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Contents, continued

Editorial: Towards improving influenza surveillance in Australia

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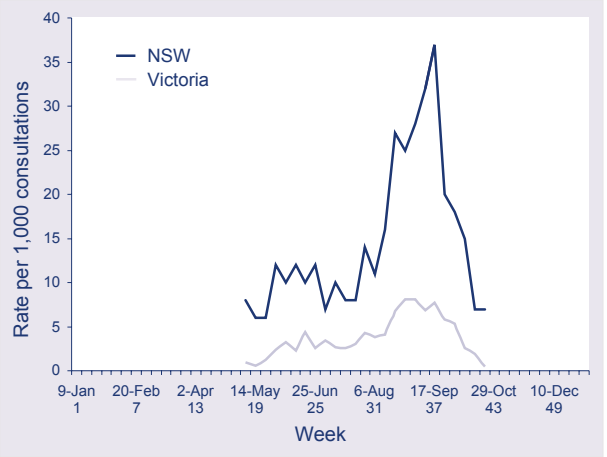
As winter and the influenza season approaches, the article by Watts and Kelly[1] is timely in highlighting significant deficiencies in the surveillance of influenza in Australia. Watts and Kelly conducted a telephone survey of sentinel practice schemes in August 2001 and found that sentinel influenza surveillance schemes vary in their definition of influenza-like illness (ILI) and in their access to laboratory support. The impact of this is illustrated in a comparison between data from New South Wales and Victorian sentinel practice schemes for 2000 (Figure). In Victoria the rates per 1,000 consultations were almost an order of magnitude lower than in New South Wales, although the number of laboratory reports of influenza in the two States during the same period were very similar.[2]

Figure: Rates of influenza-like illness in sentinal practice schemes, NSW and Victoria, 2001



While State-based influenza surveillance does provide timely relevant local information for public health action, national influenza surveillance is needed to provide a broader perspective on circulating viral strains and the impact of the disease on the community. Measuring rates of influenza-like illness in sentinel practices is important to establish the size of the annual epidemic but consistent case definitions need to be used. National collating and reporting on circulating influenza virus strains, based on sampling from a wide geographic area is essential to detect emergence and migration of new viral strains in Australia.

The Influenza Pandemic Planning Committee of the Communicable Diseases Network Australia (CDNA) made recommendations on national influenza surveillance in the report A framework for an Australian influenza pandemic plan (June 1999).[3]

"A national surveillance system should be established using a nationally agreed definition of influenza-like illness (ILI), consistent surveillance methods and national coordination of data collection, analysis and dissemination. The system should comprise community based surveillance of influenza based on sentinel practices during the intra-pandemic period, complemented by institutional surveillance with enhanced measures during a pandemic."

To date there has been no truly national influenza surveillance system but a conglomeration of data from laboratories, sentinel practice schemes and absenteeism data from a major national employer. Data from these systems have been analysed and published in *Communicable Diseases Intelligence* during the winter months since 1994. These data have also been used to produce an influenza surveillance annual report.

While there are obvious needs for improvements, it is important to note recent changes to national influenza surveillance, which will begin to bring surveillance to the standards set in the Pandemic Plan.

The agreement by the CDNA to make laboratory-confirmed influenza a nationally notifiable disease from January 2001, gives Australia a national influenza surveillance system for the first time. There will be a legal obligation to report laboratory-confirmed cases from all Australian medical practices, hospitals and laboratories to the National Notifiable Diseases Surveillance System (NNDSS). Up to now laboratory-confirmed influenza cases have only been reported through the Virology and Serology Reporting Scheme (LabVISE), however, the number of participating laboratories in LabVISE has been declining. From now, all Australian laboratories will be under legal

obligation to notify all influenza diagnoses and the resulting data will be more representative although cases undergoing laboratory testing are often those with more severe illness. CDNA is developing national case definitions and the Public Health Laboratory Network is developing detailed laboratory definitions and testing guidelines, both of which will improve the consistency of data reported to NNDSS.

In addition, NNDSS has been undergoing extensive revisions and from this year a larger set of data will be reported for each notifiable disease. For all cases of influenza it will be possible to record the virus type and strain, the vaccination status of the case and to identify cases linked in an outbreak.

The need for timely and national reporting of influenza data noted by Watts and Kelly is also being addressed. The new NNDSS data acquisition system will allow near real time data transfer from the States and Territories to the Commonwealth. This system provides flexibility to allow rapid revision of data records to include new information. The result should be a national data set for all diseases which is more accurate and up-to-date than ever before.

While the Commonwealth Department of Health and Ageing has been collating reports from sentinel schemes (ASPREN, New South Wales, Victoria, Northern Territory and Western Australia) and LabVISE and publishing these in *CDI* and on the Web, the regularity of reports changed with changes to publication of *CDI*. Since June 2001 these reports have included data from the NNDSS and were published on the Communicable Diseases Australia Website at: http://www.health.gov.au/pubhlth/cdi/ozflu/flucurr.htm; weekly during the winter months and fortnightly during the non-influenza season. These postings will continue throughout the year to monitor influenza activity in the tropical regions of Australia as well as baseline levels of influenza activity during non-epidemic periods in temperate regions. However, these influenza data are as limited in representativeness, comparability and timeliness as the systems from which the information is drawn and the data must be interpreted with care. A commentary on the data and comparison of current year's data with the preceding year go some way to providing meaningful interpretations of emerging trends.

The WHO Collaborating Centre for Reference and Research on Influenza is publishing reports on circulating influenza strains in Australia and outbreaks in the region on its Website http://www.influenzacentre.org/. This provides essential, timely information on changes in the frequency of influenza strains circulating in Australia.

However, there is still room for much improvement to influenza surveillance in Australia. Areas for further work include harmonising surveillance methods used and improving the representativeness of the sentinel schemes; improving surveillance of influenza vaccination and utilising other surveillance such as morbidity and mortality data.

The differences in case definitions of influenza-like illness (ILI), surveillance practices and reporting formats between different sentinel practice schemes need to be resolved. The clinical signs and symptoms of influenza may vary between different age groups. Infants and children may present with symptoms that are indistinguishable from that caused by other respiratory diseases and influenza may cause non-respiratory symptoms.[4] There is a need for laboratory support of influenza sentinel surveillance systems to allow an estimate of the proportion of influenza-like illness that are actually caused by influenza, which varied in one study in Victoria from 49 to 54 per cent.[5] There is a need for CDNA to develop a consensus clinical case definition of influenza-like illness which is simple but specific and an agreement between the sentinel practice schemes to use this case definition in their surveillance.

Sentinel practice surveillance schemes find it difficult to maintain a consistent number of practices reporting to their schemes. In 2000, the number of practices reporting influenza-like illness to ASPREN varied from 52 to 77, from 8 to 41 in the New South Wales scheme, from 25 to 47 in the Victorian scheme and from 9 to 14 in the Northern Territory scheme.[2] Improvements in reporting may require offering inducements to participating practices. A recent report from Hawaii has shown that offering rapid testing kits for influenza to physicians ordering viral cultures resulted in an increase in samples sent for culture from 396 to 2,169 in consecutive influenza seasons.[6] Clearly physicians found the availability of rapid influenza diagnostic tests in the consultation room useful, and the feasibility of influenza diagnostic tests in Australian practices should be investigated. Finally, the representativeness of sentinel schemes in Australia needs to be improved by recruitment of practices in rural and regional towns and in areas outside the south east of the country.

As influenza vaccination becomes more widespread, there is a need to incorporate accurate measures of vaccine coverage into our

surveillance systems. A recent study shows that 74 per cent of over 65-year-olds in Australia were vaccinated against influenza in 2000.[7] The impact of influenza vaccination on the size and severity of the influenza season in Australia should be assessed. As noted above, vaccination data on all influenza cases can now be recorded in the NNDSS. Better data collection will enable more informative modelling of cases prevented by vaccination and other interventions.

There is also a need to have a timely access to hospitalisation and mortality data to measure the annual impact of influenza epidemics. The United States has used measurements of excess mortality due to influenza to develop a severity index,[8,9] which can be used to measure the impact of annual influenza epidemics on hospitalisation.[10] In a recent study of excess winter mortality in the United Kingdom, the proportion due to influenza was observed to be falling in recent years, probably due to increasing vaccination and decreasing variation in the circulating virus.[11] Australian hospitalisation and mortality data, if available in a timely manner, for example from sentinel hospitals, would be useful to give warning of severe epidemics due to major antigenic shifts in the influenza virus and to measure the disease burden due to influenza.

The latest draft (March 2001) of the Australian Action Plan for Pandemic Influenza[12] comments on surveillance.

"An effective national surveillance system is an essential component of a program for the control of influenza to ensure the provision of timely information to public health departments, health care providers and the general public about levels of influenza activity and circulating strains."

Highlighting deficiencies in sentinel systems and working toward consistent national reporting of influenza through the NNDSS are important steps toward achieving an effective influenza surveillance system in Australia.

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Fragmentation of influenza surveillance in Australia

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Abstract

Monitoring of community influenza through sentinel practice networks is essential to track the onset and progress of epidemics. In 1999, the Influenza Pandemic Planning Committee of the Communicable Diseases Network Australia New Zealand (CDNANZ) recommended that a national surveillance system be established comprising both community-based and institutional surveillance. In 2001, however, influenza surveillance remains fragmented in Australia and mainly restricted to major cities. Methods of surveillance and reporting of influenza activity vary between States and even within States. Three disparate case definitions are in use for reporting influenza-like illnesses. Many sentinel sites do not have laboratory support for confirmation of influenza or identification of circulating strains. Dissemination of information is uncoordinated and without a standardised reporting format for collation at a national level. Prompt attention to these issues is important to ensure an adequate public health response to future influenza virus epidemics or a pandemic. *Commun Dis Intell* 2002;26:8-12.

Keywords: influenza, epidemic and pandemic preparedness, surveillance

Introduction

In 1999, the Influenza Pandemic Planning Committee recommended that virological and clinical influenza surveillance programs should be conducted using sentinel general practitioner sites.[1] Virological surveillance facilitates the collection and identification of influenza strains circulating within the population, and has the potential to detect genetic shifts and drifts in influenza virus that may signify the emergence of novel strains. In some circumstances virological surveillance may alert clinicians and public health officials to the circulation of non-influenza viruses that may be contributing to significant mortality within the community. Clinical surveillance enables an estimation of the impact of influenza on the community and provides information against which to evaluate public health policies, such as the provision of free influenza vaccine to people aged 65 years and over. If timely, clinical surveillance also allows feedback to clinicians about the likelihood of the presence of influenza in the community.

State-based sentinel practice influenza surveillance programs operate in New South Wales, Victoria, Western Australia, South Australia and the Northern Territory and obtain supplementary surveillance data provided by regional and hospital-based laboratories. In addition, influenza virus activity is monitored through the Australian Sentinel Practice Research Network (ASPREN), an Australia-wide general practice sentinel reporting system. ASPREN general practitioners (GPs) recorded the attendances of patients for 14 conditions during 2001, two of which were 'influenza' and 'influenza with culture'. The aim of ASPREN is to provide information on the burden of disease in primary health care and to monitor consultation rates.[2] An independent national sentinel surveillance program is funded by Roche pharmaceuticals and is based in New South Wales.

As part of the Virology and Serology Laboratory Reporting Scheme (LabVISE), sentinel laboratories in Australia report laboratory identification of viruses and other organisms to the Commonwealth Department of Health and Ageing, on a monthly basis.[2]

The Influenza Pandemic Planning Committee was established by the Communicable Diseases Network Australia New Zealand (CDNANZ) to develop a contingency plan for pandemic influenza in Australia and in 1999 made a number of recommendations regarding influenza surveillance.[1] The committee recommended that:

"A national surveillance system should be established using a nationally agreed definition of influenza-like illness (ILI), consistent surveillance methods and national coordination of data collection, analysis and dissemination. The system should comprise community-based surveillance of influenza based on sentinel practices during the interpandemic period, complemented by institutional surveillance, with enhanced measures during a pandemic".

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Other recommendations included:

- Sentinel sites (health care providers) be at a ratio of 1 per 200,000 in metropolitan areas and 1 per 50-100,000 in rural areas.
- Information should be gathered by sentinel sites on the number of ILI seen and include age, gender, locality and vaccination status.
- The first patient seen with an ILI on a Monday, Tuesday or Wednesday should have a nose and throat swab collected for detection of influenza virus.
- Sentinel nursing homes and other institutions or closed communities should be included in surveillance.
- Year round monitoring should consist of virus detection in children and routine detection of influenza virus in laboratories.
- Data should be accumulated weekly and forwarded to a national centre on a weekly basis. State centres should provide fortnightly feedback to sentinel sites.

We undertook a survey of all Australian States and Territories to determine the extent of sentinel influenza surveillance in each jurisdiction and to compare current surveillance practices with the recommendations of the Influenza Pandemic Planning Committee 2 years after the recommendations had been made.

Methods

In August 2001, a telephone survey was conducted with co-ordinators of sentinel practice State-based schemes in New South Wales, Western Australia and the Northern Territory. In Queensland and Tasmania representatives were identified through contact with respective State health departments. The Department of Human Services in South Australia was contacted in February 2002. Co-ordinators of programs with multi-State sentinel sites, ASPREN and the Roche National program, were also contacted. Agencies were asked to provide information on influenza surveillance, the number of general practitioners involved in surveillance, the number and location of surveillance sites, the frequency of reporting and surveillance issues that were perceived as important to the responders. Results of the survey were summarised in tabular form and distributed to each agency that had provided information so that a representative from the agency could check the accuracy of the summary. Permission was obtained from each agency to publish summary information.

Results

Table 1 summarises the current sentinel surveillance programs throughout Australia. State-based sentinel influenza surveillance programs operate in New South Wales, Victoria, Western Australia, South Australia and the Northern Territory. There is no state-specific monitoring of influenza activity in Queensland, or Tasmania. The New South Wales, Victorian, Western Australian and the Roche National scheme operate from May until September inclusive. In South Australia, the Northern Territory, and in ASPREN practices, influenza activity is monitored year round. There is some overlap between schemes, as some ASPREN practitioners may report to both ASPREN and state-based surveillance schemes.

Influenza surveillance is not representative of the population by region because a significant majority of sentinel sites in state-based, ASPREN and the Roche programs are in metropolitan locations. There are 120 registered ASPREN sites; most sites are located in the major cities of Sydney, Melbourne, Adelaide and Brisbane with a few sites in Perth and Hobart and some rural areas. The Roche national program has similar coverage to ASPREN, excluding the rural sites and including Newcastle. Data from ASPREN sites are collated to produce a national figure with no attempt to analyse the data by region. Only in Victoria is surveillance approaching the site ratio per head of population recommended by the Influenza Pandemic Planning Committee. In Queensland, follow-up of persons with laboratory-confirmed influenza commenced in August 2001 and a protocol for follow-up of laboratory-confirmed cases is being developed in Tasmania.

The variation in surveillance schemes highlighted above has resulted in methodological differences, both within and between States, for the collation and dissemination of data. For example, only the first visit for an episode of ILI is recorded in some States, while both the first and subsequent visits are recorded in other States. This may result in distortions in ILI consultation data between regions. Consultation data is split into metropolitan and rural regions in some States but not others. Reporting varies from weekly to monthly intervals. While laboratories in all States and Territories have access to facilities for detection of respiratory viruses, differences occur in the laboratory support and range of diagnostic tests offered to GPs participating in sentinel surveillance programs (Table 1). Many states conduct laboratory surveillance independent of sentinel surveillance.

Table 2 demonstrates the variations in case definitions for reporting ILI currently in use. The ASPREN case definition is used by GPs in the New South Wales, Northern Territory and South Australian surveillance programs and GPs throughout Australia who are registered with, and collect data, for ASPREN. Following an analysis of Western Australian and Victorian data which evaluated the predictive value of various symptoms of ILI for laboratory-confirmed influenza,[3] a common case definition was adopted for use in Western Australian and Victoria. GPs in the Roche National scheme use yet another case definition for recording ILI consultations.

Discussion

Surveillance can be an effective tool in assessing influenza activity, indicating the early detection of epidemics and identifying circulating strains. In Australia, as elsewhere, the lack of standardisation of information and use of various case definitions make assessment of severity and the comparison of influenza data between States over time problematic.[4,5] Ascertaining which signs and symptoms are most predictive of influenza has been the subject of a number of recent studies.[3,6-10] Fever and cough have been found to be predictors of influenza in 3 studies,[6-8] another study identified fever, cough and acute onset,[9] and another fever, cough and fatigue.[3] One study found that clinical signs varied with the virus subtype.[10] Agreement on a simple and reliable case definition for ILI would provide a uniform format for data collation and might increase the accuracy of the clinical diagnosis of influenza.

Year round reporting of laboratory-confirmed cases through the sentinel laboratory (LabVISE) program provides further data on circulating viruses. Although reports from laboratories are submitted monthly for collation, problems have been identified with LabVISE data because of delays in specimen collection and reporting, variations in numbers of laboratories reporting and changing diagnostic tests.[4,11]

Participation of GPs is paramount to the success of sentinel surveillance. Community-based practitioners are well positioned to observe increasing consultations for ILI due to rising levels of respiratory pathogens.[5] This surveillance can serve as an early warning system of influenza epidemics if increases in presentations of ILI are monitored and circulating viruses identified.[12-14] Laboratory support should therefore be accessible to all sentinel sites so that influenza can be distinguished from other circulating viruses.

If Australia is to respond to an influenza epidemic or pandemic, all elements of surveillance must be operational during interim periods.[1] In the 2 years since the publication of the Australian Influenza Pandemic Plan, many of the recommendations for pandemic preparedness appear incomplete at both a state and national level. A framework for pandemic preparedness cannot exist without an agreed definition on ILI, laboratory support for surveillance sites and surveillance methods that provide for timely analysis and dissemination of data.

Acknowledgements

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Table 1. Sentinel influenza surveillance programs in Australia

State	Organisation responsible	Number of general practitioners	Number of metropolitan sites	Number of rural sites	Record influenza-	Virological surveillane with like illness support[1]	Collation of data laboratory	Reporting
NSW State program (1)	Communicable Diseases Surveillance and Control Unit, NSW Health	32	4	5	No	Yes PCR, serology, IF	Weekly	Weekly
NSW State program (2)	Communicable Diseases Surveillance and Control Unit, NSW Health	37-47[2]	1 public health unit	2 public health units	Yes	No	Weekly	Weekly
Victoria State program	Victorian Infectious Diseases Reference Laboratory	70	21	20	Yes	Yes PCR (respiratory multiplex)	Weekly	Fortnightly
WA State program	PathCentre	8-12	6-8	2-4	Yes	Yes PCR, rapid culture, cell culture	Weekly	Weekly
SA State program	Department of Human Services	Not stated			Yes	No	Weekly	Weekly
NT State program	Centre for Disease Control, Territory Health Services	17	11	2	Yes	Yes throat gargle(WHO)[3] rapid kit	Monthly	Monthly
ASPREN[4] national program	Research & Health Promotion Unit, RACGP[5]	55	Not available[6]	-	Yes	No (may request)	Weekly	Fortnightly
Roche national program	Centre for Virus Research, Westmead Hospital	65	2-10 per major city[7]	-	Yes	No rapid kit	Weekly	Weekly

1. Laboratory support includes testing of the presence of virus by polymerase chain reaction (PCR) or antigen detection including immunofluorescence (IF), and/or testing for specific antibodies.
2. 17 GPs also report to ASPREN
3. Throat gargles sent to World Health Organization Collaborating Centre for Influenza Reference and Research, Parkville, Victoria for analysis.
4. ASPREN Australian Sentinel Practice Research Network
5. RACGP Royal Australian College of General Practitioners
6. Predominantly Melbourne, Sydney, Adelaide and Brisbane, but registered GPs in Perth, Hobart and some rural locations also contribute data. On average 55 reports from 120 registered sites are received each week.
7. Sites in Sydney, Melbourne, Adelaide, Brisbane, Perth, Hobart and Newcastle.

Table 2. Case definition for influenza like illness (ILI) used in different States

State	Fever, cough, fatigue	ASPREN	Sudden onset fever or chills myalgia +/- headache +/- dry cough +/- fatigue
NSW State program (2)		✓	
NSW sites of ASPREN national program		✓	
NSW sites of Roche national program			✓
Victorian State program	✓		
Victoria sites of ASPREN national program		✓	
Victoria sites of Roche national program			✓
SA State program		✓	
SA sites of ASPREN national program		✓	
SA sites of Roche national program			✓
WA State program	✓		
WA sites of ASPREN national program		✓	
WA sites of Roche national program			✓
NT surveillance program		✓	
Queensland sites of ASPREN national program		✓	
Queensland sites of Roche national program			✓
Tasmania sites of ASPREN national program		✓	
Tasmania sites of Roche national program			✓

ASPREN Australian Sentinel Practice Research Network

ASPREN criteria: sudden onset (<12 hours), cough, rigors/ chills, fever, prostration and weakness, myalgia, redness of mucous membranes, influenza in close contacts

ASPREN for ILI during epidemic: 4 criteria, outside epidemic: 6 criteria

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Correction

Invasive meningococcal disease and HIV coinfection

There was an omission in the article Couldwell D. Invasive meningococcal disease and HIV coinfection *Commun Dis Intell* 2001;25:279-280. A reference was inadvertently omitted from end of the second paragraph of the Introduction. The Introduction is reprinted below with the missing reference.

Introduction

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

Numerous encapsulated bacteria cause sepsis at increased rates in HIV-infected individuals; higher rates of mortality also occur.[2] The commonly involved pathogens vary with geographic location as well as patient risk factors. Although there have been a number of reports of meningococcal disease in HIV-infected patients,[3,4,5] an increased risk in HIV-infected people has not been demonstrated.[6,7] However, a population-based study of sporadic meningococcal disease from Atlanta in the United States identified immune compromise due to conditions including HIV-infection in two-thirds of affected adults over 24 years of age.[8]

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Outcomes from the first two years of the Australian hepatitis C surveillance strategy

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Abstract

The objectives of national hepatitis C surveillance are to identify those at risk in order to appropriately target prevention and care programs, and to evaluate the impact of these approaches. In 1998 the Communicable Diseases Network Australia New Zealand (CDNANZ) appointed the Hepatitis C Surveillance Committee to develop and implement approaches for improved hepatitis C surveillance in Australia. The Australian Hepatitis C Surveillance Strategy was endorsed in 1999 and provides a framework for improvements to national hepatitis C surveillance. The strategy covers two main surveillance activities: surveillance of incident and prevalent hepatitis C, and the long-term outcomes of hepatitis C. The committee (now the CDNA Viral Hepatitis Surveillance Committee) has continued to facilitate the implementation of the recommendations proposed. Progress towards improvement of hepatitis C surveillance in Australia includes the development of standard case reporting for hepatitis C, collation of data on incident and prevalent hepatitis C from a range of populations at lower and higher risk of hepatitis C, and collation of data from liver transplant registries. Advances in the implementation of the strategy are incremental. While there is enthusiastic commitment towards improving hepatitis C surveillance in Australia, the number of cases, the capacity and competing priorities of State and Territory health departments has meant that implementation has been challenging, highlighting the difficulties in introducing new systems into an already complex situation. *Commun Dis Intell* 2002;26:14-22.

Keywords: hepatitis C, surveillance

Introduction

Since 1989, when infection with the hepatitis C virus was identified as the main cause of non-A non-B viral hepatitis in most of the industrialised world, it has become the most frequently notified communicable disease in Australia. In 2000, just over 20,000 cases of hepatitis C infection were reported across the 8 public health jurisdictions. Surveys have found that the highest prevalence in Australia occurs in people with a history of injecting illicit drugs. Other groups with higher levels of hepatitis C are people with haemophilia, prisoners and people from countries with a high prevalence of hepatitis C.

In recognition of the public health importance of hepatitis C infection, the Communicable Diseases Network Australia (CDNA), formally CDNANZ, established the Hepatitis C Surveillance Committee in 1998. The committee was given the responsibility of improving the national capacity to monitor the occurrence of the infection and its consequences, through the development and implementation of a national surveillance strategy. This review reports on the outcome of this process to date.

Challenges presented by surveillance for hepatitis C

There are a number of aspects of hepatitis C infection that have presented challenges to surveillance activities. First, detection of incident cases of infection is difficult because less than 10 per cent of people who are exposed to the virus develop symptoms of acute hepatitis, and an even smaller proportion seek medical advice. New infection can also be detected serologically, but requires serial testing of individuals within a limited time period, to determine that antibodies have developed. Second, because hepatitis C infection in Australia is strongly associated with the illegal and socially stigmatised practice of injecting drug use, it is difficult to undertake monitoring of a large group of people who are at risk of infection. Finally, the long (over decades) and variable time course of chronic infection complicates the assessment of outcomes such as liver failure and hepatocellular carcinoma (HCC).

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Development of the National Hepatitis C Surveillance Strategy

The Hepatitis C Surveillance Committee was established under the chairmanship of Dr Linda Selvey with a broad membership, including a representative with experience in hepatitis C surveillance from each State and Territory, and representatives from the Australian Hepatitis Council, the Australian National Council on AIDS, Hepatitis C and Related Diseases Hepatitis C Committee, the Macfarlane Burnet Centre for Medical Research, the National Centre in HIV Epidemiology and Clinical Research (NCHECR), the Public Health Laboratory Network, the Commonwealth Department of Health and Ageing and the NSW Users and AIDS Association.

The terms of reference agreed for the committee were to:-

1. Develop a strategy for national hepatitis C surveillance in Australia, to incorporate routine case reporting of hepatitis C diagnoses as well as other methods of data collection.
2. Oversee the implementation of the strategy at a national level.
3. Consider mechanisms for integrating hepatitis C surveillance activities with the corresponding activities for HIV.

Following a series of meetings, the Australian Hepatitis C Surveillance Strategy was drafted by the committee and endorsed in 1999 by the CDNA. Since then the committee has continued to facilitate the implementation of the recommendations proposed in the Strategy. In 2001, its terms of reference were extended to include the development of national surveillance for hepatitis B infection, and the name was changed to CDNA Viral Hepatitis Surveillance Committee.

Specific elements of the strategy for the surveillance of hepatitis C are summarised in Table 1. In implementing the strategy, the committee recognised that finite resources and differing surveillance structures within jurisdictions would mean that not all of the agreed activities could be introduced at once, and that an incremental approach would be required. In the remainder of this review, we outline the developments that have taken place in key areas of the strategy.

Routine reporting of incident hepatitis C via NNDSS

In reviewing existing procedures in the course of developing the surveillance strategy, the committee identified the lack of standard case definitions across jurisdictions, and the absence of information on risk factors for hepatitis C as key weaknesses in the national surveillance system. The jurisdictions recognised that they would be likely to continue to use somewhat different procedures for identifying and reporting on incident and prevalent cases of hepatitis C infection, but they endorsed standard case definitions, and a set of risk categories that would be used to classify exposure for all cases determined to be incident (Table 2). Currently the case definitions are awaiting CDNA approval. The enhanced data collection for hepatitis C infection was incorporated in broader changes that were taking place in the reporting procedures of the National Notifiable Diseases Surveillance System.

Incident hepatitis C cases have been separately reported by all jurisdictions (except Queensland and Northern Territory) since 1997 (Table 3). The numbers of incident cases detected are likely to be affected by the mechanisms for detecting cases in these years (Table 4). In the largest jurisdictions, classification of incident cases is determined by passive reporting. In smaller jurisdictions, where all (or the majority) of hepatitis C notifications were actively investigated to determine if they were incident or prevalent during this time period, a much higher proportion of cases has been determined to have been incident. Changes in surveillance practices within jurisdictions (e.g. the introduction of enhanced surveillance in Western Australia and in some New South Wales Public Health Units) has contributed to an increased number of incident cases reported at various times between 1997 and 2000.

In 2001, the Australian Capital Territory, the Northern Territory, South Australia and Tasmania undertook enhanced surveillance on all HCV notifications. NSW Health had developed a procedure for enhanced hepatitis C surveillance which was implemented via the Public Health Units. Victoria chose to undertake enhanced surveillance on 10 per cent of all new notifications of hepatitis C infection. Western Australia developed new procedures to coincide with the introduction of the revised national system for reporting notifiable communicable diseases, including a review of 30 per cent of all notifications for the assessment of risk factors. All jurisdictions found that there was a considerable amount of extra work involved in processing cases under the strategy, both in order to identify incident cases and to collect risk factor information.

Table 1. Recommendations from the Hepatitis C Surveillance Strategy

Focus of surveillance activities			Recommendation	Priority
1. Surveillance for HCV transmission	1.1 Incident HCV infections	1.1.1 Routine reports	Common data collection Enhanced surveillance and NIHCR	High
		1.1.2 Sentinel populations	Set up sites based on CDC model	High
		1.1.3 Serially tested populations	Collate data from • People who inject drugs • Blood donors	High
	1.2 Prevalent HCV infections	1.2.1 Routine reports	Common data collection	High
		1.2.2 Populations at higher risk	Collate data from • Needle-syringe program • Methadone clinics • Prison entrants	High
		1.2.3 Populations at lower risk	Collate data from • Blood donors • Pregnant women/newborns • ADF entrants • Primary care and STI clinics	High
2. Surveillance for the long termoutcomes of chronic HCV infection	2.1 Existing data sources	2.1.1 Cancer registries	Collate data from HCV positive registrants	Moderate
		2.1.2 National Liver Transplant Register	Collate data from HCV positive registrants	Moderate
		2.1.3 Hospital discharge data	Collate data from HCV positive admissions	Moderate
	2.2 Clinical networks		Build collaborative networks	Moderate
	2.3 Incident case register		Develop a national register	High
	2.4 Morbidity and mortality in research cohorts of people with advanced liver disease		Build collaborative networks with researchers who study advanced liver disease	Moderate-low
	2.5. HCV related ABS mortality data		Collate data from HCV related deaths	Moderate

Table 2. Risk categories for incident HCV infections

Associated with injecting drug use	Other risk factors
Injecting drug use only in the previous two years	Blood/blood products/tissues in Australia
Injecting drug use more than 2 years ago	Blood/blood products/tissues overseas
Injecting drug use unknown	Haemodialysis
Never injected drugs	Needlestick/biohazardous injury in healthcare worker
	Needlestick/biohazardous injury in non-healthcare worker
	Surgical work
	Major dental surgery
	Tattoos
	Acupuncture
	Ear or body piercing
	Perinatal transmission
	Sexual partner with HCV
	Imprisonment
	Health care worker with no documented exposure
	Household contact with HCV
	Non IDU remote risk (non IDU associated risk identified, but not in one/two years prior to diagnosis)
	Other risk (specify in other risk details)
	Risk unable to be determined
	Unknown (not recorded)

Table 3. Number of incident hepatitis C notifications reported to NNDSS in Australia, 1997-2000*

State or Territory of diagnosis	1997	1998	1999	2000
ACT	3	8	20	20
NSW	19	110	100	139
SA	48	67	80	89
Tas	2	18	18	31
Vic	9	21	70	87
WA	73	126	108	75
Total	**154**	**350**	**396**	**441**

* Analysis by onset date

Table 4. Summary of State and Territory HCV notification systems

State or territory	Source of HCV notifications	Passive or enhanced surveillance for incident cases
ACT	Doctor, laboratory, hospital	Enhanced
NSW	Laboratory, doctor notification possible, but rare	Passive
NT	Doctor, laboratory	Passive
Qld	Doctor, laboratory, hospital	No surveillance of incident cases
SA	Doctor, laboratory	Enhanced
Tas	Doctor, laboratory	Enhanced
Vic	Doctor, laboratory	Passive
WA	Doctor*	Enhanced system operational between 1995-1999

* No public health legislation requiring laboratories to report HCV notifications. Informal agreement with largest pathology laboratory to notify cases operational in recent years.

Assessment of HCV incidence via other surveillance mechanisms

The Commonwealth Department of Health and Ageing provides funding to NCHECR for HCV surveillance activities. A number of agencies responsible for HCV testing provide regular tabulations of testing results to the NCHECR. Methods and results are in turn made publicly available through the NCHECR Annual surveillance report.[1]

Incidence in serially tested populations

The surveillance strategy recognised that there are several population groups in Australia that undergo repeat or regular testing for hepatitis C infection and could be used to further monitor HCV transmission in Australia, particularly among people at higher risk. Possible sites for monitoring of this kind include primary care facilities that provide services to people who inject drugs, prison medical services, and blood transfusion services. So far, systematic information on repeat testing has been available only from the Kirketon Road Centre, a primary care clinical service in central Sydney (Table 5). The hepatitis C incidence rate among injecting drug users attending the Kirketon Road Centre between 1996 and 2000 varied between 12 and 21 per 100 person years, with higher rates among the younger age group (less than 20 years).[1] Monitoring of hepatitis C incidence among people at higher risk will assist in the development and evaluation of prevention strategies and identification of factors within these groups that are associated with an increased risk of infection.

Incidence in other populations

Detailed scrutiny of incident cases detected through blood donor screening or other testing in people at lower risk could lead to insight into the sources of HCV transmission through modes other than injecting drug use in Australia. Although the number of such cases is likely to be very small, they may have significant implications for public health practice. In particular, any cases that may be healthcare associated will require a very thorough investigation of possible breaches in infection control that may have occurred.

Table 5. Hepatitis C incidence among clients of Kirketon Road Centre, Sydney

Age	1996		1997		1998		1999		2000	
Under 20	5	(31)[1]	6	(42)	8	(74)	4	(67)	0	(0)
20-29	8	(11)	11	(19)	10	(21)	6	(20)	4	(27)
Over 30	2	(6)	3	(7)	2	(5)	2	(6)	2	(11)
Total	**15**	**(12)**	**20**	**(18)**	**20**	**(21)**	**12**	**(17)**	**6**	**(17)**

1. Numbers in brackets represent incidence per 100 person years

Monitoring of hepatitis C prevalence

Prevalence is a less effective indicator of HCV transmission patterns than incidence, but it has considerable value as an indicator of the extent of infection in the population, the current burden to the health care system and the levels of risk in different populations.

Prevalence data are available from a number of specific lower risk populations that are routinely tested for hepatitis C. Mandatory screening takes place for all blood donors through the Australian Red Cross Blood Service (Table 6) and Australian Defence Force (ADF) entrants (Table 7). A survey conducted by the NCHECR in 2001 found that many antenatal clinics in Australia routinely offer hepatitis C testing to pregnant women,[2] but it is difficult to derive prevalence information from these clinics in a comprehensive way. Development of a national network of antenatal clinics for hepatitis C (and hepatitis B) surveillance will commence in 2002.

Sexual health clinic attenders represent another population that is routinely offered hepatitis C testing, often as a result of self reported risk behaviours such as injecting drug use. A national network of sexual health clinics currently provide information on HIV testing,[1] and could provide additional data on the extent and outcomes of hepatitis C testing. Development of a national network of sexual health clinics for hepatitis C is a goal for 2002.

Monitoring of hepatitis C prevalence among people who inject drugs has been undertaken through surveys of attenders at needle and syringe programs that have been undertaken annually at a number of sites in Australia over a one week period since 1995.[3,4] The survey includes a questionnaire to ascertain demographic and behavioural information, and collects finger prick blood samples for analysis of anti-HCV antibody (Table 8). Monitoring of hepatitis C prevalence in survey participants who have recently commenced injecting (e.g. less than 3 years duration) provides a measure of recent HCV transmission levels among injecting drug users in Australia (Table 9).

Table 6. Hepatitis C prevalence among blood donors,* by State or Territory, 2000

State or Territory	Number screened for HCV antibody	Number positive for HCV antibody	Prevalence per 100 000 donations
ACT and NSW	307,690	40	13
NT	8,715	6	69
Qld	195,940	41	21
SA	87,828	7	8
Tas	-	-	-
Vic	258,014	39	15
WA	99,718	19	19
Total	**955,984**	**152**	**16**

* First time or repeat donors

Table 7. Hepatitis C prevalence among Australian Defence Force entrants, 1997 to 2000

	Jun to Dec 1997	Jan to Dec 1998	Jan to Dec 1999	Apr[1] to Mar 2001	Total
Number of entrants tested	1,676	3,352	4,379	4,384	13,791
Number positive for HCV antibody	1	2	9	4	16
HCV prevalence per 100 000 entrants	60	60	205	91	116

1 Data not available for first quarter of 2000

Table 8. Hepatitis C prevalence data from the 2000 Needle and Syringe Survey, according to sex and State or Territory of participating needle and syringe program

State or Territory	Percentage of anti-HCV antibody positive (number tested)		
	Males	Females	Total
ACT	54 (120)	64 (42)	57 (162)
NSW	65 (535)	69 (325)	66 (865)
NT	46 (70)	32 (19)	42 (90)
Qld	37 (464)	44 (249)	39 (719)
SA	48 (200)	46 (92)	47 (294)
Tas	53 (17)	13 (8)	40 (25)
Vic	64 (177)	59 (115)	62 (293)
WA	46 (56)	26 (19)	41 (75)
Total	**52 (1,639)**	**55 (869)**	**53 (2,523)**

Table 9. Hepatitis C prevalence data in injecting drug users reporting less than three years injecting from the Needle and Syringe Survey, 1995-2000

	Percentage of anti-HCV antibody positive (number tested)		
	Males	Females	Total
1995	18 (77)	28 (53)	22 (131)
1996	11 (162)	16 (74)	13 (238)
1997	12 (193)	16 (126)	13 (320)
1998	15 (273)	20 (182)	17 (457)
1999	16 (238)	28 (155)	20 (393)
2000	25 (207)	28 (127)	26 (334)

Surveillance of therapy uptake in people with chronic hepatitis C

Antiviral therapy for chronic hepatitis C has improved markedly in recent years, with combination interferon and ribavirin therapy producing a sustained response (indeed, a probable cure) in approximately 40 per cent of treated patients. Currently people with chronic hepatitis C who have progressed to moderate-severe hepatic fibrosis or cirrhosis are eligible for government funded combination interferon and ribavirin therapy through the Highly Specialised Drugs program. From 2002, information will be collated on the number of people in Australia receiving government funded antiviral therapy for chronic hepatitis C. If the current treatment criteria are maintained, these data may provide some information on trends in people with chronic hepatitis C and progressive liver disease.

Surveillance of the long-term outcomes of chronic hepatitis C

Progress towards the recommended activities in relation to the long term outcomes of hepatitis C include an analysis of liver transplant register data and cancer data. Morbidity and mortality data remain as other possible data sources, which could be used to further examine the long term outcomes of HCV infection. While all of these data sources may have biases, taken together, they do serve to provide some understanding of the long term burden of HCV.

Although the majority of people who acquire HCV infection develop chronic hepatitis C, a minority will progress to advanced complications, including liver failure and hepatocellular carcinoma. Estimates and projections of the hepatitis C epidemic in Australia indicate that the incidence of these disease complications is likely to double over the next decade.[5] In order to monitor long-term outcomes of chronic hepatitis C, data are being collected from the Australian and New Zealand Liver Transplant Registry on the incidence of liver transplantation in Australia and its underlying causes, including hepatitis B and hepatitis C. In 2002, data will also be collected on the number of people with liver failure who are awaiting (as opposed to undergoing) liver transplantation.

Analyses of trends in HCC mortality in Australia have recently been performed, with evidence of increasing mortality, in particular among overseas-born men.[6] Although these trends almost certainly represent increased incidence of long-term complications of chronic viral hepatitis (hepatitis B and C), no information on causation was available from the Australian Institute of Health and Welfare data source used for the analyses. In order to assess the incidence of HCC among people with hepatitis C infection, a proposal is under development to cross-match de-identified hepatitis C notifications with national death and cancer registries. Matching of this kind has previously been undertaken between the HIV/AIDS and cancer registration systems nationally.

In people with chronic hepatitis C who obtain a sustained response to antiviral therapy the risk of advanced liver disease complications is markedly reduced. However, for people with chronic hepatitis C and progressive disease, risk of advanced liver disease complications will be maintained in three distinct population groups: those with undiagnosed hepatitis C; those unable or not willing to access therapy; and those who fail antiviral therapy. A hepatitis C observational database with input from a collaborative network of hospital and primary care sites will be established in 2002. One of the objectives of the observational database will be to monitor progression to advanced liver disease complications among people who fail antiviral therapy.

Discussion

Despite clear commitment towards improving hepatitis C surveillance in Australia, the capacity and competing priorities of State and Territory health departments has inevitably led to lower levels of implementation than had been initially envisaged. Implementation of new policies and procedures to coordinate national hepatitis C surveillance in Australia is dependent on reaching a high level of agreement between the jurisdictions and recognition of the differing capacities, responsibilities and priorities of the States, Territories and the Commonwealth. Progress has highlighted the difficulties in introducing new systems into an already complex situation. The challenge for the Hepatitis C Surveillance Strategy is to recommend improvements to national HCV surveillance that are both feasible and sustainable, and to support the implementation of these recommendations. Consultation and continued communication with all stakeholders is essential for this process.

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OzFoodNet: enhancing foodborne disease surveillance across Australia: Quarterly report, July to September 2001

The OzFoodNet Working Group[1]

Introduction

OzFoodNet is a collaborative network of epidemiologists and microbiologists 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 third quarterly report of OzFoodNet summarises the incidence of foodborne disease in the 6 States of Australia and specific foodborne outbreaks identified between July and September 2001.

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Historical comparisons use notifications by date of onset. All other data are reported using the date the report was received by the health agency.

During the third quarter of 2001, Australia experienced an outbreak of *Salmonella* Stanley. The CDNA requested that OzFoodNet coordinate the national investigation, which identified contaminated peanuts from China as the food vehicle. The investigation also alerted health authorities in Canada and the United Kingdom to human cases of salmonellosis associated with the same brand of peanuts. *Salmonella* Typhimurium phage type 126 also emerged as a national problem during the quarter. In September 2001, OzFoodNet began a national survey to estimate the incidence of diarrhoeal disease, and a pilot of the national case control study into *Campylobacter* infections. During this quarter, the Australian Capital Territory joined OzFoodNet and the Northern Territory participated as an observer. Data are only included for the Territories where specified.

Notifications in the third quarter

During the third quarter 2001, OzFoodNet sites reported 4,014 notifications of campylobacteriosis, which represented a 25 per cent increase over the mean for the same quarter for the years 1998 to 2000.[1] The median age of cases ranged between 27 to 33 years old. All States reported that the male to female ratio of cases ranged from 1.1:1.0 to 1.4:1.0. There was one small outbreak of *Campylobacter* infection in Queensland that was associated with eating duck livers in a restaurant.

OzFoodNet sites reported a total of 1,081 cases of salmonellosis during the third quarter and identified the source of 4 *Salmonella* outbreaks. As for previous reports, Queensland reported a lower median age of reported cases (9.0 years old) compared to other States (range of medians: 17.0–23.1 years old). OzFoodNet sites reported that *Salmonella* Typhimurium (phage types 126, and 135), and S. Stanley were the most commonly notified infections during the quarter.

The major feature of *Salmonella* epidemiology during this quarter was the emergence of *Salmonella* Typhimurium phage type 126 in jurisdictions across Australia. The National Enteric Pathogen Surveillance Scheme reported that *S.* Typhimurium 126 was among the five most common infections in five different jurisdictions (Joan Powling, The University of Melbourne, 14 January 2002, personal communication) (Table 1). The South Australian Department of Human Services conducted a case control study of this serovar, which implicated chicken products. There were also concurrent epidemics of this organism in chicken flocks.

The Tasmanian OzFoodNet site continued to report that the most common serovar was *Salmonella* Mississippi, which is endemic in that State. Queensland reported that the distribution and rates of salmonellosis changed depending on geographical location, with higher rates in the north of the State. Jurisdictions reported an increase in the incidence of *Salmonella* Stanley between July and September, which was related to the national outbreak.

State health departments received 14 notifications of listeriosis during the third quarter of 2001, five of which were from Western Australia. Median ages for cases not associated with pregnancy ranged from 43 to 83 years. Tasmania reported one maternal-foetal infection during the quarter.

OzFoodNet sites reported seven cases of shiga toxin producing *E. coli* infections during the quarter; four were from South Australia and three from Queensland. Investigators did not identify any sources and all cases appeared sporadic. The median age of cases were 22 years in South Australia and 7 years in Queensland. The South Australian Health Department was notified of one case of haemolytic uraemic syndrome in a 21-year-old male on holiday from the United Kingdom.

There were 11 notifications of yersiniosis for the third quarter of 2001 (Figure). The Communicable Diseases Network Australia agreed to remove yersiniosis from the list of nationally notifiable disease, but most jurisdictions still receive reports. The decline in yersiniosis has occurred over several years and follows similar trends in other countries. OzFoodNet sites reported that during the quarter there were 86 cases of shigellosis, and 13 cases of typhoid.

Foodborne disease outbreaks

During the third quarter of 2001, OzFoodNet sites reported 17 outbreaks that were potentially related to food (Table 2). These outbreaks affected approximately 244 people, of whom 7 were hospitalised. There were no reported deaths from these outbreaks. Ten outbreaks were associated with meals served at restaurants, and three with takeaway food or catered functions.

There were three community-wide epidemics occurring during the quarter, two of which crossed State and Territory boundaries. One of these was a small outbreak of cryptosporidiosis associated with unpasteurised pets' milk that was not intended for human consumption.

Table 1. Top five *Salmonella* infections reported to OzFoodNet sites, July to September 2001, by date of receipt of notification at the Health Department

OzFoodNet Site	Top 5 *Salmonella* infections	Number of cases				
		3rd Qtr 2001	3rd Qtr 2000	Year to date 2001	Total 2000	Ratio*
Queensland	S. Typhimurium 135	22	14	99	83	1.6
	S. Virchow 8	22	18	145	153	1.2
	S. Typhimurium 126	16	0	52	2	
	S. Saintpaul	15	23	121	157	0.7
	S. Aberdeen	13	2	69	42	6.5
Hunter	S. Typhimurium 126	4	1	7	3	4.0
	S. Typhimurium 170	2	0	5	1	-
	S. Birkenhead*	1	0	1	9	-
	S. Bovismorbificans 30	1	0	2	0	-
	S. Bredeney	1	0	1	0	-
New South Wales	S. Typhimurium 135	41	20	155	115	2.1
	S. Typhimurium 9	18	8	107	138	2.3
	S. Stanley	14	1	30	8	14.0
	S. Typhimurium 126	14	6	64	56	2.3
	S. Enteritidis 4	12	5	20	19	2.4
South Australia	S. Typhimurium 126	49	1	88	3	49.0
	S. Typhimurium 108	6	2	12	8	3.0
	S. Stanley	5	0	0	6	-
	S. Infantis	4	1	8	12	4.0
	S. Typhimurium 43	4	0	5	0	-
	S. Typhimurium 12A	4	1	11	9	4.0
Tasmania	S. Mississippi	4	0	90	69	-
	S. Typhimurium 9	1	1	10	21	1.0
	S. Typhimurium 135	1	1	2	5	1.0
	S. Enteritidis 4	1	0	3	5	-
	S. Infantis	1	0	1	4	-
Western Australia	S. Typhimurium 135	16	7	0	68	2.3
	S. Chester	10	1	0	12	10.0
	S. Kiambu	10	0	0	9	-
	S. Stanley	10	0	0	5	-
	S. Typhimurium 4	7	0	0	1	-
Victoria	S. Typhimurium 99	99	95	438	539	1.0
	S. Typhimurium 9	16	9	33	35	1.8
	S. Typhimurium 135	15	17	79	109	0.9
	S. Stanley	11	3	11	11	3.7
	S. Typhimurium 104	12	0	19	0	-

* Ratio of cases for the third quarter 2001 to the third quarter 2000.

Table 2. Outbreaks reported by OzFoodNet sites, July to September 2001

State	Month of Outbreak	Setting	Agent responsible	Number exposed	Number affected	Evidence*	Responsible vehicles
Australia	Jul—Sep	Community	*Salmonella* Stanley	Unknown	27	D, M	Imported dried peanuts
ACT	Sep	Conference	Probably Norwalk virus	115	25	D	Suspected salad
Hunter	Jul	Restaurant	Unknown	25	10	D	Suspected honey chicken
	Jul	Fast food outlet	Unknown	Unknown	2	D	Suspected takeaway chicken
Qld	Jul	Restaurant	*Clostridium perfringens*	15	8	S	Beef curry
	Jul	Restaurant	*Clostridium perfringens*	7	7	D	Unknown
	Jul	Restaurant	*Campylobacter*	Unknown	2	D, M	Duck liver
	Aug	Community	Cryptosporidiosis	Unknown	6	S, M	Unpasteurised pets milk (cow)
	Jul	Functions x 2	Norwalk virus	90	56	S	Salads, steak sandwiches
SA	Jun	Household	*S.*Typhimurium 135a	n/a	2	S, M	Homemade italian sausage
Vic	Jul	Hotel restaurant	*S.* Typhimurium 99	91+	19	S	Lamb's fry
	Aug	Restaurant	Butterfish diarrhoea	15	4	D	Escolar
	Aug	Restaurant	*S.* Typhimurium 99	316+	50	S	Eye fillet meal
	Aug	School camp	Unknown (suspect *Campylobacter*)	27	6	D	Suspected unpasteurised milk
	Sep	Restaurant	Unknown	17	7	D	Unknown
WA	Jul	Restaurant	Unknown	11	6	D	Suspected undercooked turkey
	Sep	Restaurant	Unknown	10	7	D	None identified

*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.

The Communicable Disease Network Australia requested that OzFoodNet coordinate the national investigation into an outbreak of *Salmonella* Stanley amongst people of Asian ethnicity. OzFoodNet held national teleconferences of State and Territory investigators to generate hypotheses about the reasons for this national increase. The Victorian Department of Human Services and the Microbiological Diagnostic Unit (MDU) sampled dried peanuts originating from China after 2 cases gave a history of consumption during interviews. MDU identified *Salmonella* Stanley in the peanuts with a molecular pattern that was indistinguishable from patient isolates. The Australia New Zealand Food Authority coordinated a nation-wide recall of the contaminated product. OzFoodNet sites reported 27 cases of salmonellosis associated with these peanuts. The Australian investigation triggered product recalls and outbreak investigations in Canada and the United Kingdom.[1]

The South Australian Department of Human Services continued investigations into a State-wide outbreak of *Salmonella* Typhimurium phage type 126. Since reporting this outbreak in the previous OzFoodNet report other jurisdictions around Australia have identified cases of this emerging infection.[2] South Australian investigators completed a case-control study that showed that illness was associated with consumption of chicken. The Department also identified corroborating evidence for this link, including descriptive epidemiology and microbiological evidence.

This outbreak is one of a number in 2001 that were possibly associated with chicken.[2,3,4] It is concerning that cases of this serovar are now occurring in other Australian States and Territories. It once again raises the difficult question about the role that contaminated chicken plays in the epidemiology of *Salmonella* and *Campylobacter* infections in humans in Australia.

Applied research

In September 2001, the Tasmanian OzFoodNet Site piloted the national *Campylobacter* case control study. This study aims to examine the risk factors for infection with sporadic *Campylobacter* infection. *Campylobacter* is the most common enteric disease reported to health agencies, and is a cause of significant morbidity in Australia. This study will recruit approximately 1,200 cases and 1,200 controls across Australia during the next 12 months. The case control study will use the results of a comparison of 8 *Campylobacter* typing methods that is being coordinated by the OzFoodNet-Hunter Site and Hunter Area Pathology.

During this quarter, the National Centre for Epidemiology and Population Health started the national OzFoodNet gastroenteritis survey. The aim of this cross-sectional survey is to measure the prevalence of gastrointestinal illness across all States and Territories of Australia. Interviewers use Computer Assisted Telephone Interviews (CATI) to ask respondents about demographic details and whether they have experienced an episode of gastrointestinal disease in the last month. If participants mention that they have had an episode of gastroenteritis, interviewers record symptom details and the patients' use of health services. This study includes residents of the Northern Territory where many people living in remote areas would not have telephone. Despite this, in the month of September Northern Territory residents reported the highest crude proportion of people experiencing gastroenteritis in the previous month, and South Australian residents reported the lowest (Table 3).

The population survey covers all States and Territories and will run for a year. It will provide important information about the burden of gastrointestinal disease and will supplement information that States and Territories collect about the causes of foodborne illness. OzFoodNet aims to combine these data to learn more about the causes and burden of foodborne illness in Australia.

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Figure: Notifications of yersiniosis in OzFoodNet sites, 1998 to September 2001, by month of offset.

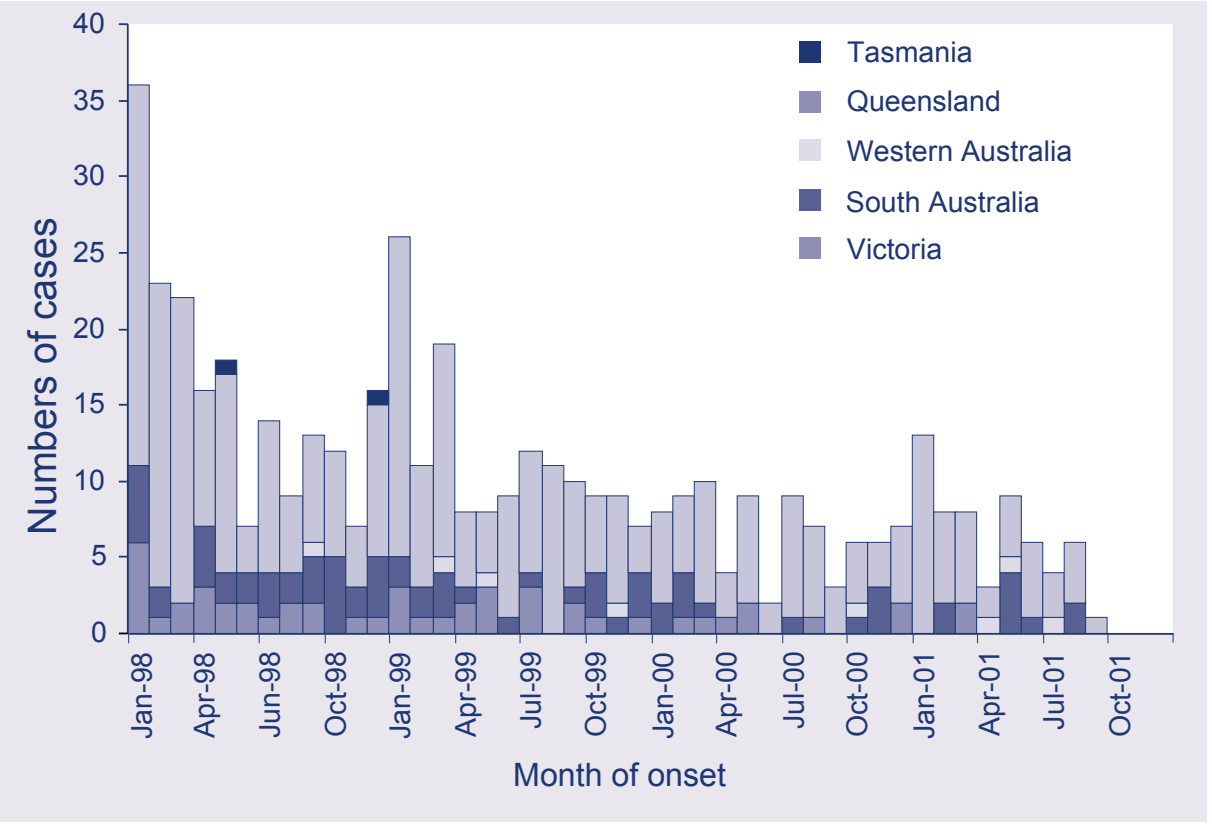

Table 3. Unweighted results of the national OzFoodNet gastroenteritis survey during September 2001 showing the proportion of respondents reporting an episode of gastroenteritis in the previous month, and the response rates by jurisdiction

State or Territory	Number of respondents	Proportion with gastroenteritis (%)	Response rate (%)
New South Wales*	110	9.3	64
Victoria	93	11.8	64
Tasmania	97	9.3	71
Queensland	89	14.6	62
South Australia	90	8.8	68
Western Australia	83	10.8	65
Northern Territory	68	23.5	63

* Includes an over sample of the Hunter region of New South Wales

Australia's Imported Food Program – a valuable source of information on micro-organisms in foods

Ann L Bull,[1,2] Scott K Crerar,[1] Mary Y Beers[2]

Abstract

Foods imported into Australia are subject to laboratory testing for microbiological and chemical hazards under the Imported Food Program (IFP) for the purposes of protecting public health and safety. The program, operating under the Imported Food Control Act 1992, is jointly administered by the Australian Quarantine Inspection Service (AQIS) and the Australia New Zealand Food Authority (ANZFA). Foods that fail under the IFP are subsequently subjected to appropriate treatment to rectify the problem, or are destroyed or re-exported. This article presents a limited analysis of IFP test results on selected foods imported between 1995 and 1999. As corrective action is taken immediately on the basis of failing test results, regular analysis of collated data is not considered a priority. Nonetheless these data potentially represent an important source of information on the nature of food microorganisms detected in imported foods. For example, IFP data could be used to focus local and state-based food surveillance efforts, provide information to importers, to inform national initiatives such as OzFoodNet, and to better target investigative and preventative efforts concerning foodborne illness. *Commun Dis Intell* 2002;26:28-32.

Keywords: OzFoodNet, foodborne disease, Imported Food Program, quarantine, Listeria, Salmonella, E. coli

Introduction

The Imported Food Program (IFP) was established in 1990. The Program arose due to several serious incidents of food poisoning overseas during the 1980s[1,2] and its aim was to ensure the safety of food imported into Australia. The national program initially concentrated on foods considered a high public health risk. In 1992, through the *Imported Food Control* Act, the IFP was given specific legislative backing and expanded to cover all imported foods and beverages. The IFP ensures that food entering Australia complies with Australian food law. ANZFA undertakes scientifically based risk assessments for the program while AQIS conducts the operational aspects of the program.

Within IFP, foods are classified as Risk or Surveillance category foods. Risk category foods are initially determined and periodically reviewed by ANZFA on the basis of scientific risk assessments. Current Risk food groups and the full list of tests carried out are listed in the Table. These are foods which are considered to pose an inherent or historical high risk to public health, based on the likelihood of them being contaminated with harmful bacteria. Surveillance category foods are divided into two categories: Active and Random. Foods in the Active surveillance category are considered to pose a moderate public health risk, and for which more information is needed to make a definite categorisation. Foods in the Random Surveillance category are considered to pose a low level of risk to health and safety.

The Australian Customs Service (ACS) refers 100 per cent of Risk foods to AQIS for inspection on entry into Australia. Tests on Risk foods are specific to the food and testing rates depend on the compliance history of the producer. The first five shipments of a Risk food sourced from a particular producer are initially inspected. After five consecutively cleared shipments, the inspection intensity drops to the next level which is one in four shipments.

Following 20 cleared inspections, the inspection level drops to one in 20 shipments. Any failure at any stage results in elevation to a 100 per cent inspection rate until five consecutive shipments are cleared. Risk category foods are not released onto the market until test results are known.

Foods in the Active Surveillance category are inspected at the rate of 10 per cent by the supplying country. Foods in the Random Surveillance category are inspected at a rate of 5 per cent of shipments based on the volume entering by the respective tariff code.

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New products which have not been previously imported into Australia must first be assessed to ensure that they meet quarantine requirements. For example, unprocessed foods such as some raw meats are presently not allowed into Australia due to quarantine laws. Provided a food meets Australia's quarantine requirements, a risk assessment is then undertaken by ANZFA which concentrates on the public health and safety aspects of the food. Due to the recent problems with bovine spongiform encephalopathy (BSE) and the risk of acquiring variant Creutzfeldt-Jakob disease from the consumption of beef products, all beef products are now classified as Risk category foods. Importing countries are now assessed by ANZFA as to their level of BSE risk. Depending on the risk category that a country is allocated, various levels of certification are required to satisfy the Australian authorities that products are derived from animals not exposed to BSE risk.

All inspection and testing of foods under the IFP is carried out by AQIS. On entry into Australia, ACS refers all foods at the relevant rates of inspection, to AQIS. An AQIS officer then attends the premises of the importer and carries out an inspection. This involves examining packaging for defects or indications of contamination, and for appropriate labelling. The AQIS officer may also take samples of the foods for analytical testing if appropriate.

National standards stipulating allowable levels of microorganisms in foods are set out in the Australian Food Standards Code.[3] The levels are determined through risk assessment methodology which is consistent with established international practices such as those established by the International Commission on Microbiological Specifications for Foods (ICMSF), and the Codex Alimentarius Commission.[4]

The aim of this analysis was to assess whether IFP data can contribute to public health policy and be used in a broader and more strategic manner to help inform efforts in the control of and policy on foodborne illness. At present these data are accessible only by AQIS and used almost exclusively for operational purposes.

Methods

Microbiological data for Risk foods were obtained from the AQIS database as Microsoft Excel files. Data fields included date of entry, producer, country of origin, quantity, test type, test result (pass/fail) and details of organisms detected. Data were transferred to Microsoft Access and analysed by calculating rates of failures by organism and food type. Results were transferred to Microsoft Excel for production of graphs.

Results

Overall failure rates

Of the 17,685 microbiological tests performed on Risk foods for the five-year period 1995 to 1999, there were 486 failures (2.7%). Yearly failure rates ranged from 2.3 to 4.0 per cent.

Failure rates (failed tests/total tests 100) over the five-year period were calculated for Risk foods (Table 1). Of all Risk foods, smoked vacuum-packed fish had the highest failure rate of 8.6 per cent for *Listeria* contamination.

Failures for contamination with *Listeria*, *Salmonella* and *E. coli*

Foods tested for *Listeria* include smoked vacuum-packed fish, soft cheeses, chicken and mussels (Table). The percentage of total *Listeria* failures increased sharply until 1998 and then declined in 1999 (Figure 1). When examined separately, the results for soft cheeses and smoked fish are similar, in that there is an upward trend for failures until 1998 (results not shown).

Salmonella testing is carried out on spices, seafood items, pork, chicken and coconut (Table). No failures have been recorded in chicken or pork. A wide range of *Salmonella* serotypes were isolated from these foods including *S.* Weltevreden, *S.* Muenchen, *S.* Typhimurium, *S.* Mbandaka, *S.* Stanley and *S.* Hvittingfoss. The overall failure rate for *Salmonella* can be seen in Figure 1.

Foods tested for *E. coli* contamination include seafood, chicken and pork (Table). All failures for *E. coli* occurred in seafood (Figure 1). Limits for *E. coli* differ slightly for different foods.

For all 3 organisms the number of tests carried out each year remained fairly constant.

Standard plate count failures

Standard plate counts are carried out on seafood, pork and chicken. All failures were from seafood (Figure 2). Allowable levels differ slightly for different groups of foods (Table), however generally the SPC must be $< 10^5$/g.

Table. Failures by food type, 1995 to 1999

Risk food type	Test	Allowable limit	Overall failure rate %	Total number of tests
Smoked Fish	*Listeria monocytogenes*	Nil detect	8.6	388
Peanuts	Aflatoxins	15 µg/kg	7.1	1,438
Paprika	*Salmonella*	Nil detect	4.5	369
Marinara mix	*E. coli* Standard plate count Domoic acid *Salmonella* PSP	10/g 5 x 10^5 20 mg/kg Nil detect 0.8 mg/kg	3.7	283
Molluscs	*E. coli* Standard plate count Domoic acid *V. cholerae* PSP	2.5/g 10^5/g 20 mg/kg Nil detect 0.8 mg/kg	2.5	2,440
Crustaceans (cooked and chilled)	*E. coli* Standard plate count SET *Salmonella* Standard plate count	10/g 10^6/g Nil detect Nil detect 10^5/g	2.0	5,187
Pepper	*Salmonella*	Nil detect	1.6	1,584
Soft cheeses	*Listeria monocytogenes*	Nil detect	1.1	1,440
Selected Fish	Mercury	0.5 mg/kg	1.0	2,185
Coconut	*Salmonella*	Nil detect	0.8	524
Tuna	Histamines	200 mg/kg	0.4	1,162
Pork (cooked and chilled)	*E. coli* Standard plate count *Salmonella* CPS Standard plate count	10/g 10^6/g Nil detect 100/g 10^5/g	0.0	29
Chicken (cooked and chilled)	*Listeria monocytogenes* *E. coli* Standard plate count *Salmonella* CPS *E. coli* Standard plate count	Nil detect 10/g 10^6/g Nil detect 100/g 9/g 10^5/g	0.0 0.0	22
Cinnamon	*Salmonella*	Nil detect	0.0	10

SET = Staphylococcal enterotoxin
CS = commercial sterility
PSP = paralytic shellfish poisoning
CPS = Coagulase positive *Staphylococcus*

Figure 1: Percentage failures for *Listeria*, *Salmonella*, and *E. coli* contamination, 1995 to 1999

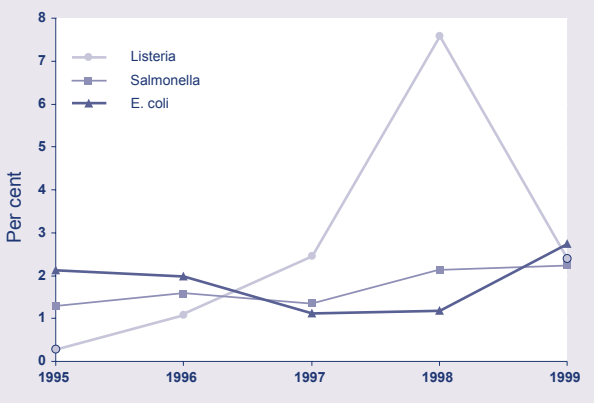
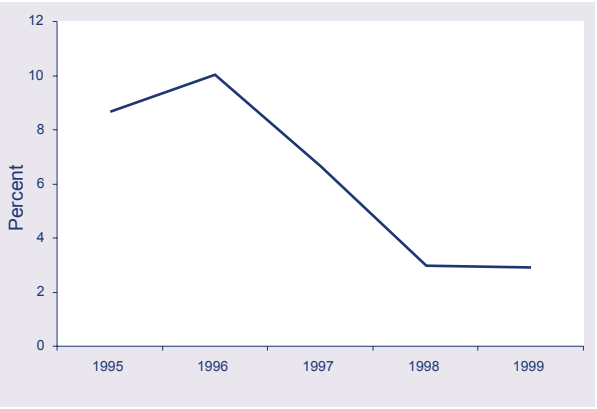


Figure 2. Percentage failed tests for high standard plate counts, 1995 to 1999



Numbers in parentheses represent number of tests performed

SPC = Standard Plate Count

Discussion

The IFP conducts the only ongoing systematic surveillance of imported foods for microbiological hazards in Australia. Collated data from the program could be utilised better to assist research into foodborne microorganisms and facilitate the identification of potential links between food items and illness due to specific human pathogens.

Routine analysis and interpretation of such data would provide important information for food importers and public health agencies to assist efforts towards the control of and policy development regarding foodborne illness. For example *Listeria* and *E. coli* contamination, as well as high standard plate counts may indicate poor production processes. For this reason awareness of trends such as the increase in *Listeria* failures from 1995 to 1998 is useful and could lead to actions to help improve the overall quality of food entering Australia.

The IFP database could also be used more effectively to assist some epidemiological foodborne diseases investigations. If, for example, clusters of disease due to unusual *Salmonella* serotypes are diagnosed with no apparent source, the IFP test results may be scanned to determine whether there have been previous associations between the particular pathogen and imported food types.

There were two notable multi-State outbreaks of diseases during 2001 solely attributed to imported food items.[5] One of these outbreaks was caused by halva, a sweet manufactured from sesame seeds which was contaminated with *Salmonella*. As a result of this outbreak, this product was elevated to Risk category for 3 months and 100 per cent was tested for *Salmonella* contamination. Following this time, during which there were no failures, it will be recommended that halva and other sesame seed products be tested for *Salmonella* under the Random surveillance category. The other outbreak was from peanuts contaminated with *Salmonella*. Peanuts are not currently tested for *Salmonella* at the Risk level of inspection. Part of the difficulty with linking failures in Risk category foods to particular outbreaks relates to the operation of the program and the recall system. Risk foods are not normally released until test results are known, resulting in foods which have failed for *Salmonella* contamination, for example, not reaching the marketplace. Of course this will only apply where the food has been tested.

Since 1995 there have also been 16 food recalls prompted by a failed test under the IFP. Such recalls are precautionary in nature, aiming to prevent the risk of illness occurring. As already mentioned, foods may also been elevated to Risk status as a result of them being linked to illness, as occurred for imported halva.

There are many limitations in trying to better utilise these data. These include the difficulty of determining exact denominators, in terms of volumes of different foods entering Australia. The number of tests is used here as a proxy, however this means that foods with low failure rates will be tested less and will consequently be under-represented in the results. Other limitations include the data quality which is variable, as data, particularly in terms of entry, occurs at many sites around Australia and the process is not harmonised. Timeliness is a particular issue of this system, and in some cases test results may not be entered into the database for several months after testing of the food.

The IFP surveillance, in conjunction with other food monitoring and intelligence needs to be more effectively utilised to allow the possible elucidation of links between food and disease and eventually lead to remedial action in food production processes. In summary, as well as being used to monitor individual shipments of imported food, these data represent an additional and valuable resource for public health agencies to aid in the investigation, prevention and control of foodborne illness.

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An outbreak of *Salmonella* Typhimurium PT135 gastroenteritis associated with a minimally cooked dessert containing raw eggs

Mohinder Sarna,[1,2] Gary Dowse,[1] Greg Evans,[3] Charles Guest[2]

Abstract

In April 2000, we investigated an outbreak of gastroenteritis amongst attendees of a local community dinner in a Perth suburb. Of the 98 people interviewed (response rate 98%), 53 reported gastrointestinal symptoms (attack rate 54%). Faecal cultures from 11 cases, 2 food preparers, 1 waitress and leftover mock ice-cream dessert grew *Salmonella* Typhimurium PT135. Of the 3 food handlers, one was asymptomatic, another gave an unclear history of onset of illness and the waitress claimed illness onset 9 days after the dinner. A cohort study implicated fruit salad (RR 1.64 [95% CI: 1.05-2.58], p=0.017) and/or mock ice-cream dessert (RR 1.78 [95% CI: 0.91-3.52], p=0.045). Eggs used to make the mock ice-cream dessert were supplied directly from the producer who used inappropriate shell cleaning methods. The method of preparation of the dessert encouraged contamination. *Salmonella* species were not isolated in poultry faecal samples collected from the implicated egg farm. The cause of this outbreak was almost certainly the ice-cream dessert with contamination most likely resulting either from the eggs used to make the dessert or one or both of the food preparers, coupled with inadequate cooking of the dessert. Eggs used in preparing food for mass consumption should be sourced from distributors with approved cleaning procedures. Furthermore, pasteurised egg products or egg pulp should be used in the preparation of uncooked or minimally cooked dishes. *Commun Dis Intell* 2002;26:32-37.

Keywords: Salmonella; *outbreak investigation; gastroenteritis*

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Introduction

On 26 April 2000, the Communicable Disease Control Branch (CDCB) of the Health Department of Western Australia received notification that 20 or more persons who had attended a function in the preceding week had experienced acute gastroenteritis characterised predominantly by diarrhoea, abdominal cramps, headaches, nausea and vomiting. All cases had attended a dinner celebrating a religious festival on one or both nights of 19 and 20 April 2000. A local community caterer had catered for the dinner.

Methods

Epidemiological investigation

A structured questionnaire was developed based on information from a set menu and preliminary findings. A retrospective cohort study was conducted among staff and dinner attendees in order to describe the course of the outbreak and determine its cause. Telephone interviews were performed by 2 trained staff of the CDCB between 26 and 30 April 2000.

A case was defined as any person who ate the dinner on either 19 or 20 April 2000 or both, and who reported the onset of gastrointestinal symptoms in the period from the time of the dinner to 25 April 2000. Gastrointestinal symptoms included diarrhoea (defined as two or more loose bowel motions within a 24 hour period) or at least two of the following symptoms: abdominal pain or discomfort; nausea; vomiting; or chills/sweats. An epidemic curve of time of onset of illness following the dinner was generated and relative risks of illness associated with the consumption of individual food items were calculated using Epi Info 6 software.

Environmental health investigation

Site investigation

The kitchen of the community centre was visited by an Environmental Health Officer (EHO) from the local government authority on 28 April 2000 (earlier access was not possible). The kitchen facilities, including food storage and preparation areas were inspected. Temperatures of refrigerators and freezers were measured. The flow of work was assessed through detailed interviews with the food handlers in an effort to identify risky food handling procedures and opportunities for cross-contamination. Samples of leftover food stored in the freezers of the community hall kitchen and in the household of one dinner guest, were obtained for testing. Environmental sampling was not carried out for a number of reasons. Access to the kitchen was not possible until 10 days after the dinner was held. In addition, in keeping with religious customs, the kitchens had been rigorously cleaned after the dinners were held. It was felt by the EHOs from the local authority and the CDCB that environmental sampling would yield little in this situation.

Investigation of the egg farm

In an effort to determine the source of the *Salmonella* infection, the egg farm where the eggs were purchased was inspected. As the likelihood of isolating *Salmonella* species from whole shell eggs was very low, samples of chicken litter and faecal matter were collected from 2 chicken sheds on 7 June 2000 and cultured at PathCentre for *Salmonella* species.

Laboratory investigation

A total of 13 cases, 2 food preparers and 2 waitresses provided faecal specimens which were cultured for *Salmonella*, *Shigella* and *Campylobacter* species, and examined for parasites with direct and concentrate microscopy. Several items of leftover food were submitted to the Food and Waters laboratory at PathCentre, Perth for testing. These included soup, beef, carrot bake, potato bake, bread, mock ice-cream, wine, grape juice and lemon cordial. Foods were examined for total bacterial plate count, and tested for specific organisms, namely *E. coli*, coagulase positive *Staphylococcus*, *Clostridium perfringens*, *Bacillus cereus* and *Salmonella* species. *Salmonella* isolates were further identified by serotyping. Phage typing was performed at the Microbiological Diagnostic Unit, University of Melbourne.

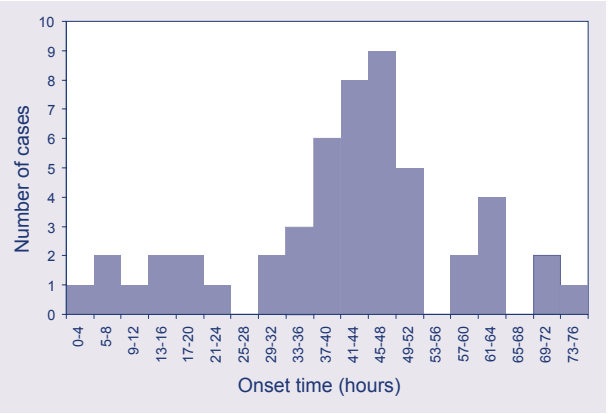
Results

Epidemiological investigation

A total of 107 people attended the dinner over 2 nights: 39 on 19 April and 71 on 20 April 2000, with 3 people attending on both nights. Seven people who attended on the first night did not eat any food and were not ill. Of the remaining 100 people, 98 were interviewed (98% response). Fifty-three fulfilled the case definition, giving an overall attack rate (AR) of 54 per cent. Of these, 22 were female (AR 42%) and 31 were male (AR 58%). Of

those interviewed, 21 of 30 people who attended the dinner on the first night were ill (AR 70%), and 32 of 68 people from the second night were ill (AR 47%) (chi2=4.41, p=0.036). The age range of the cases was 1 to 73 years old (median 27.5 years).

Figure. Number of cases of gastrointestinal illness categorised by onset time after consumption of food



The median onset time of gastrointestinal symptoms was 43 hours after eating the dinner (range 4–75 hours) (Figure). Diarrhoea was the most common symptom (96%), followed by abdominal pain or discomfort (85%), tiredness or weakness (81%), headache (51%), chills (47%), fever (45%), sweats (42%), nausea (40%), abdominal distension (36%) and vomiting (11%). Three cases experienced bloody diarrhoea (6%). The median duration of gastrointestinal symptoms was 4 days (range 1–12 days). Nineteen cases (36%) visited a doctor, and one case was taken to hospital but not admitted. Most cases were ill over the Easter holidays and did not have any time off work.

An analysis of foods eaten on the night found that the mock ice-cream dessert showed the strongest association with illness, with a relative risk (RR) of 1.78 (95% C.I: 0.91-3.52, p=0.045), followed by fruit salad with a RR of 1.64 (95% CI: 1.05-2.58, p=0.017). There was also an elevated RR associated with eating parsley (RR 1.74, 95% CI: 0.91-3.33, p=0.06). The food specific attack rates and relative risks are shown in the Table.

Food for both nights was prepared in one batch and the menu was identical for both nights. There were two food handlers actively involved in food preparation, plus two other staff employed for waitressing. All of them had tasted the mock ice-cream dessert either on the day it was prepared or served, as well as taking home other leftover food items. Faecal specimens were submitted by both food preparers and both waitresses. One of the preparers was asymptomatic. The other food preparer reported an enteric illness, but gave an inconsistent history of onset of illness, ranging from the time of food preparation to 7 days later. One waitress was asymptomatic and the other experienced gastroenteritis 9 days after the function (when the faecal specimen was collected) and denied consumption of any leftover food during the intervening period.

Environmental investigation

Site investigation

The kitchen appeared to be clean on the day of inspection. There was a defined separation of work areas. Refrigerator and freezer temperatures complied with requirements and adequate handwashing facilities were provided.

Food preparation and handling

Food for the dinner was prepared in the kitchen of the community centre on 17, 18 and 19 April 2000 and stored in the refrigerator. This food was reheated and served on the evenings of the dinner. Vegetables were stored uncooked in the chiller, prior to being cooked on the day of the dinner. Salads were prepared on the day of the dinner. The meat was prepared and roasted the morning it was purchased. It was then cooled, cold sliced and refrigerated until the evening of the dinner.

The mock ice-cream dessert was made with fresh eggs purchased directly from a local egg farm. The food preparers maintained that only clean, unbroken eggs were used. To prepare the ice-cream base, hand separated yolks were mixed with sugar and whipped with a small electric hand blender. The whipping was done in approximately 10 batches. Lemon juice and hador (a soy milk-based non-dairy substitute) were then added, and the mixture poured into trays and frozen. The whites were kept for the next day to be whipped into a meringue topping with sugar and added to the lemon base. The entire tray was then placed in a hot oven at 225˚C until the top was just brown, taken out, cooled and placed back in the freezer until served. The cooking method was designed to deliver small amounts of heat to the top to brown the egg white, while leaving the base, containing uncooked egg yolks, frozen.

While the salads and cold items were put on the table, all hot food and the mock ice-cream and fruit salad dessert were served in the kitchen, where they were collected by guests.

Investigation of the egg farm

The egg farm consisted of 2 sheds of laying chickens. An interview with the egg farmer revealed that at the time the eggs were sold for this dinner, the usual practice was for visually dirty eggs to be soaked in a large container of water on the egg farm to clean them. It was unclear how long the eggs were soaked or how often the water was changed.

Laboratory investigation

Faecal specimens from 11 of the 13 cases, both food preparers and one waitress (whose illness onset was 9 days after the function), as well as a sample of the leftover mock ice-cream dessert were positive for *Salmonella* Typhimurium PT135. No organisms were isolated from other leftover foods tested. *Salmonella* species were not cultured from any poultry faecal matter or chicken litter samples obtained from the egg farm.

Table. Food specific attack rates and relative risks

Food	Persons who ate item			Persons who did not eat					
	No. ill	Total	Attack rate (%)	No. ill	Total	Attack rate (%)	Relative risk	(95% CI)	p value
Boiled egg	48	85	56	5	12	42	1.36	0.68-2.72	0.34
Soup	49	91	54	4	6	67	0.81	0.44-1.47	0.69
Beef	44	76	57	9	21	43	1.35	0.80-2.29	0.22
Carrot bake	31	55	56	22	42	52	1.08	0.74-1.56	0.70
Potato bake	39	66	59	14	31	45	1.31	0.85-2.03	0.20
Cabbage salad	30	48	63	23	49	47	1.33	0.92-1.93	0.13
Bread (n=96)	52	95	55	0	1	0	-	-	-
Green salad	33	53	62	20	44	45	1.37	0.93-2.01	0.1
Herbs (n=87)	34	63	54	9	24	38	1.44	0.82-2.53	0.17
Haroset (n=83)	34	65	52	7	18	39	1.35	0.72-2.51	0.32
Parsley (n=87)	36	65	55	7	22	32	1.74	0.91-3.33	0.06
Mock ice-cream	47	79	59	6	18	33	1.78	0.91-3.52	0.045
Fruit salad	39	61	64	14	36	39	1.64	1.05-2.58	0.018
Wine	34	62	55	19	35	54	1.01	0.69-1.48	0.96
Grape juice	35	62	56	18	35	51	1.10	0.74-1.62	0.63
Other juice	42	73	58	11	24	46	1.26	0.78-2.02	0.32

(n=97 except where indicated)

Discussion

This report describes the investigation of a well-defined *Salmonella* Typhimurium PT135 point source outbreak with a high attack rate (54%), suggestive of a high level of contamination.[1] Two food preparers and one waitress were also infected. The range and sequence of symptoms, and the incubation period (6 to 72 hours) are consistent with *Salmonella* infection. It is not clear why higher attack rates were seen in those who attended on the first night. Food for both nights was prepared in one batch and the menu was identical for both nights. Speculatively an extra day's storage at appropriate temperatures may have resulted in some diminution in the number of microorganisms in the suspect foods.

An analysis of foods eaten on the 2 nights of the dinner showed non-significant elevations in risk for several foods. The highest risk and significant associations were seen with consumption of the ice-cream dessert and fruit salad. A portion of the ice-cream was served together with a helping of fruit salad to each person on the same plate and diners were not offered a choice of fruit salad or ice-cream separately. Hence the common association of these foods with risk of illness is not surprising. Unfortunately, no leftover fruit salad was available for testing. Furthermore, laboratory confirmation that the *Salmonella* Typhimurium PT135 cultured from faecal samples and the mock ice-cream dessert were indistinguishable, provides strong corroborative evidence of the causative agent and the contaminated food. The near significant association of parsley is puzzling and could reflect chance or cross-contamination within the kitchen.

The environmental health inspection did not detect any clear breaches in food handling or food safety practices, although the method of preparing the mock ice-cream dessert encouraged contamination as it involved much manual handling with hundreds of eggs being separated by hand. The origin of the bacterial contamination for the outbreak remains unclear. As meat was prepared and cooked on the morning it was purchased, any *Salmonella* originating from the meat had little opportunity to multiply or contaminate other foods. Isolation of *S.* Typhimurium from the mock ice-cream dessert implicates the eggs as a possible source of contamination, although shedding by either or both of the food preparers, one of whom was asymptomatic and possibly a carrier pre-dating the outbreak, cannot be excluded. Moreover, the other food preparer gave an inconsistent history regarding the time of onset of illness.

The investigation also revealed that the batch of eggs bought for the dinner was soaked in a tub of water on the farm to clean them of any poultry faecal matter. *Salmonellae* are widespread in the poultry industry through the food chain, and therefore can be present on the outside of eggs.[2,3] It was unclear how long the eggs were soaked or how often the water was changed. This manner of cleaning the eggs is inappropriate as it is thought to make the eggshell more permeable and thus more susceptible to bacterial contamination, especially if the water used for soaking has a high microbial load.[4] The practice has now been discouraged by the Egg Board and currently, eggs that are not too dirty are scraped clean with a knife or grinding wheel and sold as unwashed eggs through a retail outlet on the premises. Any badly soiled eggs are sold to the local Egg Board, who then pulp and pasteurise the mixture.

Whatever the mode of transmission, once the egg mixture was contaminated, bacteria had ample opportunity to multiply given the method of preparing the dessert over 2 successive days, with minimal cooking. This combination of circumstances may have led to the contamination of the mock ice-cream dessert, resulting in the outbreak. Although *Salmonellae* were not isolated from chicken faecal matter from the farm, this does not rule out contamination of the egg mixture with *S.* Typhimurium PT135 originating from shell eggs.

Between 400 and 900 notifications of salmonellosis are received annually in Western Australia,[5] with the predominant serovar being *S.* Typhimurium (42% in 2000), most frequently phage type 135.[5] *Salmonella* Typhimurium PT135 has strong epidemiological associations with the cattle and poultry industry.[2] To date the most common serovars found in cloacal and drag swabs in the poultry industry in Western Australia have been *S.* Sofia and *S.* Infantis, and more recently, *S.* Kiambu (Kim Leighton, Health Department of Western Australia, personal communication).

Outbreaks of foodborne disease originating from the use of either unpasteurised whole shell eggs or cracked and dirty eggs have been reported both in Australia[6,7] (Dr Jeffrey Hanna, Tropical Public Health Unit, Queensland, personal communication), and elsewhere.[8] The first 2 reports detailed investigations that found the incorporation of raw eggs into dishes that received minimal or no further cooking. The lack of a national outbreak register in Australia makes it difficult to accurately assess the

incidence of foodborne disease attributable to eggs as a vehicle for transmission.

An inquiry report by the Australia New Zealand Food Authority in 1996, reviewed egg and egg products and proposed to prohibit the retail sale and use of unpasteurised egg products for catering purposes and include a mandatory warning and advisory statement regarding all cracked eggs and unpasteurised egg products.[9] However, no nationally consolidated data are available regarding the levels of important pathogens in eggs with intact shells or egg products.[10]

In summary, two key points result from the investigation of this outbreak. Firstly, eggs used in preparing food for mass consumption should be sourced from distributors with approved cleaning procedures. A distinction should be made between eggs bought through large-scale commercial suppliers where the eggs undergo a rigorous and controlled sanitising and cleaning process, and farm-direct outlets that may not have adequate procedures in place to clean shell eggs. Secondly, caterers should be educated about good food handling practices when handling shell eggs. A survey of raw egg use by home caterers highlighted the level of ignorance among food handlers when handling raw eggs.[11] Over 40 per cent of caterers reported not washing their hands after handling intact shell eggs. Eggs should be included as one of the foodstuffs where thorough handwashing after handling is mandatory, as is the case with raw meat and vegetables. Caterers should be encouraged to opt for the use of pasteurised egg products or egg pulp in the preparation of dishes where the dish is subsequently not cooked or minimally cooked.

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An outbreak of *Salmonella* Typhimurium phage type 135 infection linked to the consumption of raw shell eggs in an aged care facility

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In March 2001, the Communicable Diseases Control Branch and local government investigated an outbreak of gastroenteritis in an aged-care facility in rural South Australia. Initial reports indicated 12 residents and 1 staff member had experienced gastrointestinal illness; 3 residents had been hospitalised. An epidemiological and environmental investigation sought details of illness, food consumption, food purchasing practices and social activities for the 3 day period prior to the onset of illness in the first case. In total, 18 (16 residents, 2 nursing staff) were ultimately linked to this outbreak (Figure). The predominant symptoms were diarrhoea in 18 cases (100%) and vomiting in 10 cases (55.5%). Of the 15 cases for whom the time of onset was available, the median incubation period was 45.5 hours (range: 12 to 94 hours). Thirteen stool specimens provided by residents and staff yielded *Salmonella* Typhimurium phage type 135. One secondary case in a health care worker was identified. This case is believed to have acquired the infection by person-to-person transmission. There were no reports of gastrointestinal illness in kitchen food handlers.

Attack rates (AR) for food items served at the nursing home in the 3 days prior to the outbreak were calculated. A rice pudding (AR=42%) and meat-based potato pie (AR=48.5%) were identified as possible sources for the infections. The environmental investigation established that 8 raw shell eggs had been whisked into the cooked rice pudding immediately prior to serving. In addition, raw egg had been incorporated into the potato topping of a meat pie and lightly browned. Indeed, the incorporation of raw shell eggs into dishes that received minimal or no further cooking was a plausible explanation for the outbreak. Microbiological sampling of both the leftover meat-based potato pie and a frozen serve of the rice pudding confirmed the presence of *Salmonella* Typhimurium phage type 135.

The first staff member to become ill did not consume the potato pie or rice pudding. Nonetheless, the staff member reported handling and cracking the raw eggs included in the rice pudding. The staff member reported that the exterior surfaces of the shell eggs were visibly contaminated, however, there were no reported breaks in the integrity of the eggshells.

A trace-back investigation identified the producer that supplied the nursing home with shell eggs. An environmental investigation identified that whole and crushed grains used to feed the chickens were securely stored, however, an intermediate step, the chicken feed crusher, provided rats with access to crushed grain. Rat faeces were observed in the chicken feed crusher and crushed grain stored in sealed bins. Microbiological sampling of the chicken feed, chicken feed crusher and the exterior surface and interior contents of shell eggs collected after the outbreak were negative for *Salmonella* spp. However, microbiological sampling of chicken manure from the farm yielded *Salmonella* Typhimurium phage type 135.

This outbreak illustrates the dangers associated with eating inadequately cooked shell eggs. The elderly and immunocompromised persons are particularly susceptible to infection with small numbers of *Salmonellae* and in turn are more likely to suffer more severe disease than healthy adults. In nursing homes, pasteurised egg products should be used in dishes that are not thoroughly cooked. Hands, cooking utensils and food preparation surfaces should be washed thoroughly after contact with shell eggs to prevent cross contamination. The economic impact of this outbreak was considerable. Three residents were hospitalised and the need for additional staff resources presented an overwhelming burden for the local community.

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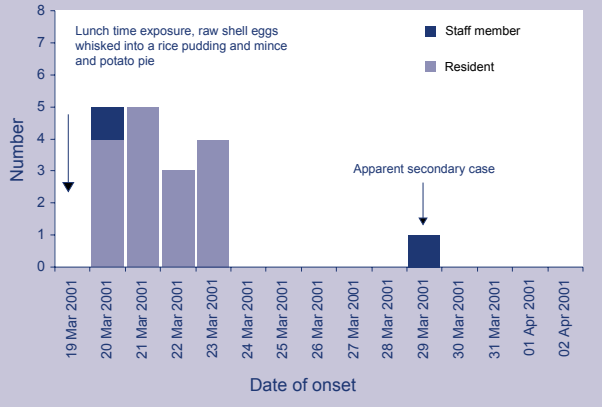
With the cooperation of the nursing home, local government implemented changes to food handling procedures. Consequently, no further cases of *Salmonella* Typhimurium phage type 135 infections have been reported from this aged-care facility.

Editor's note: This outbreak was briefly reported in the OzFoodNet January to March 2001 quarterly report published in *Commun Dis Intell* 2001;25:105.

Figure **Outbreak of *Salmonella* Typhimurium phage type 135 in an aged care facility in South Australia**



Reappearance of human cases due to Murray Valley encephalitis virus and Kunjin virus in Central Australia after an absence of 26 years

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Abstract

Murray Valley encephalitis (MVE) and Kunjin virus disease are endemic in the tropical parts of the Northern Territory and Western Australia, but have been absent from Central Australia since 1974. In 2000, 5 laboratory-confirmed cases of encephalitis occurred over a short period in the normally dry inland region of Central Australia. The sudden occurrence of cases in March and April 2000 followed unusually high rainfall in the preceding months and evidence of flavivirus activity in the endemic areas in the Kimberley region of Western Australia. Further cases were reported in the following wet season, without preceding human cases in known endemic areas. These findings indicate the reintroduction of these viruses into Central Australia and establishment of local cycles of infection with an ongoing risk to the local population. This area may also act as a potential source for reintroduction of MVE into south-eastern Australia. *Commun Dis Intell* 2002;26:39-44.

Keywords: Murray Valley encephalitis, Kunjin, encephalitis, flavivirus, Central Australia

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Introduction

Murray Valley encephalitis (MVE) and Kunjin (KUN) are mosquito-borne flaviviruses found in Australia.[1] Human infections are usually asymptomatic, though a small percentage develop clinical disease manifesting as febrile illness with headache, as polyarthralgic illness or as encephalitis.[2,3,4,5] Encephalitis has high mortality and morbidity. Nearly all cases of encephalitis have been due to MVE and relatively uncommonly due to KUN.[3,5,6] Both viruses are believed to survive in cycles of infection between birds and mosquitoes in enzootic foci in the Kimberley region of Western Australia, the Top End of the Northern Territory and possibly northern Queensland.[7,8] Spread outside these areas is thought to occur when flooding allows migration of infected waterbirds that introduce the virus into local populations. This is thought to be the mechanism for epidemic disease in areas of Western Australia south of the Kimberley, south of the Top End in the Northern Territory and for the rare occasions of spread into south-eastern Australia.[1,7,9,10,17]

Culex annulirostris is the major vector of MVE virus, although other mosquitoes, such as *Aedes normanensis* may also be involved.[1,9]

The last national epidemic of MVE encephalitis occurred in 1974,[2] beginning in the Murray Valley region of south-eastern Australia. That epidemic included 5 Northern Territory cases, two of which were in the Alice Springs area.[1] A recent review[8] report that a further 13 cases of MVE disease had been recorded in the Northern Territory between 1975 and 1999, none of which occurred in Central Australia. A single presumptive case from Alice Springs was reported in 1997, but could not be confirmed because of the death of the patient.[11] No further cases have been recorded in south-eastern Australia since 1974.

The epidemiology of KUN is less well documented but appears to be more widespread than MVE. It is found throughout northern Australia, with occasional spread to south-eastern Australia,[7] but cases have never been identified in Central Australia.

Sentinel chicken flocks are employed as a means of early warning of MVE and KUN virus activity by testing for general and specific flavivirus seroconversion.[12,13] In the months of March and April 2000, a series of media alerts were issued by the Territory Health Services following seroconversion of sentinel chickens in the Northern Territory.

During the same period, clinicians at Alice Springs Hospital reported several cases of undiagnosed neurological illness in both paediatric and adult patients. Eventually 5 cases of MVE and KUN encephalitis were diagnosed in Central Australia.

We report the epidemiology of human disease in Central Australia in 2000, discuss the environmental indicators and public health surveillance systems, and highlight the potential for an ongoing risk to human health.

Patients and methods

Ethical approval was sought from and supported by the Central Australian Human Research Ethics Committee for the publication of the results of this investigation.

The medical records of all 5 cases were reviewed and data on geographical location, age, gender, ethnicity, clinical features, laboratory and radiological investigations were collected.

Serological testing was performed at the Division of Microbiology and Infectious Diseases, Western Australian Centre for Pathology and Medical Research in Perth and in the Arbovirus Research and Surveillance Laboratory, Department of Microbiology, University of Western Australia. A standard flavivirus haemagglutination inhibition (HI) test and an indirect immunofluorescence assay (IFA) were used for IgG and IgM detection in serum and cerebrospinal fluid (CSF) samples.[3,4] Specific IgG to MVE or KUN was determined using an epitope-blocking enzyme immunoassay (EIA).[12] MVE-RNA in CSF was detected using reverse transcription, then DNA amplification by polymerase chain reaction (RT-PCR).[14] All were done in duplicate and only reported as positive or negative if both results were the same. If the results were discrepant the test was repeated. Samples unable to be resolved on repeat testing were called equivocal.

Data on sentinel chicken seroconversion to MVE and Kunjin viruses in Central Australia were obtained from the Sentinel Chicken Surveillance Programme of the Northern Territory and mosquito vector prevalence data from the Medical Entomology Branch, Northern Territory Department of Health and Community Services. Sentinel flocks are maintained at major population centres by officers of the Northern Territory Department of Primary Industry and Fisheries or by volunteers in a co-operative programme with the Northern Territory Department of Health and Community Services.

They are bled monthly and samples are sent to the Department of Microbiology at the University of Western Australia and tested with the epitope-blocking EIA.[12] Seroconversion of one or more of the flock to MVE virus initiates a media alert by the Northern Territory Department of Health and Community Services.

Mosquito monitoring in the Alice Springs area is carried out on a weekly basis by the Medical Entomology Branch of the Department of Health and Community Services and the Alice Springs Council using 6 sites in urban, semi rural, and rural locations. Mosquito numbers and species are recorded. Increases of potential vector species trigger public health warnings.

Results

The first indication of MVE virus activity in the Northern Territory in 2000 was seroconversion in a sentinel flock in a rural setting near Darwin bled on 24 February. This was followed by sentinel flock seroconversions in Tennant Creek on 3 March and in Alice Springs on 22 March 2000.

From January 2000, there was a sharp rise in *Cx. annulirostris* mosquito numbers at multiple rural sites near Alice Springs in Central Australia. The numbers remained high to very high in most sites until early April, after which numbers began to decline. At the single Alice Springs urban trap site numbers were low to moderate at all times.

All the cases were Aboriginal and came from remote communities. Demographic details are provided in Table 1. Two paediatric patients from a single remote community presented to hospital on the same day, one with MVE encephalitis, and the other with KUN encephalitis. All patients presented with 24-48 hours of prodromal symptoms, including malaise, irritability, fever, vomiting, headache and neck stiffness. In 4 cases there was progression to significant neurological manifestations. Three of these developed severe disease and required ventilation for refractory seizures or deteriorating level of consciousness. One infant was left with residual quadriparesis and one adult with persisting altered consciousness and generalised weakness. A second infant had persistent hypotonia and mild left-sided weakness at 3 month follow-up and has an uncertain neurological outlook. Of the 2 patients considered to have recovered fully, the paediatric patient is neurologically intact, while the adult patient is abnormal but as a result of chronic substance abuse.

Cases 1, 2 and 3 were confirmed as acute MVE encephalitis and case 4 as acute KUN encephalitis (Table 2). Case 5 had a rapid rise in antibody to both viruses with the presence of both KUN and MVE antibody on the epitope-blocking EIA. The CSF showed predominantly polymorphs in children and monocytes in adults. The samples analysed biochemically showed elevated protein and normal glucose.

No cases of non-encephalitic MVE or KUN infection were identified in Central Australia during this period.

Table 1. Demographic and clinical characteristics of the cases

Case no.	Date of onset	Age	Sex	Symptoms/signs	Outcome	Agent
1	25/3/2000	69 years	M	Fever, headache, neck stiffness, deteriorating sensorium	Severe cognitive impairment	MVE
2	27/3/2000	3 months	F	Fever, seizures, flaccid quadriplegia	Quadriplegia	MVE
3	3/4/2000	2 months	M	Fever, irritability, seizures	Hypotonia	MVE
4	3/4/2000	4 years	M	Fever, vomiting, neck stiffness	Complete recovery	KUN
5	13/4/2000	30 years	M	Fever, ataxia, altered sensorium, neck stiffness	Complete recovery	Unspecified (MVE/KUN)

Table 2. Serology and CSF results, 2000

	Serology						CSF					
Case	Date	MVE HI	MVE IgM	KUN HI	KUN IgM	EIA	Date	PMNs x106/L	Mono x106/L	Protein g/L	Glucose mmol/L	MVE PCR
1	30/3 24/4	1:10 1:320	Positive Positive	<1:10 1:80	Negative Positive	MVE	29/3	70	110	0.81	3.6	Equiv*
2	6/4 11/4	1:40 1:160	Positive Positive	1:80 1:40	Negative Negative	MVE	29/3	250	300	2.5	3.2	Positive
3	8/4 28/4	1:40 >1:640	Positive Positive	<1:10 1:320	Negative Not done	MVE	5/4	510	40	Not tested	Not tested	Not done
4	7/4 3/5	<1:10 <1:10	Positive Negative	1:10 1:160	Positive Positive	KUN	8/4	110	75	Not tested	Not tested	Not done
5	17/4 4/5	1:320 1:10240	Positive Positive	1:320 1:20480	Positive Positive	MVE + KUN	17/4	50	140	0.94	2.3	Negative

Discussion

There had been no confirmed cases of MVE or KUN disease in Central Australia between the last national epidemic in 1974 and this outbreak.[8] That is presumed to reflect the absence of the correct environmental conditions that would allow the reappearance of the virus, by migration of infected waterbirds into the area or other mechanisms, and the establishment of local cycles of activity.[1,7,9,10]

The first indicator of MVE activity in Australia in 2000 was seroconversion of sentinel chicken flocks from the Kimberley and Pilbara regions of northern Western Australia, in early January.[13] This was followed in the Northern Territory by seroconversions in chicken flocks in Darwin in late February, Tennant Creek in early March and Alice Springs in late March, accompanied by a rise in *Cx. annulirostris* mosquito numbers around Alice Springs in January and continued high numbers until early April. These events followed extraordinary environmental conditions, with unusually high rainfall in the preceding months and extensive flooding resulting in large contiguous bodies of water extending from northern Western Australia across to Central Australia (C Woodsworth, Bureau of Meteorology, personal communication; Monthly Weather Review, Bureau of Meteorology). The sequential seroconversion of the sentinel chickens in a south-easterly direction reflected the pattern of rain and flooding, and presumably the migration of MVE and KUN infected birds or wind blown infected vectors aided by prevailing north-westerly monsoonal winds.

The demographic and clinical features in this outbreak were similar to previous reports.[3,4,5] There was a mixture of adult and paediatric cases that is typical of epidemic disease occurring in non-immune populations,[3,5] and is consistent with rare flavivirus cases in Central Australia. Interestingly, the cases included 2 young Aboriginal infants. Previous series that included Aboriginal infants[3,4] had occurred in endemic populations, and there were no cases in children under 6 months of age. This may be due to high levels of maternal antibody expected in these populations[15,16] providing passive protection for the newborn. That would not have been the case in the Central Australian communities, and therefore very young children were susceptible. The male predominance observed in this series has been previously reported.[3,4] In adults, it is presumed to reflect higher exposure risk, but it is also seen in young children. The explanation for the latter is unknown.

It is notable that all cases came from remote communities, despite sentinel chicken seroconversion in urban Alice Springs. However, mosquito numbers were low in the urban trap, suggesting that numbers of infected mosquitoes in urban areas were too low to pose a risk to humans despite the presence of the virus in rural areas.

Previous reports from Western Australia (1978-1991) and the Northern Territory (1987-1996) have highlighted the severity of MVE encephalitis.[3,4,5] Although only 1-2 per 1,000 infections with MVE virus result in encephalitis,[1] of those that do, over 20 per cent are fatal, and permanent neurological sequelae occur in about half the survivors.[5] With the addition of our 3 definite MVE cases to those summarised previously,[5] the case fatality rate of MVE is 9/49 (18.4%). Eleven of the 49 (22.4% overall, 27.5% of the survivors) had major neurological sequelae and 12 cases (24.5% overall, 30% of survivors) had minor sequealae. Only 17 cases (34.7%) were documented to have made a complete recovery. All fatalities have occurred in patients less than 2 or over 60 years of age. As previously reported,[3,4] we found elevated CSF protein in the 3 cases in which it was measured. CSF cell counts showed no clear trends, although the predominance of monocytes in adults and polymorphs in children is of interest, perhaps reflecting earlier sampling in children.

KUN encephalitis is much less common than MVE encephalitis, with only 3 definite cases recorded in the literature since 1974, including the one in this series.[4,6] In addition, a further confirmed case occurred in Alice Springs in 2001, a 24-year-old woman with a mild illness and full recovery (A Brown and D Smith, personal communication). All of these have survived, suggesting that it may be a milder illness than MVE encephalitis, though the numbers are still too small to be confident.

One of the patients (case 5) had high levels of antibody to both KUN and MVE. It is likely that he had been previously infected with one of these viruses, and the current acute infection evoked brisk antibody responses to both the current and previous virus. We were unable to ascertain which was the current infecting virus. Case 4 showed an initial positive IgM to both MVE and KUN in the serum, but the MVE IgM was quite weak and was not seen in the second sample.

The level of susceptibility to MVE and KUN infection within the Central Australian population is not known, as there is no current local human seroprevalence data. In the absence of cases for over 25 years, it is assumed that all people who were born or moved into the area since 1974 will be susceptible, including the local Aboriginal community. That is unlike the situation in the Top End where very high levels of past infection are found in local Aboriginal communities.[15] Serosurveys have been conducted in Aboriginal coastal communities in the Cape Leveque region of Western Australia, where occasional human disease occurs.[16] These show low seroprevalence overall with the highest prevalence in the age groups likely to have resided there during the last outbreak in the late seventies and early eighties.[3] This suggests that while there may be some effective immunity in people resident in Central Australia during the 1974 epidemic, it is likely to be limited. Therefore it must be assumed that the possibility exists for further outbreaks of MVE in Central Australia in the future, involving both Aboriginal and non-Aboriginal residents and visitors.

A further two cases of MVE encephalitis, one KUN encephalitis and an undifferentiated MVE/KUN encephalitis have occurred the Central Australian region in early 2001 (A Brown and D Smith, personal communication). These occurred around Alice Springs itself and involved Aboriginals and Caucasians. As there was no evidence of spread from outside the area, it suggests that endemic activity has become established in the region. This highlights the need for increased vigilance in the future, particularly in terms of clinical and arboviral surveillance activities, when climatic conditions are ideal for virus replication and transmission. Widespread and high summer rain, high vector mosquito numbers and seroconversion in sentinel chickens in Central Australia may help to predict outbreaks of MVE in Central Australia and other regions, including more densely populated areas of south-eastern Australia. It is believed that epidemics in that area result from reintroduction of virus when 2 successive years of abnormal spring rainfall in the Murray Darling River catchment allow chains of bird-mosquito transmission from northern Australia.[9] However, if endemic activity is now established in Central Australia, this spread could occur more easily. Prompt and efficient communication between public health authorities, clinicians and the community is essential. Media warnings serve as a reminder for people to take mosquito protection measures, but may also facilitate early diagnosis of clinical cases.

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Epidemiology of invasive meningococcal disease in North Queensland, 1995 to 1999

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Abstract

This study describes all episodes of invasive meningococcal disease (n=120) acquired in north Queensland over the 5 year period 1995 to 1999. Indigenous people had a 3-fold greater risk than others of acquiring invasive meningococcal disease. There were 7 deaths, six in non-indigenous people. The majority (72.4%) of identified isolates were serogroup B. We found no evidence of significant resistance to the antibiotics recommended for treatment or chemoprophylaxis. Two outbreaks of disease were identified, one serogroup B and one serogroup C. Compared to the previous 5 years (1990 to 1994) there were far fewer cases of serogroup C disease and a lower incidence and risk of invasive meningococcal disease among indigenous people. *Commun Dis Intell* 2002;26:44-50.

Keywords: invasive meningococcal disease, Neisseria meningitidis, *indigenous people*

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Introduction

The epidemiology of invasive meningococcal disease in north Queensland from 1990 to 1994 has been reviewed previously.[1] In that period there were 69 cases with invasive meningococcal disease, giving an annual incidence of 3.3 per 100,000 population.[1] The incidence rate in indigenous people was 12.6 times that of non-indigenous people. From 1990 to 1994 seventy per cent of cases were caused by serogroup C *Neisseria meningitidis*, and there were 5 group C outbreaks. There were 3 deaths, all of indigenous people and all caused by group C organisms. In this paper we review the epidemiology of invasive meningococcal disease in north Queensland for the period 1995 to 1999, and make comparisons with the earlier study.[1]

Materials and methods

Case definition

Only cases of invasive meningococcal disease diagnosed and acquired in north Queensland between the beginning of 1995 and the end of 1999 were included in the study.

A confirmed case of invasive meningococcal disease was defined as a clinically compatible illness and at least one of the following:

- isolation of *N. meningitidis* from a normally sterile site;
- a positive PCR test for meningococcal DNA in a specimen from a normally sterile site;
- detection of Gram-negative intracellular diplococci in a specimen from a normally sterile site; or
- the detection of meningococcal antigen in CSF.

The PCR test became available in Queensland for the first time in 1999; a serological test for detecting meningococcal IgM did not become available until 2000.

A probable case of invasive meningococcal disease was defined as a clinically compatible illness with at least one of the following:

- a haemorrhagic rash;
- isolation of *N. meningitidis* from a throat swab; or
- close recent contact with a confirmed case.

Probable cases were notified for the first time in Queensland in 1999.

Since 1995, the geographical area included in the surveillance jurisdiction of north Queensland has been expanded to include the Mackay and Moranbah Health Service Districts (Figure 1). In 1996, the total population of north Queensland was 592,000, of whom approximately 48,000 (8%) were Indigenous.[2]

Figure 1. The Northern Public Health Zone of Queensland showing the 11 Health Service District jurisdictions

Case ascertainment

All cases of invasive meningococcal disease notified to The Tropical Public Health Unit in Cairns during the period 1 January 1995 to 31 December 1999 were collated. These data were supplemented with a search of the state-wide computerised notifiable diseases database for invasive meningococcal disease; one additional case was ascertained in this way. Information on indigenous status, clinical presentation and mortality for this case was also collected from these sources, and supplemented from hospital medical records.

Bacteriological data and methods

All laboratory data on meningococcal isolates examined by the Public Health Microbiology Laboratory, Queensland Health Scientific Services, Brisbane, were accessed. These data were searched and information on serogroup, serotype, serosubtype and antibiotic sensitivities was

collated. Serogrouping was performed on each isolate using commercially available antisera (Murex Biotech, England). Monoclonal reagents for sero/subtyping were obtained from the National Institute of Public Health and the Environment, Netherlands. Antibiotic sensitivities were determined using an agar plate dilution method as part of the set methodology adopted by the National *Neisseria* Network around Australia.[3]

Analysis

Data were entered into a MS Access database and analysed using MS Excel, the Statistical Package for the Social Sciences (SPSS) and Epi Info (Version 6.04b).

Incidence rates were calculated for each year for defined age groups, and for each Health Service District. Denominator populations were calculated from annual estimated resident populations. To compare risks in different age groups the incidence rate ratio (IRR), the ratio of the incidence in the age cohort of interest to the incidence in the reference age cohort (40+ years) was calculated[4] using Epi Info. The IRR was also calculated for indigenous people relative to non-indigenous people based on 1996 census data.[2]

Results

During the study period there were 120 notifications, 113 confirmed and 7 probable cases. Two notified cases were tourists, both of whom had been in north Queensland for at least 2 weeks before symptom onset, and therefore it was assumed that they had acquired their disease in the region.

The annual incidence of invasive meningococcal disease for the north Queensland zone varied between 2.9 and 5.0 cases per 100,000 population (Table 1). The incidence in 1999 when probable cases first became notifiable was 3.8 cases per 100,000 population for confirmed cases only.

Nearly three quarters of the infections were acquired within the Health Service Districts containing major population centres: Cairns, Townsville and Mackay. There was no significant difference in risk between the different Health Service Districts, except Cape York, which had no cases (Table 2).

Almost half the notified cases were females (58, 48.3%). The largest number of cases and the highest risk occurred in the 0-4 year age group, which had about 15 times the risk of those aged over 40 years (Table 3).

Table 1. Incidence rates for invasive meningococcal disease, north Queensland, 1995 to 1999, by year

Year	Number of cases	Incidence per 100,000 per annum
1995	27	4.9
1996	16	2.9
1997	21	3.7
1998	27	4.7
1999*	29	5.0
Total	**120**	**4.2**

* includes probable as well as confirmed cases. The rate in confirmed cases only was 3.8 cases per 100,000 population.

Twenty-five (20.8%) cases were in Aboriginal or Torres Strait Islander people yielding an incidence of 10.4 cases per 100,000 population. The incidence for non-indigenous people was 3.5 cases per 100,000 population. Indigenous people were three times more likely to develop invasive meningococcal disease than non-indigenous people (IRR = 3.0, 95% CI = 1.9-4.7).

There were 15 cases for which the serogroup was not determined; 7 of these were probable cases. During the study period a single case was diagnosed using PCR. At this time serogrouping using PCR was not being performed. Of the remaining seven, six had Gram-negative diplococci seen in cerebral spinal fluid (CSF), and one had *N. meningitidis* cultured in a district hospital laboratory, but the isolate was neither serogrouped nor forwarded to the reference laboratory. Ninety-two per cent of the 105 isolates that were serogrouped were caused by serogroup B (72.4%, 76) and serogroup C organisms (20.0%, 21). In all 5 years the majority of isolates were serogroup B *N. meningitidis*. There were no serogroup A isolates (Figure 2). Twenty-two isolates from the 25 indigenous cases were serogrouped; the majority (17, 77.3%) were serogroup B and three (13.6%) were serogroup C.

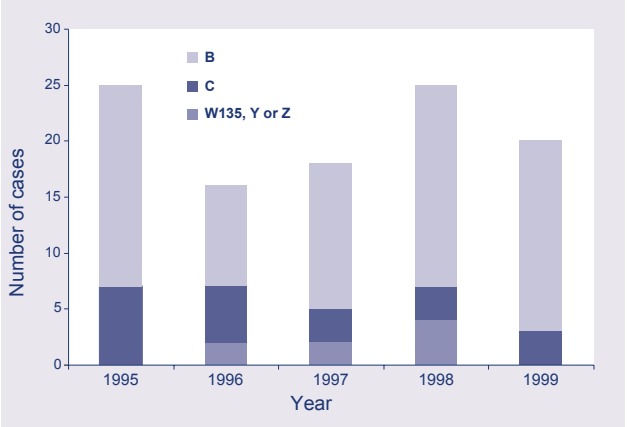
Table 2. Invasive meningococcal disease, north Queensland, 1995 to 1999, by Health Service District of acquisition

Health Service District	Number of cases	Per cent	Incidence per 100,000 per annum
Cairns	37	30.8	5.7
Townsville	28	23.3	3.7
Mackay	23	19.2	4.5
Bowen	7	5.8	4.4
Mount Isa	6	5.0	3.8
Moranbah	5	4.2	4.7
Charters Towers	4	3.3	5.0
Innisfail	4	3.3	2.5
Tablelands	4	3.3	2.2
Torres and northern peninsula area	2	1.7	4.5
Cape York	0	0.0	0.0

Table 3. Invasive meningococcal disease, north Queensland, 1995 to 1999, by age group

Age group	Number of cases	Incidence per 100,000 per annum	IRR and 95% CI	
0-4	48	21.1	15.4	8.5-28.0
5-9	17	7.5	5.5	2.7-11.1
10-14	7	3.2	2.3	0.9-5.8
15-19	14	6.8	4.9	2.4-10.4
20-29	16	3.4	2.5	1.2-5.1
30-39	4	0.8	0.6	0.2-1.9
40+	14	1.4	1.0	(Reference)

Figure 2. Invasive meningococcal disease, north Queensland, 1995 to 1999, by serogroup and year



Full serogroup, serotype and serosubtype could not be determined for all isolates. In part, this was because serotyping and subtyping was not performed during 1995 and 1996, but also because 41 isolates were non-typeable when serotyped and/or serosubtyped. Among the isolates there were three of the phenotype B:4:P1.4, however, these were tested using pulse-field gel electrophoresis, and were not genotypically of the New Zealand epidemic strain.[5] There were no isolates of the 'outbreak' virulent C strain C:2a:P1.5,[6] but there were 3 isolates with the C:2b:P1.2 phenotype,[1] all from non-indigenous cases.

There were 2 clusters in north Queensland during the study period, one each caused by serogroup B and serogroup C organisms. These have been described elsewhere.[7,8] Neither of these clusters occurred in indigenous communities.

There were 7 deaths due to invasive meningococcal disease. Three deaths occurred in patients with meningitis and four in patients with septicaemia. The case fatality rate for disease caused by serogroup C organisms was 9.5 per cent (2/21) and 6.6 per cent (5/76) for cases with serogroup B infections. Only one of the deaths occurred in an indigenous person, a one-year-old male who became unwell in Mt Isa in August 1998. The other 6 deaths were of 3 females aged between 3 months and 18 years, and 3 males aged between two and 20 years. One death occurred in 1995, two in 1998 and four in 1999.

Penicillin sensitivity was available for 83 (69.2%) of the 120 notified cases. None of these were 'relatively resistant' (MIC ≥ 1 mg/L). Nineteen (22.9%) were 'sensitive' (MIC ≤ 0.03 mg/L) and 64 (77.1%) were 'less sensitive' (MIC 0.06-0.5 mg/L).[9] Eighty-three isolates were tested for sensitivity to tetracycline; all were inhibited at 8 μg/ml. These isolates were also all inhibited at 5 μg/ml of spectinomycin, 0.008 μg/ml of ceftriaxone and 0.03 μg/ml of ciprofloxacin. Eighty isolates were tested for their sensitivity to rifampicin. Ten of these were not inhibited at the standard breakpoint of 0.125 μg/ml.

Discussion

This study and the earlier one by Hanna *et al*[1] describe relatively large numbers of cases of a rare disease over a considerable period of observation. Therefore, it is possible to make meaningful comparisons regarding changes in the epidemiology of invasive meningococcal disease in north Queensland over a decade. Two of the major changes observed are:

1. a decline in the proportion of cases caused by serogroup C *N. meningitidis* with a concomitant decline in the number of serogroup C clusters; and
2. a decline in risk for indigenous people in the second relative to the first period, manifest both as a lower IRR and fewer deaths (3.1 versus 12.6 and 1 versus 3, respectively). The differences between 1990 to 1994 and 1995 to 1999 are summarised in Table 4.

In Australia 63 per cent[9] and in Queensland 70 per cent[8] of *N. meningitidis* isolates obtained during 1999 were serogroup B. Our findings on serogroup are therefore consistent with the epidemiology of meningococcal disease in the State and the nation. The relative disappearance of serogroup C *N. meningitidis* is intriguing but fortunate because it resulted in relatively fewer outbreaks.

Considering the poor living conditions for indigenous people in Cape York and the high prevalence of cigarette smoking by indigenous adults in this area (65.2%, Well Person's Health Check, unpublished report, Tropical Public Health Unit, Cairns), the absence of cases of invasive meningococcal disease from Cape York was surprising. However, there was a marked decline in the overall incidence of invasive meningococcal disease in indigenous people in north Queensland in 1995 to 1999 compared to the previous 5 years (Table 4). A large proportion (43.8%) of people living in Cape York in 1996 were Indigenous.[2] Therefore, the causes of the reduced incidence of invasive meningococcal disease in indigenous

Table 4. Invasive meningococcal disease, north Queensland, 1990 to 1994 and 1995 to 1999

Parameter	1990 to 1994	1995 to 1999
Incidence (per 100,000 per annum)	3.3	4.2
Indigenous incidence (per 100,000 per annum)	20.2	10.4
IRR for indigenous people relative to others	12.6	3.0
Serogroup B (%)	26	72
Serogroup C (%)	70	20
Number of clusters	5	2
Number of serogroup C clusters	5	1
Number of serogroup C clusters in indigenous communities	3	0
Case fatality rate (%)	4.3	5.8
Case fatality rate for serogroup C (%)	6.3	9.5

people in north Queensland are likely, at least in part, to account for the absence of cases from Cape York. It should also be noted however, that the population of Cape York Health Service District was only 8,387 (1.4% of the North Queensland Health Zone)[2] and therefore one would not expect many cases from this area. The remoteness of much of the district may lead to under-reporting. No comparison with 1990 to 1994 is possible as cases were not reported by the Health Service District in the earlier study.[1]

Demographic changes are unlikely to account for the observed change in indigenous incidence. The 1991 census recorded only 7.5 per cent of the population in the North Queensland Health Zone as indigenous people (Fiona Tulip, data manager, Tropical Public Health Unit, Cairns, personal communication, 2001). In the 1996 census it was 8.1 per cent.[2] The major cause for the decline in the indigenous incidence is likely to be the decline in serogroup C disease, due to the decline or disappearance of clones that have a propensity to cause outbreaks. In the period 1990 to 1994, three of 5 outbreaks involved indigenous people, and two of these outbreaks involved C:2b:P1.2 *N. meningitidis*. These 2 outbreaks accounted for 13 (33.3%) of the 39 indigenous cases of invasive meningococcal disease.[1] By contrast, in 1995 to 1999 only 3 cases involved C:2b:P1.2 *N. meningitidis*, and none of these were in indigenous people. Of 22 people involved in 5 outbreaks caused by serogroup C organisms during 1990 to 1994, 16 (72.7%) were Indigenous. During 1995 to 1999, 2 outbreaks (one serogroup B and serogroup C) involved 6 people,[7,8] none of whom were Indigenous.

The incidence rate for invasive meningococcal disease in north Queensland during 1995 to 1999 (4.2 per 100,000 per annum) was higher than that reported for Sydney (2.3 per 100,000 per annum)[10] and other New South Wales areas (2.8 per 100,000 per annum)[10] in 1991 to 1999, and for Queensland during 1999 (2.8 per 100,000 per annum).[8] Indeed, for 1999 the incidence rate for north Queensland (5.0 per 100,000 per annum) was much higher than that for Queensland, and the rates both include probable cases and hence can be compared.

During the course of this study Queensland Health was using monoclonal antibodies to perform serosubyping and this resulted in many isolates being only partially typeable or non-typeable. This has also been seen by other researchers.[11] Queensland Health is currently introducing molecular serosubtyping, and this will reduce the non-typeable rate.

The prevalence of reduced sensitivity to penicillin (77%) was similar to that for Australia as a whole (74%) in 1999.[9] No north Queensland isolates were resistant (MIC ≥ 1 mg/L) to penicillin. All 83 isolates were fully sensitive to ceftriaxone and ciprofloxacin. Only one isolate had a raised MIC to rifampicin (MIC ≥ 1 mg/L). Our findings on

sensitivity to penicillin and ceftriaxone mean that current recommendations on treatment (benzylpenicillin with ceftriaxone or cefotaxime initially for bacterial meningitis, benzylpenicillin once penicillin sensitive *N. menigitidis* is isolated, ceftriaxone or cefotaxime if the patient is allergic to penicillin)[12] remain appropriate for north Queensland. The rarity of significant resistance to rifampicin and universal sensitivity to ceftriaxone and ciprofloxacin means that these antibiotics can all be safely recommended for chemoprophylaxis in north Queensland, as in current guidelines.[12]

Acknowledgements

Tropical Public Health Unit Network nurses are thanked for their contribution to data collection. Fiona Tulip is thanked for assistance with data entry.

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Farewell to Angela Merianos

It is with regret that we farewell Dr Angela Merianos as Editor of *Communicable Diseases Intelligence (CDI)*. Angela has been Editor since February 2000 and this era has seen a number of notable developments for *CDI*, including a change from monthly to a quarterly publication and strengthening of the format and associated production quality resulting in a journal of the highest standard.

Angela also leaves her role as Director of the Surveillance and Epidemiology Section and Medical Adviser for the Communicable Diseases and Health Protection Branch of the Commonwealth Department of Health and Ageing. She has also been the Commonwealth representative to the Communicable Disease Network Australia. Her contribution and dedication will be greatly missed.

Dr Jenean Spencer will take up the role as head of the Surveillance and Epidemiology Section and Editor of *CDI*.

We thank Angela for all her contributions and wish her well for the future.

Rising prevalence of genital *Chlamydia trachomatis* infection in heterosexual patients at the Sydney Sexual Health Centre, 1994 to 2000

Basil Donovan[1,2]

Abstract

This study sought to investigate trends in the prevalence of genital *Chlamydia trachomatis* infection in heterosexual patients attending an urban sexual health service. Data from cases of *C. trachomatis* in all new self-referred heterosexual patients who were tested at the Sydney Sexual Health Centre from 1994 to 2000 were extracted from the Centre's database. Female sex workers and homosexually active men were excluded. Over the study period the prevalence of *C. trachomatis* infection doubled from 1.8 per cent to 3.5 per cent among the women (p=0.004) and tripled from 2.1 per cent to 6.6 per cent among the men (p<0.001) who were tested. Both men and women reported an increasing overall trend in the mean (but not median) number of sexual partners during the previous 3 months (p=0.039 and p=0.001, respectively). There were modest increases in the proportion of men and women that reported unprotected vaginal or anal sex in the previous 3 months, from 76.5 per cent to 81.7 per cent for males (p=0.122) and from 65.1 per cent to 70.2 per cent (p=0.01) for females. The introduction of more sensitive DNA-based testing probably only accounted for 8 per cent of the rise in prevalence among women and 16 per cent among men. These findings complement the rising trends in national notifications of *C. trachomatis* infection. Further investigation and interventions on a national scale to reduce the prevalence of *C. trachomatis* seem timely. Commun Dis Intell 2002;26:51-54.

Keywords: Chlamydia trachomatis; *heterosexual; genital infection*

Introduction

Chlamydia trachomatis infection is the most common bacterial sexually transmissible infection (STI) in the world. Often under-estimated because of its clinical subtlety, it is the leading cause of pelvic infections, preventable infertility and ectopic pregnancy in wealthier nations. In men, *C. trachomatis* is a common cause of frequently asymptomatic urethritis, which may lead to epididymitis.[1]

Notifications of *C. trachomatis* infection in Australia more than doubled between 1995 (36 cases per 100,000 population) and 2000 (91 cases per 100,000 population)[2] making it the most commonly reported bacterial infection. The extent to which this rise in notifications represents a real increase in incidence and prevalence, the introduction of easier and more sensitive DNA-based tests, or improved surveillance, is unknown. New South Wales was the last jurisdiction to begin contributing *C. trachomatis* notifications to the National Notifiable Diseases Surveillance System (end of 1998). The reporting rate for New South Wales (57 cases per 100,000 population) is lower than the overall national rate.

Specialist sexual health services offer the potential to contribute longitudinal sentinel site data to complement population-based surveillance.[3] These services can provide greater patient demographic and risk behaviour detail, have consistent STI screening practices and the testing technologies that they use are known. The Sydney Sexual Health Centre (SSHC) is based at Sydney Hospital in the Central Business District. Because of its proximity to public transport and to where many people work or socialise, the Centre's patients are drawn from throughout metropolitan Sydney. This study sought to investigate trends in *C. trachomatis* infection among heterosexuals attending SSHC between 1994 and 2000.

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Methods

Study population

All patients who attended SSHC for the first time between January 1994 and December 2000 were eligible for the study with the following exclusions:

- patients not tested for *C. trachomatis* at SSHC (consistently in the range 15-20% during the study period), because of prior testing or treatment elsewhere; because they were at negligible risk and presented for reasons such as counselling, contraception or cervical cytology; or because they declined the offer of testing;
- patients referred by other doctors, because of the unpredictable effects of concentrating patients with known infections and of previous screening or treatment for *C. trachomatis*;
- men who reported having sex with other men in the previous 12 months, because this group have sexual networks that differ from the bulk of the community so they are the subject of a separate study;
- women who reported that they were currently engaged in sex work, because they were a mixture of heavily screened local sex workers (diminishing the prevalence) and women who may have arrived from overseas with infections acquired in high prevalence countries (inflating the prevalence).[4]

Thus the study population comprised a mixture of all symptomatic and asymptomatic heterosexuals who had referred themselves to an inner city sexual health service.

Data collection

Patients attending SSHC have their demographics, medical history, sexual behaviour, drug use, tests performed, and diagnoses, routinely recorded on a proforma medical record which is then transferred to a database after quality checking.

Laboratory tests

Male patients between 1994 and November 1996 provided a first-void urine which was tested for *C. trachomatis* by enzyme immunoassay. Thereafter, polymerase chain reaction (PCR; Amplicor, Roche) testing of urine was used. For women, cell culture of an endocervical swab was used to the end of December 1995: thereafter all swabs were tested by in-house PCR at the South Eastern Area Laboratory Service at the Prince of Wales Hospital.

Analysis

The Mantel-Haenszel chi-square test for trend was used to compare proportions of positive tests for *C. trachomatis* year-by-year over the study period, with a SPSS package.

Results

Between 1994 and 2000, 14,020 self-referred, heterosexual, non-sex working, new patients were tested for *C. trachomatis* at SSHC. While there were some year-to-year fluctuations there was no overall trend in the age of female or male patients, nor in the likelihood that they presented with anogenital symptoms (Table). The symptoms were not necessarily due to *C. trachomatis* infection.

The frequency distribution of the number of sexual partners is shown in Figure 1. There was no change in the median (1) number of sexual partners reported by women in the previous 3 months though there was a marginal increase in the mean between 1994 and 2000, from 1.23 to 1.35 (p=0.039). The proportion reporting unprotected vaginal or anal sex increased slightly from 76.5 per cent to 81.7 per cent (p=0.122), while the small proportion that presented as contacts of *C. trachomatis* or non-gonococcal urethritis (NGU) did not vary (p=0.909) (Table).

For men, there was no change in the median (1) number of sexual partners in the previous 3 months but a significant increase in the mean over the study period from 1.65 to 1.79, p=0.001. There were also modest increases in the proportions who reported unsafe sex (from 65.1% to 70.2%, p=0.010), and the proportion who presented because of known contact with *C. trachomatis* (p=0.006) (Table).

During the study period there were 357 diagnoses of *C. trachomatis* in men and 157 diagnoses in women. The percentage yield of positive *C. trachomatis* tests increased among women from 18 of 997 tests (1.8%) in 1994 to 28 of 800 tests (3.5%) in 2000 (p=0.004). Among men the positive yield rose from 36 of 1,685 tests (2.1%) in 1994 to 74 of 1,114 tests (6.6%) in 2000 (p<0.001) (Figure 2).

Making the assumption that the real prevalence of *C. trachomatis* did not rise in the first year that PCR testing was introduced, then the additional yield over culture would have been 8 per cent (from 2.2% in 1995 to 2.4% in 1996 among women) and a 16 per cent additional yield over enzyme immunoassay (from 3.6% in 1996 to 4.3% in 1997 among men).

Table **Self-referred heterosexual patients tested for *Chlamydia trachomatis* at the Sydney Sexual Health Centre, 1994 to 2000, by sex, sexual behaviour and reason for presentation**

	Women						
	1994	1995	1996	1997	1998	1999	2000
Median age in years	26	25	25	26	26	25	25
Median/mean number of sexual partners in the last 3 months*	1/1.23	1/1.21	1/1.29	1/1.30	1/1.30	1/1.28	1/1.35
Range	0-10	0-7	0-7	0-7	0-11	0-7	0-20
Per cent reporting any unprotected vaginal or anal sex in last 3 months	76.5	81.4	78.4	79.0	77.9	79.3	81.7
Per cent presenting as NGU or chlamydia contacts	6.1	5.8	5.4	6.6	5.3	6.3	6.4
Per cent presenting with genital or anal symptoms	NA	NA	43.0	38.0	47.1	44.1	39.6

	Men						
	1994	1995	1996	1997	1998	1999	2000
Median age in years	29	29	29	30	29	30	28
Median/mean number of sexual partners in the last 3 months*	1/1.65	1/1.60	1/1.75	1/1.88	1/1.83	1/1.92	1/1.79
Range	0-36	0-20	0-20	0-40	0-30	0-40	0-41
Per cent reporting any unprotected vaginal or anal sex in last 3 months	74.6	78.6	76.2	74.5	76.0	76.1	80.3
Per cent presenting as chlamydia contacts	1.3	2.1	2.6	1.9	2.7	3.8	2.3
Per cent presenting with genital or anal symptoms	NA	NA	55.1	54.0	56.9	54.9	52.8

* 3 outliers (100, 120 and 150 partners) excluded.

NGU non-gonococcal urethritis

NA = not available

Figure 1. The frequency distribution of the number of sexual partners of self-referred heterosexual patients at the Sydney Sexual Health Centre, 1994 to 2000, by gender

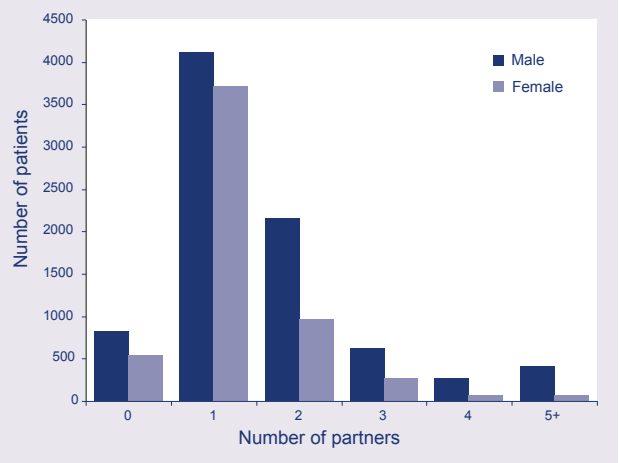
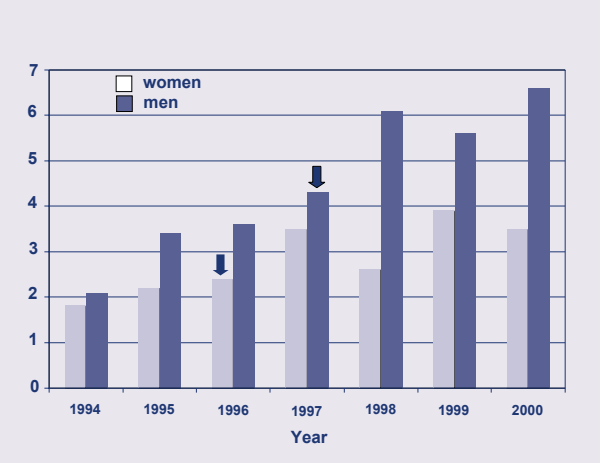


Figure 2. Prevalence of *Chlamydia trachomatis* in self-referred heterosexual patients at the Sydney Sexual Health Centre, 1994 to 2000, by gender



* Arrows signify the first full year of polymerase chain reaction testing for each gender

Discussion

In this carefully selected study population of sexually active heterosexuals attending a sentinel site in Sydney, the prevalence of *C. trachomatis* almost doubled in women and tripled in men between 1994 and 2000. Up to 16 per cent of the increase in men and 8 per cent of the increase in women could be explained by the introduction of more sensitive DNA-based testing, which is consistent with our understanding of the increased sensitivity of DNA-based tests for *C. trachomatis*.[5] Thus most of the increase was attributable to a rising prevalence of *C. trachomatis* infection in the population sampled.

The basis for the increasing prevalence of *C. trachomatis* in this clinical population is unclear and could not be explained by age variation or increasing symptoms. Over the study period both women and men reported modest increases in the mean number of sexual partners and in the likelihood that they reported unprotected sex in the previous 3 months. Whether these sexual behaviour changes reflected trends in the community or just this self-selected population is unknown. Nevertheless, rising STI notifications in Britain have been temporally associated with increasing risk in repeated population-based sexual behaviour surveys.[6]

While sentinel site data have limitations,[3] in the case of STIs the experience of large sexual health services generally reflects what is happening in the community provided appropriate sub-populations are selected.[6,7] In this case the findings were consistent with the increase in national population-based notifications of *C. trachomatis*, including an increasing proportion of diagnoses being made in men.[2]

The major difference between sexual health service and population-based data is that *C. trachomatis* diagnoses are at least as common in men as in women in the former while the male:female ratio in the national reports is around 1:1.5.[2] In part, the prevalence among men would be expected to be higher in the clinic setting because men are more likely to develop symptoms and thus selectively seek health care. However, the difference is also attributable to equal testing rates for men and women in sexual health clinics but much more frequent testing of women than men elsewhere.[7,8] In this respect sexual health service data may better reflect the distribution of *C. trachomatis* in the community – most of which probably remains undiagnosed.[6,9] Consideration should be given to including high risk male patients in any campaign to encourage more testing for *C. trachomatis* by general practitioners.

Further investigation into the underlying causes of the increasing prevalence of *C. trachomatis* and interventions on a national scale to reduce the prevalence seem timely.

Acknowledgement

Thanks to Richard Rohrsheim for data management and to Virginia and Wynne-Markham for preparation of data.

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Public Health Laboratory Network (PHLN)

The Public Health Laboratory Network (PHLN) is a collaborative group of laboratories nominated by State and Territory health departments, which have expertise and provide services in public health microbiology. National bodies involved in animal health and epidemiology of communicable diseases are also represented, as is the major public health laboratory in New Zealand. Participating laboratories and organisations are listed at the end of this article.

The PHLN was established in 1997 as part of the implementation of the National Communicable Diseases Surveillance Strategy (NCDSS) to complement the activities of the then Communicable Diseases Network Australia New Zealand, now the Communicable Disease Network Australia (CDNA). PHLN aims to provide a national source of expertise in the laboratory aspects of communicable disease surveillance and control, and to take an active role in strategic planning in these areas.

Professor Lyn Gilbert was the inaugural chair of the PHLN, and was succeeded by Dr David Smith in 2001, with Dr Gary Lum as Deputy Chair. The group meets at monthly teleconferences supplemented by annual face-to-face planning meetings. Various formal and informal working groups hold teleconference and exchange information as required between the full PHLN meetings.

During its first 4 years, PHLN has been very active in providing strategic advice to CDNA and the Commonwealth Department of Health and Ageing on a coordinated national approach to public health microbiology for surveillance and control of communicable diseases. In addition it has commented on public health microbiology to a wide range of other organisations, usually following an invitation from that organisation. However, all matters relevant to public health microbiology are considered, and PHLN has been prepared to initiate input where appropriate.

One of the major undertakings has been to assist the CDNA and Department of Health and Ageing to improve the quality and timeliness of laboratory based communicable disease surveillance to introduce laboratory surveillance standards. The PHLN has now produced a set of Laboratory Case Definitions. They are designed to provide nationally consistent criteria for significant laboratory results and cover all of the current notifiable diseases. They have also been used by CDNA as part of the process of developing national case definitions for notifiable diseases. PHLN is providing ongoing input with the aim of creating complementary laboratory and public health definitions. In addition, more detailed case definitions are being produced for each of the notifiable infections that provide an up-to-date summary of testing methods and performance. PHLN is collaborating in a review of the Serology and Virology Laboratory Reporting Scheme (LabVISE) reporting system, with the aim of producing a suitable national laboratory basis for non notifiables communicable disease surveillance.

PHLN has provided a laboratory perspective on a wide range of other microbiological and public health issues, including laboratory testing, surveillance, specimen transport and financing of public health testing. Members of the PHLN have been instrumental in providing information for the current review *Financing of Public Health Laboratory Services* commissioned by the National Public Health Partnership (NPHP). It is also represented on committees involved in the review of regulations related to the transport of infectious materials, influenza pandemic planning, hepatitis C surveillance, pneumococcal surveillance, nucleic acid testing for sexually transmitted infections and many others. More recently a Laboratory Infection Containment Working Party has been formed at the request of the National Public Health Partnership to provide advice on guidance and containment of pathogenic organisms.

The PHLN also acts as a first point of contact to identify individuals or laboratories with appropriate expertise for unusual outbreak investigations. In the recent past this was effectively applied to the white powder incidents following the anthrax incidents in the USA in October 2001. The PHLN was able to exchange information and rapidly develop protocols for handling suspect materials. This is being progressed further to develop plans for the handling of future bioterrorist threats. Members have previously been involved in establishing plans for the Sydney Olympics.

PHLN has taken an active role in a number of areas related to the implementation of quality assurance (QA) measures for laboratories. It was represented on the working group of the National Pathology Accreditation Advisory Committee to develop standards for laboratories undertaking nucleic acid detection tests, and has collaborated in serology QA exercises. There has also been an active interest in proposals for regulation of laboratory tests being developed by the Therapeutic Goods Administration. PHLN has acted with the Royal College of Pathologists of Australasia and other organisations to represent and promote the interests of laboratories in the process, and now sits on an Australian Health Ministers Advisory Committee (AHMAC) working group that is considering the role and impact of these proposed regulations.

In addition to the PHLN Summary Laboratory Definitions mentioned above, PHLN has also produced a document on *Laboratory Precautions for Samples Collected from Patients with Suspected Viral Haemorrhagic Fevers*. It was recognised that most suspected cases of viral haemorrhagic fever will be managed in hospitals without very high-level containment facilities in their laboratory, but there were no guidelines available to assist with processing of these specimens under such conditions. These were written to address that deficiency.

This article has provided a brief overview of the history and range of activities of the PHLN. It has been a valuable addition to the Australian scene and has provided a mechanism for consensus on a wide range of issues related to the laboratory aspects of public health. Members of PHLN also participate in a variety of other public health related activities at the local, national and international level. Through PHLN they are able to effectively provide a national perspective on issues discussed and to disseminate information from these other sources. We look forward to continuing and expanding our activities in the future.

Contact details

For further information on the Public Health Laboratory Network including Public Health Laboratory Network publications contact:

Secretariat, Public Health Laboratory Network, MDP6, GPO Box 9848, Canberra, Australian Capital Territory, 2601. Telephone: +61 2 6289 7401. Facsimile +61 2 6289 7791. E-mail: PHLN@health.gov.au.

You may also contact one of the participating laboratories if you want matters raised at PHLN.

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Surveillance systems reported in *CDI*, 2002

Surveillance has been defined by the World Health Organization as the 'continuing scrutiny of all aspects of the occurrence and spread of disease that are pertinent to effective control'. It is characterised by 'methods distinguished by their practicability, uniformity, and frequently by their rapidity, rather than complete accuracy.'[1] Although some surveillance schemes aim for complete case ascertainment, others include only a sample of all cases of the conditions under surveillance, and these samples are subject to systematic and other biases. Results generated from surveillance schemes must be interpreted with caution, particularly when comparing results between schemes, between different geographical areas or jurisdictions and over time. Surveillance data may also differ from data on communicable diseases gathered in other settings.

In Australia, communicable diseases surveillance systems exist at national, state and local levels. State and local surveillance systems are crucial to the timely and effective detection and management of outbreaks and in assisting in the effective implementation of national policies. The national surveillance system combines some of the data collected from State and Territory-based systems to provide an overview at a national level. Specific functions of the national surveillance system include: detection and management of outbreaks affecting more than one jurisdiction; monitoring of the need for and impact of national control programs; guidance of national policy development; and resource allocation and description of the epidemiology of rare diseases for which there are only a few notifications in each jurisdiction. National surveillance also assists in quarantine activities and facilitates agreed international collaborations such as reporting to the World Health Organization.

This article describes the surveillance schemes that are routinely reported on in *Communicable Diseases Intelligence (CDI)*.

The major features of the surveillance schemes for which *CDI* publishes regular reports are described below. Other surveillance schemes for which *CDI* publishes occasional reports include the National Mycobacterial Surveillance System (*Commun Dis Intell* 2001;25:254-260), the Australian Mycobacterium Reference Laboratory Network (*Commun Dis Intell* 2001;25:261-265), and the National Neisseria Network (*Commun Dis Intell* 2001;25:54-58).

National Notifiable Diseases Surveillance System

National compilations of notifiable diseases have been published intermittently in a number of publications since 1917.[2] The National Notifiable Diseases Surveillance System (NNDSS) was established in 1990 under the auspices of the Communicable Diseases Network Australia (CDNA).

The system coordinates the national surveillance of more than 50 communicable diseases or disease groups endorsed by the CDNA. Under this scheme, notifications are made to the State or Territory health authority under the provisions of the public health legislation in their jurisdiction. Computerised, de-identified unit records of notifications are supplied to the Department of Health and Ageing for collation, analysis and publication in *CDI*.

Data provided for each notification include a unique record reference number, State or Territory, disease code, date of onset, date of notification to the relevant health authority, sex, age, Aboriginality, postcode of residence, and the confirmation status of the report (as defined by each State or Territory).

From 2002 additional data are being collected on the infecting organism and subtype, the diagnosis method, full details of vaccination where appropriate, resident location as defined in the National Localities Index, dates of onset, specimen collection, notification and date when notification was received by health authorities, indigenous status defined as per the ABS format, outbreak reference number, how the case was found, whether the case was confirmed, and whether the case was imported from overseas.

The data are presented on the *Communicable Diseases - Australia* Internet site each fortnight. They are also published in *CDI* every quarter. Cases reported to State and Territory health authorities for the current reporting period are listed by State or Territory, and totals for Australia are presented for the current period, the year to date, and for the corresponding periods of the previous year. HIV infection and AIDS notifications are not included in this section of *CDI*. Surveillance for these conditions is conducted separately by the National Centre for HIV Epidemiology and Clinical Research and is reported in the HIV and AIDS surveillance reports (see below).

A commentary on the notification data is included with the tables in each issue and graphs are used to illustrate important aspects of the data.

Australian Sentinel Practice Research Network

The Research and Health Promotion Unit of the Royal Australian College of General Practitioners operates the Australian Sentinel Practice Research Network (ASPREN). ASPREN is a national network of general practitioners who report presentations of defined medical conditions each week. The aim of ASPREN is to provide an indicator of the burden of disease in the primary health care setting and to detect trends in consultation rates.

There are currently about 66 general practitioners participating in the network from All States and Territories. Seventy-five per cent of these are in metropolitan areas and the remainder are rural based. Between 4,000 and 6,000 consultations are recorded each week.

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published.

In 2002, 10 conditions are being monitored, six of are related to communicable diseases issues. These include influenza, gastroenteritis, acute cough in four sub-categories: with chest and systemic signs; with chest signs; with systemic signs; without signs. The other recordable conditions are treated hypertension at target levels; treated hypertension above target levels; treated hyperlipidaemia at target levels and treated hyperlipidaemia at above target levels.

Data for communicable diseases are published in CDI every quarter. Data are presented in tabular form together with the rate of reporting per 1,000 consultations. The conditions are defined as follows:

Influenza

(a) Viral culture or serological evidence of influenza virus infection; or

(b) influenza epidemic, plus four of the criteria in (c); or

(c) six of the following:

1. sudden onset (within 12 hours);
2. cough;
3. rigour or chills;
4. fever;
5. prostration and weakness;
6. myalgia, widespread aches and pains;
7. no significant respiratory physical signs other than redness of nasal mucous membrane and throat;
8. influenza in close contacts.

Gastroenteritis

Intestinal disease presumed or proven to be infective in origin. A stool sample is not carried out and one episode only is recorded per patient.

Acute cough

Reports of acute cough defined below, are classified into 4 sub-categories according to whether chest or systemic signs are present or absent.

Includes any patient 2 years or older, who presents with acute cough of less than 14 days duration and at least one other symptom of a respiratory infection, such as symptoms of URTI, sore throat, sputum production, dyspnoa, wheeze or chest pain for which there is no other explanation.

Excludes patients that have a history of chronic respiratory illness that requires ongoing treatment, such as COPD, bronchiectasis or asthma.

Chest signs are focal or generalised signs such as crepitations, crackles, coarse breath sounds or wheezes in non-asthmatics. Excludes patients with signs of consolidation (pneumonia).

Systemic signs are:

Adult or child >12 years	**Child 2-12 years**
Temperature >38˚C	Temperature >38˚C
Respiratory rate >20	Respiratory rate >30; or
Pulse rate >100, or	Pulse rate >110
Being confined to bed	

Acute cough with chest and systemic signs

Acute cough defined as above with one or more chest signs and one or more systemic signs. Each episode is recorded.

Acute cough with chest signs

Acute cough as defined above with one or more chest signs but no systemic signs. Each episode is recorded.

Acute cough with systemic signs

Acute cough as defined above with one or more systemic signs but no chest signs. Each episode is recorded.

Acute cough without signs

Acute cough as defined above but without any chest or systemic signs.

HIV and AIDS surveillance

National surveillance for HIV and AIDS is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR) within the University of New South Wales, in collaboration with State and Territory health authorities and the Commonwealth of Australia.

Cases of HIV infection are notified to the National HIV Database on the first occasion of diagnosis in Australia, either by the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania and Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia and Western Australia). Cases of AIDS are notified through the State and Territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Currently, two tables presenting HIV infection diagnoses, AIDS diagnoses and AIDS deaths are published in each issue of *CDI* when available.

Tabulations of diagnoses of HIV infection and AIDS are based on data available 3 months after the end of the reporting period, to allow for reporting delay and to incorporate newly available information.

Each year from 1997, the NCHECR has published *HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia annual surveillance report.* The annual surveillance report, available through www.med.unsw.edu.au/nchecr, provides a comprehensive analysis and interpretation of surveillance data on HIV/AIDS, viral hepatitis and sexually transmissible infection in Australia.

National Influenza Surveillance Scheme

Influenza surveillance in Australia is based on several schemes collecting a range of data that can be used to measure influenza activity. In 2001, four sentinel general practitioner schemes contributed reports of influenza-like illness: the Australian Sentinel Practice Research Network, Tropical Influenza Surveillance from the Northern Territory, the New South Wales Sentinel General Practice Scheme and the Victorian Sentinel General Practice Scheme. The Virology and Serology Laboratory Reporting Scheme (LabVISE) contributes laboratory reports of influenza diagnoses including virus type. From autumn to spring, the results of each of the schemes are

published together fortnightly on the Communicable Disease Australia Website as the National Influenza Surveillance Scheme.

Annual reports on influenza in Australia are published in CDI each year (Commun Dis Intell 25:107-112). These reports include the above data as well as absenteeism data from a major national employer and influenza typing data from the WHO Collaborating Centre for Influenza Reference and Research.

Sentinel Chicken Surveillance Programme

The Sentinel Chicken Surveillance Programme is used to provide an early warning of increased flavivirus activity in Australia. The main viruses of concern are Murray Valley encephalitis (MVE) and Kunjin. 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 parts of the north-east Kimberley region of Western Australia and the Top End of the Northern Territory but are epizootic in other areas of the Kimberley, Pilbara, Gascoyne and Mid-west regions of Western Australia, in north Queensland and in Central Australia. MVE virus is also responsible for occasional severe epidemics of encephalitis in eastern Australia. The most recent was in 1974 when there were 13 fatalities and cases were reported from all mainland States. Since then, 68 cases of MVE have been reported, 61 from the north of Australia and seven from central Australia. In addition, one case of encephalitis caused by MVE and/or Kunjin virus(es) was reported from the north of South Australia in 2000.

Since 1974, a number of sentinel chicken flocks have been established in Australia to provide an early warning of increased MVE virus activity. These programs are supported by individual State health departments. Each State has a contingency plan which will be implemented if one or more chickens in a flock seroconverts to MVE virus.

Currently, 29 flocks are maintained in the north of Western Australia, 9 in the Northern Territory, 10 in New South Wales and 10 in Victoria (Figures 1,2, 3 and 4). Two additional flocks will be set up in northern Queensland (at Mt Isa and Normanton) early in 2002. The flocks in Western Australia and the Northern Territory are tested all year round but those in New South Wales, Victoria and Queensland are tested only in the summer months, during the main MVE risk season. Results are coordinated by the Arbovirus Laboratory in Perth and reported bimonthly.

Figure 1. Sentinel chicken flocks in Western Australia

Figure 2. Sentinel chicken flocks in the Northern Territory

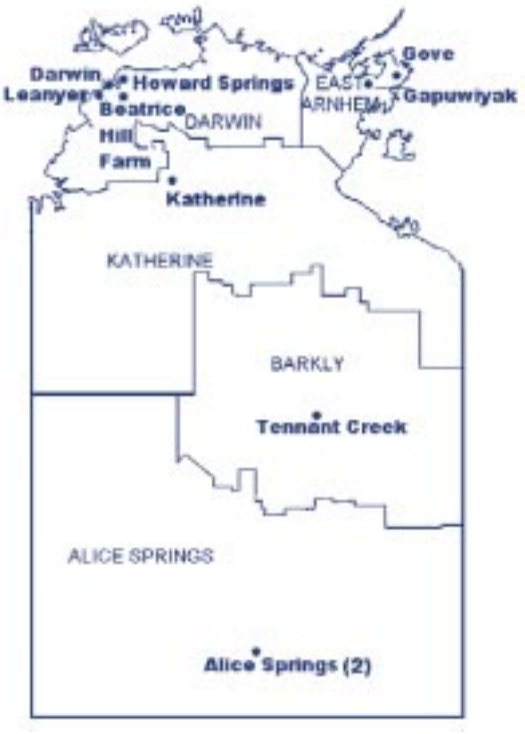

Figure 3. Sentinel chicken flocks in New South Wales

Figure 4. Sentinel chicken flocks in Victoria

Australian Gonococcal Surveillance Programme

The Australian Gonococcal Surveillance Programme (AGSP) includes 10 reference laboratories in all States and Territories and in New Zealand. These laboratories report data on sensitivity to an agreed core group of antimicrobial agents on a quarterly basis. The antibiotics which are currently routinely surveyed are the penicillins, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens. When *in vitro* resistance to a recommended agent is demonstrated in 5 per cent or more of isolates, it is usual to reconsider the inclusion of that agent in current treatment schedules. Additional data are also provided on other antibiotics from time to time. At present all laboratories also intermittently test isolates for the presence of high level resistance to the tetracyclines and azithromycin. Comparability of data is achieved by means of a standardised system of testing and a program-specific quality assurance process. Expanded annual reports are published in *CDI (Commun Dis Intell* 2001;25:59).

Virology and Serology Laboratory Reporting Scheme (LabVISE)

The Virology and Serology Laboratory Reporting Scheme began operating in 1977. The scheme comprises 18 laboratories from all States and the Australian Capital Territory. Contributors submit data on the laboratory identification of viruses and other organisms. Each record includes mandatory data fields (laboratory, specimen collection date, a patient identifier code, and organism), and optional fields (patient's sex, date of birth or age, postcode of residence, specimen source, clinical diagnosis, and the method of diagnosis).

Reports are collated, analysed and published quarterly. Each report includes 2 summary tables. The delay between date of specimen collection and date of publication ranges from 2 weeks to several months. A commentary on the laboratory reports includes the observation of recent trends with accompanying graphical presentation.

Data derived from this scheme must be interpreted with caution. The number and type of reports received is subject to a number of biases. These include the number of participating laboratories, which has varied over time. The locations of participating laboratories also create bias, as some jurisdictions are better represented than others. Also changes in diagnostic practices, particularly the introduction of new testing methodologies, may affect laboratory reports. The ability of laboratory tests to distinguish acute from chronic or past infection must also be considered in interpretation of the data. Although changes in incidence cannot be determined from this data, general trends can be observed, for example with respect to seasonality and the age-sex distribution of patients.

References

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CDI Instructions for authors

Communicable Diseases Intelligence (*CDI*) is a quarterly joint publication of the Surveillance and Epidemiology Section, Communicable Diseases and Health Protection Branch, Commonwealth Department of Health and Ageing and the Communicable Diseases Network Australia. Its aim is to provide timely information about communicable diseases in Australia to those with responsibility for their control. *CDI* has a particular emphasis on public health issues.

CDI invites contributions dealing with any aspect of communicable disease epidemiology, surveillance or prevention and control in Australia. Submissions can be in the form of original articles, short reports, surveillance summaries, reviews or correspondence.

The approximate publication schedule for *CDI* is February, May, September and November. It is finalised for publication at the start of the publication month. Very topical brief contributions (for example reports of current outbreaks) may be published in the publication month, by arrangement with the editorial staff.

Submission procedure

Manuscripts submitted to the CDI for peer review must be offered exclusively to the Journal.

Paper submission

Two copies of the manuscript should be submitted (one complete and one for the reviewers without author(s)' names, affiliations or acknowledgements), printed with double-spacing.

Electronic submission

Manuscripts may also be submitted by e-mail. For e-mail submissions no hard copies are required.

Submission addresses and contact details

Contributions and requests for further information should be sent to:

The Editor
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Manuscript

Articles and short reports

The text of articles and short reports should be structured as far as is possible to contain: abstract; introduction; methods; results; discussion; acknowledgments; and references. Structured abstracts are not acceptable. Short contributions may need fewer subsections. Manuscripts of 2,000 words or less are preferred for articles and 1,000 words or less for short reports. Include a separate word count of the main text and of the abstract on the title page.

The title page should also include, for each author:

- full name, including middle initial;
- title and address of position held when the article was produced; and
- current postal address, direct telephone number, facsimile number and e-mail address.
- Identify one author as correspondent.

The covering letter should include:

- signatures of all authors; and
- confirmation that the manuscript content (in part or in full) has not been submitted or published elsewhere.

Articles or reports which describe an outbreak investigation that involved the issue of a media alert or was listed on an electronic Mail list server such as PROMED, should include a reference to these documents.

Copyright

All authors are asked to transfer copyright to the Commonwealth before publication.

Authors

Authorship should be based on substantial contribution to the article; each author should have participated sufficiently to take public responsibility for the article. Others contributing to the work should be recognised in the acknowledgments.

Style

Use abbreviations sparingly (spell out the first use). Reference and footnote numbers should be after any punctuation marks.

Tables

Submit all tables on separate pages; simplify the information as much as possible, keeping the number of columns to a minimum and the headings short. Information in tables should not be duplicated in the text.

Tables are to be submitted without borders, blank rows or blank columns for spacing. Avoid using paragraph returns if possible, using rows or columns to separate data instead. Use separate columns for each information type; e.g. percentage and number should be in separate columns rather than having one in parentheses in the same column.

Figures and illustrations

Supply a copy of all figures on a separate page, labelled on the back with the figure number and title. Histograms and graphs should be produced in Microsoft Excel and created on a separate worksheet. The numerical data on which these are based should also be provided to enable editing for in-house style. Worksheets should be appropriately titled to distinguish each figure. Do not include the graph heading on the Excel worksheet.

All other figures should be provided in an appropriate graphic format (see electronic copies). Do not embed figures or graphs in the manuscript text document.

All table and figure headings should be provided in the manuscript at the end of the text. All tables and figures should be referred to within the result section and should not duplicate information in the text.

References

References should be identified consecutively in the text by the use of superscript numbers without brackets. Do not use automatic referencing or footnotes.

Accuracy of references is the responsibility of authors. Use the Vancouver reference style (see International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. *Ann Intern Med* 1997; 1126:36-47) and abbreviate journal names as in Medline. Give surnames and initials of all authors (or only the first five authors (et al) if there are more than six). Cite first and last page numbers, and specify the type of reference (eg, a letter, an editorial, an abstract, a supplement). Cite personal communications and unpublished papers in the text, not in the reference list, with the exception of material that has been accepted for publication (in press). Obtain written permission from people cited, and give their titles, positions and affiliations.

Protection of patients' rights to privacy

Identifying details about patients should be omitted if they are not essential, but data should never be altered or falsified in an attempt to attain anonymity.

Ethics approval

All investigations on human subjects must include a statement that the subjects gave their written informed consent, unless data collection was covered by public health legislation or similar studies have been considered by a relevant ethics committee and a decision made that its approval was not required. The name of the ethics committee that gave approval for the study should be included in the text. Alternatively, if approval is not required a statement to this effect should also appear in the manuscript. When informed consent has been obtained it should be included in the article.

Ethical approval may also be required for case reports.

Electronic copies

Authors are asked to provide an electronic copy by e-mail or on a computer disk (on 3.5 inch diskette). Microsoft Word for Windows 97 (or earlier version) is preferred, or alternatively Rich Text Format (RTF) files should be used. Arial font is preferred, and if not available; Times New Roman. Do not use headers or footers, or automatic referencing for bullets; references; footnotes; or numbered paragraphs.

Label disks with the title of the article, authors' names, and the word-processing format. Black and white illustrations or photographs can be included if required. Electronic copies of computer-generated illustrations should be saved in Adobe Photoshop, JPEG, EPS, GIF, or TIFF formats. Electronic versions of photos need to be at least 300 dpi.

Review process

Short reports, surveillance summaries, reviews and correspondence are not subject to peer review.

On receipt of a manuscript authors will be sent a brief acknowledgment indicating whether it will be considered for publication. The articles then undergo a review process that may include peer review by two experts in the topic area. Articles may be rejected without peer review. Occasionally, reports of urgent public health importance may be published on the CDA website, at the discretion of the Editor. Authors may be asked to revise articles as a result of the review process before the final decision about publication is made by the Editor. Revised articles are to be returned with a letter addressing the response to the reviewers' comments. On acceptance of the article all authors are required to sign a copyright release form transferring copyright to the Commonwealth. Accepted manuscripts are edited and final proofs returned for checking prior to printing.

Citation of *CDI*

Citations referring to this journal should use the abbreviation *Commun Dis Intell* to be consistent with that used by Medline citation.

Composition of Australian influenza vaccine for the 2002 season

In order to select virus strains for the manufacture of Influenza Vaccine for 2002 Season, a meeting of the Australian Influenza Vaccine Committee (AIVC) on Influenza Vaccines was convened on 11 October 2001.

Having considered the information on international surveillance by WHO and up-to-date epidemiology and strain characterisation presented at the meeting, the Committee considered that the WHO recommendations on the composition of vaccines for 2002 Southern Hemisphere Season should be followed:

H1N1 strain:	an A/New Caledonia/20/99 (H1N1)-like strain A/New Caledonia/20/99 (IVR-116) is recommended as a suitable vaccine strain.	15 µg HA per dose.
H3N2 strain:	an A/Moscow/10/99 (H3N2) -like strain A/Panama/2007/99 (RESVIR-17) is recommended as a suitable vaccine strain.	15 µg HA per dose.
B Strain:	A B/Sichuan/379/99-like strain B/Johannesburg/5/99, B/Victoria/504/00 or B/Guangdong/120/2000 are recommended as the suitable vaccine strain.	15 µg HA per dose.

The SRID reagents for testing the potency of influenza vaccines for A/New Caledonia/20/99 (IVR-116) and A/Panama/2007/99 (RESVIR-17) are available from NIBSC and CBER/FDA.

Communicable Diseases Surveillance

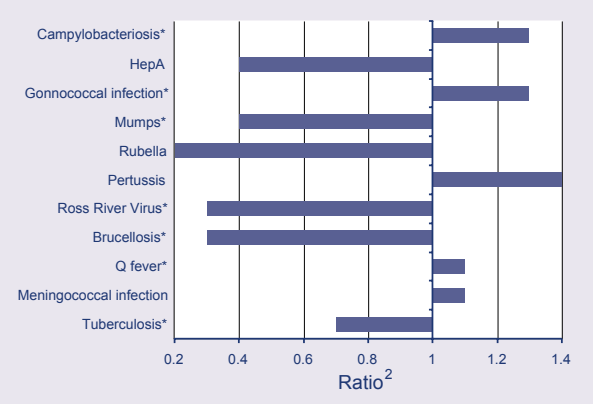
Highlights for 4th quarter, 2001

Communicable Disease Surveillance Highlights report on data from various sources, including the National Notifiable Diseases Surveillance System (NNDSS) and several disease specific surveillance systems that provide regular reports to Communicable Diseases Intelligence. These national data collections are complemented by intelligence provided by State and Territory communicable disease epidemiologists and/or data managers who have formed a Data Management Network. This additional information has enabled the reporting of more informative highlights each month.

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia, and the CDI Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme. In this report, data from the NNDSS are referred to as 'notifications' or 'cases', and those from ASPREN are referred to as 'consultations' or 'encounters' while data from the LabVISE scheme are referred to as 'laboratory reports'.

Figure 1 shows the changes in disease notifications with an onset date in the final quarter of 2001, compared with the 5-year final 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 include campylobacteriosis, gonococcal infection and Q fever. Diseases where the number of reports were two standard deviations below the 5 year mean include Ross River virus infection, brucellosis, tuberculosis and mumps. It should be noted, however, that these data are provisional and subject to revision. Delayed reporting of chronic or late presenting diseases may occur, and updating of notification data in 2002 may increase the numbers of these diseases in the future. These and other disease trends are discussed below, with additional commentary provided by representatives from 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 October to 31 December 2001 with historical data[2]



1. Selected diseases are chosen each quarter according to current activity
2. Ratio of current quarter total to the mean of the 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

Carolein Giele from Communicable Disease Control, Health Department of Western Australia reported that the increase of incident hepatitis C cases from Western Australia in this quarter reflected a recent download of anti-HCV results from a large laboratory in Perth. This enabled the search and linkage of previous negative anti-HCV results, used to identify recent seroconversions.

Gastrointestinal diseases

Haemolytic uraemic syndrome

One case of HUS with an onset date in the final quarter of 2001 was reported to the NNDSS. The case was an 11-month-old infant from New South Wales. Robert Menzies from the Communicable Disease Surveillance and Control Unit, NSW Department of Health indicated that an infective cause was not laboratory-confirmed. Consumption of sausage was the suspected source.

Campylobacteriosis

As for the third quarter of 2001, notifications of campylobacter infections were above the mean of the same period for the previous 5 years. There were 4,693 cases reported with an onset date in the final quarter of 2001, giving an overall rate of 146 cases per 100,000 population. Jurisdictions with reporting rates above the Australian rate included South Australia (247 cases per 100,000 population), Tasmania (197 cases per 100,000 population) and Western Australia (169 cases per 100,000 population).

Foodborne illness is notoriously under-reported. In the United Kingdom it has been estimated that only 1 in 8 cases of camplyobacteriosis is reported.[1] Notification rates for camplyobacter infections may be affected by microbiology laboratory screening protocols. This issue is being investigated by OzFoodNet.

Hepatitis A

There were 155 cases of hepatitis A reported with an onset date in the final quarter of 2001, giving a national notification rate of 3.2 cases per 100,000 population. The highest reporting rates were received from the Northern Territory (10.1 cases per 100,000 population), the Australian Capital Territory (7.6 cases per 100,000 population) and New South Wales (4.8 cases per 100,000 population).

Kerry-Ann O'Grady from Communicable Diseases Section, Department of Human Services Victoria, reported that cases from that jurisdiction were predominantly travel related. In New South Wales there was an increase in hepatitis A notifications reported in the last quarter of 2001; 80 cases compared with 57 in the third quarter. The increase was in men living in central and south-eastern Sydney (13 in the third quarter, up to 40 in the fourth quarter). The most commonly reported risk exposures during the quarter were male-to-male sex (16 cases, 20%), eating in a restaurant or gathering (16 cases, 20%), overseas travel (13 cases, 16%) and recreational drug use (8 cases, 10%). Risk exposures were unknown or not available for 23 cases (29%). Robyn Pugh, from the Communicable Diseases Unit, Queensland Department of Health, reported that there were fewer notifications of hepatitis A in 2001 than in the previous 4 years. Of the 99 notifications from Queensland in 2001 where information about injecting drug use was recorded, 7 (7%) of 99 were injecting drug users. In the last quarter of 2001, there were 3 injecting drug users (12%) who acquired hepatitis A in Queensland.

Hepatitis E

In late October the Department of Human Services Victoria received one notification of hepatitis E for a non-pregnant female who had arrived in Australia from India. The woman was well on the plane, but within 2 days of arrival experienced nausea, vomiting, and abdominal pain, developing jaundice 6 days later. She presented to hospital 10 days after onset of illness and was admitted for 3 days. Blood tests were negative for hepatitis A, B and C. Serological testing by Victorian Infectious Diseases Reference Laboratory confirmed the diagnosis with a strongly positive hepatitis E IgG titre. Other family members in India, her travel companions and partner remained well.

Typhoid

Fifteen cases of typhoid were reported with an onset date in the reporting period, including 7 from New South Wales, 3 from Victoria, 3 from Queensland and 1 case from both South Australia and Western Australia. Of the 15 cases, 6 were males and 9 were females, and the age range was 5 to 50 years.

All typhoid infection reported in the period were associated with overseas travel. The Department of Human Services Victoria reported 2 cases which were acquired in Indonesia, and a third acquired in Pakistan. All 3 cases were unrelated and of different strains. Of the 3 cases of typhoid notified in Queensland one was acquired in Papua New Guinea, one in Bangladesh and one in Indonesia. All 7 typhoid cases reported in New South Wales were acquired overseas, and there were no links between the cases.

Gary Dowse, from Communicable Disease Control, Health Department of Western Australia reported that in Western Australia a single case of typhoid was notified during the quarter, in a 31-year-old asylum seeker in detention on Christmas Island. It is most likely that the organism was acquired in Indonesia. Of 14 cases of typhoid notified in Western Australia during 2001, 9 (64%) were in asylum seekers travelling via Indonesia. Other cases reported in 2001 included 2 overseas students returning from Indonesia, a visiting seaman, a refugee from Africa, and a child returning from a visit to Pakistan.

Listeria

There were 14 reports of listeriosis, including 5 cases from Queensland, 3 cases from both New South Wales and Western Australia, 2 cases from Victoria and a single case from South Australia.

Of the 3 cases of listeriosis notified in December in Western Australia, two involved foetal death-in-utero (at 18 and 23 weeks, respectively) in women with febrile illnesses. Both the latter cases were serogroup 4, but no commonalities in food histories were identified.

Salmonellosis

There were 1,825 notifications of salmonellosis infections received nationally in the final quarter of 2001. *Salmonella* reports of note in the final quarter of 2001 included an increase in *Salmonella* Typhimurium phage type 170 in a number of jurisdictions, an outbreak of *Salmonella* Typhimurium phage type 126 in South Australia, and the appearance of a rare serovar in the Northern Territory. OzFoodNet was invited to further investigate these outbreaks, with the assistance of State and Territory health authorities. Further information can be obtained from OzFoodNet. (contact Martyn Kirk, Coordinating Epidemiologist, OzFoodNet, c/o National Public Health Partnership, 589 Collins St, Melbourne 3000, Australia, telephone: +61 3 9616 1522, facsimile: +61 3 9616 1500, E-mail: martyn.kirk@dhs.vic.gov.au).

In previous quarters of 2001 the emergence of *Salmonella* Typhimurium phage type 126 in jurisdictions across Australia has been noted (see OzFoodNet quarterly report, this issue). Cases continue to be reported in the current reporting period. Jane Raupach, from the Communicable Disease Control Branch of the Department of Human Services, South Australia, reported 23 cases of *Salmonella* Typhimurium phage type 126 with dates of onset from 1 October to 31 December 2001. A case control study found an association between cases and the consumption of chicken. The association was supported by descriptive epidemiology and microbiological evidence.

In Queensland other *Salmonella* clusters of note included a small cluster of *Salmonella* Singapore cases predominantly among adult females who reside in Brisbane and surrounding metropolitan areas. There were 12 cases of *S*. Singapore notified during the final quarter of 2001, 8 of whom were female, and seven of the 8 cases were older than 18 years. Six of the cases were notified over a one week period in December. A common link has not yet been identified and investigations are continuing.

Peter Markey, from Centre for Disease Control, Northern Territory Department of Health and Community Services, reported that from October to November there was a cluster of 15 cases of *Salmonella* Mgulani. This serovar is rarely identified in the Northern Territory. In 2000 the National Enteric Pathogen Surveillance System identified 44 cases with this serovar, 31 of whom resided in Queensland, 9 from New South Wales, 3 from Victoria and 1 from the Australian Capital Territory.[2] The majority of cases in Queensland are thought to be sporadic, although in New South Wales clusters have been described.[3]

Cases of *Salmonella* Mgulani reported from the Northern Territory in the current period were dispersed over a wide geographical area in the Top End. Cases ranged in age from 5 months to 52 years, and 13 of the 15 cases were non-Aboriginal people. The outbreak was investigated using telephone interviews and a standard questionnaire; 14 cases were interviewed but no particular source was identified. There have been no further cases since November.

Cryptosporidiosis

David Coleman and colleagues, from the Department of Health and Human Services, Tasmania, investigated a cluster of 45 cases of *Cryptosporidium* infection amongst residents in northern Tasmania in November 2001. Case patients were primarily children aged between 1 and 9 years and adults between 20 and 34 years (range: 1 to 41 years). The distribution of cases was spread over 7 local council districts. Investigations suggest that in the majority of cases farm animals were the likely source of infection. While cases reported various settings where animal contact occurred, the majority had attended an animal nursery at a local agricultural show. Of the first 19 cases, 16 (84%) attended the agricultural show with 14 of the 16 (88%) also reporting visiting the animal nursery. Person-to-person secondary transmission appears to be the likely mode of spread for later cases, with 10 of the 13 cases (77%) in a second peak reporting that at least one member of the household or a close contact had been ill prior to their own illness.

Sexually transmitted infections

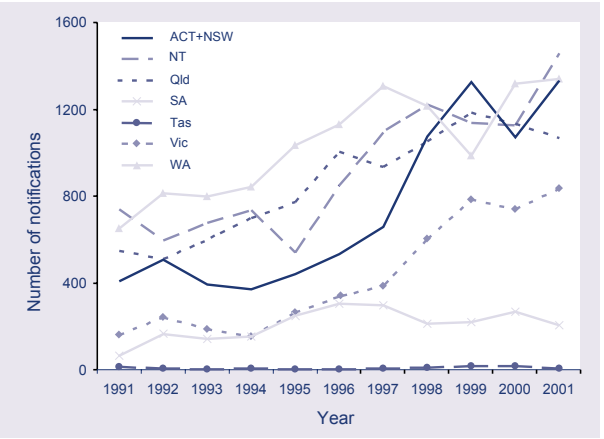
Congenital syphilis

A case of congenital syphilis was reported from the Kimberley region of Western Australia in a baby born in September. The mother had been diagnosed with infectious syphilis late in the third trimester of pregnancy. The last case of congenital syphilis notified in Western Australia was in 1992. The Kimberley region has experienced a resurgence of syphilis over the past several months.

Gonococcal infection

A total of 1,539 cases of gonococcal infection were notified in the final quarter of 2001, giving a notification rate of 32 cases per 100,000 population. This is an increase above the 5 year mean for the same reporting period (Figure 2). The highest notification rates were seen in the Northern Territory (757 cases per 100,000 population) and in Western Australia (79 cases per 100,000 population). There was no increase in case numbers in Tasmania and a decrease compared to 2000 in South Australia and Queensland. The increase seen in the Northern Territory reverses a decreasing trend from 1998 to 2000. Jan Savage from the AIDS/STD program of the Centre for Disease Control, Northern Territory Department of Health and Community Services, reported that the increases observed since 1995 in the Northern Territory are believed to reflect more acceptable (less invasive) methods of specimen collection, improved test sensitivity with the availability of PCR testing, and increased screening as part of 'well persons' health checks'.

Figure 2. Notifications of gonococcal infections, Australia, 1991 to 2001, by jurisdiction

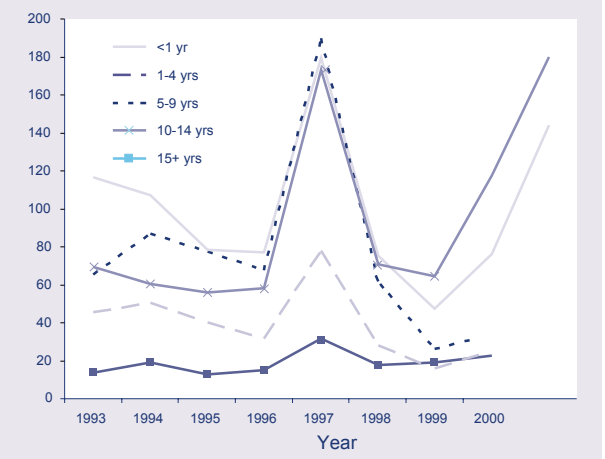


Vaccine preventable diseases

Pertussis

To date, a total of 3,210 cases of pertussis with an onset in the last quarter of 2001 have been reported to NNDSS, giving a national rate of 66 cases per 100,000 population. All jurisdictions showed an increase in pertussis notifications in the second half of 2001, apart from the Australian Capital Territory, where the three-year cyclic peak in notifications was observed in 2000. Highest notification rates for the final quarter of 2001 were received from South Australia (152 cases per 100,000 population), the Northern Territory (142 cases per 100,000 population), New South Wales (82 cases per 100,000 population) and Queensland (77 cases per 100,000 population). At the end of November 2001 the Communicable Diseases Network Australia issued a media release regarding pertussis, in response to the large number of notifications.

Figure 3. Rate of notification for pertussis, Australia, 1993 to 2001, by age group



Previous highlights for 2001 have noted the increase in pertussis notifications from all jurisdictions (apart from the Australian Capital Territory) for the current year. The notification rates by age group, from 1993 to 2001 are shown in Figure 3 (figure and commentary prepared by Heather Gidding, National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases). The three-year cycle of peaks in the notification rate for pertussis is evident, with high reporting rates in 1997 and 2001. As in 1997, all age groups have shown an increase in pertussis notifications in 2001, with the increase most evident in infants less than one year of age, and in the 10-14 year age group. The 1-4 year age group have shown a modest rise since 1999, attributed to

vaccine (DTP) given as part of the infant vaccination schedule. As at 31 December 2001, DTP uptake in Australian children 12-14 months inclusive was 92.2 per cent (3 doses) and 90.3 per cent for children 24-26 months (4 doses).[4] The rate for the 5- 9 year age group has not increased as dramatically in 2001 compared with the rise in 1997. This may be attributed to the introduction of a booster dose for 4-year-olds in 1994.

In Victoria, 852 notifications of pertussis were received in 2001, 85 per cent of which were received in the second half of the year. Notifications began to increase rapidly in September/October. Of the 622 notifications received in this 6 month period, 30 were in infants aged less than 6 months. There was one death in a 6-week-old infant in July.

In Queensland the largest number of pertussis cases for the 4-year period 1998 to 2001 occurred in 2001. There were 3 pertussis related deaths; one in the final quarter of 2001. Of the deaths, two were children under 1 year of age and one was a child in the 1-5 age range.

In the Northern Territory there were 72 cases of pertussis in the final quarter, including one death. This diagnosis was made on microbiological evidence at postmortem in a 5-month-old child for whom diagnosis of SIDS was being considered.

There have been no pertussis related deaths in Western Australia in the current reporting period and New South Wales reported there have been no pertussis related deaths in 2001.

Tetanus

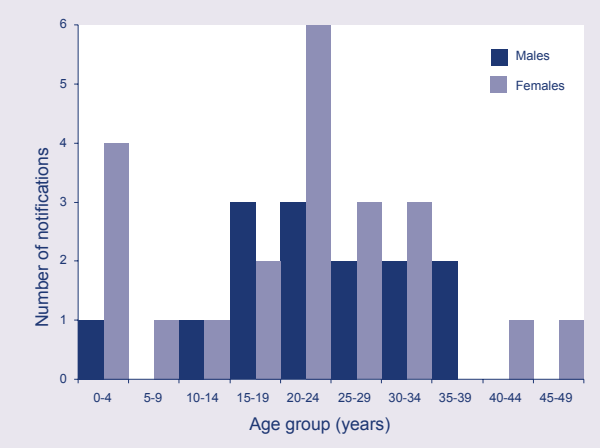
Two cases of tetanus with an onset in the final quarter of 2001 were reported to NNDSS, including an 82-year-old female from South Australia, and a 49-year-old male from Victoria. The vaccination history of both cases was uncertain.

Measles

There were 37 cases of measles reported with an onset of disease in the final quarter of 2001. The age and sex distribution of the 36 cases where the gender was recorded is shown in Figure 4. One additional case (whose gender was unrecorded) was in the 20-24 year age group.

The majority (22 of the 36 cases, 61%) were notified in Victoria. In that jurisdiction between 21 October and 31 December, 18 laboratory-confirmed measles cases were notified, of whom nine (50%) were hospitalised. All but one case (whose infection was acquired overseas) were epidemiologically linked, and were of the same genotype (D5), thus considered to be part of the same outbreak. Of the total number of cases from this jurisdiction, 88 per cent were aged 18 to 34 years, none of whom had a documented history of measles vaccination. A source for the outbreak was not identified.

Figure 4. Notification of measles, Australia 1 October to 31 December 2001, by age and sex



An outbreak of measles occurred in Western Australia after a 25-year-old woman became ill in late November, shortly after returning from a holiday in Bali. This woman infected 4 other individuals, who in turn each infected one other person. Of the 9 cases, 5 were aged 20-25 years, and the remaining 4 were older teenagers who had not been vaccinated. Four of the cases required hospitalisation.

There has been no endemic measles circulation in Western Australia for 3 years. In this period all 41 cases of measles occurring in Western Australia have been either imported by overseas visitors or returning holiday-makers, or transmitted from these imported cases to local residents. There have been 14 separate importations responsible for these cases.

While 2 cases of measles were recorded from New South Wales in the reporting period, they were not epidemiologically linked.

Mumps

There were 13 cases of mumps notified in the final quarter of 2001, including 7 cases from New South Wales, 4 cases from Western Australia and 2 cases from South Australia. Of the 13 cases, nine were males, and four were females. All but one (a 4-year-

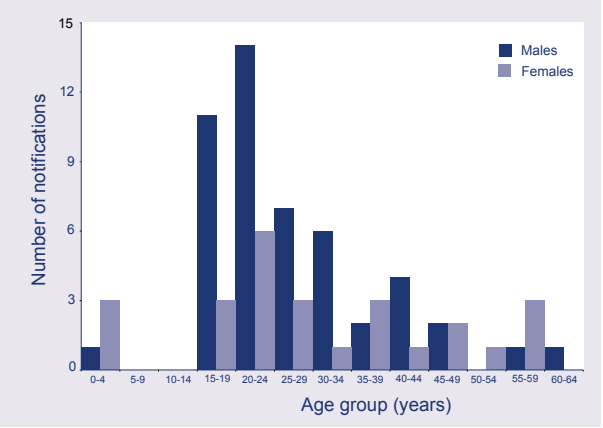
old male case notified in New South Wales) were aged over 20 years. The number of notifications in the current reporting period is lower than the average number of notifications received nationally in the same reporting period for the previous 5 years.

Rubella

There were 75 notifications of rubella with an onset in the final quarter of 2001. The majority of cases (65%) were males. The age/sex distribution of cases is shown in Figure 5. Seventeen cases (23% of all rubella notifications) were females of child bearing age (aged between 15 and 45 years). None of the 4 cases in the 0-4 year age group were congenitally acquired.

The notification rate of rubella for Queensland remains higher than all other jurisdictions, and 44 of the 75 cases (59%) reported during this period were from that jurisdiction. Of the 44 notifications from Queensland, 11 were in the age range 15-19 years and the remaining 33 were over 20 ages of age.

Figure 5. Notification of rubella, Australia 1 October to 31 December 2001, by age and sex



Influenza

Laboratory-confirmed influenza cases are now reported to NNDSS by all jurisdictions. While New South Wales and Victoria have ceased sentinel GP influenza surveillance for the season, national surveillance data consisting of laboratory reports through NNDSS and LabVISE and national and sentinel general practice schemes in the Northern Territory are reported fortnightly on the Communicable Diseases Australia web site http://www.health.gov.au/pubhlth/cdi/ozflu/flucurr.htm

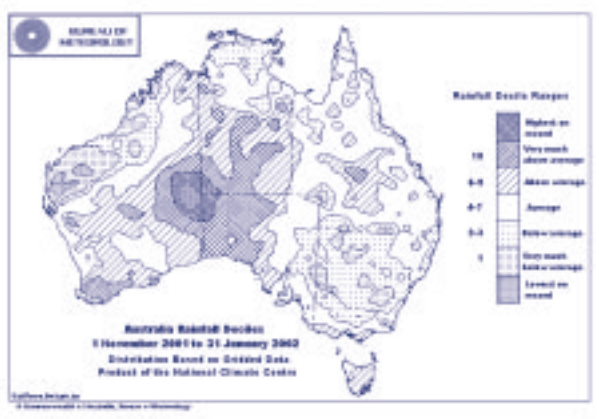
Haemophilus influenzae type b

There were 2 cases of *Haemophilus influenzae* type B infection (Hib) with an onset in the final quarter of 2001. Both cases were from Victoria. Further information was provided on one of these cases. A 5-year-old non-Aboriginal child from rural Victoria had documented evidence of 4 doses of Hib vaccine. The child initially had fever and red cheeks, and presented to a GP 2 days later with sore throat, dyspnoea and drooling. They were transferred to hospital with a provisional diagnosis of epiglottitis. While the child's throat swab was negative, blood cultures were positive for Hib (septicaemia). The child was treated successfully with intravenous ceftriaxone.

Vectorborne diseases

A number of jurisdictions have reported that the expected rise in arbovirus infections did not occur at the end of 2001. This was attributed to the cooler weather conditions and lack of rain in some

Figure 6. Australia rainfall deciles, 1 November 2001 to 31 January 2002



areas of the country (Figure 6).

Source: Australian Bureau of Meterology Website: http://www.bom.gov.au

According to the Bureau of Meterology:

'November to January rainfall shows above to well above average over the southern two-thirds of the Northern Territory, the western two-thirds of South Australia, and the southern half of Western Australia. The highest on record rainfall area covering the tri-state border area of Western Australia, the Northern Territory, and South Australia derives mainly from

record rains in December. Victoria's far south and Tasmania were also wetter than average. There were two significant areas of below average falls for the three months. The first covered the northern areas of Western Australia and the far north of the Northern Territory. After a promising start to the monsoon in November, both December and January were generally much drier than average. The second region covers most of New South Wales and northern Victoria, together with some parts of eastern South Australia.'

Figure 7 shows the number of Ross River and Barmah Forest virus disease notifications. Comparisons to the previous 4 years are shown in Figure 8 and 9 for Ross River and Barmah Forrest, respectively. Interestingly, it does appear that notifications of Ross River virus disease are down compared to previous years, but numbers of Barmah Forest virus disease, which peak later in the season compared with Ross River virus cases do not appear unusually low.

No reports of Murray Valley encephalitis virus infection were recorded with an onset in the final quarter of 2001. Information regarding sentinel chicken activity in the reporting period is available in this issue (see page 86).

Figure 7. Notifications of Ross River and Barmah Forrest virus, Australia, 2001, by month

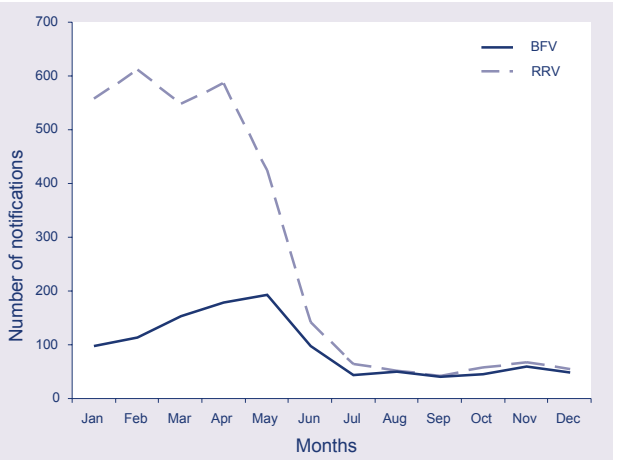


Figure 8. Notifications of Ross River virus, Australia, 1997 to 2001, by month

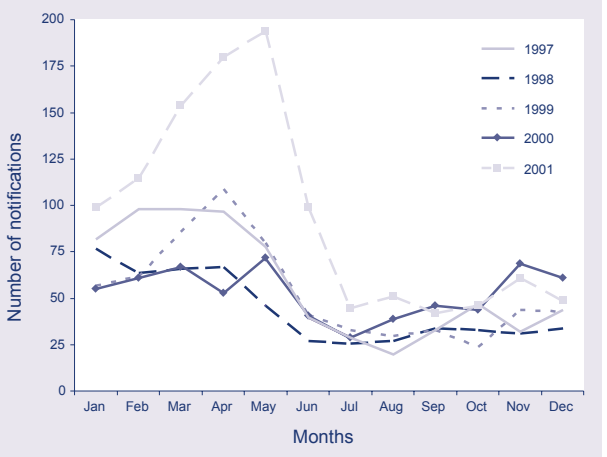
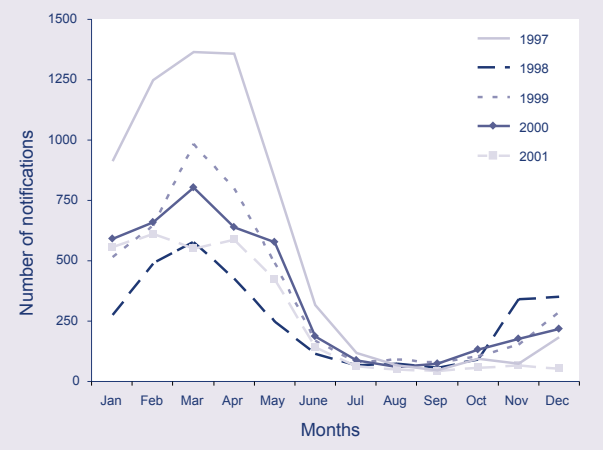


Figure 9. Notifications of Barmah Forrest virus, Australia, 1997 to 2001, by month



Other bacterial infections

Legionellosis

There were 76 cases of legionellosis reported to NNDSS with an onset in the final quarter of 2001, including 22 cases from Victoria. In this jurisdiction an outbreak of 3 cases of *Legionella pneumophila* serogroup 1 (diagnosed by urinary antigen) was detected in October 2001. Two of the cases were confirmed by culture. Despite extensive environmental investigation, no source was identified. Enhanced surveillance was undertaken but no further cases were identified.

In the Northern Territory there was one case of legionellosis in a 66-year-old Aboriginal man. The organism was identified as *L. longbeachae*.

While there were no outbreaks of legionellosis in New South Wales in the last quarter of 2001, there were 16 sporadic cases including 7 cases identified as *L. longbeachae*, 1 *L. pneumophila* type 2, 6 *L. pneumophila* not further specified, and two with typing information unavailable.

Meningococcal infection

The increasing number of meningococcal cases reported nationally continued in the final quarter of 2001, with 148 cases received nationally. Overall, the national reporting rate for this period was 3.1 cases per 100,000 population. Jurisdictions with rates higher than the national rate included Tasmania (7.7 cases per 100,000 population), the Northern Territory (6.1 cases per 100,000 population), South Australia (4.8 cases per 100,000 population), Queensland (3.7 cases per 100,000 population) and Victoria (3.3 cases per 100,000 population).

Queensland reported the highest proportion of cases (27%) in the reporting period. The number of notifications of invasive meningococcal diseases was the highest Queensland has recorded for 4 years. Apart from a small cluster of 2 cases in a boarding school, all were sporadic cases.

In Tasmania during the period 23 September to 15 October 2001 a total of 10 confirmed cases of invasive meningococcal disease were reported from greater Hobart (population 194,000, giving a cumulative incidence of 52 per 100,000 population for this period). Three patients presented with clinical meningitis while the remainder developed septicaemia. Three female patients aged 18, 21 and 60 years subsequently died. The age range was 18 to 60 years with 8 cases aged between 18 and 22 years. Blood cultures from 6 cases were confirmed as serogroup C. All had identical pulse field gel electrophoresis (PFGE) patterns and have been shown to belong to a hyperinvasive strain C (2a:P1.5,2). Two cases were diagnosed on the basis of PCR from cerebrospinal fluid and two using serology alone. Molecular typing of 5 serogroup C isolates from patients earlier in 2001 (also from greater Hobart) showed that these were identical to the outbreak strain. One of these cases (a female aged 25) also died. Group C isolates obtained from cases from elsewhere in Tasmania were different than the Hobart cases. Since October there have been 2 further cases (including one death) of invasive group C meningococcal disease. Molecular typing is in progress on these isolates. In the September/October period a common factor was attendance at nightclubs in Hobart and numerous public warnings were made in relation to the sharing of drinks, cigarettes and other smoking activities.

Other non-notifiable diseases

VRE outbreak, Perth

Western Australia experienced Australia's largest yet recorded outbreak of vancomyin resistant *Enterococcus* (VRE) in the latter part of 2001. In late July, 2001 a vancomycin resistant *Enterococcus* spp was isolated, from a patient in the Intensive Care Unit at Royal Perth Hospital. Screening of the patients' contacts revealed further VRE colonised individuals, leading to the investigation of contacts of VRE colonised patients. A specialised computer system was used to track approximately 4,000 patients who had contact with colonised individuals during the outbreak. In October, contact tracing was broadened to include screening of all hospital patients.

Vancomycin resistant *Enterococcus faecium* (vanB) was isolated among 165 patients (4 infections, 161 colonised), the vast majority of which were detected through an active screening program. No deaths were associated with VRE infection. Cases were originally detected among renal and intensive care patients with later spread to other wards such as the Haematology Unit. Antibiotic usage among this patient population appeared to be high. Patient cohorting, screening and extensive ward cleaning was employed to control the outbreak. A case control study is now in progress to assess the risk factors for VRE acquisition during this outbreak.

The outbreak had been terminated by early January 2002, following implementation of a co-ordinated control program at Royal Perth Hospital and across other Western Australian health-care facilities. VRE was first detected in Western Australia in 1996, and only around 20 isolates, mostly sporadic, had previously been identified.

Restaurant-associated Norwalk-like virus outbreaks in Western Australia

Two separate outbreaks, a week apart, of apparent foodborne illness were reported in December by work groups that had attended the same Perth restaurant. The nature of illness in both groups was similar, with onset of nausea, vomiting and diarrhoea within 12-48 hours of the meal, and relatively short duration of illness of 1-2 days.

Initial microbiological investigations, including PCR for calicivirus genotype 2 in food and faecal specimens, did not reveal a cause. However, several faecal specimens were subsequently re-tested at the Victorian Infectious Disease Reference Laboratory, revealing a genotype 1 Norwalk-like virus was responsible for illness in both outbreaks. Epidemiological investigations did not conclusively indicate a suspect food source.

LabVISE

There were 6,212 reports to LabVISE from 14 laboratories in final quarter of 2001 (Table 4). In this reporting period, there were 4,171 viral infections recorded (67% of all reports) and 2,041 reports of bacteria and other microorganisms (33% of all reports). Rotavirus was the most regularly identified virus in this period, with a total of 531 reports, followed by varicella zoster (524 reports). Among the bacterial isolates the largest numbers of reports were of *Chlamydia* spp (928 reports) and *Treponema pallidum* (347 reports). The reports of *Treponema pallidum* equate well with the number of syphilis notifications (n=342) received by NNDSS during the same period while the number of *Chlamydia* infections reported to NNDSS was approximately 4 times that received via LabVISE. This may be due to the majority of diagnoses of syphilis being undertaken in public health laboratories that report to LabVISE. In comparison, the availability of nucleic acid tests for *Chlamydia* in a large number of public and private laboratories leads to a large proportion of diagnoses being undertaken outside the LabVISE network.

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4. Australian Childhood Immunisation Register statistical tables. Table 4 http://www.hic.gov.au/annualreport/statistics/acir_table4.htm.

Erratum

Australia's Notifiable Disease Status, 1999: Annual report of the National Notifiable Diseases Surveillance System

The following corrections to this report (*Commun Dis Intell* 2001;25:190-245) should be noted.

Under *Results* (p207) the total notifications should read 88,229 not 88,239.

Results (p208). The sentence 'Measles notifications fell by more than 50 per cent compared with 1998' should read 'Measles notifications fell by more than 50 per cent compared with the 5 year mean (Figure 3).'

Tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 24,853 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 October and 31 December 2001 (Table 2). The notification rate of diseases per 100,000 population for each State or Territory is presented in Table 3.

There were 5,647 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 October and 31 December 2001 (Tables 4 and 5).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 40-43 to 48-52, 2001, ending 30 December 2001, are included in this issue of *Communicable Diseases Intelligence* (Table 6).

Table 1. Reporting of notifiable diseases by jurisdiction (4th quarter 2001)

Disease	Data received from:*
Bloodborne diseases	
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)	All jurisdiction, except NT
Hepatitis C (incident)	All jurisdictions except Queensland
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacterosis	All jurisdictions except NSW
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listerosis	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 may not yet be reporting a disease either because legislation has not yet made that disease notifiable in that jurisdiction, or because notification data for that disease are not yet being reported to the Commonwealth

† In the Australian Capital territory, infections with Murray Valley encephalitis virus and kunjin virus are combined under Murray Valley encephalitis

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 October to 31 December 2001, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 4th quarter 2001[1]	Total 3rd quarter 2001[1]	Total 4th quarter 2000[1]	Last five years mean 4th quarter	Year to date 2001	Last 5 years YTD mean	Ratio†
Bloodborne diseases															
Hepatitis B (incident)	0	25	1	7	6	2	23	8	72	116	83	67	402	289	1.1
Hepatitis B (unspecified)	12	1,021	NN	170	60	2	484	86	1,835	2,333	2,003	1,696	8,141	7,137	1.1
Hepatitis C (incident)	2	28	0	NN	15	0	5	41	91	137	99	77	536	266	1.2
Hepatitis C (unspecified)	57	1,177	55	713	139	89	1,297	200	3,727	4,129	4,491	4,267	15,898	17,813	0.9
Hepatitis D	0	0	0	0	0	0	4	0	4	3	12	7	21	18	0.6
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	2	0	2	1	0.0
Campylobacterosis[2]	116	-	63	1,016	928	231	1,532	807	4,693	4,214	3,748	3,693	16,062	12,675	1.3
Cryptosporidiosis‡	0	54	42	151	12	61	76	49	445	243	232	N/A	1,612	N/A	N/A
Haemolytic uraemic syndrome	0	1	0	0	0	0	0	0	1	2	8	4	5	11	0.2
Hepatitis A	6	79	5	24	6	1	25	9	155	150	131	372	530	1,999	0.4
Hepatitis E	0	0	0	0	0	0	1	0	1	1	3	1	10	5	0.7
Listerosis	0	3	0	5	1	0	2	3	14	15	16	15	62	65	0.9
Salmonellosis	22	435	95	628	158	33	275	179	1,825	1,189	1,543	1,632	7,075	6,732	1.1
Shigellosis	1	18	21	25	8	0	17	27	117	132	122	149	564	620	0.8
SLTEC,VTEC[3]	0	0	0	4	5	0	0	2	11	7	6	7	46	23	1.5
Typhoid	0	7	0	3	1	0	3	1	15	20	12	15	79	71	1.0
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	2	0	0	3	3	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0

Table 2 continued. Notifications of diseases received by State and Territory health authorities in the period 1 October to 31 December 2001, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 4th quarter 2001[1]	Total 3rd quarter 2001[1]	Total 4th quarter 2000[1]	Last five years mean 4th quarter	Year to date 2001	Last 5 years YTD mean	Ratio†
Sexually transmissible diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0.0
Chlamydial infection	80	1,024	314	1,324	347	88	1,053	680	4,910	5,066	4,413	3,042	20,052	11,991	1.6
Donovanosis	0	0	2	2	NN	0	0	2	6	10	2	6	29	31	1.0
Gonococcal infection[4]	3	320	374	226	27	6	207	376	1,539	1,583	1,137	1,191	6,371	5,123	1.3
Syphilis[5]	6	135	120	14	6	7	1	35	324	352	439	384	1,320	1,655	0.8
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0.0
Haemophilus influenzae type b	0	0	0	0	0	0	2	0	2	4	7	8	25	41	0.2
Influenza‡	3	40	32	34	34	0	10	47	200	1,005	55	N/A	1,317	N/A	N/A
Measles	1	2	0	1	1	0	22	10	37	16	26	116	139	393	0.3
Mumps	0	7	0	0	2	0	0	4	13	19	35	37	112	179	0.4
Pertussis	15	1,336	70	695	570	45	351	128	3,210	3,028	1,846	2,250	9,339	6,281	1.4
Pneumococcal disease‡	3	105	20	79	25	19	94	43	388	651	164	N/A	1,622	N/A	N/A
Rubella[6]	1	13	0	44	3	0	13	1	75	70	142	321	258	1,078	0.2
Tetanus	0	0	0	0	1	0	1	0	2	0	3	2	3	5	1.3
Vectorborne diseases															
Arbovirus infection NEC	0	0	0	0	0	0	0	0	0	8	3	9	37	54	0.0
Barmah Forest virus infection	0	51	0	91	0	0	3	14	159	140	172	124	1,138	674	1.3
Dengue	1	13	3	3	0	0	2	4	26	47	11	70	180	231	0.4
Japanese encephalitis	0	0	0	0	0	0	0	0	0	0	0	N/A	0	N/A	N/A
Kunjin virus infection	-	0	0	0	0	0	0	0	0	0	0	N/A	2	N/A	N/A
Malaria	0	40	11	52	4	1	14	8	130	160	176	146	698	789	0.9
Murray Valley encephalitis	0	0	0	0	0	0	0	0	0	0	0	N/A	3	N/A	N/A
Ross River virus infection	0	35	12	94	14	1	7	21	184	160	514	530	3,223	5,250	0.3

Table 2 continued. Notifications of diseases received by State and Territory health authorities in the period 1 October to 31 December 2001, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 4th quarter 2001[1]	Total 3rd quarter 2001[1]	Total 4th quarter 2000[1]	Last five years mean 4th quarter	Year to date 2001	Last 5 years YTD mean	Ratio†
Zoonoses															
Anthrax‡	0	0	0	0	NN	0	0	0	0	0	0	N/A	0	N/A	N/A
Australian bat lyssavirus‡	0	0	0	0	0	0	0	0	0	0	0	N/A	0	N/A	N/A
Brucellosis	0	0	0	4	0	0	0	0	4	4	10	13	19	40	0.3
Leptospirosis	0	12	3	9	1	2	14	1	42	41	65	56	240	220	0.7
Other lyssavirus‡	1	9	0	0	6	0	18	3	37	0	0		0	N/A	N/A
Ornithosis	0	0	0	0	0	0	0	0	0	25	40	28	131	74	0.0
Q fever	0	39	0	99	5	0	6	6	155	141	146	139	671	559	1.1
Other bacterial infections															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0.0
Legionellosis	0	16	1	6	14	0	22	17	76	60	85	66	292	268	1.1
Leprosy	0	1	0	0	0	0	0	0	1	1	1	1	4	7	0.7
Meningococcal infection	0	30	3	34	18	9	40	14	148	242	168	130	672	509	1.1
Tuberculosis	3	73	4	6	2	2	77	12	179	241	259	252	887	991	0.7
Total	**333**	**6,149**	**1,251**	**5,563**	**2,419**	**599**	**5,701**	**2,838**	**24,853**	**25,767**	**22,430**	**20,927**	**99,833**	**84,170**	**1.2**

1. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
2. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
3. Infections with Shiga-like toxin (verotoxin) producing E. coli (SLTEC/VTEC).
4. Northern Territory, Qld, SA , Vic and WA: includes gonococcal neonatal ophthalmia.
5. Includes congenital syphilis.
6. Includes congenital rubella.

* Date of notification = a composite of three dates: (i) the true onset date from a clinician, if available, (ii) the date the laboratory test was ordered, or (iii) the date reported to the public health authority.

† Ratio = ratio of current month total to mean of the same reporting period in last 5 years calculated as described above.

‡ Notifiable from January 2001 only.

NA Not calculated as only notifiable for under 5 years.

NN Not Notifiable

NEC Not elsewhere classified.

\- Elsewhere classified.

Table 3. Notification rates of diseases by State or Territory, 1 October to 31 December 2001. (Rate per 100,000 population)

	State or Territory								
Disease	**ACT**	**NSW**	**NT**	**Qld**	**SA**	**Tas**	**Vic**	**WA**	**Australia**
Bloodborne diseases									
Hepatitis B (incident)	0.0	1.5	2.0	0.8	1.6	1.7	1.9	1.7	1.5
Hepatitis B (unspecified)	15.3	62.5	NN	18.7	16.0	1.7	40.1	18.0	38.3
Hepatitis C (incident)	2.5	1.7	0.0	NN	4.0	0.0	0.4	8.6	2.3
Hepatitis C (unspecified)	72.6	72.1	111.3	78.6	37.0	75.7	107.4	41.9	76.9
Hepatitis D	0.0	0.0	0.0	0.0	0.0	0.0	0.3	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
Campylobacterosis[2]	147.7	-	127.5	112.0	247.1	196.5	126.9	169.0	146.0
Cryptosporidiosis[‡]	0.0	3.3	85.0	16.6	3.2	51.9	6.3	10.3	9.2
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis A	7.6	4.8	10.1	2.6	1.6	0.9	2.1	1.9	3.2
Hepatitis E	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Listerosis	0.0	0.2	0.0	0.6	0.3	0.0	0.2	0.6	0.3
Salmonellosis	28.0	26.6	192.3	69.2	42.1	28.1	22.8	37.5	37.7
Shigellosis	1.3	1.1	42.5	2.8	2.1	0.0	1.4	5.7	2.4
SLTEC,VTEC[3]	0.0	0.0	0.0	0.4	1.3	0.0	0.0	0.4	0.2
Typhoid	0.0	0.4	0.0	0.3	0.3	0.0	0.2	0.2	0.3
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
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	101.9	62.7	635.7	146.0	92.4	74.9	87.2	142.4	101.3
Donovanosis	0.0	0.0	4.0	0.2	NN	0.0	0.0	0.4	0.1
Gonococcal infection[4]	3.8	19.6	757.1	24.9	7.2	5.1	17.1	78.8	31.8
Syphilis[5]	7.6	8.3	242.9	1.5	1.6	6.0	0.1	7.3	6.7

Table 3 continued. Notification rates of diseases by State or Territory, 1 October to 31 December 2001. (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	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0
Influenza‡	3.8	2.4	64.8	3.7	9.1	0.0	0.8	9.8	4.1
Measles	1.3	0.1	0.0	0.1	0.3	0.0	1.8	2.1	0.8
Mumps	0.0	0.4	0.0	0.0	0.5	0.0	0.0	0.8	0.3
Pertussis	19.1	81.8	141.7	76.6	151.8	38.3	29.1	26.8	66.2
Pneumococcal disease‡	3.8	6.4	40.5	8.7	6.7	16.2	7.8	9.0	8.0
Rubella[6]	1.3	0.8	0.0	4.9	0.8	0.0	1.1	0.2	1.5
Tetanus	0.0	0.0	0.0	0.0	0.3	0.0	0.1	0.0	0.0
Vectorborne diseases									
Arbovirus infection NEC	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Barmah Forest virus infection	0.0	3.1	0.0	10.0	0.0	0.0	0.2	2.9	3.3
Dengue	1.3	0.8	6.1	0.3	0.0	0.0	0.2	0.8	0.5
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
Malaria	0.0	2.4	22.3	5.7	1.1	0.9	1.2	1.7	2.7
Murray Valley encephalitis‡	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	0.0	2.1	24.3	10.4	3.7	0.9	0.6	4.4	3.8
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	0.4	0.0	0.0	0.0	0.0	0.1
Leptospirosis	0.0	0.7	6.1	1.0	0.3	1.7	1.2	0.2	0.9
Other lyssavirus‡	1.3	0.6	0.0	0.0	1.6	0.0	1.5	0.6	0.8
Ornithosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Q fever	0.0	2.4	0.0	10.9	1.3	0.0	0.5	1.3	3.2
Other bacterial infections									
Legionellosis	0.0	1.0	2.0	0.7	3.7	0.0	1.8	3.6	1.6
Leprosy	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection	0.0	1.8	6.1	3.7	4.8	7.7	3.3	2.9	3.1
Tuberculosis	3.8	4.5	8.1	0.7	0.5	1.7	6.4	2.5	3.7

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 for the reporting period of 1 October to 31 December 2001[1], and total reports for the year[2]

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2001	This period 2000	Year period 2001[3]	Year to date 2000
Measles, mumps, rubella												
Measles virus	2	3	-	-	1	-	-	-	6	25	44	172
Mumps virus	-	-	1	-	-	-	1	7	9	12	49	58
Rubella virus	-	4	-	6	3	-	3	2	18	32	51	145
Hepatitis viruses												
Hepatitis A virus	-	3	2	10	2	-	1	7	25	80	146	375
Hepatitis D virus	-	1	-	-	-	-	2	-	3	3	9	8
Hepatitis E virus	-	-	-	-	-	2	-	-	2	-	4	1
Arboviruses												
Ross River virus	-	3	12	54	81	-	2	17	169	250	1,268	1,423
Barmah Forest virus	-	2	1	31	5	-	-	9	48	46	169	180
Dengue not typed	-	-	2	-	-	-	-	6	8	41	175	85
Murray Valley encephalitis virus	-	-	1	-	-	-	-	-	1	-	20	2
Flavivirus (unspecified)	-	-	-	-	-	-	1	-	1	6	40	27
Adenoviruses												
Adenovirus type 1	-	-	-	-	2	-	-	-	2	6	8	14
Adenovirus type 3	-	-	-	-	1	-	1	-	2	13	18	35
Adenovirus type 4	-	-	-	-	1	-	-	-	1	1	5	15
Adenovirus type 5	-	-	-	-	1	-	-	-	1	2	8	6
Adenovirus type 6	-	-	-	-	3	-	-	-	3	-	3	-
Adenovirus type 7	-	-	-	-	-	1	2	-	3	3	8	7
Adenovirus type 8	-	-	-	-	-	-	2	-	2	-	3	1
Adenovirus type 37	-	-	-	-	-	-	1	-	1	1	11	11
Adenovirus type 40	-	-	-	-	-	-	-	4	4	14	86	74
Adenovirus not typed/pending	4	56	3	9	106	1	42	62	283	354	1,039	1,132
Herpes viruses												
Herpes virus type 6	-	-	-	-	-	-	-	2	2	5	6	16
Cytomegalovirus	2	69	5	88	141	9	87	38	439	380	1,312	1,220
Varicella-zoster virus	5	25	8	170	48	3	76	189	524	489	1,494	1,658
Epstein-Barr virus	-	19	5	113	209	-	39	75	460	669	1,926	2,196
Other DNA viruses												
Papovavirus group	-	-	-	-	-	-	-	1	1	1	7	12
Molluscum contagiosum	-	-	-	-	-	-	-	2	2	2	11	15
Parvovirus	-	2	5	78	18	-	6	32	141	90	389	437
Picornavirus family												
Coxsackievirus A9	-	2	-	-	-	-	-	-	2	2	11	10
Coxsackievirus A16	-	-	-	-	-	-	2	-	2	-	8	15
Coxsackievirus B1	-	-	-	-	1	-	2	-	3	-	4	1
Coxsackievirus B4	-	-	-	-	-	-	10	-	10	3	16	3
Coxsackievirus B5	-	-	-	-	1	-	1	-	2	2	5	7
Coxsackievirus B untyped/pending	-	-	-	-	-	-	1	-	1	-	1	-
Echovirus type 3	-	1	-	-	-	-	-	-	1	2	2	5
Echovirus type 11	-	1	-	-	-	-	-	-	1	33	7	166
Echovirus type 18	-	1	-	-	-	-	-	-	1	-	1	-
Echovirus type 30	-	2	-	-	-	-	5	-	7	9	121	17
Poliovirus type 1 (uncharacterised)	1	10	-	-	-	-	-	-	11	9	22	26
Poliovirus type 2 (uncharacterised)	-	3	-	1	-	-	-	-	4	7	8	16

Table 4 continued. Virology and serology laboratory reports by State or Territory for the reporting period of 1 October to 31 December 2001[1], and total reports for the year[2]

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2001	This period 2000	Year period 2001[3]	Year to date 2000
Picornavirus family												
Poliovirus type 3 (uncharacterised)	-	3	-	-	-	-	-	-	3	2	8	8
Poliovirus not typed/pending	-	-	-	-	-	-	1	-	1	1	1	1
Rhinovirus (all types)	2	80	2	1	7	-	6	39	137	181	420	501
Enterovirus type 71 (BCR)	1	4	-	-	-	-	-	1	6	7	6	15
Enterovirus not typed/pending	-	4	4	6	1	6	38	89	148	153	815	753
Ortho/paramyxoviruses												
Influenza A virus	10	26	6	12	218	1	28	163	464	199	1,499	1,898
Influenza B virus	4	30	-	8	63	1	15	26	147	34	580	279
Parainfluenza virus type 1	-	1	-	-	9	-	-	-	10	8	230	44
Parainfluenza virus type 2	-	-	-	-	3	-	-	2	5	18	36	114
Parainfluenza virus type 3	-	62	-	19	50	-	29	117	277	306	516	803
Respiratory syncytial virus	-	52	1	11	45	1	19	38	167	394	2,735	3,059
Ortho/paramyxoviruses												
Influenza A virus	10	26	6	12	218	1	28	163	464	199	1,499	1,898
Influenza B virus	4	30	-	8	63	1	15	26	147	34	580	279
Parainfluenza virus type 1	-	1	-	-	9	-	-	-	10	8	230	44
Parainfluenza virus type 2	-	-	-	-	3	-	-	2	5	18	36	114
Parainfluenza virus type 3	-	62	-	19	50	-	29	117	277	306	516	803
Respiratory syncytial virus	-	52	1	11	45	1	19	38	167	394	2,735	3,059
Other RNA viruses												
HTLV-1	-	-	1	-	-	-	-	3	4	3	9	12
Rotavirus	1	191	-	-	192	6	89	52	531	624	1,771	2,246
Norwalk agent	-	-	-	-	7	-	58	-	65	8	82	59
Other												
Chlamydia trachomatis not typed	10	133	35	222	204	4	28	292	928	933	3,154	3,295
Chlamydia psittaci	1	-	-	-	-	2	30	2	35	16	102	78
Mycoplasma pneumoniae	-	17	5	65	56	3	53	37	236	342	686	1,125
Mycoplasma hominis	-	2	-	-	-	-	-	-	2	1	8	5
Coxiella burnetii (Q fever)	-	3	-	21	2	-	10	11	47	54	101	221
Rickettsia prowazeki	-	-	-	-	-	-	1	-	1	-	2	-
Rickettsia australis	-	-	-	-	-	-	1	-	1	-	2	2
Rickettsia tsutsugamushi	-	-	-	-	-	-	-	-	1	-	11	2
Rickettsia - Spotted fever group	-	-	-	-	-	5	-	-	23	-	44	1
Rickettsia spp - other	-	-	-	-	-	-	-	3	3	2	12	13
Streptococcus group A	-	5	10	64	-	-	21	-	100	156	348	368
Yersinia enterocolitica	-	3	-	2	-	-	-	1	6	1	15	10
Bordetella pertussis	-	40	2	21	87	-	80	10	240	290	689	845
Bordetella parapertussis	-	-	-	-	-	-	1	-	1	-	1	-
Legionella pneumophila	-	-	-	-	1	-	8	2	11	1	44	17
Legionella longbeachae	-	-	-	-	9	-	1	6	16	30	59	51
Legionella species	-	-	-	-	-	-	3	-	3	-	5	-
Cryptococcus species	-	1	-	-	6	-	-	-	7	3	18	9
Leptospira species	-	-	1	7	10	-	-	3	21	20	63	55
Treponema pallidum	-	50	75	80	130	-	-	12	347	332	909	774
Entamoeba histolytica	-	-	-	2	-	-	4	-	6	4	17	7
Toxoplasma gondii	-	-	-	-	1	-	3	-	4	4	16	9
Echinococcus granulosus	-	1	-	-	-	-	-	1	2	3	18	4
Total	**43**	**915**	**187**	**1,101**	**1,725**	**45**	**814**	**1,363**	**6,212**	**6,722**	**23,547**	**26,274**

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.
2. From January 2000 data presented are for reports with report dates in the current period. Previously reports included all data received in that period.
3. Totals comprise data from all laboratories. Cumulative figures are subject to retrospective revision, so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

– No data received this period.

Table 5. Virology and serology laboratory reports by laboratories for the reporting period 1 October to 31 December 2001[1]

	Laboratory	October 2001	November 2001	December 2001	Total this period
Australian Capital Territory	The Canberra Hospital	-	54	-	**54**
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	115	143	91	**349**
	New Children's Hospital, Westmead	102	72	32	**206**
	Royal Prince Alfred Hospital, Camperdown	55	25	8	**88**
	South West Area Pathology Service, Liverpool	131	112	21	**264**
Queensland	Queensland Medical Laboratory, West End	521	244	366	**1,131**
	Townsville General Hospital	-	-	-	**-**
South Australia	Institute of Medical and Veterinary Science, Adelaide	883	744	501	**2128**
Tasmania	Northern Tasmanian Pathology Service, Launceston	14	10	3	**27**
Victoria	Monash Medical Centre, Melbourne	14	-	-	**14**
	Rickettsia Reference Laboratory, Geelong	-	-	-	**-**
	Royal Children's Hospital, Melbourne	54	-	-	**54**
	Victorian Infectious Diseases Reference Laboratory, Fairfield	107	61	112	**280**
Western Australia	PathCentre Virology, Perth	428	205	231	**864**
	Princess Margaret Hospital, Perth	65	37	11	**113**
	Western Diagnostic Pathology	-	39	36	**75**
Total		**2,489**	**1,746**	**1,412**	**5,647**

1. The complete list of laboratories reporting for the 12 months, January to December 2001, 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 40-43 to 48-52, 2001

week number: / ending on:	40-43 28 October 2001		44-47 25 November 2001		48-52 30 December 2001	
Doctors reporting:	174		193		151	
Total encounters:	20,910		23,493		17677	
Condition	**Reports**	**Rate per 1,000 encounters**	**Reports**	**Rate per 1,000 encounters**	**Reports**	**Rate per 1,000 encounters**
Influenza	61	2.9	59	2.5	23	1.3
Influenza with culture	1	0.0	1	0.0	0	0.0
Chickenpox	40	1.9	46	2.0	41	2.3
Shingles	20	1.0	44	1.9	17	1.0

Additional reports

Australian encephalitis: Sentinel Chicken Surveillance Programme

Sentinel chicken flocks are used to monitor flaivirus activity in Australia. The main viruses of concern are Murray Valley encephalitis (MVE) and Kunjin. 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. Currently, 30 flocks are maintained in the north of Western Australia, 9 in the Northern Territory, 10 in New South Wales and 10 in Victoria. Two additional flocks will be set up in northern Queensland (at Mt Isa and Normanton) early in 2002. The flocks in Western Australia and the Northern Territory are tested all year round but those in New South Wales, Victoria and Queensland are tested only in the summer months, during the main MVE risk season.

Results are coordinated by the Arbovirus Laboratory in Perth and reported bimonthly. For more information see Commun Dis Intell 2002;26:57.

A Broom,[1] J Azuolas,[2] D Dwyer,[3] L Hueston,[3] JS Mackenzie,[4] L Melville,[5] D W Smith,[6] PI Whelan[7]

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2. *Veterinary Research Institute, Victoria*
3. *Virology Department, Westmead Hospital, New South Wales*
4. *Department of Microbiology, The University of Queensland*
5. *Berrimah Agricultural Research Centre, Northern Territory*
6. *Perth PathCentre, Western Australia*
7. *Department of Health and Community Services, Northern Territory*

November/December 2001

Sentinel chicken serology was carried out for 28 of the 29 flocks in Western Australia in November and December 2001. There were 2 confirmed seroconversions (1 MVE and 1 flavivirus) from Kununurra in the north-east Kimberley. There were also 2 seroconversions (1 MVE and 1 MVE/Kunjin) from Fitzroy Crossing, one MVE from Derby and one MVE from Broome (all sites in the West Kimberley) in December but these have yet to be confirmed.

Serum samples from six of the nine Northern Territory sentinel chicken flocks were tested at the University of Western Australia in November and December 2001. There was one seroconversion to MVE virus in the Katherine chickens in November.

The sentinel chicken programs in New South Wales and Victoria commenced in November 2001. There have been no flavivirus seroconversions reported in November or December 2001.

The State and Territory Health Departments provide funding for the sentinel chicken surveillance programs in Western Australia, the Northern Territory, New South Wales and Victoria.

Editor's note: This is the last Sentinel Chicken Surveillance Programme bi-monthly report to be published in Communicable Diseases Intelligence. From 2002 a Sentinel Chicken Surveillance Programme annual report will be published in Communicable Diseases Intelligence and future bimonthly reports will be published on the Communicable Diseases Australia Website at: http://www.health.gov.au.

Gonococcal surveillance

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

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

Reporting period 1 July to 30 September 2001

The AGSP laboratories examined a total of 913 isolates in this quarter. Another 16 strains were

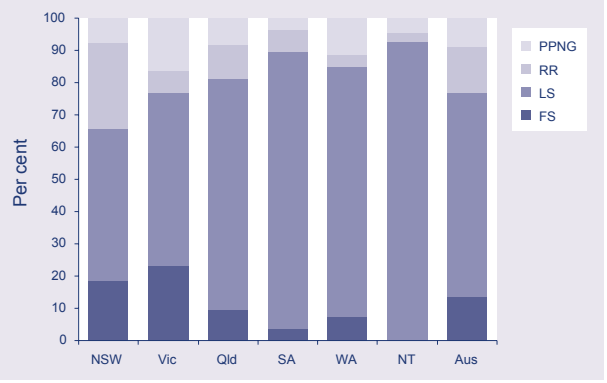
non-viable. This number is a considerable increase over the 794 examined in the same period in 2000. About 40 per cent of this total were from New South Wales, 22 per cent from Victoria, 15 per cent from Queensland, 13 per cent from the Northern Territory, 6 per cent from Western Australia and 2.5 per cent from South Australia. There were few isolates from other centres.

Penicillins

Figure 1 shows the proportions of gonococci fully sensitive (MIC ≤ 0.03 mg/L), less sensitive (MIC 0.06 – 1 mg/L), relatively resistant (MIC ≥ 1 mg/L) or else penicillinase producing (PPNG) aggregated for Australia and by State and Territory. A high proportion those strains classified as PPNG or else resistant by chromosomal mechanisms fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

In this quarter about 26 per cent of all isolates were penicillin resistant by one or more mechanisms – 7 per cent PPNG and 19 per cent by chromosomal mechanisms (CMRNG). The proportion of penicillin resistant strains ranged from 3 per cent in the Northern Territory to 36 per cent in Queensland.

Figure 1. Categorisation of gonococci isolated in Australia by penicillin susceptibility and by region, 1 July to 30 September 2001



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=66) was slightly less in this quarter than in the corresponding period in 2000 (n=70). The highest proportion of PPNG was found in isolates from South Australia (14%), Western Australia (13%) and Victoria (12%). PPNG were present in most jurisdictions including 1 (0.8%) in the Northern Territory. South and south-east Asian countries were the main source of external acquisition, but included an isolate acquired in Ireland. Local acquisition was prominent in Victoria.

More isolates were resistant to the penicillins by separate chromosomal mechanisms (n=173). These CMRNG were concentrated in Queensland (30% of isolates there), New South Wales (22%) and Victoria (21%). Three CMRNG were detected in the Northern Territory.

Ceftriaxone

Low numbers of isolates with decreased susceptibility to ceftriaxone were present in Victoria, New South Wales, Queensland and the Northern Territory. The persistence of these isolates in Australia and their presence in nearby countries[2,3] suggests that continued monitoring of this phenomenon is warranted. There is no evidence thus far that these strains with higher ceftriaxone MICs have been associated with treatment failure when third generation cephalosporins are used.

Spectinomycin

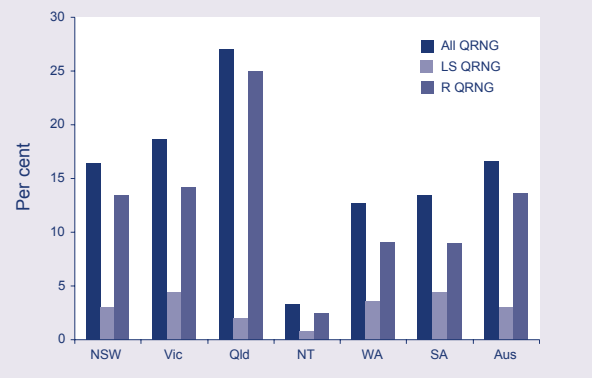
All isolates were susceptible to this injectable agent.

Quinolone antibiotics

Quinolone resistant *N. gonorrhoeae* (QRNG) are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06 – 0.5 mg/L) or resistant (MIC ≥ 1 mg/L) groups.

The total number of all QRNG (n=151) was again high and little changed from the previous quarter (n=165) and the September quarter in 2000 (n=142). QRNG were 16.6 per cent of all strains examined and this percentage was slightly lower than preceding periods in 2001 and the corresponding quarter in 2000. QRNG were again widely distributed. High rates were maintained in Queensland (27%), Victoria (18%), New South Wales (16%), South Australia (13%) and Western Australia (13%). Four QRNG were detected in the Northern Territory.

Figure 2. Distribution in Australia of *N. gonorrhoeae* showing quinolone resistance, 1 July to 30 September 2001



LS QRNG = Ciprofloxacin MICs 0.06 – 0.5 mg/L

R QRNG = Ciprofloxacin MICs ≥ 1 mg/L

In this quarter most of the QRNG exhibited higher levels of resistance as measured by MICs (Figure 11) and this is a continuation of a significant shift in the distribution of QRNG on the basis of MICs. In both New South Wales and Victoria in particular there has been a significant decrease in the number of 'less sensitive' QRNG in recent quarters.

Local acquisition was again prominent and MICs ranged up to 16mg/L.

High level tetracycline resistance

The number (n=89) and proportion (9%) of high level tetracycline resistance (TRNG) detected rose in this quarter from 56 (6.5%) in the June quarter. TRNG represented 12 per cent of isolates from Queensland and Victoria, 11 per cent from Western Australia, 9 per cent from New South Wales, and 2 per cent from the Northern Territory.

References

1. World Health Organization. Guidelines for the management of sexually transmitted infections. WHO/HIV_AIDS/(2001).01;WHO/RHR/o1.10:pp 1-5 World Health Organization, Geneva 2001.

2. WHO Western Pacific Region Gonococcal Antimicrobial Surveillance Programme. Surveillance of antibiotic susceptibility of *Neisseria gonorrhoeae* in the WHO Western Pacific Region 2000. *Commun Dis Intell* 2001;25:274-276.

3. Muratani T, Akasaka S, Kobayashi T, et al. Outbreak of cefozopran (penicillin, oral cephems and aztreonam) -- resistant *Neisseria gonorrhoeae* in Japan. *Antimicrob Agent Chemother* 2001:45:3603-3606.

HIV and AIDS surveillance

National surveillance for HIV disease is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with State and Territory health authorities and the Commonwealth of Australia. Cases of HIV infection are notified to the National HIV Database on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the State and Territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in HIV/AIDS and related Diseases in Australia Annual Surveillance Report. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Internet: http://www.med.unsw.edu.au/nchecr. Telephone: +61 2 9332 4648.
Facsimile: +61 2 9332 1837.

HIV and AIDS diagnoses and deaths following AIDS reported for 1 July to 30 September 2001, as reported to 31 December 2001, are included in this issue of Communicable Diseases Intelligence (Tables 7 and 8).

Table 7. Number of cases of newly diagnosed HIV infection and AIDS and number of deaths following AIDS occurring in the interval 1 July to 30 September 2001, and reported by 31 December 2001 by sex and State/Territory

	Sex	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Totals for Australia			
										This period 2001	This period 2000	Year to date 2001	Year to date 2000
HIV diagnoses	Female	0	7	0	2	3	0	7	3	22	22	67	**65**
	Male	3	99	1	7	9	0	28	7	154	159	467	**524**
	Not reported	0	0	0	0	0	0	0	0	0	0	2	**0**
	Total[1]	3	106	1	9	12	0	35	10	176	181	537	**591**
AIDS diagnoses	Female	0	2	0	1	1	0	3	0	7	7	12	**20**
	Male	0	14	0	6	4	0	9	0	33	34	89	**15**
	Total[1]	0	16	0	7	5	0	12	0	40	41	102	**174**
AIDS deaths	Female	0	2	0	1	0	0	2	0	5	1	8	**7**
	Male	2	10	0	3	5	0	4	0	24	27	52	**92**
	Total[1]	2	12	0	4	5	0	6	0	29	28	60	**99**

1. Persons whose sex was reported as transgender are included in the totals.

Table 8. Number of cases of newly diagnosed HIV infection and AIDS, and number of deaths following AIDS, cumulative to 30 September 2001 and reported by 31 December 2001 by sex and State/Territory

	Sex	State or Territory								
		ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Australia
HIV diagnoses	Female	27	664	10	175	69	5	243	130	**1,323**
	Male	230	11,486	111	2,114	715	80	4,140	967	**19,843**
	Not reported	0	244	0	0	0	0	24	0	**268**
	Total[1]	257	12,415	121	2,296	784	85	4,423	1,103	**21,484**
AIDS diagnoses	Female	9	208	0	51	26	3	76	27	**400**
	Male	88	4,791	37	876	360	45	1,720	363	**8,280**
	Total[1]	97	5,011	37	929	386	48	1,805	392	**8,705**
AIDS deaths	Female	4	118	0	35	16	2	53	17	**245**
	Male	70	3,271	24	587	241	29	1,311	256	**5,789**
	Total[1]	74	3,397	24	624	257	31	1,371	274	**6,052**

1. Persons whose sex was reported as transgender are included in the totals.

Childhood immunisation coverage

Tables 9 and 10 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR).

The data show the percentage of children fully immunised at age 12 months for the cohort born between 1 July to 30 September 2000 and at 24 months of age for the cohort born between 1 July to 30 September 1999 according to the Australian Standard Vaccination Schedule.

A full description of the methodology used can be found in Commun Dis Intell *1998;22:36-37.*

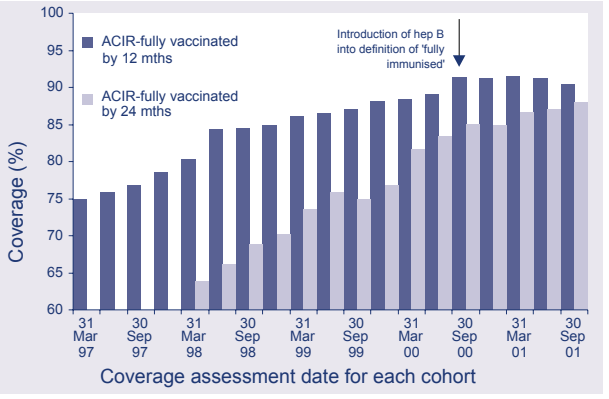
Commentary on the trends in ACIR data are provided by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases. For further information please contact the ACIR at: telephone: +61 2 9845 1255, E-mail: brynleyh@chw.edu.au.

The new National Health and Medical Research Council Australian Standard Vaccination Schedule, including universal hepatitis B vaccination commencing at birth, began for all children born on or after May 2000. This cohort (children born 1 July to the 30 September 2000) are the first eligible to follow the new schedule, which now requires receipt of 2 or 3 hepatitis B vaccines by 12 months of age to qualify for full immunisation.

Vaccination coverage for 'fully immunised' by 12 months for Australia has decreased from the last quarter by 0.8 percentage points but is still above 90 per cent (Table 1). Coverage decreased in all states and territories except in Tasmania where coverage increased by 0.3 percentage points to 91.3 per cent. Coverage is now below 90 per cent in 3 States, New South Wales, the Northern Territory and Western Australia. This decrease should not be a consequence of the introduction of hepatitis B vaccination, as hepatitis B is combined with the Diphtheria, Tetanus, Pertussis (DTP) vaccine or the *Haemophilus Influenzae* type B (Hib) vaccine in all jurisdictions. Unpublished analysis of the same 1 year olds by the Health Insurance Commission (HIC), has revealed no differences in 'fully immunised' coverage estimates when calculated with or without hepatitis B. This suggests that the decrease in 'fully immunised' coverage is not directly related to the introduction of hepatitis B vaccination. Nevertheless, as coverage for all individual vaccines for 12-month coverage is above 90 per cent in all jurisdictions, there must either be some parents who are selectively failing to immunise with some vaccines or a data problem with either notifications or data processing to the ACIR or both. In their regular parent surveys, the HIC have found that some parents have an objection to particular vaccines. It must also be remembered that the cohort reported on here is the first full 3-month cohort eligible to follow the new schedule. So, whilst the introduction of hepatitis B vaccination appears to have had little effect on 'fully immunised' coverage estimates, it is possible that changes in the administration and timing of the Hib and DTP vaccines in the new schedule may have had some effect on parents decisions to immunise or providers understanding of the new schedule.

In contrast, estimates of 'fully immunised' by 24 months for Australia (for which the requirements have not changed) has increased from the last quarter by one percentage point and is now 88 per cent (Table 2). Coverage increased in all States and Territories except for Western Australia with the largest increase occurring in the Northern Territory from 79.8 per cent to 83.5 per cent.

Figure 1. Trends in vaccination coverage, Australia, 31 March 1997 to 30 September 2001, by age cohorts



Source: Australian Childhood Immunisation Register

Table 9. Percentage of children immunised at 1 year of age, preliminary results by disease and State for the birth cohort 1 July to 30 September 2000; assessment date 31 December 2001

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	AUSTRALIA
Number of children	1,049	21,750	792	12,373	4,362	1,477	15,190	6,256	63,249
Vaccine									
Diphtheria, Tetanus and Pertussis (%)	92.4	91.8	88.8	92.7	92.5	92.6	93.1	90.7	92.2
Poliomyelitis (%)	92.2	91.7	89.1	92.6	92.4	92.4	93.1	90.6	92.1
Haemophilus influenzae type b (%)	93.7	93.8	93.1	94.7	94.5	95.5	94.8	93.9	94.3
Hepatitis B (%)	90.9	89.9	87.3	91.5	90.5	91.3	91.0	89.1	90.4
Fully Immunised (%)	93.7	93.8	93.1	94.7	94.5	95.5	94.8	93.9	94.3
Change in fully immunised since last quarter (%)	+1.8	-0.8	-2.1	-0.3	-1.1	+0.3	-1.0	-0.4	-0.8

Table 10. Percentage of children immunised at 2 years of age, preliminary results by disease and State for the birth cohort 1 July to 30 September 1999; assessment date 31 December 2001[1]

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	AUSTRALIA
Number of children	1,065	22,173	786	12,660	4,578	1,534	15,838	6,245	64,879
Vaccine									
Diphtheria, Tetanus and Pertussis (%)	92.0	89.1	85.8	91.9	91.7	92.6	91.0	88.3	90.3
Poliomyelitis (%)	95.3	93.7	93.9	94.4	95.4	96.0	95.3	93.1	94.3
Haemophilus influenzae type b (%)	96.4	95.0	91.9	95.0	96.2	96.6	96.2	94.0	95.3
Measles, Mumps & Rubella (%)	93.3	92.3	93.2	93.3	95.1	93.9	93.5	92.8	93.1
Fully Immunised (%)[2]	90.1	86.4	83.5	90.2	89.9	90.1	88.8	85.5	88.0
Change in fully immunised since last quarter (%)	+3.5	+0.7	+3.7	+1.6	+0.8	+1.4	+1.3	-0.5	+1.0

1. The 12 months age data for this cohort was published in *Commun Dis Intell* 2001;25:30
2. These data relating to 2 year old children should be considered as preliminary. The proportions shown as 'fully immunised' appear low when compared with the proportions for individual vaccines. This is at least partly due to poor identification of children on immunisation encounter forms.

Acknowledgment: These figures were provided by the Health Insurance Commission (HIC), to specifications provided by the Commonwealth Department of Health and Ageing. For further information on these figures or data on the Australian Childhood Immunisation Register please contact the Immunisation Section of HIC on telephone 02 6124 6607.

Bulletin board

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8th National Public Health Association of Australia (PHAA)
Immunisation conference
16-17 May 2002
Hilton on the Park
Melbourne, Victoria
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Facsimile: +61 2 6282 5438
E-mail: conference@phaa.net.au
Website: http://www.phaa.net.au

Australasian Sexual Health Conference
Come West 2002!
29 May-1 June 2002,
Sheraton Perth Hotel
Western Australia
Contact: Dart Associates
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E-mail: dartconv@mpx.com.au

Institute for Microbiology of Medical Faculty of Masaryk University & St Anna's Faculty Hospital
11th Tomasek Days
Annual conference of young microbiologists
5-7 June 2002
Brno, Czechia
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The Communicable Diseases Intelligence *bulletin board is provided as a service to readers. Every effort has been made to provide accurate information, but readers are advised to contact the relevant organisation for confirmation of details. Information about the availability of resources is included when space allows. Inclusion of a resource on the bulletin board does not imply endorsement by either the Communicable Diseases Network Australia or the Commonwealth Department of Health and Ageing.*

Contributions to the bulletin board are invited from those organisations with forthcoming events relevant to communicable diseases control. Details can be e-mailed to cdi.editor@health.gov.au.

Overseas briefs

World Health Organization

This material has been summarised from information on the World Health Organization Internet site. A link to this site can be found under 'Other Australian and international communicable diseases sites' on the Communicable Diseases Australia homepage.

Cholera in Nigeria

As of 26 November 2001, the World Health Organization (WHO) has reported a total of 2,050 cases of cholera and 80 deaths in Kano State in Kano Metropolis. WHO is working with the State Ministry of Health to control the outbreak. A cholera camp has been set up and mobilisation teams have been formed to trace contacts, carry out disinfection of houses and other areas, and provide health education. WHO has also supplied cholera kits.

One hundred and twenty cases of cholera have also been reported in Jigawa State. WHO is working with the Federal Ministry of Health and a team from Kano State to investigate this outbreak.

Ebola haemorrhagic fever outbreak now under control

The outbreak of Ebola haemorrhagic fever that has killed 34 people in Gabon and the Republic of Congo is under control, the World Health Organisation (WHO) said on 31 January 2002. "We are satisfied the epidemic has calmed," said David Heymann, head of WHO's Communicable Disease Division, who credited "a great government commitment" in Gabon for containing the outbreak. A total of 23 people have died in Gabon. The other 11 victims were in the neighbouring Republic of Congo. The United Nations health agency has relied on Gabonese efforts to halt the epidemic but would send international experts back to the region if necessary, Heymann said.

Tularemia - Kosovo

As of 17 January 2002 the Institute of Public Health in Pristina has reported 282 cases of tularemia since the outbreak began on 1 November 2001, 59 of which have been laboratory confirmed. There have been no deaths to date. The majority of cases have been detected in rural areas, mainly in the Lipjlan, Ferijaz, and Pristine municipalities. The patients have been between 16 and 44 years of age.

Tularemia is endemic in many parts of the world, including North America, eastern Europe, China, Japan, and Scandinavia. It is a bacterial disease normally transmitted from animal hosts and has various clinical manifestations. Symptoms include high fever and body aches, swollen glands and difficulty with swallowing, which continue for about 2 weeks.

The investigation of the outbreak continues, and measures for case management, environmental control, and health education are in place. The last outbreak of tularemia in Kosovo occurred in April 2000.

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.

Vaccine for some exposed to anthrax

Source: Reuters Online, 15 December 2001 (edited)

The United States government may offer anthrax vaccinations to some people exposed to the bio warfare agent in mail attacks so they would not get sick once they stop taking antibiotics. People exposed to high doses may still have potentially deadly anthrax spores in their lungs after taking the recommended 60-day course of antibiotics. As many as 3,000 people are at risk of having lingering spores and might be candidates for an anthrax vaccine. (This was approved on 18 December 2001; http://www.nytimes.com/ 2001/ 12/19/national/19VACC.html).

The vaccine is approved for preventing anthrax before exposure. Giving it after someone comes in contact with the biological warfare agent would be done on an experimental basis only. Alternatives to vaccination include advising people to watch for symptoms and to keep in close touch with their

doctors after their 60-day course ends or extending antibiotic treatment to 90 days. About 10,000 people were urged to take the 60-day treatment because they were exposed to anthrax through tainted letters sent to media outlets and 2 United States senators. No-one who has finished the 60-day therapy has developed anthrax.

Milk replacement possible source of BSE, Denmark

Source: Danish Veterinary and Food Administration, 21 December 2001

Denmark is investigating a possible link between bovine spongiform encephalopathy (BSE) and milk replacements. So far detailed reports have been completed on the first 3 cases of the 7 BSE cases in cattle born in Denmark. Two of these cases were born after the ban on the use of mammalian meat and bonemeal (MBM) to ruminants was put into force. It appears that the only feed which has been used in all 3 BSE-positive herds, is a German milk replacement, but contamination with MBM in other feed products used in the herds cannot be ruled out. According to the producer of the milk replacement, the products contain fat derived from bones from cattle and swine delivered from slaughterhouses in several European Union (EU) member states. At the time of production of the batches of milk replacements that were used in BSE infected herds, it was not obligatory to remove specified risk material in the slaughterhouses in all EU member states. Therefore there is a risk that the product might have been contaminated with fat produced from parts of spinal cord and brain from BSE positive cattle.

Denmark's BSE reference laboratory, the Danish Veterinary Laboratory (DVL), is currently conducting a risk evaluation on the use of animal fat as feed for ruminants and the risk entailed using the milk replacements on the market in Denmark. The risk evaluation is made with reference to a possible tightening of the rules concerning production, import, and use of these feed stuffs. DVL is also conducting an epidemiological investigation into the possible causal association of BSE and milk replacements. If the product in question has been used by most Danish farmers, a causal relation between a case of BSE and the use of this milk replacement on the farm is less likely. It is expected that the results of the above mentioned investigations might be available in mid-2002.

BSE cattle born after feed curbs cause concern

Source: The Guardian, 25 January 2002 (edited)

Scientists are trying to explain a sudden rise in the number of bovine spongiform encephalopathy (BSE) infected cattle born after tough feed controls were meant to throttle the disease. Over 6 weeks, 4 such animals have been diagnosed, bringing the total to 10, and more are likely now that the government has stepped up testing. The European commission has signalled it would consider imposing new export controls on British beef if the number rose to more than 50 in 12 months.

The latest case to be announced was a Friesian cow which had spent its entire life in Leicestershire, but there has been a wide geographical spread of cases, including 2 in Northern Ireland, causing difficulties for officials trying to pinpoint causes of infection, likely to have been well over 5 years ago.

Although officials say there is no food safety risk because the animals are too old to go into the food chain, frustration is growing among government advisers. There is increasing suspicion that mammalian meat and bone meal continued to reach calves after August 1996, when feed laws were strengthened, either through supplies being kept on farms or through cross contamination.

Scientists have hypothesised that some animals might get the disease when in the womb, but it is understood maternal transmission appears unlikely in most confirmed cases. The possibility exists that BSE is being spread on a small scale through pastures being contaminated by excrement from infected cows.

Opinions about the origin of BSE and other BSE-related issues

Source: European Commission Press Releases, 5 December 2001 (edited)

The Scientific Steering Committee (SSC), which advises the European Commission about bovine spongiform encephalopathy (BSE) and other multidisciplinary issues, published new opinions on the origin and transmission of BSE, on the BSE cases found in the United Kingdom (UK) among

cattle born after the ban on feeding meat-and-bone meal (MBM), and on the surveying requirements for obtaining reliable data on the prevalence of BSE and transmissible spongiform encephalopathies (TSE) in cattle, sheep, and goats. The committee also updated a standing opinion on the sourcing of ruminant materials for medical devices.

The opinion on the origin and transmission routes for BSE mainly confirms the standing scientific consensus hypotheses of a prion of unknown origin as the agent for transmitting the disease mainly via feed and cross contamination of feed, and to a lesser extent, via maternal transmission. The SSC considers that not one of the alternative hypotheses about a 'third' transmission route has so far been substantiated by scientific evidence. Evidence is equally very limited if not absent for hypotheses about factors influencing the susceptibility of cattle to BSE.

The 6 BSE cases found so far in the UK among cattle born after the August 1996 ban on feeding MBM to cattle currently give the SSC no reason to assume there is a higher BSE risk in the UK than previously assumed. Therefore there is no need to revise scientific advice on the UK Data Based Export Scheme (DBES) of any other BSE-related opinions.

The committee further adopted an opinion on the surveying and testing requirements for obtaining statistically authoritative and reliable data on the prevalence of BSE and TSE in cattle, sheep, and goat populations in the European Union. The opinion sets out the technical criteria for sample design, sample size, confidence intervals, etc. Sampling of the cattle population should be targeted on the group of so-called risk animals (for example, fallen stock). This also means the sample size can be kept significantly lower than in the case of sampling healthy animals sent for slaughter. In the goat and sheep population risk animals are much more difficult to identify. Therefore, the survey for most countries will need to be targeted at healthy animals sent for slaughter, and will need to cover a larger number of animals. Surveys would have to be accompanied by measures ensuring that animals suspected of being infected with TSE are not deliberately kept outside the testing program.

The SSC further updated its opinion on the safe sourcing of medicinal products from countries where BSE is highly unlikely to be present. The use of catgut sourced from such countries does not present a risk according to the scientists.

Scientists widen BSE checks to deer

Source: The Guardian, 24 December 2001 (edited)

Government scientists are to check deer to see whether they harbour BSE-like diseases under a research program designed to close loopholes in the battle against a menace that has probably killed more than 100 Britons since 1995 and dogged agriculture for nearly 10 years longer.

Precautionary steps to reassure officials about the safety of venison will involve collecting specimens from farmed animals to check their brains and tonsils for both BSE and a similar killer of deer and elk in the United States, chronic wasting disease (CWD). Britain's large wild deer population may also be monitored for the 2 diseases, although no laboratory experiment to ascertain whether BSE in cattle can be transmitted by injection or feed to deer has been attempted.

A Department of the Environment spokesman said that veterinary laboratories already cross-checked some deer samples collected for other purposes and had found no evidence of BSE-like diseases.

First case of BSE in Finland

Source: Ministry of Agriculture and Forestry, 7 December 2001 (edited)

The Finnish Ministry of Agriculture and Forestry has announced that Finland has its first confirmed case of bovine spongiform encephalopathy (BSE) in a cow. The case was confirmed in the EU reference laboratory on 7 December 2001. The cow had shown clinical signs of disorder and was slaughtered. The initial Western Blot examinations in the national reference laboratory had already shown strong evidence of a positive case.

The 6-year-old cow was born in Finland. No MBM has been used on the farm for over 20 years. There is not evidence as yet of the source of the infection. All bovine animals on the farm of origin, all cohort animals, and the progeny of the positive animal have been destroyed.

The identification of a clinical case of BSE in Finland underlines the great importance of clinical veterinary surveillance. Finland has carried out a

relatively low number of BSE tests in slaughtered cattle; figures can be obtained at: <http://www.bsereview.org.uk/data/cattle-tested-oct01.htm>.

Finland was identified, within the European Union assessment of the geographical BSE-risk (GBR) of European and other countries, to belong to 'Category II', where BSE is unlikely but it cannot be excluded that cattle is infected (clinical or sub-clinical) with the BSE agent. The report on Finland's GBR was published in July 2000: <http://europa.eu.int/comm/food/fs/sc/ssc/out118_en.pdf>.

Of the other European countries in Category II group, several have indeed been found infected since the publication of their relevant reports. During 2001, BSE has been identified for the first time in local cattle in Italy, the Czech Republic, the Slovak Republic, Slovenia and Greece, bringing the number of infected European countries to 17. So far, Japan is the only non-European country where BSE has been identified in non-imported cattle.

First case of BSE in Austria

Source: Office International des Epizooties, 14 December 2001 (edited)

On 6 December 2001, the preliminary rapid test for bovine spongiform encephalopathy (BSE) showed a positive result for a cow from the federal province of Lower Austria (Niederosterreich). On the same day that the BSE contingency plan came into effect, the provincial authorities were informed and movement restrictions were placed on all the animal carcasses at the slaughterhouse. The test was repeated on 7 December 2001 and followed by the immunohistochemical test as an additional method. Both the rapid test and the immunohistochemical test showed positive results.

Since 1 January 2001, all slaughtered bovines aged more than 30 months are subject to examination in the course of the all-Austrian Surveillance Programme. Furthermore, all suspect animals are also examined for BSE. The total number of examinations carried out up to 9 December 2001 was 217,970.

Quinacrine treatment of vCJD patient unsuccessful

Source: BBC News Online, 2 December 2001 (edited)

A British woman who became the first human case in trials to find a cure for variant Creutzfeldt-Jakob disease has died. The patient's condition improved dramatically after she received a course of the antimalarial drug quinacrine. It is thought that the 21-year old woman was taken off quinacrine after she showed signs of hepatotoxicity.

Reference

Korth C, May BC, Cohen FE, Prusiner SB. Acridine and phenothiazine derivatives as pharmacotherapeutics for prion disease. Proc Natl Acad Sci 2001;98:9836-9841.

Cholera hits Zanzibar

Source: TOMRIC News Agency (Dar es Salaam), 5 December 2001 (edited)

An outbreak of cholera is threatening the Zanzibaris. "At least 10 people have died in the latest cholera outbreak and the epidemic is spreading from urban to rural areas," the Director of the Preventive Service in the Zanzibar Ministry of Health, told reporters here yesterday.

Cholera in South Africa (KwaZuluNatal Province)

Source: Business Day, 4 January 2002 (edited)

The KwaZuluNatal health department has stepped up the provision of fresh water and has intensified its health education campaign following another cholera outbreak in the province. At least 119 out of the 140 new cholera cases in the province are in the Ladysmith area.

Cholera in Malawi

Source: Reuters, 4 January 2002 (edited)

A cholera outbreak in central and southern Malawi killed at least 28 people in December 2001. Controller of Preventive Health Services Habib Somanje said the country had recorded 28 deaths

out of a total of 1,394 cholera cases during the month.

The highly contagious waterborne disease first appeared in early December in the southern lakeside district of Mangochi, which borders Mozambique, before it spread to central Malawi. Cholera outbreaks are common in Malawi during the rainy season between October and April because of poor sanitation and limited access to potable water.

Dengue fever cases in the Cook Islands

Source: Pacific Islands News, Thursday 13 December 2001 (edited)

The Cook Islands recorded 3 cases of dengue fever in November 2001. These cases were reported between 26 and 28 November 2001 and all 3 victims contracted the illness in the capital, Rarotonga.

News that mosquitoes carrying the disease were at large in the Cook Islands came almost a month after 2 tourists arriving from French Polynesia were hospitalised with the illness. It is the second time this year that local cases have been reported after imported cases were discovered.

The Health Ministry said it had carried out an eradication program to wipe out the disease-carrying insects in the areas where the cases were reported. However, the recent bad weather delayed the spraying operations by several days. Other parts of the Pacific have experienced high levels of the potentially fatal disease this year.

A total of 9 cases have been reported to authorities in the Cook Islands this year, although 5 of these have been imported from abroad. The situation in the Cook Islands is far less serious than in other Pacific Nations. Samoa and French Polynesia have recently suffered from major outbreaks of the mosquito-borne disease.

Last foot and mouth disease infected area is classified disease free

Source: DEFRA press release 14 January 2002

Midnight 14/15 January saw a landmark in the fight against foot and mouth disease (FMD), when Northumberland was reclassified as FMD-free. It is not the official end of the FMD outbreak though it is a most welcome landmark.

The reclassification of Northumberland as FMD-free follows the reclassification at the start of the year of North Yorkshire, Cumbria and Durham and means that all counties in Britain now have FMD-free status so far as livestock movements are concerned.

The move follows the completion of a huge surveillance operation with over 3 million sheep tested for signs of the disease. The reclassification of Northumberland had been delayed due to the need for further detailed investigations into some blood test results which suggested that sheep could have been exposed to disease. These investigations indicated that no active virus was present, thus allowing Northumberland to attain FMD-free status.

The change in classification eases restrictions on animal movements. Livestock from Northumberland will now be able to move under local authority licence throughout the country.

Restrictions remain on some individual farms across GB which were culled out as infected premises or dangerous contacts until cleansing and disinfection work is complete or until 12 months has elapsed since preliminary disinfection if secondary cleansing and disinfection is not undertaken. Most cleansing and disinfection has been completed or will be done by the end of February 2002. A very small number are likely to remain under restriction in the coming months.

Restrictions also remain on exports. Meat and live pigs can be exported under control, though not yet from all counties. The EU Standing Veterinary Committee will consider over the coming weeks further easing of export controls.

Additional serological surveillance was undertaken in several counties, including Northumberland, where there were high sheep populations and a history of heavy infection, in order for the Chief Veterinary Officer to be confident about the disease status of flocks in those counties.

Ban on measles vaccination lifted

Source: El Moudjahid, 25 December 2001 (edited)

The study commissioned by the Minister of Health on the cause of the deaths of 7 out of 37 infants who had received measles vaccinations in the district of Mascara concluded that the deaths were due to 'a non-respect of rigorous procedures on preparation' of vaccines to be administered.

An epidemiological study was conducted by a team of specialists. The doctors who studied the

incident concluded that it was 'an isolated case of programmatic error'. There was an error in reconstitution of the vaccine with use of a solvent (as a diluent) other than that supplied by the Algerian Pasteur Institute.

The first investigations showed in effect that the lyophilised vaccine was reconstituted at the time of vaccination, and due to an error 'related to service management' was reconstituted with an inappropriate liquid, which was not sterile water and which – once administered to the infants – acted as a mortal poison. The exact nature of the product, its composition and the conditions behind its utilisation will be announced publicly once the epidemiological and toxicological investigations in Algeria and outside the country are completed.

Malaria cases increase sharply nationwide, Indonesia

Source: The Jakarta Post, Jakarta, 19 January 2002

Cases of malaria have been increasing since 1998, and have so far occurred in 13 provinces, 16 districts, and 106 villages throughout the country, affecting 15 million Indonesians.

The resurgence of malaria in Indonesia has been observed since 1997. In Java the incidence rose from 12 per 100,000 population in 1997 to 38 per 100,000 population in 1999. The increase in Java has been attributed to an increase in ponds used for fish farming. However, the World Health Organization (WHO) Regional Malaria Advisor stated in a WHO paper that 'adverse impact of economic crisis in the region has paralysed malaria control and caused focal outbreaks, more pronounced in Thailand and Indonesia. Conflict and civil unrest has exacerbated malaria in many countries of the region. Health services are scarce for people in need, particularly those who live in remote and border areas. Decentralisation inspired from political changes in Indonesia has caused confusion at implementation level and weakened the health services including malaria control'.

Rats may transmit hepatitis E virus in US

Rats roaming the streets of some United States (US) cities appear to be infected with a virus that is similar to the human hepatitis E virus (HEV), California researchers report. The investigators believe that rats might be the elusive source of this disease among urban residents who show signs of past exposure. In general, infection with HEV is not life threatening and lasts for one to 4 weeks. Other types of liver-infecting viruses, including hepatitis B and C viruses, can linger in the body for years and cause serious damage to the liver. HEV is relatively common in central Asia, but it is rare in the US and other developed nations.

'Although HEV infection is very, very rare in the US, there's a fair amount of antibodies for the disease among the derelict, inner city population and this research suggests that rats may be a source,' said study co-author Robert Purcell, head of the Hepatitis Viruses Section of the National Institute of Allergy and Infectious Diseases. A study of homeless people living in Los Angeles showed that 15 per cent had antibodies to HEV, which indicates they had been exposed to hepatitis E virus at some point in the past. "It certainly doesn't prove a connection, but if you're dealing with someone who's living in a region who might have contact with wild rats, it's something that doctors have to keep in mind," Purcell told Reuters Health.

Purcell pointed out that in many parts of the world the virus has developed into a long standing and serious problem. The infection can be life threatening in pregnant women. "HEV has become the single most important cause of acute clinical hepatitis among adults in much of Central Asia and South East Asia," he noted. "So although right now you can count American HEV cases on the fingers of one hand, the virus has risen from the exotic to the topical."

Yellow fever vaccine shortage threat

Source: BBC News Online, 21 December 2001 (edited)

Researchers have warned that there is too little yellow fever vaccine to cope with future outbreaks of the disease. Nicolas Nathan and colleagues highlight the problems encountered by Guinea in Africa, when it faced an outbreak in 2000.[1]

Yellow fever is a viral disease transmitted by mosquitoes. In the most severe outbreaks, over half of those affected can die. WHO estimates that 200,000 people in 34 countries across Africa and America are infected with yellow fever every year, leading to around 30,000 deaths. The disease can be prevented by vaccination, which protects for at least 10 years.

In their paper, Nicolas Nathan and colleagues[1] from Epicentre, Paris, France, state that: "Yellow fever epidemics are re-emerging in Africa and America, and the occurrence of repeated rural outbreaks increases the risk for major urban epidemics.

However, the international stocks of yellow fever vaccines are not sufficient to provide an adequate and rapid response to large outbreaks." He highlighted an yellow fever scare in Kano city, Nigeria, which has 1.5 million inhabitants, in 2000, and said if that had developed into an epidemic, there would not have been enough vaccine to cope.

The International Coordinating Group on Vaccine Provision for Epidemic Meningitis Control suggested that UNICEF's stockpile of 2 million doses should only be used in response to outbreaks. He said stockpiling would limit shortages, but added: "Prevention of yellow fever epidemics can only be addressed by organising pre-emptive mass vaccination campaigns or by large and effective introduction of yellow fever vaccination in the expanded programs of immunisation of the countries at risk, as recommended by the WHO."

Reference

1. Nathan N, Barry M, Van Herp M, Zeller H. Shortage of vaccines during a yellow fever outbreak in Guinea. Lancet 2001;358:2129-2130.

Vaccination-associated deaths in Brazil

Source: Virology, 290, No.2, 309-391, 25 Nov 2001 (Abstract, edited)

The yellow fever (YF) 17D virus is one of the most successful vaccines developed to date. Its use has been estimated to be over 400 million doses with an excellent record of safety. In the past 3 years, yellow fever vaccination was intensified in Brazil in response to higher risk of urban outbreaks of the disease. Two fatal adverse events temporally associated with YF vaccination were reported. Both cases had features similar to yellow fever disease, including hepatitis and multi-organ failure.

Two different lots of YF 17DD virus vaccine were administered to the affected patients and also to hundreds of thousands of other individuals without any other reported serious adverse events. The lots were prepared from the secondary seed, which has been in continuous use since 1984.

Nucleotide sequencing revealed minor variations at some nucleotide positions between the secondary seed lot virus and the virus isolates from patients; these differences were not consistent across the isolates, represented differences in the relative amount of each nucleotide in a heterogeneous position, and did not result in amino acid substitutions.

Inoculation of rhesus monkeys with the viruses isolated from the 2 patients by the intracerebral (ic) or intrahepatic (ih) route caused minimal viraemia and no clinical signs of infection or alterations in laboratory markers. Central nervous system histological scores of rhesus monkeys inoculated ic were within the expected range, and there were no histopathological lesions in animals inoculated ih.

Altogether, these results demonstrated the genetic stability and attenuated phenotype of the viruses that caused fatal illness in the 2 patients. Therefore, the fatal adverse events experienced by the vaccines are related to individual, genetically determined host factors that regulate cellular susceptibility to yellow fever virus. Such increased susceptibility, resulting in clinically overt disease expression, appears to be extremely rare.

Other

Reports from other sources.

Doctors' smoking cohort study ends

Source: News roundup, BMJ 2001;323:1270, 1 December 2001

After running for 50 years, the cohort study on the smoking habits of 40,000 British doctors, which helped to established the link between smoking and lung cancer, has ended with a valedictory thank you letter to the surviving doctors who were recruited in 1951.

Professor Sir Richard Doll, emeritus professor of medicine at Oxford University, who wrote the letter and was involved in the study since its inception, said it was devised by Sir Austin Bradford Hill to achieve maximum publicity for the critical relationship between smoking and lung cancer. The link was first established by him in a trial in 1947-49, but rejected by the Department of Health cancer committee, and not believed by a public, in which 80 per cent of men smoked.

Dr Maurice Gaba, was one of those recruited at the beginning of the study. Dr Gaba, said: "I was a forty a day man, when I received a letter in 1951 from a professor asking me about my smoking history, ending with a request to view my death certificate. I thought this doctor cares more about my health than I do, and I have never smoked since."

Adult deaths from chickenpox increasing in UK

Source: BMJ 2001;323:1091-1093. In: Reuters Health eLine, 12 November 2001

The number of adult deaths from chickenpox is increasing in England and Wales, according to the results of a study.

Professor Norman Noah from the London School of Hygiene and Tropical Medicine and colleagues reviewed the 1995 to 1997 death certificates from England and Wales in which chickenpox or varicella (the virus responsible for the disease) were mentioned. Of the 119 death certificates obtained, the study team estimated that 94, or 79 per cent, were genuinely attributable to chickenpox.

According to the authors, chickenpox is responsible for approximately 25 deaths annually, with fatality of around one in 10,000 cases. Deaths in adults accounted for 48 per cent of all deaths from chickenpox in 1967 to 1977, but have risen to make up 81 per cent of all deaths by 1986 to 1997, according to the report.

"General practitioners (GPs) see 4 children to every one adult with chickenpox, but the ratio of deaths is reversed, with 4 adults to every one child dying from the disease. Adults with chickenpox should see their GP sooner rather than later," Noah said. Deaths were twice as likely amongst men than women, and individuals born outside the United Kingdom (UK) were three times more likely to die than those born within the UK, according to the report.

"Further studies will be needed to reveal whether underlying conditions are responsible for the increased mortality in men," said Noah. "There is some evidence that individuals from tropical and temperate countries have a different experience of chickenpox. Adults from these countries are less likely to be immune to chickenpox, possibly because fewer get the disease during childhood," he added. "Chickenpox is responsible for more deaths than measles, mumps, whooping cough and (Haemophilus influenzae type B) meningitis combined. It is not a mild disease," he said.

WHO votes for smallpox reprieve

Source: New Scientist http://www.newscientist.com/news/news.jsp?id=ns9999180, 17 January 2002

The 32 nations who govern the World Health Organization have voted to put off destroying the last official stocks of smallpox virus. They have asked the WHO to set a new deadline for destroying the stocks in May, at the assembly of all 191 members of the organisation.

The virus samples were to have been destroyed this year, but in November the United States (US) decided it would keep its stocks to help develop new drugs and vaccines for smallpox. The US was considered unlikely to reverse that decision if WHO members had voted to destroy the virus.

Smallpox was declared eradicated in 1980, following a global vaccination campaign led by the WHO. The only officially remaining virus is in freezers at the Centers for Disease Control and Prevention in Atlanta, and at Vector, the Russian viral research institute at Koltsovo in Siberia.

These stocks were supposed to be destroyed in 1999, making the smallpox virus officially extinct, but the US president at the time, Bill Clinton, persuaded WHO members to postpone destroying them until December this year, so more research could be done on new vaccines and drugs, and on smallpox genes. The reason for the delay was a growing fear of smallpox as a weapon in a world no longer vaccinated for the disease.

Weaponised stocks

Not all smallpox may be in official hands. The Soviet Union weaponised 100 tonnes of the virus in the 1980s, and some may have escaped destruction. Jonathan Tucker of the Monterey Institute of International Studies in Washington says Iraq and North Korea are suspected of possessing the virus, partly because they have vaccinated their troops against smallpox.

In 1999, WHO members agreed to a smallpox research plan as part of the agreement to postpone destroying the virus. However, in December 2001, the WHO Secretariat reported that two of its goals for 2002; new anti-smallpox drugs, and an animal model for smallpox, would not be ready in time. It also said live smallpox virus would be needed to test any new drugs or vaccines.

In the wake of the anthrax attacks in the US, the US Department of Defence decided in November that smallpox stocks should not be destroyed before two anti-smallpox drugs, and a new, safer vaccine are licensed, along with new methods for detecting the virus and diagnosing infection.

CDI 2001 Subject index

A

B

C

D

E

F

G

H

I

J

K

L

M

N

O

P

Q

R

S

T

U

V

W

Y

Z

CDI Author Index 2001

M

O

P

R

S

T

V

W

Z

CDI reviewers 2001

The *CDI* staff wish to thank the following reviewers for their valued assistance throughout the year.

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Communicable Diseases Network Australia Website

Information on the Communicable Diseases Network Australia (CDNA) is now available on the Communicable Diseases Australia Website.

The Communicable Disease Network Australia was established in 1989 as the Communicable Diseases Control Network, as a joint initiative of the National Health and Medical Research Council and Australian Health Ministers' Advisory Council. Its aim was to oversee, the co-ordination of national communicable disease surveillance, the response to communicable disease outbreaks of national importance; and field training of communicable disease epidemiologists.

The CDNA Website can be accessed at: http://www.health.gov.au/pubhlth/cdi/cdna/index.htm