The study of family contacts of ascertained cases, which are highly selected populations, may introduce bias. If vaccine uptake is non-random, then most or all of the vaccinated individuals in the study will be included because of a prior vaccine failure in the household (i.e. the index case). Possible risk factors for vaccine failure are likely to be shared by members of the household thus introducing a bias against the vaccine.

Again assuming non-random vaccine uptake and the likelihood that household contacts share the same vaccination status of the primary case, studies of situations in which pertussis is introduced to the household by a vaccinated case may be biased in favour of the vaccine.[5] This arises from the reduced severity of disease in vaccinated persons which may result in close contacts being exposed to fewer bacilli than the contacts of unvaccinated individuals, thus reducing the risk of infection preferentially among vaccinated contacts and raising VE estimates.

Households with larger rather than smaller numbers of cases are more likely to be identified and included in a study.[5] This ascertainment bias is likely to lower vaccine effectiveness as households in which the vaccine is working best would be selectively excluded from the study.[5]

Case-control studies

In a case-control study, cases are selected on the basis of having the disease of interest, and controls on the basis of being comparable to cases but without having the disease so that the odds ratio of vaccination can be calculated. The traditional VE equation cannot be used in case-control studies[1] as cases represent one sampling fraction of all cases and the controls represent a different sampling fraction of the population that is not ill.[18] As the sampling fraction is unknown, the total populations of vaccinated and unvaccinated people cannot be calculated and therefore neither can attack rates.[1] However, for rare diseases, the odds ratio approximates relative risk and so can be used to estimate VE. Although the VE estimate will be erroneously high when the attack rate in vaccinated persons is greater than 10 per cent,[1] in non-outbreak situations this is usually not the case and therefore the error will not be of an important magnitude.[1] In Australia case-control studies have been used to estimate VE for measles in Western Sydney[19] and for *Haemophilus influenzae* type b infection in Aboriginal children in Western Australia.[20]

Screening method

Using the screening method, VE is estimated by comparing the proportion of cases who are vaccinated (PCV) with the proportion of a comparable group in the population who are vaccinated (PPV). The standard VE equation can be rearranged to give the screening method equation, which is:[21]

$$VE = 1 - \left[\left(\frac{PCV}{1 - PCV} \right) \left(\frac{1 - PPV}{PPV} \right) \right]$$

The screening method is a simple and rapid way of estimating VE which has been used to estimate pertussis VE in the Netherlands,[22] the United States,[23] Nova Scotia,[24] the United Kingdom,[25] and New Zealand.[26] In Australia it has been used to estimate the VE of *Haemophilus influenzae* type b[27] and, more recently, pertussis.[28] The screening method is particularly useful for routine monitoring of VE or in circumstances where data on the

vaccination status of cases only are available. Provided that any biases remain reasonably constant, the screening method may be used for monitoring changes in VE over time. It should not be relied upon for precise VE estimates.[1] An overestimate in PPV will result in an overestimate of VE and this error is particularly noticeable when vaccine coverage is greater than 80 per cent.[1]

Care must be taken to stratify the data by possible confounding variables such as age and location. If different population groups have different coverage figures then the groups should be analysed individually. Farrington[21] illustrates the effect of pooling population coverage figures in an example of two cohorts, A and B, of equal size. In cohort A there are 100 cases, 50 of whom are vaccinated and the PPV is 0.9. In cohort B there are 10 cases, one of whom is vaccinated and PPV is 0.5. The screening method VE estimate is 89 per cent in each cohort. However, if the cohorts are combined, then there are 110 cases, 51 of whom are vaccinated, while the combined value of PPV is 0.7 which produces a VE estimate of only 63 per cent.[21]

Case-cohort

This study type is also known as case-base and is similar to the screening method except that vaccination status is sampled in population controls rather than using an assumed true value of PPV.[21]

Discussion

In summary, there are a variety of observational methods which can be used to estimate VE, none of which is perfect. The screening method is the most economical and rapid means of determining whether there is a major problem with the vaccine, as all that is required is a reliable estimate of the proportion of cases who are vaccinated and an estimate of the vaccine coverage in the population at risk.[1] If the screening method results suggest that VE is lower than expected, this should be confirmed by more rigorous methods. Of the more accurate observational methods available, cohort studies undertaken during an outbreak investigation offer the simplest means of VE estimation and is the preferred study design where the situation permits.[1] The most appropriate study design will depend upon the specifics of the particular situation such as availability of resources, access to records, the number and distribution of cases and the availability of population coverage data.

Whilst results obtained using observational methods may be distorted due to unavoidable bias, it may still be possible to calculate a sufficiently good estimate of VE for operational purposes.[29] Potential biases should be considered in the design phase of a VE study and steps taken to minimise them if possible. All reports of VE studies should include a discussion of the biases which may have been operating and their possible effects on VE estimates. Provided that these steps are taken, observational methods provide valuable tools for the evaluation of vaccination programs. To date, few observational VE studies have been undertaken in Australia, suggesting the underutilisation of these methods.

Acknowledgments

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Reduction in the hepatitis B related burden of disease — measuring the success of universal immunisation programs

Alison Williams

Introduction

Infection with hepatitis B virus (HBV) continues to be an important cause of morbidity and mortality throughout the world. At present, there are approximately 350 million chronic HBV carriers worldwide, with rates ranging from country to country as well as within different regions of a country.[1] For example, carrier rates are low among Caucasian populations in the United States of America (USA), Northern Europe and Australia (0.1–0.2%), but are much higher (up to 10%) in some Australian Aboriginal, Central African, and South-East Asian populations. Most east and South-East Asian and Middle Eastern countries have intermediate to high endemicity.[2]

The burden of disease resulting from HBV infection includes acute infection and chronic hepatitis B and its sequelae including chronic hepatitis, cirrhosis and hepatocellular carcinoma (HCC).[3] Acute infection with HBV is often asymptomatic, especially in infants and children, but the risk of an acute infection becoming chronic is greatest for those infected during infancy. In addition, infants who acquire infection from their mothers may rarely develop a devastating fulminant hepatitis in the first 6 months of life.[4]

Hepatitis B vaccine is one of the most effective vaccines available and 130 countries now include hepatitis B vaccine in their routine vaccination schedules for infants and/or adolescents.[5] In addition, babies born to HBV positive mothers can be protected from infection by a combination of active immunisation and passive immunisation with hepatitis B immunoglobulin (HBIG), which is most effective if given within 12 hours of birth. There is also some evidence that vaccination alone, when started at birth and at appropriate doses, provides similar protection to the HBIG plus vaccine.[6]

Many countries now recommend universal vaccination of all newborn infants for hepatitis B, regardless of the HBV status of the mother. This initiative has prompted fears about the possible adverse effects of immunising such a young and seemingly vulnerable group. However, passive surveillance (post-marketing) since the introduction of universal infant vaccination in the USA in 1991, has shown no unexpected adverse events in neonates and infants given hepatitis B vaccine, despite use of at least 12 million doses of vaccine given in these age groups.[7]

Rates of HBV sero-prevalence have dropped dramatically during the 10–15 years of follow-up after introduction of routine hepatitis B immunisation programs.[8] In some countries the drop has been by up to a factor of ten. While some of this decline may be attributable to behavioural changes in high risk groups such as needle-exchange programs, there is also evidence that protection by infant immunisation programs has played a significant role in this reduction. For example, when trends in HBV-related morbidity and mortality are taken as indirect measures of the impact of vaccination, the effect of universal infant HBV vaccination is already evident in countries such as Taiwan, where rates of hepatocellular carcinoma have been halved in some age groups.

The Taiwanese experience

Taiwan, a country known to have high prevalence of HBV infection in the past, was one of the first countries to initiate a universal infant vaccination program. Since this began in July 1984, there has been a reduction in surface antigen carriage in children from 10 per cent to less than 1 per cent, and a 50 per cent decrease in HCC in children aged 6–14 years. These spectacular reductions have been achieved within a relatively short time frame after initiation of the vaccination program. A marked decline in rates of fulminant hepatitis in infants has also been reported following introduction of universal infant vaccination in Taiwan in 1986.[4] The study, published recently in the *Journal of Pediatrics*, found that the ratio of average mortality associated with fulminant hepatitis in children less than one year of age after introduction of universal infant HBV vaccination, was one third that of the pre-vaccination period.[4]

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The USA experience

Although the USA has a comparatively 'low' rate of endemicity for HBV, it still carries a heavy disease burden with approximately 1.25 million carriers, of whom up to 5,000 die yearly.[8] Routine infant vaccination for HBV has been recommended in the United States of America since 1991, with the first dose to be given no later than 2 months of age. More recently, the Advisory Committee for Immunization Practices has recommended that the first dose be given at birth. A similar trend towards reduction in acute hepatitis B among children aged 3–14 years has been noted with a remarkable fall in the incidence of acute HBV by 80 per cent over 10 years.[9] The overall drop in reported new cases of hepatitis B has been from 13.8 per 100,000 population to 3.3 per 100,000 population over the period 1987 to 1998, with the most marked decline in those aged 10–19 years.

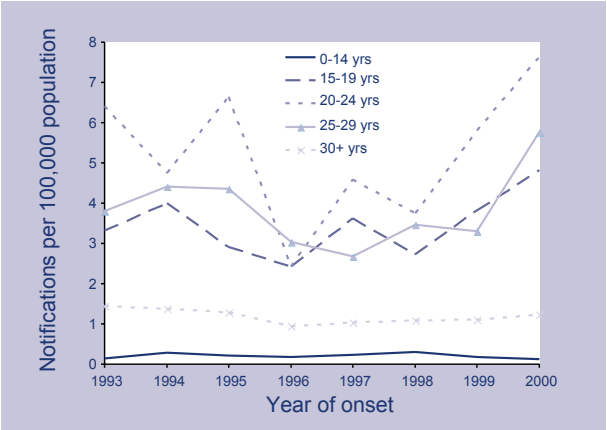
However, the success of the infant vaccination program floundered temporarily during 1999, when concerns on the thiomersal content in HBV vaccines resulted in a recommendation to delay the first dose until 6 months of age.[10] With the current availability of thiomersal-free HBV vaccines, this recommendation has been revoked, nevertheless, there remains some ongoing and unfounded reluctance to administer the birth dose of hepatitis B vaccine.[11,12]

The Australian experience

It has been estimated that there are currently 250,000 people living with chronic HBV infection in Australia.[13] HBV vaccines first became available in 1982, and were initially administered to 'at risk' groups, such as health care workers, only. Immunisation of 'at risk' infants (born to hepatitis B surface antigen positive mothers) was commenced in 1987, with the exception of South Australia which commenced in 1996. Routine adolescent immunisation commenced in 1997 and universal infant vaccination commenced in May 2000.[2]

Currently, the impact of vaccination on disease burden is less clear in the Australian setting, compared with other countries with longer-standing vaccination policies. A somewhat surprising finding was that the overall number of notifications of acute hepatitis B during 2000 was greater than at any other time since notifications began.[14] Analysis by age groups shows that although annual notifications for acute hepatitis B have stabilised or fallen in those aged under 9 years, there was a marked increase in notifications in the 15–29 year age range. This observation may have been due to the fact that this particular cohort had not been vaccinated during adolescence, and may have additional behavioural risk factors for infection (Figure). It is too early to detect the effect of universal infant vaccination on HBV infection in Australia.

Figure. Acute hepatitis B notification rate, Australia, 1993 to 2000 by age group, and month of onset[14]



Source: McIntyre P, Gidding H, Gilmour R, Lawrence G *et al.* Vaccine preventable diseases and vaccine coverage in Australia, 1999–2000. *Commun Dis Intell* 2002;26 Suppl May:19.

An Australian report of rates of hepatocellular carcinoma has shown an increase in this measure of HBV-related morbidity, in both Australian-born men and overseas-born men and women, over the past 20 years.[13] This probably reflects historical HBV prevalence patterns among these groups, and indicates that longer term follow-up is required before an effect can be demonstrated.

Why is the birth dose important? Why does it need to be universal?

In the USA, approximately 19,000 women known to have chronic hepatitis B give birth each year.[15] It has been estimated that up to 2,000 hepatitis B surface antigen (HBsAg) positive women give birth in Australia each year (A Tucker, personal communication, 2000). Assuming that a policy for universal HBV vaccination were not in place, babies may still become infected by their mother if any of the following scenarios occur:[15,16]

- the mother has been tested and found to be HBsAg positive but her status is not communicated to and acted upon by staff in the newborn nursery;
- the mother has not been tested for HBsAg prenatally and her infant does not receive the hepatitis B vaccine, even though it is recommended within 12 hours of birth;

- the mother has been tested in early pregnancy and was found to be HBsAg negative but she develops HBV infection later in pregnancy or when breastfeeding and is not re-tested; or
- the mother is HBsAg negative but the infant is exposed post-natally by another family member or care-giver. This occurs in two-thirds of cases of childhood transmission.[15]

Summary

There is collective evidence from countries of both low and high endemicity that administration of hepatitis B vaccination at birth saves lives and reduces the burden of disease from acute and chronic infection.[16] However, a discussion on the cost-effectiveness of vaccination for HBV is beyond the scope of this article.[17,18] In Australia, longer term follow-up of HBV disease burden is required following the more recent introduction of routine and universal infant vaccination.

Universal vaccination for HBV at birth can be seen as a 'safety-net' against infection at a very young age. However, it is estimated that the effect of universal infant vaccination will not be evident for at least another 15 years in Australia.

The obstacles to vaccination with HBV, which have historically included fears that the vaccine may be linked to multiple sclerosis,[19] should be put to rest, and concerns about the thiomersal content allayed by communicating the current availability of thiomersal-free vaccines to all providers and parents or care-givers. Furthermore, ongoing adverse events surveillance should be in place to detect any rare adverse events which may be related to the vaccine.

Currently, more than one half of the world's infants are still not being immunised for HBV, and the need for a global initiative for universal infant hepatitis B vaccination is apparent. This is especially true for countries with high prevalence, and the costing issues and logistics of such an initiative still remain to be addressed. In addition, there is a need to address the implementation of guidelines for screening and vaccination of families who have immigrated to Australia from countries with a high prevalence of hepatitis B.

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A review of national legionellosis surveillance in Australia, 1991 to 2000

Janet S Li,[1,2] Eddie D O'Brien,[2] Charles Guest[1]

Abstract

A study was undertaken to analyse legionellosis notifications for the period 1991 to 2000 to establish the distribution of legionellosis in Australia with the aim of identifying risk factors amenable to public health intervention. Legionellosis notification rates ranged from 0.6 cases per 100,000 population in 1991 to 2.5 cases per 100,000 population in 2000. Notifications were highest in autumn (March to May). Sixty-nine per cent of cases were males. At-risk population included those aged over 50 years. The upward trend in notification rates of legionellosis indicated that this disease remains a significant public health problem particularly among older people. Seasonal differences in notification rates require further investigation to develop appropriate prevention and control strategies. To have a better understanding of the epidemiology of legionellosis, further information is needed on smoking history, chronic illnesses, whether the notification is outbreak-related and the species of *Legionella* isolated. *Commun Dis Intell* 2002;26:462-470.

Keywords: legionellosis, Legionella longbeachae, Legionella pneumophila, *surveillance*

Introduction

Legionellosis is a collective name for the clinical syndromes caused by members of the genus, *Legionella*. It was first recognised after an outbreak of pneumonia in Philadelphia in 1976.[1] In 1978, the first Australian case was described.[2]

Legionellosis is an environmentally acquired bacterial pneumonia with no person-to-person spread. It is caused by members of the genus, *Legionella,*[3] which are aquatic organisms, widely distributed in natural and man-made habitats.[4] Transmission of the disease may, in susceptible people, follow inhalation of aerosol contaminated with pathogenic *Legionellae*. Known sources of infective aerosols include evaporative cooling towers, showers, water taps, nebulisers and whirlpool spas.[3] Outbreaks have mostly been associated with exposure to aerosols from evaporative cooling towers and complex domestic water systems.[3] However, many cases are sporadic[5,6] and for these the source of infection is seldom found.[7,8]

Legionnaires' disease is the most severe form of legionellosis and it is an important cause of community-acquired pneumonia. European and North American studies have estimated that *Legionella* species may cause between 2 and 15 per cent of all community-acquired pneumonia requiring hospitalisation.[9] The case fatality rate has been reported as ranging from 5 to 30 per cent, depending on the underlying risk factors of the patients.[10]

This paper presents an overview of the frequency and distribution of notified cases of legionellosis in Australia for the period 1991 to 2000, with the objective of identifying demographic characteristics of the disease.

Methods

Notification data of legionellosis received by the National Notifiable Diseases Surveillance System (NNDSS) between 1 January 1991 and 31 December 2000 were collated. Data were analysed using Stata statistical software (version 6).

Collection of notification data of legionellosis

Legionellosis is a notifiable disease under the public health legislation of each state and territory. All states and territories require medical practitioners to notify legionellosis and all except

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Western Australia also require laboratories to do so. Nationally, notifications of legionellosis have only been routinely collected since 1991.

The NNDSS receives de-identified notification data from each state and territory. These data include fields for sex, age in years, postcode, species details, date of onset and date of notification to the relevant health authority.[11]

Case definition

Legionellosis can be identified on clinical grounds and through laboratory diagnosis. Most states and territories use the National Health and Medical Research Council case definition,[12] a clinically compatible illness (fever, cough or pneumonia) and at least one of the following:

- isolation of *Legionella* species from lung tissues, respiratory tissue, respiratory secretions, blood or other tissues; or
- demonstration of *Legionella* species antigens in lung tissue, respiratory secretions or pleural fluid; or
- a fourfold or greater rise in indirect immunofluorescent antibody 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.

Notifications related to legionellosis outbreaks

To estimate the number of notifications related to outbreaks, legionellosis outbreaks reported in the literatures in Australia during the study period were identified by searching the Medline database. However, many outbreaks may have been unpublished. To address this issue alternative data sources that included state and territory public health bulletins, environmental health bulletins, annual surveillance reports, Master of Applied Epidemiology theses of the Australian National University and personal communication with individual departments of health were used. The number of cases by year, month of onset, season of onset, place of outbreak, putative sources and causative species from each identified report were extracted.

Statistical analyses

Calculations of crude notification rates were made using the Australian Bureau of Statistics (ABS) estimates of mid-year populations as the denominator. The trend of the crude notification rates, with and without the inclusion of the outbreak cases, over the 10-year period were examined using Poisson regression. The data were analysed to investigate differences in the incidence of legionellosis notifications according to period (year of notification) after allowing for the different population sizes. The ABS 1996 population figures were used as the denominator to calculate the age and gender-specific notification rates during the study period. To examine seasonal changes in notifications between 1996 and 2000, the number of notifications by season of onset were combined. Unpaired t-tests were used to compare the mean number of notifications in autumn versus other seasons. Degrees of freedom of seasonal comparisons were equal to n_1+n_2-2 where n_1 was the number of observations in one season, and n_2 was the number of observations in the comparing season. Sub-analyses of comparisons of seasons in *Legionella longbeachae* species and *Legionella pneumophila* species were also carried out.

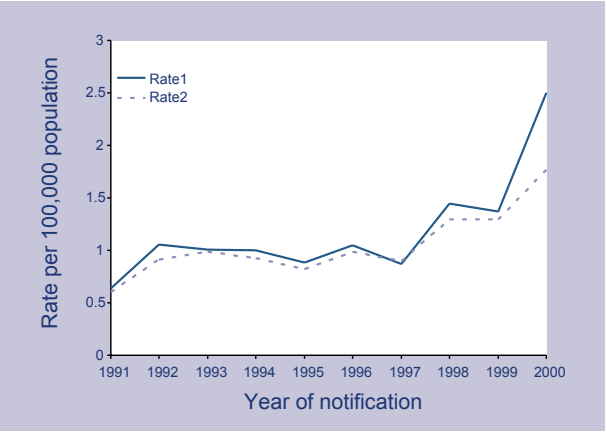
The study explored the impact of the outbreak that occurred at the Melbourne Aquarium as the large number of cases may have affected the dynamics of the seasonal comparison. To fulfil this objective the number of cases of this outbreak were removed from the data set and the sub-analysis of seasonal comparison in *Legionella pneumophila* was repeated.

Results

Trend analysis

There were 2,170 notifications of legionellosis to the NNDSS with an onset date in the study period. The trend in the period from 1991 to 1997 was flat, with the crude notification rate ranging from 0.6 to 1.1 cases per 100,000 population (Rate 1 in Figure 1). The crude notification rate raised to approximately 1.4 cases per 100,000 population in 1998 and 1999 (Rate 1 in Figure 1). The highest crude notification rate was recorded as 2.5 cases per 100,000 population in 2000 (Rate 1 in Figure 1). After excluding cases related to outbreaks, the trend remained unchanged between 1991 and 1999 except that the crude rate in 2000 dropped to 1.8 cases per 100,000 population (Rate 2 in Figure 1). The upward trend of Rate 1 (Regression coefficient of incidence rate ratio (IRR)=0.11 (95%CI: 0.10-0.13); IRR=1.12 (95%CI: 1.10-1.13); $p<0.0005$)) and Rate 2 (Regression coefficient of incidence rate ratio (IRR)=0.08 (95%CI: 0.07-0.10); IRR=1.09 (95%CI: 1.07-1.11); $p<0.0005$)) were statistically significant.

Figure 1. Crude notification rates of legionellosis, Australia, 1991 to 2000, by year



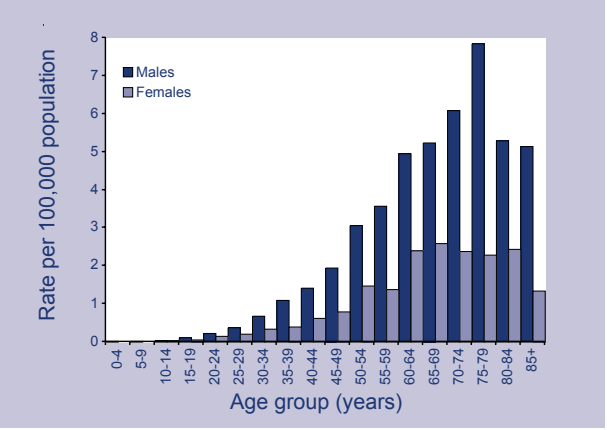
Rate1. Crude rate derived by including all legionellosis notifications during the study period.

Rate2. Crude rate derived by excluding cases related to outbreaks from legionellosis notifications during the study period.

Distribution by age and gender

There was a clear over-representation of males and elderly in notifications for legionellosis. During this period, 69 per cent of notifications were for males and 73 per cent of notifications were for people older than 50 years. The largest proportion of notifications was for males in the 75–79 years age group (Figure 2). The notification rate for males increased gradually over the adult years while the notification rate for females started to increase for those aged over 50 years, however the magnitude was much smaller than that observed in males.

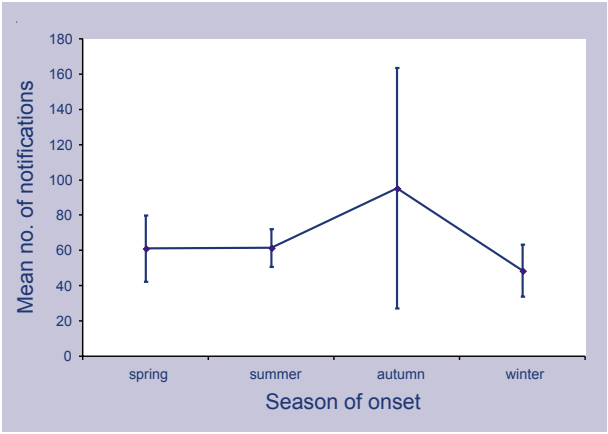
Figure 2. Notification rates for legionellosis, Australia, 1991 to 2000, by age and sex



Seasonality

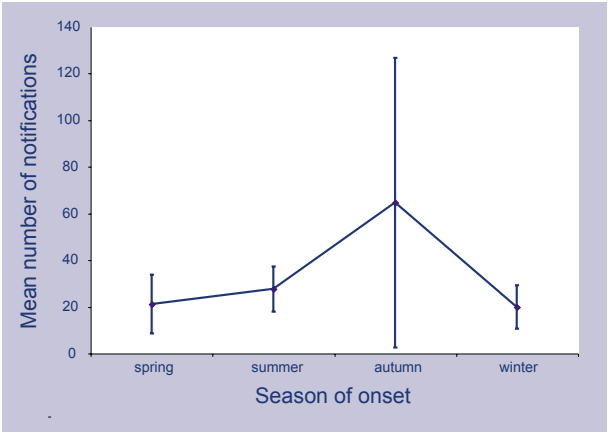
There was a seasonal pattern of legionellosis notifications between 1996 and 2000, with the mean number of notifications peaking in autumn (March to May) (Figure 3). The mean number of notifications in autumn was higher than for all other seasons however, the seasonal differences of notifications between autumn and each of the other seasons were not significant.

Figure 3. Mean number of legionellosis notifications, Australia, 1996 to 2000, by season of onset



The sub-analysis of seasonal difference of notifications in *Legionella pneumophila* species showed a similar peak in autumn (Figure 4). The difference between autumn (mean=65) and spring (mean=21) in *Legionella pneumophila* species was not significant (t_8=1.6, p=0.09). Similarly the difference between autumn (mean=65) and winter (mean=20) was also not significant (t_8=1.4, p=0.09).

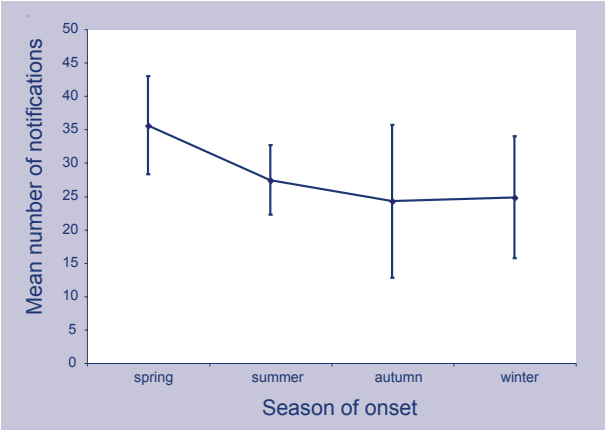
Figure 4. Mean number of *Legionella pneumophila* notifications, Australia, 1996 to 2000, by season of onset



The repetition of the sub-analysis of the a seasonal comparison in *Legionella pneumophila* which excluded the cases related to the Melbourne Aquarium outbreak increased the precision of the comparison. The difference between autumn (mean=42) and spring (mean=21) was significant (t_8=3.4, p=0.01), as was the difference between autumn and summer (mean=27.6) (t_8=2.5, p=0.03), and between autumn and winter (mean=19.8) (t_8=3.2, p=0.02).

The sub-analysis of the seasonal difference of notifications in *Legionella longbeachae* species showed a peak in spring (Figure 5). The differences between spring and summer (t_8=1.8, p=0.05), and between spring and winter (t_8=1.8, p=0.05) were significant whereas the difference between spring and autumn was not significant (t_8=1.6, p=0.07).

Figure 5. Mean number of *Legionella longbeachae* notifications, Australia, 1996 to 2000, by season of onset



Species types

Speciation of *Legionella* has only been recorded in the NNDSS since 1996. For this reason, only information on species types between 1996 and 2000 were examined. Species type data in the NNDSS were incomplete with 52 per cent of records not including this information. The NNDSS data were supplemented with data sources from individual states and territories and the results showed that *Legionella pneumophila* was the most common species (51%), followed by *Legionella longbeachae* (42%), unspecified species (4%), *Legionella bozemanii* (1%), and *Legionella micdadei* (1%).

Notifications related to legionellosis outbreaks

A total of 22 outbreaks of legionellosis were identified (Table). *L. pneumophila* serogroup 1 was the most likely cause of all of the outbreaks. These outbreaks included at least 246 cases during the 10-year period. Eight outbreaks occurred in autumn, six in summer, five in winter and one in spring. Contaminated cooling towers were suspected to be the source for 15 outbreaks while spa pools were suspected to be the source for 4 outbreaks.

Discussion

The trend of crude annual notification rates

There has been an upward trend in the number of reports of legionellosis since 1997 (Figure 1). There were several factors which contributed to this increase. Firstly, the continued increase in case ascertainment of legionellosis was partly attributable to continuing publicity about the disease and the increasing use of the urinary antigen test (UAT) since mid 1990s. The use of UAT has made the diagnosis of Legionnaires' disease caused by *L. pneumophila* serogroup 1 easier for mild cases. Nevertheless, the increased use of UAT was not solely responsible for the upward trend of notifications since 1997, but without any sign of a change in the virulence of *Legionella*,[13] it can be speculated that rapid diagnosis and increased case ascertainment were major contributors to the upward trend. Secondly, the increased notification rates in 1998 and 2000 were affected by the size of outbreaks during the period. Two *L. pneumophila* 1 related outbreaks in 1998[14] and 2000[15] accounted for 7 per cent and 24 per cent respectively, of total notifications for those years.

Distribution by age and gender

There was a marked association between age, gender and legionellosis. Overall, the ratio of disease in men to women was 2:1. Only 4 per cent of patients were 30 years or under and 73 per cent of patients were aged over 50 years. The patterns in age and sex distribution were consistent with the generally recognised epidemiology for the disease.[14,16] Males are normally regarded to be at greater risk of legionellosis, partly because they are more likely to be heavier smokers[17] and therefore may tend to have inferior respiratory and general health. Ultimately, the sex ratio of the notified cases depends upon the demography of the exposed population.[18] Also, advancing age leads to the deterioration of general health, which makes the elderly more vulnerable to the disease when exposed to *Legionellae* during gardening and shopping.

Table. Notable outbreaks of legionellosis, Australia, 1991 to 2000

No.	Year	Month	Season	Place	Area	Likely source	Species	No. of cases	Ref
1	1992	Apr	Autumn	Fairfield, Sydney	Shopping centre	*	LPsg1	26	22
2	1993	Apr	Autumn	Paramatta, Sydney	Hotel	*	LPsg1	4	23
3	1994	Apr	Autumn	Western Sydney	Hotel car park	*	LPsg1	4	19
4	1994	Jun	Winter	Western Sydney	A club	*	LPsg1	7	34
5	1994	Aug	Winter	Sunshine coast, Queenland	Holiday apartment unit	Private spa pool	LPsg1	3	35
6	1995	Jan	Summer	Sydney	Shopping centre	*	LPsg1	11	36
7	1996	Apr	Autumn	Melbourne	Metropolitan area	Not identified	LPsg1	7	37
8	1996	May	Autumn	Kangaroo Island, South Australia	Tourist resort	Spa pool	LPsg1	4	38
9	1998	Jun	Winter	Moonee Valley, Victoria	Suburban shopping district	*	LPsg1	4	39
10	1998	Oct	Spring	Thomstown, Victoria	Industrial area	*	LPsg1	18	39
11	1998	Nov	Summer	Western Sydney	Work place	*	LPsg1	3	40
12	1998	Not reported	Not	Victoria	Supported accommodation hostel	Not identified	LPsg1	2	39
13	1998	Not reported	Not reported	Victoria	Hospital	*	LPsg1	3	39
14	1999	Jan	Summer	Melbourne	Community	*	LPsg1	3	41
15	1999	Feb	Summer	Wentworth & Western Sydney	Source unclear	Not identified	LPsg1	7	42
16	1999	Jun	Winter	Melbourne	Social club	Spa pool	LPsg1	2	41
17	2000	Feb	Summer	Carlton-Fitzroy, Victoria	Community	*	LPsg1	6	15
18	2000	Mar	Autumn	Melbourne	Metropolitan area	*	LPsg1	5	15
19	2000	Apr	Autumn	Melbourne	Aquarium	*	LPsg1	125	15
20	2000	May	Autumn	Cobram, Victoria	Community	*	LPsg1	6	15
21	2000	Jun	Winter	Collingwood, Victoria	Football club	Spa pool & shower	LPsg1	4	15
22	2000	Dec	Summer	Melbourne	Private hospital	*	LPsg1	5	15

* Small cooling tower

LPsg1 *Legionella pneumophila* serogroup 1

Seasonality

Peak seasons for onset of disease appeared to be autumn followed by summer. This seasonal peak of notifications coincided with outbreaks related to small cooling towers in Australia.[15,19-23] In this study 64 per cent (14/22) of the outbreaks in the 10-year period occurred between summer and autumn. Colbourne and Dennis reported a summer to early autumn prevalence of sporadic cases of the disease in the United Kingdom.[24] Bhopal and Fallon reported an autumn peak in incidence of cooling tower-associated outbreaks in Scotland,[25] and outbreaks in the United States of America have also generally occurred in the late summer to autumn period and were generally related to small cooling towers.[24,26-28]

The effect of one autumn outbreak (the Melbourne Aquarium) on the results of the seasonal analysis of *L. pneumophila* species merits discussion because the large size of this outbreak may exaggerate the seasonal importance of autumn (n=125, with 113 of these notified in Victoria).[15] By removing this outbreak from the seasonal analysis of *L. pneumophila* the autumn peak reduced only in magnitude. The statistical significance of the comparison between autumn and other seasons were improved. It is clear that autumn had a significantly higher number of *L. pneumophila* notifications than other seasons.

Interestingly, *Legionella* colonisation of cooling towers also appears to vary seasonally.[29,30] A 12-month field study of more than 30 cooling towers in Adelaide showed that 80 per cent of towers were colonised in summer.[30] A 21-month study of 9,904 cooling tower water samples collected from New South Wales, Queensland and the Northern Territory showed a similar seasonal trend.[31] The isolation rate of *Legionellae* from these water samples peaked in the summer and autumn months.

The autumn coincidence of *Legionella* counts in tower water, outbreaks related to small cooling towers and peak notification of legionellosis merits further discussion. A model to explain this autumn coincidence has been proposed.[32] In autumn, *Legionella* populations in cooling towers are likely to be well established after the summer period. If towers are shut down, as ambient temperatures fall below 20°C, *Legionella* populations will decline until temperatures rise again in spring. However, if systems are put back into service in autumn after a short period of inactivity in response to a period of warm weather ('Indian summer'), a sudden and very rapid increase in *Legionellae* count could be expected. The increased relative humidity and reduced sunlight intensity in autumn would prolong the viability of *Legionellae* entrained in aerosols.[33] This may explain outbreak occurrence in autumn in states with a temperate climate, such as New South Wales and Victoria, but not in jurisdicitons with a tropical climate, such as the Northern Territory. During the 10-year period the Northern Territory reported between zero and 5 cases per year.

Measuring the association between the risk level of cooling towers (*Legionella* count and level of cleanliness) and onset of illness caused by *Legionella pneumophila* 1 may validate this autumn hypothesis of legionellosis. Time series data of maintenance records from a representative sample of cooling towers and information including species type, of every notification would need to be collected for this analysis.

Limitations of notification data

The epidemic potential of *Legionella pneumophila* 1 is well known, but outbreaks contribute to only a moderate proportion of all legionellosis notifications. Relatively little is known about the risk factors for sporadically occurring legionellosis. The current study cannot add to our understanding of sporadic cases.

Only larger outbreaks are likely to be published. The study was unable to account for those notifications associated with outbreaks that were not published in the literature and the number of notifications associated with outbreaks is probably higher than reported in literature.

To understand the epidemiology of sporadic legionellosis, information is needed on age, sex, onset date, causative organism, smoking history and chronic illnesses of non-outbreak related cases. The NNDSS cannot currently distinguish between outbreak-related and other notifications. Enhanced surveillance data for *Legionella* cases could include information on whether the notification was outbreak-related and whether the persons notified are smokers or have a history of chronic illnesses. Moreover, all states and territories should be encouraged to complete the data field of *Legionella* species in the NNDSS as 52 per cent of notifications had no information on reporting causative species. It is difficult to design appropriate prevention strategies for different regions since adequate information is not available to estimate which species is more prevalent in a particular region.

Conclusions and implications

Legionellosis may be an increasing public health concern in Australia. Contamination of cooling towers was a putative source for 69 per cent (15/22) of all of the outbreaks, and 36 per cent (8/22) of the outbreaks related to cooling towers in the 10-year period, occurred in autumn. While outbreak-related cases represent only a small proportion of all notifications, it is important that prompt identification and control of the sources occurs. Alerts to building managers to ensure the proper maintenance of cooling towers could also help prevent outbreaks. Since the ecology of legionellosis varies by type of infection, i.e. whether outbreak-related; travel-associated; nosocomial; occupationally-acquired; or community-acquired, national surveillance data could be improved by accurately recording such information along with the species type so that there can be a better understanding of the epidemiology of legionellosis. Future research should focus on identifying the factors contributing to the peak notification in autumn and in furthering the development of effective preventive measures.

Acknowledgments

We thank Linda Halliday for her insightful and valuable comments on the manuscript. Special thanks to Dr Chin Kei Lee for his statistical assistance and comments on the study. We also thank the Commonwealth Department of Health and Ageing for provision of the notification data for this study. Thanks also to the ACT Department of Health and Community Care, the NSW Department of Health, the Victorian Department of Human Services, the Northern Territory Department of Health and Community Services, the Western Australia Department of Health and Queensland Health for the provision of health bulletins, annual surveillance reports and notification data at state level.

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Intergovernmental Committee on HIV/AIDS, Hepatitis C and Related Diseases

History

The Intergovernmental Committee on HIV/AIDS, Hepatitis C and Related Diseases (IGCAHRD) has passed through three incarnations since its original form, the Intergovernmental Committee on AIDS (IGCA). The original committee was established in 1987 to respond to the emerging HIV epidemic. In 1997, IGCA decided to adopt related diseases such as sexually transmissible infections, invite community sector representation and to actively strengthen its links with surveillance and communicable diseases networks. IGCA became the Intergovernmental Committee on AIDS and Related Diseases (IGCARD). In 2000, given the emerging hepatitis C epidemic, IGCARD decided to specifically adopt hepatitis C as part of its mandate, and became IGCAHRD. This followed a similar change to the Australian National Council on AIDS, Hepatitis C and Related Diseases (ANCAHRD).

Role

IGCAHRD's current terms of reference were updated at the end of 2001. Its primary role is to act as a key advisory body to the National Public Health Partnership through the Communicable Diseases Network Australia (CDNA) on policy, program, social issues and activities related to HIV/AIDS, hepatitis C, sexually transmissible infections and related diseases. The committee comprises representatives from states, territories, the Commonwealth and the community sector. It aims to:

- contribute to the development and implementation of Australia's policies and programs on HIV/AIDS, hepatitis C and related diseases through jurisdictional and sector collaboration;
- ensure that national, state and territory policies reflect and address the personal, social and community aspects of HIV/AIDS, hepatitis C, sexually transmitted infections, and related diseases;
- contribute to the monitoring and surveillance of HIV/AIDS, hepatitis C and related diseases;
- provide a national network of expertise on HIV/AIDS, hepatitis C and related diseases; and
- foster the development of effective collaborative responses to HIV/AIDS, hepatitis C and related diseases.

IGCAHRD maintains links to other key committees such as the Australian National Council on AIDS, Hepatitis C and Related Diseases and the Communicable Diseases Network Australia and its other sub-committees, to ensure that effective communication is maintained on issues relevant to the sector.

Membership

Membership of IGCAHRD currently comprises:

- a Chairperson, nominated by IGCAHRD, endorsed by the CDNA, and appointed for a period of 2 years;
- one representative for both HIV/AIDS and hepatitis C matters from each State and Territory department of health;
- one HIV/AIDS and one hepatitis C representative from the Commonwealth Department of Health and Ageing, and one representative from the Office of Aboriginal and Torres Strait Islander Health, Department of Health and Ageing;
- one representative each from the Papua New Guinea National AIDS council and the New Zealand Ministry of Health;
- one representative from an appropriate national hepatitis C peak body (e.g. the Australian Hepatitis Council);
- one representative from an appropriate national peak body on injecting drug use (e.g. the Australian Injecting and Illicit Drug Users' League);
- one representative from an appropriate national peak body representing people living with HIV/AIDS (e.g. the National Association of People Living with HIV/AIDS); and

- the Chair of the Australian National Council on AIDS, Hepatitis C and Related Diseases as an ex-officio member.

In addition, the IGCAHRD may appoint subcommittees comprising either members of the IGCAHRD or other persons nominated by the IGCAHRD, to address particular issues.

Currently, three subcommittees report to IGCAHRD:

- the National HIV Surveillance Committee;
- the Viral Hepatitis Surveillance Committee; and
- the Sexually Transmitted Infections Surveillance Committee.

The Chair of IGCAHRD is Dr Linda Selvey, Manager, Communicable Diseases Unit, Queensland Health. The Deputy Chair is Ms Kim Petersen, Manager, HIV/Hepatitis C and Related Programs, South Australian Department of Human Services.

Current issues

Issues currently on IGCAHRD's agenda include:

- injecting drug use and transmission of bloodborne viruses, including in Indigenous communities — IGCAHRD has established an Indigenous Injecting Drug Use working group;
- prisoner health and bloodborne virus transmission issues in correctional settings;
- growing numbers of people seeking HIV treatment who are not eligible for Medicare coverage;
- evidence of increasing incidence of unprotected anal intercourse in Australia and implications for HIV transmission;
- cross border travel into Australia, particularly into northern Australia, of HIV-positive foreign nationals, often unaware of their HIV status;
- the national World AIDS Day campaign;
- development of a National Sexually Transmissible Infections Strategy; and
- collaboration with and links to ANCHARD and its committees.

For further information, please contact the IGCAHRD Secretary:

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Communicable diseases — a fight we can win?

The Communicable Diseases Network Australia
The Public Health Laboratory Network

Communicable Diseases Control Conference 2003

The Communicable Diseases Network Australia in conjunction with the Public Health Laboratory Network will hold their biennial 2-day Communicable Diseases Control Conference on 31 March and 1 April 2003 in Canberra at the Hyatt Hotel. The annual Master of Applied Epidemiology conference will take place on the preceding 2 days.

Aims

The conference aims to promote evidence-based discussion around emerging communicable disease themes, including:

- communicable diseases considered to be under control (e.g. poliomyelitis, measles);
- communicable diseases which are poorly controlled or re-emerging (e.g. pertussis);
- communicable diseases which are newly emerging (e.g. arboviral diseases);
- antibiotic resistance; and
- the threat of bioterrorism.

The conference is relevant to public health professionals working in all aspects of communicable disease control as well as to students in these areas.

Keynote speakers

Noted Australian and international experts working in various disciplines of communicable disease control will provide keynote addresses introducing each theme to conference participants.

Format

The conference will include keynote addresses, scientific paper presentations and poster presentations related to the themes of the conference, and will aim to deliver recommendations for improvements in communicable disease control in Australia, New Zealand and the region. A hypothetical is planned for the second day. A conference dinner will take place on the night of 31 March 2003.

Abstracts

Abstracts for oral and poster presentations will be accepted from 1 October 2002 to 7 February 2003. Early submission would be appreciated and no late submissions will be accepted. Details about submission of abstracts can be found on the conference website.

Presentations should address one or more of the following aspects of communicable diseases:

- surveillance;
- epidemiology;
- laboratory aspects;
- prevention and control; or
- public health policy.

Contact

Further information is available on the conference website at: http://www.diseases.consec.com.au. The conference email address is diseases@consec.com.au.

Communicable Diseases Surveillance

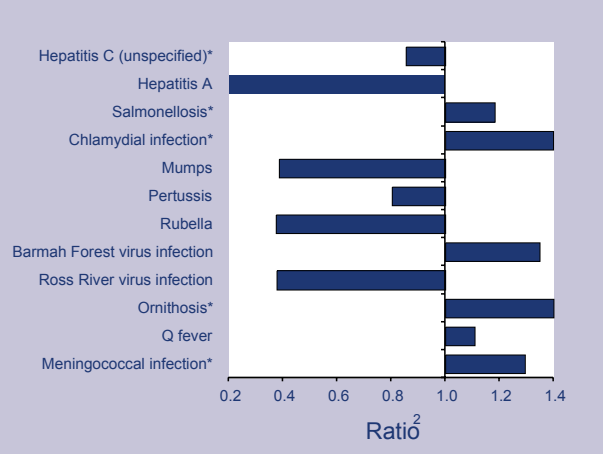
Highlights for 2nd quarter, 2002

Communicable Disease Surveillance Highlights report on data from various sources, including the National Notifiable Diseases Surveillance System (NNDSS) and several disease specific surveillance systems that provide regular reports to Communicable Diseases Intelligence. *These national data collections are complemented by intelligence provided by State and Territory communicable disease epidemiologists and/or data managers. This additional information has enabled the reporting of more informative highlights each quarter.*

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia. NNDSS collates data on notifiable communicable diseases from State or Territory health departments. The Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme which collates information on laboratory diagnosis of communicable diseases. In this report, data from the NNDSS are referred to as 'notifications' or 'cases', and those from ASPREN are referred to as 'consultations' or 'encounters' while data from the LabVISE scheme are referred to as 'laboratory reports'

Figure 1 shows the changes in disease notifications with an onset in the second quarter of 2002, compared with the 5-year second quarter mean. Disease notifications above or below the 5-year mean, plus- or minus- two standard deviations are marked with an asterisk. Diseases where the number of cases reported was two standard deviations above the mean of the same reporting period in the last 5 years in the current quarter were salmonellosis, chlamydial infections, ornithosis and meningococcal infections. The reports of unspecified hepatitis C were two standard deviations below the 5-year mean in this quarter. These and other disease trends are discussed below with additional commentary provided by state and territory health authorities.

Figure 1. Selected[1] diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 April to 30 June 2002 with historical data[2]



1. Selected diseases are chosen each quarter according to current activity.
2. Ratio of current quarter total to mean of corresponding quarter for the previous five years.

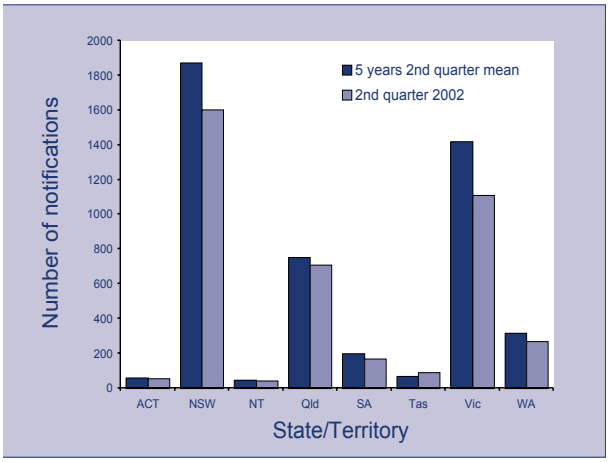
* Notifications above or below the 5-year mean for the same period plus- or minus- two standard deviations.

Bloodborne viruses

The number of unspecified hepatitis C infections in Australia has been stable since 1995 when this disease began to be separately notified. Figures from this quarter indicate a decline in unspecified hepatitis C infection, from 4,745 cases in the last 5-year mean for the second quarter to 4,058 reports for the second quarter this year. The number of notifications of unspecified hepatitis C infections are lower than the 5-year mean for every State and Territory, except Tasmania (Figure 2). The reasons for the decline are still unclear, however the decreases may be related to the improvement of surveillance practices, such as more frequent checking for duplication. It may also be that there is a smaller pool of infected individuals who have not been previously diagnosed. More time is required to determine whether this trend continues.

Incident hepatitis C notifications have also decreased from the mean of 86 cases for the second quarter based on the last 5 years' data, to 63 cases for the current quarter.

Figure 2. Notifications of unspecified hepatitis C for the second quarter 2002 compared with the 5-year mean, Australia, by State or Territory



Gastrointestinal disease

Cryptosporidiosis

The number of cryptosporidiosis notifications has fallen sharply, from 2,115 cases in the first quarter of 2002 to 580 cases in the current quarter. The decline was mainly due to the easing of outbreaks of cryptosporidiosis in Queensland, where 1,635 cases were reported in the previous quarter compared with 277 cases in this quarter. Since cryptosporidiosis only became nationally notifiable in 2001, there is no 5-year mean with which to compare the data from this quarter. The national notification rate was 12 cases per 100,000 population. The highest reporting rate was received from the Northern Territory (77 cases per 100,000 population), followed by Queensland (31 cases per 100,000 population) and Western Australia (12 cases per 100,000 population). Of the 2,695 cases of cryptosporidiosis reported with an onset date in the first 6 months of this year, 1,368 (51%) were children aged under 5 years.

Salmonellosis

The number of notifications of salmonellosis for this quarter was higher (1,943 cases) than the previous 5-year average for the same period (1,642 cases) (Figure 1). The national notification rate was 40 cases per 100,000 population, and the highest rate was reported from the Northern Territory (174 cases per 100,000 population), followed by Queensland (71 cases per 100,000 population) and South Australia (41 cases per 100,000 population).

Three major outbreaks of salmonellosis occurred in Victoria in the second quarter 2002. The first outbreak occurred in an aged care hostel in Melbourne in early April. *Salmonella* Typhimurium phage type 9 was isolated from 10 faecal specimens of the 13 patients who had specimens collected. The Victorian Department of Human Services and local government staff visited the premises and identified problems with food handling and processing. Local government environmental health officers collected food samples and environmental swabs and supervised a clean-up. No bacterial pathogens were isolated from any samples collected from the premises. The source of the outbreak was not identified.

The second outbreak occurred in June 2002, when 11 cases of ampicillin resistant *Salmonella* Typhimurium 135 were identified. Two of the reporting doctors noted that their patients had consumed pork rolls prior to the onset of disease. An investigation was initiated and a total of 26 cases were confirmed, with a further 6 suspected cases identified. Of the cases interviewed, 19 were linked to a small bakery, and all had consumed pork rolls on the same day in June. The pork rolls were made in the bakery and contained chicken liver pate (made at the bakery), egg butter made with raw eggs and oil, sliced pork loaf, cucumber, carrot and coriander. Local government authorities inspected the premises, suspended the production of pork rolls and collected food samples and environmental swabs. All food samples collected from the bakery were negative for bacterial pathogens and the primary source of the outbreak was not identified.

An outbreak of *Salmonella* Typhimurium U290 occurred in rural Victoria. Ten cases were identified and all but one had eaten pastries with cream products from the same bakery, 1–2 days before the onset of illness. A case-control study supported an association between illness and the consumption of cream and custard products from the bakery (OR indeterminate, 95%CI 13.3–∞). Departmental staff visited the site and collected samples of cream products, raw eggs, raw meat and swabs from a piping bag. Some problems with food preparation were identified. Food handlers were interviewed and three reported gastrointestinal illness, one with onset approximately 3–4 weeks prior and two with onsets within 1–2 days after the confirmed cases. All had continued to work while symptomatic. No *Salmonellae* were isolated from any of the samples collected and the primary source of the outbreak was not identified. Clean-up procedures were undertaken at the premises and advice given about safe food handling and preparation.

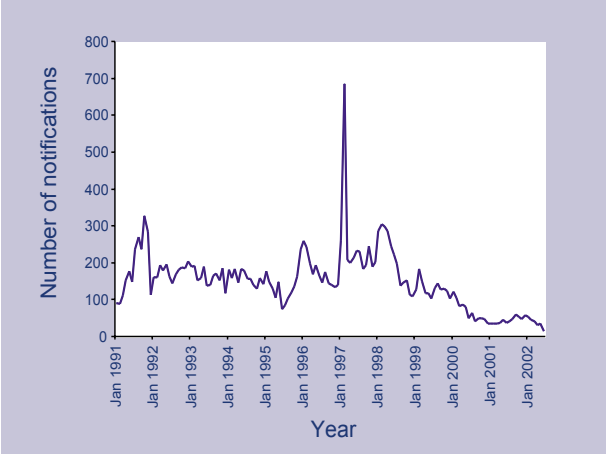
The Northern Territory has reported an increase in *Salmonella* Ball during the first 6 months of 2002 and the cause of this increase is still under investigation by the jurisdiction's health authority.

South Australia reported a large outbreak of *Salmonella* Typhimurium 8 in April 2002. A total of 78 cases were identified and 45 of those cases tested positive for S. Typhimurium 8. Food samples collected from the restaurant contained S. Typhimurium 8. A case control study was conducted and a caesar salad purchased from a restaurant in metropolitan Adelaide was implicated.

Other foodborne disease

The number of hepatitis A notifications has decreased from 141 cases in the first quarter 2002 to 80 cases reported in the current quarter, which is the lowest number of hepatitis A notifications on record (Figure 3). The national notification rate was 1.7 cases per 100,000 population with a male to female ratio of 2:1. The notifications occurred more frequently in the 20–39 years age group (38/80; 48%).

Figure 3. Notifications of hepatitis A, Australia, 1991 to 2002



In early June, a Victorian meat manufacturer recalled one of its products after tests determined the presence of *Listeria monocytogenes* in the product. Although there were 17 cases of listeriosis reported for the year to date in Australia with an onset date in the second quarter 2002, none of the cases of listeriosis were associated with consuming the meat product.

Vaccine preventable diseases

Measles

No cases of measles were reported in Tasmania, the Northern Territory, Western Australia, South Australia or the Australian Capital Territory during this quarter. Queensland reported one measles case in a partially vaccinated 2-year-old child with no history of overseas travel.

A cluster of 3 cases of measles was identified in Victoria during May and June 2002. A 29-year-old male was identified as the first case. He had not travelled in the incubation period and had an uncertain vaccination status. The second case was a 28-year-old male who was unvaccinated. Follow-up of contacts for the second case found that his 19-year-old sister had earlier reported a measles-like illness, later confirmed as measles. The sister worked in the same street where the first case lived and they may have had contact.

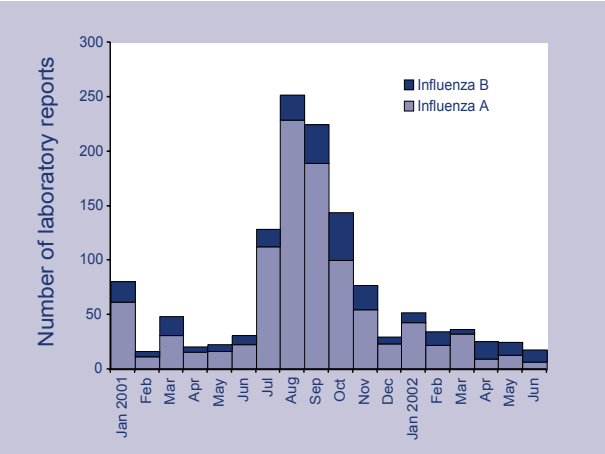
New South Wales reported 2 cases of measles this quarter. The first case was a 1-year-old unvaccinated child who had recently travelled to Pakistan and the second case was in a 1-year-old child with partial vaccination.

Influenza

There were 554 notifications of laboratory-confirmed influenza to the NNDSS during the quarter. The notification rate was highest in South Australia (31 cases per 100,000 population) where there was an outbreak of influenza type A in a health care facility. One hundred and forty-three cases were identified on a clinical basis, but only 9 were laboratory confirmed. Victoria also experienced two influenza outbreaks in schools. The first outbreak, which had onset of symptoms over 10 days, occurred in students who attended a residential camp. A total of 36 cases were identified (31 students, one teacher, one parent and 3 siblings), two of which were identified by PCR as influenza B/Hong Kong virus. The second outbreak occurred amongst secondary school students, where 100 cases of influenza-like illness were identified. Specimens were collected from 8 students and influenza B/Hong Kong virus was detected in two of the cases.

The emergence of influenza B has been observed in reports to LabVISE this quarter. The number of influenza B isolates (116 reports) has exceeded the number of reports for influenza A this quarter (Figure 4). The ratio of influenza A:B was 1:1.6 in the second quarter, but the year to date ratio is 1.4:1.

Figure 4. Laboratory reports of influenza A and B to LabVISE, Australia, 2001 to 2002, by month of specimen collection



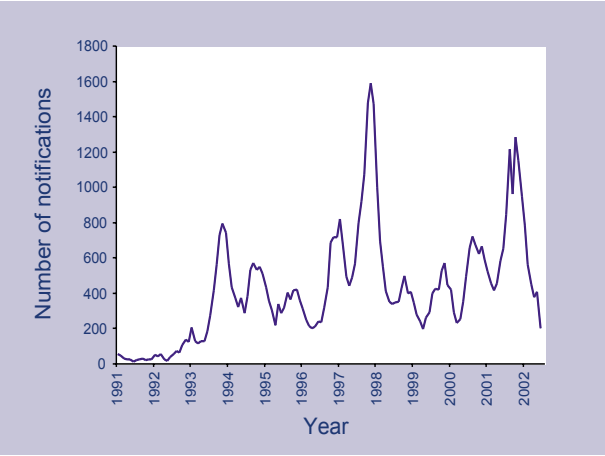
Circulation of the influenza B/Hong Kong strain has been largely absent around the world since the early 1990s. However, an outbreak of influenza B/Hong Kong strain recently occurred in Texas, United States of America.[1] The outbreak occurred late in the Northern Hemisphere influenza season with cases reported up until late May 2002. A total

of 74 laboratory-confirmed cases were identified. The exact number of cases in the community are not known as doctors are not required to report influenza in Texas. In our region, New Zealand has reported 5 cases of influenza B/Hong Kong for May and June.[2] The WHO Collaborating Centre for Reference and Research in Influenza has indicated that the current Southern Hemisphere vaccines, containing influenza B/Sichuan-like component, is expected to have reduced effectiveness against influenza B/Hong Kong-like strains. However, antibody responses to the B/Hong Kong strain have been observed in adults vaccinated with the current influenza vaccine.[3] Simonsen *et al*[4] analysed mortality data since 1972/73 season examining the number of deaths associated with influenza and pneumonia. Influenza seasons where influenza A was the dominant strain tended to be more severe (caused more deaths) than the seasons when influenza B was predominant.

Pertussis

The number of pertussis notifications received this quarter (987 cases) was lower than the 5-year mean for the second quarter (1,231 cases). The 5-year mean includes the epidemic of pertussis that occurred in 1997, but the number of notifications from this quarter is one of the lowest since mid–1993 (Figure 5).

Figure 5. Notifications of pertussis, Australia, 1991 to 2002



Vectorborne disease

This reporting period represents the third consecutive quarter with increased reporting of Barmah Forest virus (BFV) infection. A total of 329 cases of infection with BFV were reported during the current notification period, compared to a mean of 244 cases with onset dates in the same period for the previous 5 years (Figure 1). The disease was reported in five jurisdictions — New South Wales, the Northern Territory, Queensland, Victoria and Western Australia. However, the increase mainly occurred in New South Wales, where numbers rose from 93 cases of BFV infection in the previous quarter to 183 cases in the current quarter, of which 86 resided in the Hunter region.

The outbreak of Ross River virus (RRV) infection in Tasmania, reported last quarter, extended into April. A total of 53 cases were recorded during the outbreak, the majority of which were from the eastern urban fringes of Hobart. The outbreak was mainly due to an extensive rainfall in early summer period followed by a warm dry end to summer.

Zoonoses

Ornithosis

In early June, the New South Wales Health Department (NSW Health) started an investigation of an apparent outbreak of pneumonia in the Blue Mountains approximately 100km west of Sydney. The local doctors and hospitals notified NSW Health of a substantial increase in cases of pneumonia since mid-March 2002, among local residents aged between 15 and 75 years who live in the Blue Mountains.[5]

Approximately 80 cases of pneumonia were identified and the patients were asked to provide convalescent serology for testing for a range of infections, including psittacosis. Presumptive serological evidence of psittacosis was observed in 16 of 21 cases using *Chlamydia* genus IgG and IgA EIA followed by microimmunofluorescence. [5]

Q fever

During June, 9 cases of Q fever related to occupational exposure at an abattoir in south-western Victoria were notified to the Victorian Department of Human Services. The workplace had participated in a mass-screening program in the previous year but had subsequently taken on a large number of new employees. The abattoir was also receiving increased numbers of animals from Q fever endemic areas of New South Wales. Screening of new employees was organised following the outbreak with a further 11 of the 118 (9.3%) screened employees having clinical and serological evidence of recent infection. The total number of cases of Q fever related to the abattoir was twenty. There were no reports of clinical illness in previously vaccinated workers.

Other bacterial infections

Legionellosis

The total number of notifications in this quarter (79 cases) was lower than the previous 5-year mean (101 cases) for the same notification period. The cases were aged between 13 and 91 years with a male to female ration of 1.9:1. A 56-year-old male died as the result of *L. longbeachae* infection in Western Australia during this quarter.

The data on *Legionella* spp. was available for 19 cases only, of which 16 were *L. longbeachae* and three were *L. pneumophilia*. LabVISE received a total of 72 legionellosis notifications for the first 6 months of 2002, of which 21 (29.2%) were *L. longbeachae* and 46 (63.9%) were *L. pneumophilia*.

There were 2 major outbreaks of legionellosis in Victoria this quarter. During a 3-week period in April, a total of 8 cases of legionellosis (*L. pneumophila* serogroup 1) were notified in patients with recent history of exposure to a specific area within the Melbourne central business district (CBD). Cases were aged between 29 and 85 years. Three of the cases worked in the central CBD, two visited the region as a part of their work duties and the other 3 cases were casual visitors only. A total of 32 cooling towers in the area were investigated. Only one tower tested positive for *L. pneumophila* serogroup 1.

A second outbreak in Victoria occurred during a 2-week period in May. A total of 8 cases of legionellosis (*L. pneumophila* serogroup 1) were notified, all of which had recently visited a shopping district in the inner west of Melbourne. Cases were aged between 51 and 84 years, with a male:female ratio of 3:1. Four cases were local residents and another three were regular visitors. The eighth case made a single trip to the area. All premises with cooling tower systems in the shopping district and two fountains were investigated. One cooling tower tested positive for *Legionella* species but the organism isolated was identified as *L. spiritensis.* No source of the *L. pneumophila* serogroup 1 has been found.

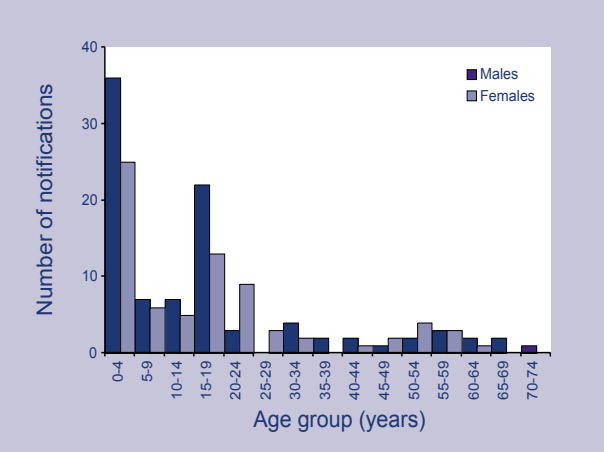
Meningococcal disease

During this quarter, there were 166 notifications of meningococcal disease reported to the NNDSS, which is an increase from the last 5-year mean for the second quarter (128 cases). The majority of notifications of meningococcal disease occurred in the 0–4 years age group and the 15–19 years age group (Figure 6). In these age groups, the number of males with meningococcal disease was higher than the number of females. However, in the 25–29 year age group, all of the reported meningococcal cases were female.

There were two clusters of meningococcal disease in Victoria. The first cluster was comprised of two university students in the same faculty who were infected with serogroup C strains. Close contacts of the students were given antibiotics and nearly 300 people at the university were given conjugate meningococcal group C vaccine. The second cluster occurred in the same child-care centre where two children were diagnosed with meningococcal disease (serogroup C). Other children in the child-care centre, staff and relatives were given antibiotics and 68 people were vaccinated with the conjugate meningococcal group C vaccine.

The national rate of meningococcal notifications was 3.4 cases per 100,000 population for this quarter. The jurisdictions with the highest rates were the Northern Territory and Tasmania (8 cases per 100,000 population). Of the 4 cases reported in the Northern Territory, two were from the same community (but with an onset date one month apart) and the other two cases were sporadic. In response to recent cases, Tasmania undertook a campaign to raise awareness about the signs and symptoms of meningococcal disease and ways to reduce the spread of the disease. A meningococcal disease immunisation campaign was implemented in Tasmania and further details are available from the Tasmanian Department of Health and Human Services.

Figure 6. Notifications of meningococcal disease, Australia, 1 April to 30 June 2002, by age group and sex



LabVISE

During the period April to June 2002, 12 participating laboratories (4 in New South Wales; 3 in Victoria; 3 in Western Australia and one in both Queensland and Tasmania) contributed 4,194

reports to LabVISE by the date of specimen collection. Although there were no contributing laboratories from the Northern Territory, samples from this jurisdiction were included in the reports from participating reference laboratories. Of the 4,194 reports received, 2,686 (64%) were of viral infections and the remainder (1,508 reports) were bacterial, spirochaete, fungal, protozoan or helminthic infections. Of the viral infections, ortho/paramyxoviruses (including influenza A and B, parainfluenza and respiratory syncytial virus) were the most frequently reported group of viral infections, accounting for 37% of viral reports. Herpesviruses (including herpes type 6, cytomegalovirus, varicella-zoster and Epstein-Barr virus) accounted for 26 per cent of viral reports. *Chlamydia* species (801 reports) accounted for more than half of all reports (53%) of non-viral infections.

During the period April to June, LabVISE received reports of 188 cases of influenza virus, 100 cases of adenovirus, 141 cases of parainfluenza, 700 cases of respiratory syncytial virus (RSV) and 79 cases of rhinovirus. Trends in the reporting of influenza and other respiratory viruses over the period 1991 to 2002 are shown in Figure 7. The patterns of seasonal variation were similar for influenza virus and RSV activities, usually with peak notifications in the winter season (June to September in Australia). The seasonal pattern of respiratory viruses shows that the peak was earlier and broader than the influenza virus peak. The distribution of reports by age shows that both RSV and influenza virus was highest in children aged 0–4 years (Figure 8).

Figure 7. Number of laboratory reports to LabVISE of influenza virus and respiratory syncytial virus, Australia, 1991 to 2002

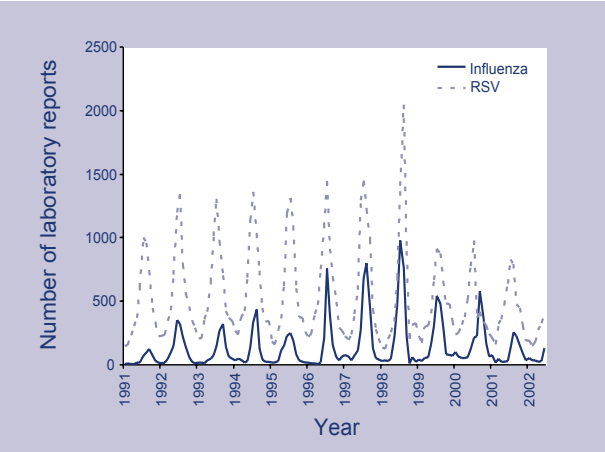
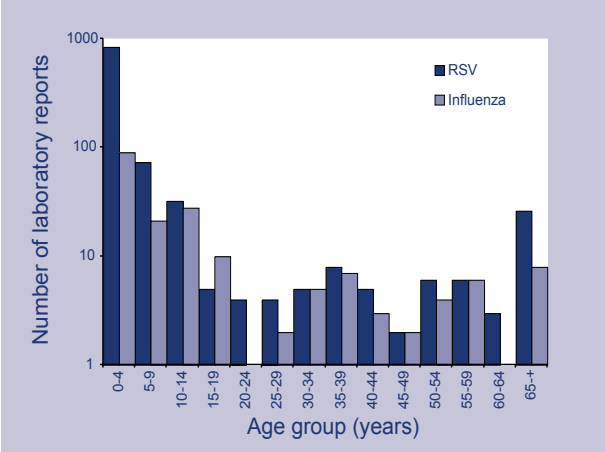


Figure 8. Number of laboratory reports to LabVISE of respiratory syncytial virus and influenza virus, Australia, 1 April to 30 June 2002, by age group and virus



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Louise Carter and Hilary McClure, Department of Health and Community Care, Australian Capital Territory.

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1. Influenza B, Hong Kong Strain — USA (Texas). ProMED mail (www.promedmail.org) 8 June 2002, Archive number 20020608.4438.

2. Influenza in New Zealand (http://www.esr.cri.nz/frame.html?content=/flu/) 28 June 2002.

3. WHO Collaborating Centre for Reference and Research on Influenza (http://www. influenza-centre.org) 28 June 2002.

4. Simonsen L, Clarke M, Williamson D, Stroup D, Arden N, Schonberger L. The impact of influenza epidemics on mortality: introducing a severity index. *Am J Pub Health* 1997;87:1944–1950.

5. Psittacosis, Gardeners — Australia (New South Wales). ProMED mail (www.promedmail.org), 13 June 2002, Archive number 20020613.4484.

Tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 22,982 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 April and 30 June 2002 (Table 2). The notification rate of diseases per 100,000 population for each State or Territory is presented in Table 3.

There were 4,194 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 April to 30 June 2002 (Tables 4 and 5).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 14-17 to 22-26, ending 30 June 2002, are included in this issue of *Communicable Diseases Intelligence* (Table 6).

Table 1. Reporting of notifiable diseases by jurisdiction

Disease	Data received from:*
Bloodborne diseases	
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)	All jurisdiction, except NT
Hepatitis C (incident)	All jurisdictions except Qld and NT
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except NSW
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Salmonellosis	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 infections	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions except SA
Gonococcal infection	All jurisdictions
Syphilis	All jurisdictions
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumoccocal disease	All jurisdictions
Poliomyeltis	All jurisdictions
Rubella	All jurisdictions
Tetanus	All jurisdictions
Vectorborne diseases	
Arbovirus infection NEC	All jurisdictions
Barmah Forest virus infection	All jurisdictions
Dengue	All jurisdictions
Japanese encephalitis	All jurisdictions
Kunjin	All jurisdictions except ACT†
Malaria	All jurisdictions
Murray Valley encephalitis	All jurisdictions†
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions except SA
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Ornithosis	All jurisdictions
Other lyssaviruses (NEC)	All jurisdictions
Q fever	All jurisdictions
Other diseases	
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, infections with Murray Valley encephalitis virus and Kunjin are combined under Murray Valley encephalitis.

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 April to 30 June 2002, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 2nd quarter 2002[1]	Total 1st quarter 2002	Total 2nd quarter 2001	Last five years mean 2nd quarter	Ratio†
Bloodborne diseases													
Hepatitis B (incident)	0	15	2	12	3	5	35	11	83	72	96	84	1.0
Hepatitis B (unspecified)	18	656	NN	182	39	10	443	96	1,444	1,835	1,487	1,759	0.8
Hepatitis C (incident)	2	21	0	NN	9	1	14	32	79	91	82	88	0.9
Hepatitis C (unspecified)	48	930	42	773	131	108	1,167	201	3,400	3,727	4,309	5,006	0.7
Hepatitis D	0	1	0	0	0	0	1	0	2	4	5	4	0.5
Gastrointestinal diseases													
Botulism	0	0	0	0	0	0	0	0	0	0	1	0	0.0
Campylobacteriosis[2]	95	_	51	949	626	188	1,234	553	3,696	4,693	3,394	3,151	1.2
Cryptosporidiosis	21	136	108	1,634	33	7	85	70	2,094	445	255	N/A	N/A
Haemolytic uraemic	0	1	0	0	0	0	1	0	2	1	2	4	0.5
Hepatitis A	3	50	9	29	3	2	34	7	137	155	96	581	0.2
Hepatitis E	0	0	0	0	0	1	0	0	1	1	1	1	0.8
Listeriosis	0	2	0	5	0	0	2	5	14	14	21	22	0.6
Salmonellosis	35	683	128	1,076	131	62	404	246	2,765	1,825	2,180	2,536	1.1
Shigellosis	0	13	33	22	12	0	16	40	136	117	116	171	0.8
SLTEC,VTEC[3]	0	0	0	1	12	0	0	3	16	11	16	11	1.5
Typhoid	0	9	0	5	1	0	10	4	29	15	33	27	1.1
Quarantinable diseases													
Cholera	0	0	0	0	1	0	0	0	1	0	0	1	1.3
Plague	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
Viral haemorrhagic fever	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

Table 2 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 April to 30 June 2002, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 2nd quarter 2002[1]	Total 1st quarter 2002	Total 2nd quarter 2001	Last five years mean 2nd quarter	Ratio†
Sexually transmissible diseases													
Chlamydial infection	125	1,067	286	1,494	383	110	1,200	745	5,410	4,910	4,696	3,495	1.5
Donovanosis	0	0	5	3	NN	0	0	0	8	6	2	6	1.3
Gonococcal infection[4]	4	310	373	238	21	5	194	334	1,479	1,539	1,457	1,432	1.0
Syphilis[5]	4	96	90	20	3	3	1	40	257	324	278	390	0.7
Vaccine preventable diseases													
Diphtheria	0	0	0	0	0	0	0	0	0	0	1	0	0.0
Haemophilus influenzae type b	0	3	1	3	1	0	2	0	10	2	5	7	1.4
Influenza	1	11	4	24	4	0	43	20	107	200	12	N/A	N/A
Measles	0	1	0	0	0	0	5	0	6	37	70	82	0.1
Mumps	0	5	0	2	1	0	6	2	16	13	31	43	0.4
Pertussis	15	505	29	555	183	22	231	85	1,625	3,210	1,217	1,464	1.1
Pneumococcal disease	0	77	11	33	15	6	61	39	242	388	87	N/A	N/A
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Rubella[6]	0	7	1	34	2	0	7	2	53	75	55	153	0.3
Tetanus	0	0	0	1	0	0	0	1	2	2	1	2	1.1
Vectorborne diseases													
Arbovirus infection NEC	0	3	0	3	0	0	1	0	7	0	9	24	0.3
Barmah Forest virus infection	0	63	12	124	3	0	36	13	251	159	324	239	1.0
Dengue	1	15	18	24	2	2	7	4	73	26	33	107	0.7
Japanese encephalitis	0	0	0	0	0	0	0	0	0	0	0	N/A	N/A
Kunjin virus infection	0	0	0	0	0	0	0	0	0	0	0	N/A	N/A
Malaria	3	40	8	57	1	2	19	12	142	130	230	251	0.6
Murray Valley encephalitis	0	0	0	0	0	0	0	3	3	0	2	N/A	N/A
Ross River virus infection	0	40	32	427	23	37	9	55	623	184	1,577	2,131	0.3

Table 2 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 April to 30 June 2002, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 2nd quarter 2002[1]	Total 1st quarter 2002	Total 2nd quarter 2001	Last five years mean 2nd quarter	Ratio†
Zoonoses													
Anthrax	0	0	0	0	NN	0	0	0	0	0	0	N/A	N/A
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	N/A	N/A
Brucellosis	0	0	0	13	0	0	0	0	13	4	6	7	1.8
Leptospirosis	0	11	1	45	0	0	4	1	62	42	70	63	1.0
Other lyssavirus	0	0	0	0	0	0	0	0	0	37	0	N/A	N/A
Ornithosis	0	3	0	2	2	0	5	1	13	0	29	17	0.8
Q fever	0	51	1	87	2	0	11	6	158	155	169	139	1.1
Other bacterial infections													
Legionellosis	0	14	0	5	7	0	22	6	54	76	65	67	0.8
Leprosy	0	0	0	0	0	0	1	1	2	1	1	1	1.4
Meningococcal infection	4	32	2	20	8	4	35	9	114	148	128	85	1.3
Tuberculosis	1	76	2	9	11	0	63	13	175	179	163	252	0.7
Total	**380**	**4,947**	**1,249**	**7,911**	**1,673**	**575**	**5,409**	**2,660**	**24,804**	**24,853**	**22,812**	**24,031**	**1.0**

1. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
2. Not reported for New South Wales because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
3. Infections with Shiga-like toxin (verotoxin) producing *E. coli* (SLTEC/VTEC).
4. Northern Territory, Queensland, South Australia , Victoria and Western Australia: includes gonococcal neonatal ophthalmia.
5. Includes congenital syphilis.
6. Includes congenital rubella.

* Date of notification = a composite of three dates: (i) the true onset date from a clinician, if available, (ii) the date the laboratory test was ordered, or (iii) the date reported to the public health authority.

† Ratio = ratio of current quarter total to mean of the same reporting period over the last 5 years calculated as described above.

N/A Not calculated as only notifiable for under 5 years.

NN Not Notifiable

NEC Not elsewhere classified.

\- Elsewhere classified.

Table 3. Notification rates of diseases by State or Territory, 1 April to 30 June 2002. (Rate per 100,000 population)

	State or Territory								
Disease[1]	**ACT**	**NSW**	**NT**	**Qld**	**SA**	**Tas**	**Vic**	**WA**	**Australia**
Bloodborne diseases									
Hepatitis B (incident)	0.0	0.9	4.0	1.3	0.8	4.3	2.9	2.3	**1.7**
Hepatitis B (unspecified)	22.9	40.1	NN	20.0	10.4	8.5	36.6	20.0	**30.0**
Hepatitis C (incident)	2.5	1.3	0.0	NN	2.4	0.9	1.2	6.7	**2.0**
Hepatitis C (unspecified)	61.1	56.8	84.8	84.9	34.8	91.9	96.4	41.9	**70.0**
Hepatitis D	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	< **0.1**
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Campylobacteriosis[2]	120.9	_	103.0	104.2	166.5	160.0	101.9	115.4	**114.8**
Cryptosporidiosis	26.7	8.3	218.2	179.4	8.8	6.0	7.0	14.6	**43.1**
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	< **0.1**
Hepatitis A	3.8	3.1	18.2	3.2	0.8	1.7	2.8	1.5	**2.8**
Hepatitis E	0.0	0.0	0.0	0.0	0.0	0.9	0.0	0.0	< **0.1**
Listeriosis	0.0	0.1	0.0	0.5	0.0	0.0	0.2	1.0	**0.3**
Salmonellosis	44.6	41.7	258.6	118.2	34.8	52.8	33.4	51.3	**56.9**
Shigellosis	0.0	0.8	66.7	2.4	3.2	0.0	1.3	8.3	**2.8**
SLTEC,VTEC[3]	0.0	0.0	0.0	0.1	3.2	0.0	0.0	0.6	**0.3**
Typhoid	0.0	0.5	0.0	0.5	0.3	0.0	0.8	0.8	**0.6**
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.3	0.0	0.0	0.0	< **0.1**
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Sexually transmissible diseases									
Chlamydial infection	159.1	65.2	577.8	164.1	101.9	93.6	99.1	155.5	**111.4**
Donovanosis	0.0	0.0	10.1	0.3	NN	0.0	0.0	0.0	**0.2**
Gonococcal infection[4]	5.1	18.9	753.5	26.1	5.6	4.3	16.0	69.7	**30.5**
Syphilis[5]	5.1	5.9	181.8	2.2	0.8	2.6	0.1	8.3	**5.3**

Table 3 (continued). Notification rates of diseases by State or Territory, 1 April to 30 June 2002. (Rate per 100,000 population)

	State or Territory								
Disease[1]	**ACT**	**NSW**	**NT**	**Qld**	**SA**	**Tas**	**Vic**	**WA**	**Australia**
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Haemophilus influenzae type b	0.0	0.2	2.0	0.3	0.3	0.0	0.2	0.0	**0.2**
Influenza	1.3	0.7	8.1	2.6	1.1	0.0	3.6	4.2	**2.2**
Measles	0.0	0.1	0.0	0.0	0.0	0.0	0.4	0.0	**0.2**
Mumps	0.0	0.3	0.0	0.2	0.3	0.0	0.5	0.4	**0.3**
Pertussis	19.1	30.8	58.6	60.9	48.7	18.7	19.1	17.7	**33.5**
Pneumococcal disease	0.0	4.7	22.2	3.6	4.0	5.1	5.0	8.1	**5.0**
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Rubella[6]	0.0	0.4	2.0	3.7	0.5	0.0	0.6	0.4	**1.1**
Tetanus	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.2	**<0.1**
Vectorborne diseases									
Arbovirus infection NEC	0.0	0.2	0.0	0.3	0.0	0.0	0.1	0.0	**0.1**
Barmah Forest virus infection	0.0	3.8	24.2	13.6	0.8	0.0	3.0	2.7	**5.2**
Dengue	1.3	0.9	36.4	2.6	0.5	1.7	0.6	0.8	**1.5**
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	3.8	2.4	16.2	6.3	0.3	1.7	1.6	2.5	**2.9**
Murray Valley encephalitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.6	**0.1**
Ross River virus infection	0.0	2.4	64.6	46.9	6.1	31.5	0.7	11.5	**12.8**
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	1.4	0.0	0.0	0.0	0.0	**0.3**
Leptospirosis	0.0	0.7	2.0	4.9	0.0	0.0	0.3	0.2	**1.3**
Other lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Ornithosis	0.0	0.2	0.0	0.2	0.5	0.0	0.4	0.2	**0.3**
Q fever	0.0	3.1	2.0	9.6	0.5	0.0	0.9	1.3	**3.3**
Other bacterial infections									
Legionellosis	0.0	0.9	0.0	0.5	1.9	0.0	1.8	1.3	**1.1**
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.2	**<0.1**
Meningococcal infection	5.1	2.0	4.0	2.2	2.1	3.4	2.9	1.9	**2.3**
Tuberculosis	1.3	4.6	4.0	1.0	2.9	0.0	5.2	2.7	**3.6**

1. Rates are subject to retrospective revision.
2. Not reported for New South Wales because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
3. Infections with Shiga-like toxin (verotoxin) producing *E. coli* (SLTEC/VTEC).
4. Northern Territory, Queensland, South Australia , Victoria and Western Australia: includes gonococcal neonatal ophthalmia.
5. Includes congenital syphilis.
6. Includes congenital rubella.

NN Not Notifiable

NEC Not Elsewhere Classified.

- Elsewhere Classified.

Table 4. Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 April to 30 June 2002, and total reports for the year[2]

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2002	This period 2001	Year to date 2002[3]	Year to date 2001
Measles, mumps, rubella												
Measles virus	-	-	-	-	-	-	4	1	5	70	5	70
Mumps virus	-	1	-	1	-	-	1	-	3	4	3	4
Rubella virus	-	-	-	10	2	-	6	-	18	10	18	10
Hepatitis viruses												
Hepatitis A virus	-	1	4	9	4	-	2	1	21	5	21	5
Hepatitis D virus	-	-	-	1	-	-	-	-	1	1	1	1
Arboviruses												
Ross River virus	-	1	18	116	16	8	3	40	202	228	202	228
Barmah Forest virus	-	5	3	49	2	-	1	8	68	59	68	59
Dengue type 2	-	-	-	-	-	-	-	1	1	-	1	
Dengue not typed	1	1	90	1	1	-	1	13	108	-	108	-
Murray Valley encephalitis virus	-	-	-	-	-	-	1	2	3	-	3	-
Kunjin virus	-	-	-	-	-	-	-	2	2	-	2	-
Flavivirus (unspecified)	-	-	1	4	-	-	4	-	9	3	9	3
Adenoviruses												
Adenovirus type 3	-	-	-	-	-	-	1	-	1	2	1	2
Adenovirus type 4	-	-	-	-	-	-	2	-	2	-	2	-
Adenovirus type 7	-	-	-	-	-	-	5	-	5	2	5	2
Adenovirus type 8	-	-	-	-	-	-	2	-	2	1	2	1
Adenovirus type 19	-	-	-	-	-	-	2	-	2	-	2	-
Adenovirus type 37	-	-	-	-	-	-	1	-	1	1	1	1
Adenovirus type 40	-	-	-	-	-	-	-	9	9	-	9	-
Adenovirus not typed/pending	-	30	-	9	41	-	35	33	148	98	148	98
Herpes viruses												
Cytomegalovirus	2	47	1	31	148	3	39	5	276	176	276	176
Varicella-zoster virus	4	48	17	144	32	2	80	149	476	248	476	248
Epstein-Barr virus	-	21	15	170	126	1	36	118	487	207	487	207
Other DNA viruses												
Molluscum contagiosum	-	-	-	-	-	-	-	5	5	-	5	-
Parvovirus	-	3	1	8	47	-	12	23	94	30	94	30
Picornavirus family												
Coxsackievirus B1	-	2	-	-	-	-	-	-	2	-	2	-
Echovirus type 6	-	9	-	1	-	-	1	-	11	-	11	-
Echovirus type 9	-	5	-	1	1	-	-	-	7	2	7	2
Echovirus type 13	-	4	-	-	-	-	-	-	4	-	4	-
Echovirus type 30	1	1	-	-	-	1	-	-	3	-	3	-
Poliovirus type 1 (uncharacterised)	-	2	-	-	-	-	-	-	2	3	2	3
Poliovirus type 2 (uncharacterised)	-	1	-	-	-	-	-	-	1	3	1	3
Poliovirus type 3	-	2	-	-	-	-	-	-	2	-	2	-
Rhinovirus (all types)	-	46	3	-	2	-	-	29	80	28	80	28
Enterovirus not typed/pending	1	-	13	2	-	1	17	76	110	29	110	29
Picorna virus not typed	-	-	-	-	-	-	12	-	12	-	12	-

Table 4 (continued). Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 April to 30 June 2002, and total reports for the year[2]

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2002	This period 2001	Year to date 2002[3]	Year to date 2001
Ortho/paramyxoviruses												
Influenza A virus	-	1	-	4	35	-	39	16	95	58	95	58
Influenza B virus	-	2	-	8	5	-	1	8	24	16	24	16
Parainfluenza virus type 1	1	32	-	5	1	-	2	2	43	4	43	4
Parainfluenza virus type 2	-	1	-	2	4	-	-	2	9	3	9	3
Parainfluenza virus type 3	-	8	-	-	35	-	1	19	63	47	63	47
Respiratory syncytial virus	-	27	2	19	21	-	10	36	115	43	115	43
Other RNA viruses												
Rotavirus	-	16	-	2	29	3	22	14	86	82	86	82
Calici virus	-	-	-	-	-	-	-	8	8	-	8	-
Norwalk agent	-	3	-	-	-	-	27	-	30	46	30	46
Other												
Chlamydia trachomatis not typed	10	116	33	303	173	4	4	271	914	442	914	442
Chlamydia psittaci	-	-	1	-	-	-	6	5	12	12	12	12
Chlamydia spp typing pending	-	-	-	-	-	-	1	-	1	1	1	1
Mycoplasma pneumoniae	-	19	4	46	81	1	87	40	278	103	278	103
Coxiella burnetii (Q fever)	1	3	1	22	9	-	7	15	58	18	58	18
Rickettsia spp - other	-	-	-	-	-	-	-	4	4	-	4	-
Streptococcus group A	-	9	8	63	-	-	16	-	96	79	96	79
Yersinia enterocolitica	-	1	-	-	-	-	-	-	1	2	1	2
Brucella species	-	-	-	2	-	-	-	-	2	-	2	-
Bordetella pertussis	-	32	11	98	121	-	78	25	365	140	365	140
Legionella pneumophila	-	2	-	-	-	-	14	-	16	3	16	3
Legionella longbeachae	-	-	-	-	2	-	3	2	7	-	7	-
Legionella species	-	-	-	-	-	-	2	-	2	-	2	-
Cryptococcus species	-	-	-	3	4	-	-	-	7	3	7	3
Leptospira species	-	2	1	7	-	-	-	1	11	3	11	3
Treponema pallidum	-	34	75	91	61	-	-	27	288	163	288	163
Entamoeba histolytica	-	-	-	1	-	-	1	4	6	1	6	1
Toxoplasma gondii	-	3	-	-	4	-	2	1	10	4	10	4
Echinococcus granulosus	-	-	-	-	4	-	2	4	10	-	10	-
Total	**21**	**541**	**302**	**1,233**	**1,011**	**24**	**593**	**1,019**	**4,744**	**2,483**	**4,744**	**2,483**

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.
2. From January 2000 data presented are for reports with report dates in the current period. Previously reports included all data received in that period.
3. Totals comprise data from all laboratories. Cumulative figures are subject to retrospective revision, so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

– No data received this period.

Table 5. Virology and serology laboratory reports for the reporting period 1 April to 30 June 2002, by laboratories[1]

	Laboratory	January 2002	February 2002	March 2002	Total this period
Australian Capital Territory	The Canberra Hospital	-	-	-	**-**
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	96	59	36	**191**
	New Children's Hospital, Westmead	24	12	55	**91**
	Royal Prince Alfred Hospital, Camperdown	30	15	9	**54**
	South West Area Pathology Service, Liverpool	21	59	68	**148**
Queensland	Queensland Medical Laboratory, West End	512	533	425	**1,470**
	Townsville General Hospital	-	-	-	**-**
South Australia	Institute of Medical and Veterinary Science, Adelaide	562	445	-	**1,007**
Tasmania	Northern Tasmanian Pathology Service, Launceston	-	-	14	**14**
Victoria	Monash Medical Centre, Melbourne	20	7	11	**38**
	Royal Children's Hospital, Melbourne	105	58	22	**185**
	Victorian Infectious Diseases Reference Laboratory, Fairfield	126	112	138	**376**
Western Australia	PathCentre Virology, Perth	391	300	320	**1,011**
	Princess Margaret Hospital, Perth	21	6	35	**62**
	Western Diagnostic Pathology	35	47	15	**97**
Total		**1,943**	**1,653**	**1,148**	**4,744**

1. The complete list of laboratories reporting for the 12 months, January to December 2002, will appear in every report regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.

\- Nil reports

Table 6. Australian Sentinel Practice Research Network reports, weeks 14-17 to 22-26, 2002

Week number Ending on	1-4 27 January 2002		5-8 24 February 2002		9-13 31 March 2002	
Doctors reporting	250		239		229	
Total encounters	26,272		26,113		25,932	
Condition	**Reports**	**Rate per 1,000 encounters**	**Reports**	**Rate per 1,000 encounters**	**Reports**	**Rate per 1,000 encounters**
Influenza	46	1.8	33	1.3	49	1.9
Gastroenteritits	239	9.1	245	9.4	239	9.2
Acute cough with chest and systemic signs	46	1.8	52	2.0	51	2.0
Acute cough with chest signs	137	5.2	162	6.2	205	7.9
Acute cough with systemic signs	45	1.7	61	2.3	75	2.9
Acute cough without signs	259	9.9	226	8.7	255	9.8

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia. The system provides the national surveillance of more than 50 communicable diseases or disease groups endorsed by the Communicable Diseases Network Australia and the National Public Health Partnership. Notifications of these diseases are made to State and Territory health authorities under the provisions of their respective public health legislation. De-identified core unit data are supplied fortnightly for collation, analysis and dissemination. For further information, see Commun Dis Intell *2002;26:58.*

LabVISE is a sentinel reporting scheme. Currently 15 laboratories contribute data on the laboratory identification of viruses and other organisms. This number may change throughout the year. Data are collated and published in Communicable Diseases Intelligence *quarterly. These data should be interpreted with caution as the number and type of reports received is subject to a number of biases. For further information, see* Commun Dis Intell *2002;26:61.*

ASPREN currently comprises about 66 general practitioners from throughout the country, not all of whom report each week. Between 4,000 and 6,000 consultations are reported each week, with special attention to 10 conditions chosen for sentinel surveillance in 2002. Communicable Diseases Intelligence *reports the consultation rates for six of these. For further information, including case definitions, see* Commun Dis Intell *2002;26:60.*

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. For more information see* Commun Dis Intell *2002;26:61.*

Reporting period 1 January to 31 March 2002

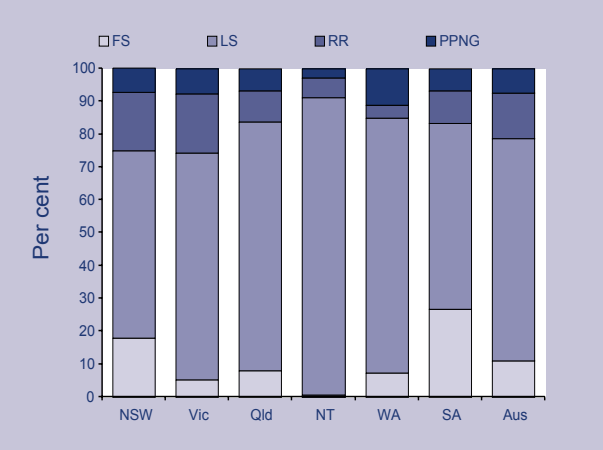
The Australian Gonococcal Surveillance Programme laboratories examined a total of 1,044 isolates in this quarter, an increase of about 10 per cent over the number in the same quarter in the past 3 years. Approximately 43.5 per cent of this total was from New South Wales where much of the increase occurred, 16.5 per cent from Victoria, 13.5 per cent from Queensland, 15.2 per cent from the Northern Territory, 8 per cent from Western Australia and 3 per cent from South Australia. Isolates from other centres were few.

Penicillins

In this quarter approximately 21 per cent of all isolates were penicillin resistant by one or more mechanisms — 7 per cent penicillinase producing (PPNG) and 14 per cent by chromosomal mechanisms (CMRNG). The proportions are relatively unchanged from the same period in 2001. The proportion of penicillin resistant strains ranged from 8.7 per cent in the Northern Territory to 26 per cent in Western Australia.

Figure 9 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 aggregated for Australia and by state and territory. A high proportion of those strains classified as PPNG or else resistant by chromosomal mechanisms fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

Figure 9. Categorisation of gonococci isolated, Australia, 1 January to 31 March 2002, by penicillin susceptibility and region



FS fully sensitive to penicillin, MIC ≤ 0.03 mg/L
LS less sensitive to penicillin, MIC 0.06 – 0.5 mg/L
RR relatively resistant to penicillin, MIC ≥ 1 mg/L
PPNG penicillinase producing *Neisseria gonorrhoeae*

The number of PPNG isolated across Australia (n = 75) continued to decline and was slightly less in this quarter than in the corresponding period in 2001 (85). The highest proportion of PPNG was found in isolates from Western Australia (17%). PPNG were present in all jurisdictions including four (2.7%) in the Northern Territory.

More isolates were resistant to the penicillins by separate chromosomal mechanisms (n = 142). These CMRNG were especially prominent in Victoria (18% of isolates) and New South Wales (17%). Nine CMRNG were detected in the Northern Territory.

Ceftriaxone

Low numbers of isolates with decreased susceptibility to ceftriaxone (MICs 0.06 mg/L) were present in New South Wales.

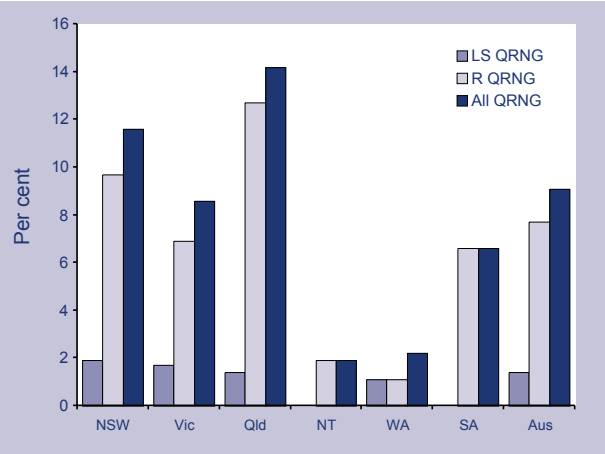
Spectinomycin

All isolates were susceptible to this injectable agent.

Quinolone antibiotics

The change seen from the same period last year was for fewer quinoline resistant *N. gonorrhoeae* (QRNG), but for those QRNG to have higher levels of resistance. The majority of QRNG (80 of 95, 84%) now exhibit higher level resistance. The total number (95) and proportion (9%) of all QRNG was about half that seen in the first quarter of 2001 (197 isolates, 21%) (Figure 10).

Figure 10. Distribution of *N. gonorrhoeae* showing quinolone resistance, Australia, 1 January to 31 March 2002



LS QRNG = Ciprofloxacin MICs 0.06 – 0.5 mg/L

R QRNG = Ciprofloxacin MICs ≥ 1 mg/L

Quinolone resistant *N. gonorrhoeae* are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06 – 0.5 mg/L) or resistant (MIC ≥ 1 mg/L) groups.

QRNG were again widely distributed. High rates were maintained in Queensland (14%) and New South Wales (11.6%). 8.6 per cent of Victorian and 6 per cent of South Australian isolates were QRNG. Forty-four of the New South Wales, 12 of the Victorian and 15 of the Queensland QRNG isolates exhibited high level resistance (MIC ciprofloxacin ≥ 1 mg/L) and higher level QRNG were also seen in the Northern Territory, South Australia and Western Australia. Local acquisition became increasingly prominent and MICs ranged up to 16mg/L.

High level tetracycline resistance

The number (136) and proportion (13%) of high level tetracycline resistant (TRNG) detected almost doubled from the corresponding period in 2001. TRNG represented between 12 and 17 per cent of isolates from Queensland, Victoria, Western Australia and New South Wales with four TRNG present in the Northern Territory.

Reference

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

HIV and AIDS surveillance

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

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in 'HIV/AIDS viral hepatitis and sexually transmissible infections Australia annual surveillance report.' The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Internet: http://www.med.unsw.edu.au/nchecr. Telephone: +61 2 9332 4648. Facsimile: +61 2 9332 1837. For more information see Commun Dis Intell *2002;26:59.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 January to 31 March 2002, as reported to 30 June 2002, are included in this issue of Communicable Diseases Intelligence *(Tables 7 and 8).*

Table 7. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 January to 31 March 2002, by sex and State or Territory of diagnosis

	Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Totals for Australia			
										This period 2002	This period 2001	Year to date 2002	Year to date 2001
HIV diagnoses	Female	0	4	1	5	3	0	9	2	24	26	24	26
	Male	1	63	1	18	3	0	56	4	146	168	146	168
	Not reported	0	1	0	0	0	0	0	0	1	0	1	0
	Total[1]	1	71	2	23	6	0	65	6	174	195	174	195
AIDS diagnoses	Female	0	0	0	1	1	0	0	1	3	4	3	4
	Male	0	6	1	8	6	0	4	3	28	31	28	31
	Total[1]	0	7	1	9	7	0	4	4	32	36	32	36
AIDS deaths	Female	0	0	0	0	2	0	0	0	2	2	2	2
	Male	0	6	0	0	2	0	0	2	10	13	10	13
	Total[1]	0	6	0	0	4	0	0	2	12	15	12	15

1. Persons whose sex was reported as transgender are included in the totals.

Table 8. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 31 March 2002, by sex and State or Territory

	Sex	State or Territory								
		ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Australia
HIV diagnoses	Female	28	678	11	185	75	5	259	134	**1,375**
	Male	232	11,640	113	2,170	730	80	4,257	984	**20,206**
	Not reported	0	242	0	0	0	0	24	0	**266**
	Total[1]	260	12,585	124	2,362	805	85	4,556	1,124	**21,901**
AIDS diagnoses	Female	9	208	0	52	29	3	79	30	**410**
	Male	88	4,827	38	890	369	45	1,729	384	**8,370**
	Total[1]	97	5,048	38	944	398	48	1,817	416	**8,806**
AIDS deaths	Female	4	122	0	35	18	2	57	19	**257**
	Male	71	3,348	25	592	244	30	1,313	266	**5,889**
	Total[1]	75	3,479	25	629	262	32	1,377	286	**6,165**

1. Persons whose sex was reported as transgender are included in the totals.

Childhood immunisation coverage

Tables 9, 10 and 11 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR).

The data show the percentage of children fully immunised at 12 months of age for the cohort born between 1 January and 31 March 2001, at 24 months of age for the cohort born between 1 January and 31 March 2000, and at 72 months of age for the cohort born between 1 January and 31 March 1996 according to the Australian Standard Vaccination Schedule. As at 30 June 2002, all children assessed for coverage at six years of age (those between 72 - <75 months of age), had a date of birth equal to or greater than the commencement date of the ACIR, enabling immunisation coverage reports to be extracted for this oldest age group.

A full description of the methodology used can be found in Commun Dis Intell *1998;22:36–37.*

Commentary on the trends in ACIR data are provided by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases. For further information please contact the NCIRS at: telephone: +61 2 9845 1256, E-mail: brynleyh@chw.edu.au.

Immunisation coverage for children 'fully immunised' by 12 months for Australia has decreased marginally from the last quarter by 0.3 per cent to 90.2 per cent (Table 9). The change in 'fully immunised' coverage varied by state and territory. The Australian Capital Territory (–1.6%) and the Northern Territory (–1.1%) experienced the greatest decreases in coverage. All other states experienced only marginal increases or decreases in coverage over the quarter. Coverage is hovering around the 90 per cent level in almost all jurisdictions with the highest level in Tasmania (91.7%) and the lowest in Western Australia (88.5%). Despite this, Western Australia was the only jurisdiction that experienced an increase (+0.1%) in Hib coverage at 12 months of age. The Australian Capital Territory (–2.0%), New South Wales (–1.4%) and the Northern Territory (–1.4%) experienced the greatest decrease in Hib coverage.

Table 9. Percentage of children immunised at 1 year of age, preliminary results by disease and State for the birth cohort 1 January to 31 March 2001; assessment date 30 June 2002

Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	1,008	21,139	902	12,141	4,376	1,460	14,882	6,082	**61,990**
Diphtheria, Tetanus and Pertussis (%)	91.2	91.7	89.7	91.7	92.1	93.0	92.5	90.2	**91.8**
Poliomyelitis (%)	91.2	91.7	89.4	91.6	92.1	92.9	92.5	90.1	**91.7**
Haemophilus influenzae type b (%)	92.7	93.1	94.7	93.7	94.3	95.2	94.3	93.2	**93.7**
Hepatitis B (%)	93.8	94.3	95.1	94.1	94.9	94.7	93.7	92.6	**94.0**
Fully immunised (%)	89.8	89.9	88.6	90.6	90.9	91.7	90.7	88.5	**90.2**
Change in fully immunised since last quarter (%)	+1.6	-0.7	-1.1	-0.2	+0.3	+0.7	-0.3	+0.5	**-0.3**

The decrease in coverage at 12 months of age for most jurisdictions and for all vaccines indicates that coverage appears to have reached a plateau for this age group. The trend in 'fully immunised' coverage at 12 months of age has been slightly downward since early 2001 when it peaked at 91.5 per cent, 1.3 per cent higher than the latest estimate. Whilst this decrease over a 12-month period is not substantial, it is of concern as some jurisdictions have now dropped back to just under 90 per cent coverage. Although there has been adverse media coverage relating to the MMR vaccine, this should not have impacted on coverage at 12 months of age in any direct way.

In comparison, immunisation coverage for measured by 'fully immunised' at 24 months for Australia increased marginally from the last quarter by 0.3 percentage points to 88.1 per cent (Table 10). Coverage increased from the previous quarter in 5 states and territories, South Australia (+2.3%), Tasmania (+2.2%), the Northern Territory (+1.4%), New South Wales (+1.1%) and the Australian Capital Territory (+0.1%). Victoria, Queensland, and Western Australia all experienced small decreases in coverage over the quarter. Tasmania was the first state to achieve greater than 90 per cent coverage for 'fully immunised' at 24 months of age. Almost all other states are approaching 90 per cent coverage, most within 1–2 percentage points of this target. Coverage for individual vaccines by 24 months for Australia, however, is much greater than for 'fully immunised' with coverage for Hib at 95 per cent and coverage for poliomyelitis (OPV) and measles-mumps-rubella (MMR) vaccines approaching 95 per cent. At the jurisdiction level, there were no important changes in coverage except for the significant increase in diptheria-tetanus-pertussis (DTP) vaccine coverage at 24 months for Tasmania (+2.7%) and the Northern Territory (+2.0%).

Table 11 shows immunisation coverage estimates for 'fully immunised' and for individual vaccines by 6 years of age for Australia and by State or Territory. These are the first official ACIR figures of immunisation coverage estimates for this age group to be published. Coverage estimates are presented for DTP (the 5th dose), OPV (the 4th dose) and MMR (the 2nd dose). 'Fully immunised' coverage at 6 years of age for Australia is 80.6 per cent. Coverage for this age group varies significantly by state or territory. The Northern Territory has the lowest coverage at 72 per cent, whilst Victoria has the highest coverage at 83 per cent. Coverage by individual vaccine also varies with coverage greater for the 5th dose of DTP (84%) and the 4th dose of OPV (84%) than for the 2nd dose of MMR (82%). This pattern exists across all jurisdictions. However, the recently published NCIRS study on MMR coverage shows that the ACIR MMR coverage figure is likely to be an under-estimate of the true coverage level at 6 years of age, which is actually 4 per cent greater at 86 per cent for an earlier birth cohort.[1]

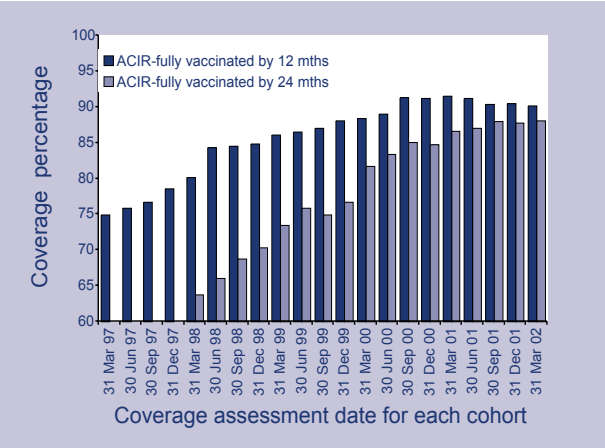
Table 10. Proportion of children immunised at 2 years of age, preliminary results by disease and State for the birth cohort 1 January to 31 March 2000; assessment date 30 June 2002[1]

Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	987	21,864	894	12,613	4,513	1,509	15,311	6,274	63,965
Diphtheria, Tetanus, Pertussis (%)	90.2	90.4	88.8	90.6	91.0	93.1	90.6	87.7	90.3
Poliomyelitis (%)	93.9	94.0	95.9	93.9	95.4	95.6	94.8	93.1	94.2
Haemophilus influenzae type b (%)	94.7	95.0	95.0	94.5	95.6	96.2	95.5	93.7	95.0
Measles, Mumps, Rubella (%)	93.4	93.1	95.2	92.9	94.2	94.3	93.7	91.4	93.2
Fully immunised (%)[2]	88.6	88.0	87.2	88.5	89.8	91.8	88.3	85.0	88.1
Change in fully immunised since last quarter (%)	+0.1	+1.1	+1.3	-0.3	+2.3	+2.2	-0.5	-1.3	+0.3

1. 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.

Figure 11 shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months and 24 months. However, the rate of increase in coverage is slowing with the curve beginning to flatten out and turn downward slightly for estimates at 12 months of age.

Figure 11. Trends in vaccination coverage, Australia, 1997 to 2002, by age cohorts



Acknowledgment

The table figures were provided by the Health Insurance Commission (HIC), to specifications provided by the Commonwealth Department of Health and Ageing. For further information on these figures or data on the Australian Childhood Immunisation Register please contact the Immunisation Section of the HIC: Telephone: +61 2 6124 6607.

Reference

1. National Centre for Immunisation Research and Surveillance (NCIRS). Immunisation Coverage: Australia 2001. Report. Canberra: Department of Health and Ageing, 2001. http://www.health.gov.au/pubhlth/immunise/report.pdf

Table 11. Percentage of children immunised at 6 years of age, preliminary results by disease and State for the birth cohort 1 January to 31 March 1996; assessment date 30 June 2002

Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	1,031	21,859	15,885	12,715	4,818	6,621	1,555	881	65,365
Diphtheria, Tetanus, Pertussis (%)	83.7	83.2	85.0	84.5	84.7	81.2	82.4	75.8	83.7
Poliomyelitis (%)	83.7	83.2	85.6	84.9	84.9	81.3	83.1	76.4	84.0
Measles, Mumps, Rubella (%)	82.7	80.0	85.0	84.1	83.4	80.6	81.4	76.4	82.4
Fully immunised (%)	81.3	78.3	83.3	82.6	81.8	78.3	79.7	72.0	80.6

National Enteric Pathogens Surveillance System

The National Enteric Pathogens Surveillance System (NEPSS) collects, analyses and disseminates data on human enteric bacterial infections diagnosed in Australia. These pathogens include Salmonella, E. coli, Vibrio, Yersinia, Plesiomonas, Aeromonas *and* Campylobacter. Communicable Diseases Intelligence *reports only on* Salmonella.

Data are based on reports to NEPSS from Australian laboratories of laboratory-confirmed human infection with Salmonella. Salmonella *are identified to the level of serovar and, if applicable, phage-type. Infections apparently acquired overseas are included. Multiple isolations of a single* Salmonella *serovar/phage-type from one or more body sites during the same episode of illness are counted once only. The date of the case is the date the primary diagnostic laboratory isolated a* Salmonella *from the clinical sample.*

Note that the historical quarterly mean count should be interpreted cautiously, and is affected by surveillance artefacts such as newly designated and incompletely typed Salmonella.

We thank contributing laboratories and scientists.

Reported by Joan Powling (NEPSS Co-ordinator) and Mark Veitch (Public Health Physician), Microbiological Diagnostic Unit — Public Health Laboratory, Department of Microbiology and Immunology, University of Melbourne. For further information please contact NEPSS at the above address or on Telephone: +61 3 8344 5701, Facsimile: +61 3 8344 5701.

Reports to the National Enteric Pathogens Surveillance System of Salmonella *infection for 1 April to 30 June 2002 are included in Table 12. Data include cases reported and entered by 20 July 2002. Counts are preliminary, and subject to adjustment after completion of typing and reporting of further cases to NEPSS. The top 25* Salmonella *types identified in each Australian State and Territory in the same period is shown in Table 13.*

During the second quarter of 2002, the 25 most common *Salmonella* types in Australia accounted for 1,310 (67%) of all 1,960 reported human infections.

Table 12. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 January to 31 March 2002, as reported to 15 April 2002

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total all *Salmonella* for quarter	20	508	84	639	150	35	359	165	1,960
Total contributing *Salmonella* types	11	100	46	108	47	10	72	65	221

Table 13. Top 25 *Salmonella* types identified in Australian States and Territories, 1 April to 30 June 2002

National	*Salmonella* type	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 2nd quarter 2002	Last 10 years mean 2nd quarter	Year to date 2002	Year to date 2001	Total 2001
1	S. Typhimurium 135	4	55	3	18	2	3	66	21	172	109	445	361	636
2	S. Typhimurium 9	1	38	1	27	1	0	61	10	139	99	445	258	399
3	S. Typhimurium 170	1	32	0	23	1	0	69	1	127	14	265	39	148
4	S. Saintpaul	0	8	3	59	7	0	11	13	101	79	261	173	289
5	S. Typhimurium 126	1	42	1	6	7	1	16	1	75	20	140	129	313
6	S. Virchow 8	0	1	0	66	0	0	2	0	69	30	225	161	245
7	S. Bovismorbificans 24	6	56	0	2	0	0	0	4	68	3	75	13	18
8	S. Birkenhead	0	25	1	27	0	0	1	1	55	50	171	163	253
9	S. Typhimurium 8	0	0	1	2	43	0	0	6	52	10	60	16	26
10	S. Chester	0	9	6	27	2	1	1	4	50	37	107	99	166
11	S. Hvittingfoss	0	2	1	39	0	1	2	0	45	15	110	57	89
12	S. Muenchen	0	10	2	16	1	0	3	9	41	33	87	88	125
13	S. Mississippi	0	0	0	1	2	24	4	0	31	13	69	97	123
14	S. Aberdeen	0	0	0	26	1	0	2	0	29	26	100	60	88
15	S. Typhimurium U290	1	11	0	1	0	1	13	2	29	<1	56	7	26
16	S. Virchow 34	0	6	0	19	1	0	2	0	28	14	69	52	87
17	S. Potsdam	0	13	0	11	0	0	2	2	28	13	54	40	60
18	S. Agona	0	16	0	7	2	1	1	1	28	13	53	30	56
19	S. Waycross	0	7	0	18	0	0	0	0	25	25	75	33	54
20	S. Montevideo	0	11	0	7	0	0	5	0	23	6	55	17	27
21	S. Typhimurium 12	0	3	0	8	4	0	5	0	20	10	42	37	62
22	S. Ball	0	0	18	2	0	0	0	0	20	6	41	16	35
23	S. Infantis	0	7	0	4	2	0	5	1	19	27	61	70	123
24	S. Mgulani	0	0	3	15	0	0	0	0	18	10	48	36	67
25	S. Typhimurium 197	2	9	0	6	0	0	1	0	18	<1	22	4	8

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.

Dengue fever: global update

El Salvador

El Salvador has reported 1,773 cases of dengue infection, six of which died. All areas (departments) of the country have been affected; based on overall rates, the worst affected departments are San Salvador, Santa Ana, Cabanas, and Cuscatlan.

Malaysia

In Malaysia, there have been 3,156 dengue fever cases in Selangor. So far, 5 people have died from the disease. 'There has been a 38 per cent increase in the total number of cases so far this year compared to the same period last year,' (an official) said. Most of the cases were dengue fever while 183 cases were the more deadly form, dengue haemorrhagic fever. Of the total number of reports, 1,222 cases or 38.7 per cent were confirmed cases. Control measures being taken include fogging at all areas where cases were reported, eliminating breeding spots, destroying *Aedes* larvae and deploying residents to get involved in clean-ups.

Honduras

Source: http://www.nacion.co.cr/ln_ee/2002/julio/15/mundo7.html

The government of Honduras announced that the number of deaths due to dengue haemorrhagic fever in the past 6 months might increase to twenty-three. The official reports indicate that as of 15 July 2002, the disease has been responsible for 9 deaths. As of 15 July 2002, there have been more than 6,262 cases of classical dengue fever and 545 cases of dengue haemorrhagic fever identified in the country. With the arrival of the rainy season at the end of May, the government has mobilised a large number of brigades with instructions to eliminate standing water where the *Aedes agypti* mosquito breeds.

Taiwan

Source: Taipei Times, 15 July 2002 (edited) http://www.taipeitimes.com/news/2002/07/15/story/0000148315

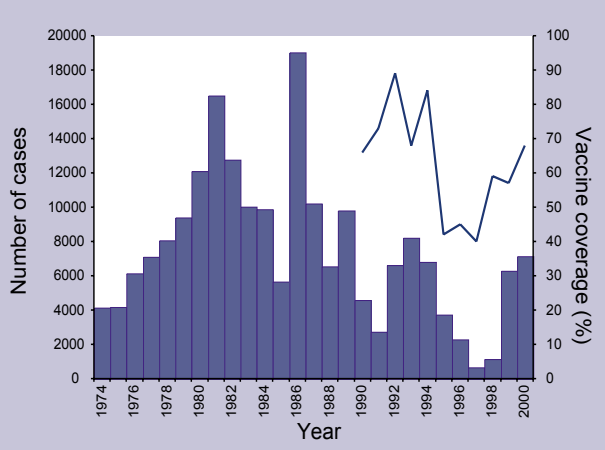
Southern Taiwan is experiencing a major outbreak of dengue fever with an average of 10 confirmed cases reported every day. As of 15 July 2002, there were 283 confirmed cases, including 202 in Kaohsiung City, 80 in Kaohsiung County, and one in Taipei County. Three of the cases have been confirmed as dengue haemorrhagic fever, which can be fatal if not recognised and treated properly.

Measles - Papua New Guinea

A measles outbreak in the Western Highlands of Papua New Guinea has killed at least 102 children in the past 4 months. Over 1,200 paediatric measles patients were admitted to the Mt Hagen hospital between January and April 2002, with 426 cases in April alone. Limited supplies of drugs to treat the disease were received from the Australian Agency for International Development on 29 April 2002, but one medical officer said that would not be sufficient to curb the outbreak.

The severity of this outbreak is unusual and suggests either low vaccination coverage in the affected area or perhaps a breakdown in the cold chain for the vaccine distribution. Historical notifications and recent rates of vaccine coverage are illustrated in the Figure. The medications would be used for treatment of complications and symptoms of measles, but would not interrupt transmission of the virus. The only intervention that would interrupt transmission of the virus would be vaccination of the high-risk population with potent measles vaccine.

Figure. Number of measles cases and measles vaccine coverage rates, Papua New Guinea



Source: ProMed

Recombinant forms of HIV spreading globally

The human immunodeficiency virus type 1 (HIV-1) is currently classified into 3 major groups: M, N, and O. Group M encompasses the global spread of HIV-1 and comprises several subtypes or clades, designated A to H, that have appeared in different geographic settings. Genetic interaction between these subtypes and the apparent evolution of new genotypes of as-yet-unknown disease-producing potential are described in two new reports.

Newly identified recombinant forms of the HIV-1 are being spread to diverse geographic regions, which could have implications for the development of an AIDS vaccine. Researchers at the Laboratoire Retrovirus in Montpellier, France, characterised the genome sequences of isolates from an individual in Senegal and one in Mali. The 2 isolates matched two others described in patients from Burkina Faso and Mali. All 4 isolates comprised a mosaic genome structure that included fragments from HIV subtypes A, G, K, and J. They matched isolates from Burkina Faso, Mali, the Ivory Coast, and Nigeria. The strains have also been isolated from patients in France and Australia, indicating global spread. The group notes the importance of seeking biological differences among subtypes and circulating recombinant forms and tracking their molecular epidemiology; 'because recombination may introduce genetic and biological consequences that are far greater than those resulting from the steady accumulation of single mutations'.[1]

In another report, Spanish researchers detail the characterisation of the first reported recombinant to originate in Western Europe. Its genome is made up primarily of subtype G, with the extracellular portion subtype B's env. The researchers also note that a parental non-recombinant subtype strain has been isolated in the same area, implying that the recombinant virus was most likely generated locally in north-western Spain or in Portugal.[2]

References

1. *J Acquir Immune Defic Syndr* 2002;29:522-30.
2. *J Acquir Immune Defic Syndr* 2002;29:536-43.

Toxic ingestion, sodium nitrite — USA (New York)

A food preservative was to blame for the sudden outbreak of illness that triggered a chemical-contamination scare in downtown New York. Six victims were taken to St Joseph's Medical Center with severe breathing problems, bluish pallors and mental confusion. Because the cause was not immediately known, police, firefighters, hazardous-materials workers and anti-terrorist agents were deployed. The victims had been at a dinner party featuring a dish that included processed food from Egypt. Officials narrowed their hunt for a cause to a substance in a packet labelled in English and Arabic, that the victims said they had sprinkled on their meal. “A sample from a packet labelled ‘refined iodised table salt’ did not contain table salt — it was 100 per cent sodium nitrite” said an official.

Accidental excessive ingestion of sodium nitrite (saltpetre) has been responsible for prior cases or outbreaks of methemoglobinemia following food ingestion. Accidental mis-labelling of powdered substances that make their way into food preparation is, unfortunately, not an uncommon event.

New ‘superbug’ found in hospital: the ESBL bacterium can break down antibiotics - UK

A new 'superbug' which can neutralise antibiotics and cause fatal blood poisoning has been found at a Lanarkshire hospital. The extended-spectrum beta-lactamases (ESBL) superbug is reported to have claimed the life of one patient at one of Scotland's most modern hospitals, which opened in 2001. Between July 2001 and April 2002, 41 patients contracted the bacteria, which produce enzymes that break down common antibiotics. A report, published by the Scottish Centre for Infection and Environmental Health claims the 'bug' could become the new methicillin resistant *Staphylococcus aureus*.[1] Last month, cardiac surgery at Edinburgh Royal Infirmary was halted after it emerged that 13 patients had contracted the superbug.

First described in Europe in the early 1980s, ESBL-containing Gram negative bacilli have become a widespread problem[2] especially in western and southern Europe.

References

1. http://www.show.scot.nhs.uk/scieh/PDF/weekly_report.pdf
2. Paterson DL. Extended-spectrum beta-lactamases: the European experience. *Curr Opin Infect Dis* 2001;14:697-701.

Poliomyelitis eradication — European Region certified

Source: World Health Organization (edited) <http://www.who.int/inf/en/pr-EURO.2002-12.02.html>

The historic decision to certify the European Region of the World Health Organization (WHO) polio-free was announced today. The European Region has been free of indigenous poliomyelitis for over 3 years. Europe's last case of indigenous wild poliomyelitis occurred in eastern Turkey in 1998. Poliovirus imported from polio-endemic countries remains a threat. A decade ago, imported poliovirus paralysed 71 people and caused 2 deaths in a community in the Netherlands that refused vaccination.

The path to a polio-free European Region began in 1988, following the call of the World Health Assembly to eradicate poliovirus. Success was achieved through an unprecedented series of coordinated national immunisation campaigns, known as Operation MECACAR. Some 60 million children under 5 years of age received 2 extra doses of polio vaccine every year from 1995 to 1998. This synchronisation of immunisation among neighbouring countries has become a model for eradicating the disease globally.

Since the Global Polio Eradication Initiative was launched in 1988, 2 WHO regions have been certified polio-free: the Americas in 1994 and the Western Pacific in 2000. Poliomyelitis cases have dropped from an estimated 350,000 in 125 countries in 1988 to just 480 reported cases in only 10 polio-endemic countries in 2001.

Influenza B virus, Hong Kong strain; late-season outbreak in Texas

A rare strain of influenza not protected against by the current United States of America influenza vaccine has caused a late-season outbreak in Houston. In what is probably an omen of the upcoming winter influenza season, the Hong Kong strain of influenza B virus made several hundred school-age children in Houston, Galveston, El Paso, and Austin ill in March, April, and May 2002.

The outbreak was noteworthy because of the lateness of its occurrence and the rarity of the strain. It is highly unusual for Texas to have outbreaks in May, and it may indicate continued transmission over the summer. Doctors confirmed that the first Texas case occurred on 6 March 2002 and the most recent case on 28 May 2002. There have been no cases in June. The outbreak was attributed to a change in the virus and susceptibility built up by years of non-exposure to the virus, which last caused an epidemic in the United States in 1988-1989. Except for South-East Asia, where it has consistently circulated, the viral strain has been absent from much of the world since the early 1990s.

Influenza B viruses generally cause less severe outbreaks of respiratory infection in man. The influenza B viruses appear to go through cycles of prevalence. In contrast to influenza A viruses, which have natural reservoirs in pigs, horses, and birds, the influenza B viruses are mainly isolated from humans.

Shigella sonnei — *Canada*

A week after the first recall of a potentially contaminated Greek pasta salad was issued across Ontario, about 500 people had fallen ill with shigellosis — 114 in Toronto alone. The current outbreak occurred after people ate Greek pasta salad bought at stores. The manufacturer issued a recall of its salad on 18 May 2002, after federal food inspectors told the company that people had complained of stomach cramping, bloody diarrhoea, headaches, and other symptoms of shigellosis. A day later, more than 40 people had fallen ill. By 27 May that number had risen to 504 ill people.

The manufacturer has been making the salad for years, shipping it to hundreds of supermarkets and stores across Ontario and eastern Canada. The salad will not be shipped out again until investigators figure out what could have caused contamination. The company continues to produce hundreds of other products, including various salads, chicken dishes, and desserts.

Poland reports first case of BSE

After a massive 2-day search, on 5 May 2002, Polish officials located the place where the country's first case of mad cow disease may have originated, but are still unsure how the animal got infected. Inadequate documentation about the 9-year-old cow's history forced police and veterinary services to conduct a village-by-village search before tracing its origin to the tiny farming community of Swiebodzin.

No other diseased animal had been found so far but 3 other cows in the same abattoir had also been destroyed as a precaution. The labour-intensive search illustrated the problems caused by Poland's antiquated agricultural infrastructure. Poland has delayed implementing the European Union (EU) livestock identification norms due to logistical problems and poor planning.

Veterinary authorities of the Ukraine and Estonia have banned beef imports from Poland after the first bovine spongiform encephalitis (BSE) case was detected in Poland on 4 May 2002.

Israel reports first case of BSE

Source: Reuters News Agency via The Globe and Mail Website, 4 June 2002 (edited)

On 4 June 2002 Israel confirmed its first case of mad cow disease, found in a cow in a kibbutz in the Golan Heights. The Agriculture Ministry said it would launch an emergency plan to examine brains of all cattle over the age of 30 months that are sent to slaughterhouses, before releasing their meat to the market. The Israeli-born animal, bred in Kibbutz Ortal, was 'probably infected by poultry fish meal imported from Europe'. In addition to the plan for examining the brains of slaughtered cattle, the internal organs of slaughtered cattle over the age of one year will be destroyed.

The Israel Dairy and Meat Board opened a phone line on 5 June 2002 to provide information to the public on questions concerning beef consumption in the wake of the confirmation of Israel's first case of mad cow disease.

The 5 cows related to the cow that died last week of BSE had been put down and cremated. Of these, two that were put down were the same age as the infected animal and came from the same herd in the Beit She'an Valley. The cows were put down because of the high likelihood that they had been fed on the same feed as the contaminated animal. The infected cow's 3 offspring were also put down.