Annual report of the National Influenza Surveillance Scheme, 1999

Jennifer Thomson,[1] Ming Lin,[1] Alan Hampson[2]

Abstract

An effective national surveillance system is an essential component of a program for the control of influenza. The National Influenza Surveillance Scheme includes data from sentinel general practice consultations for influenza-like illness, laboratory reports of influenza and absenteeism rates from a national employer. The 1999 season peaked between May and September with maximal activity between July and August. Influenza A was the dominant type in all States and Territories with influenza A H3N2 viruses predominating and influenza A H1N1 occurring sporadically. There was no evidence of significant drift among the H3N2 isolates (A/Sydney-like strains) whereas the H1N1 isolates showed significant antigenic changes from the vaccine strain A/Beijing/262/95 and were closely related to a new variant A/New Caledonia/20/99. A small peak in influenza B activity occurred towards the end of the influenza season and isolates remained closely related to the vaccine reference strain B/Beijing/184/93. *Commun Dis Intell* 2000;24:145-152.

Keywords: surveillance, influenza, vaccine, antigenic drift, case definition

Introduction

Influenza infection is mostly associated with an acute self-limiting upper respiratory tract infection. However, complications may occur and these most commonly include lower respiratory tract infection - in particular primary and secondary pneumonia, exacerbation of chronic obstructive pulmonary disease[1,2] and exacerbation of cardio-pulmonary disease.[1] Influenza-related morbidity (measured as excess hospitalisation) and mortality are most often due to these

ISSN 0725-3141
Volume 24
Number 6
June 2000

1. National Centre for Disease Control, Department of Health and Aged Care, Canberra, Australian Capital Territory
2. World Health Organization Collaborating Centre for Influenza Reference and Research, Parkville, Victoria
Corresponding author: Dr Jennifer Thomson, National Centre for Disease Control, Department of Health and Aged Care, GPO Box 9848, Canberra, ACT, Australia 2601. Fax: (02) 6289 7791. E-mail: Jenny.Thomson@health.gov.au

Contents

cont'd next page

complications.[1,2] Although influenza infection affects all age groups, the rates of serious morbidity and mortality tend to be highest amongst those aged 65 years and over and those with chronic medical conditions.[1,3,4] Young infants and pregnant women are also recognised as being at increased risk of hospitalisation from influenza.[1,2]

Since 1977 influenza A (H1N1 and H3N2) and influenza B viruses have been in global circulation.[5] Influenza outbreaks usually occur during the winter months in temperate climates (peaking between December and March in the northern hemisphere and June and September in the southern hemisphere) but may occur throughout the year in tropical regions.[1,2,4,6] During an epidemic the overall attack rate is high such that even a low frequency of complications results in an increase in the number of hospitalisations and often in mortality.[1] Every 10-30 years influenza may cause pandemics in which a quarter or more of the population may be affected over a short period and during which the rates of illness and death from influenza can increase dramatically.[2,3]

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.[5,6] The major objectives of such surveillance include:[4]

(i) early detection of epidemics to enable the implementation of public health measures such as immunisation of the at risk groups, control campaigns and provision of clinical services,

(ii) characterisation of the nature of the epidemic and evaluation of the impact of the epidemic and associated public health measures, and

(iii) isolation and antigenic characterisation of influenza virus to assist in the formulation of the following season's vaccine.

The Annual Influenza Report provides a summary of the surveillance methods and data for the previous year (1999).

Surveillance Methods

Routine surveillance of influenza in Australia comprises three systems:[4,5,6]

- laboratory diagnosis including virus isolation and serology by laboratories participating in LabVISE (Laboratory Virology and Serology Reporting Scheme);
- consultation rates for clinically diagnosed influenza illness by sentinel general practitioners;
- absenteeism data of workers from a national employer.

At the National Centre for Disease Control from May to October these data are compiled, analysed and reported in *Communicable Diseases Intelligence (CDI)* regularly and a summary is included in the annual report of the National Influenza Surveillance Scheme.

Additional information is provided by the WHO Collaborating Centre for Reference and Research on Influenza and by Australia wide surveillance programs organised by pharmaceutical companies for the last two years (1998,1999) as part of multi-centre drug trials conducted in the Southern Hemisphere.

Laboratory surveillance (LabVISE)

Laboratory reports of influenza are sent to LabVISE all year round. This is a national scheme of sentinel laboratories Australia wide: in 1999 a total of 13 sentinel laboratories contributed to the scheme, although not all provided reports each month. Influenza diagnosis and reporting for the Northern Territory was referred to laboratories in Queensland, South Australia or Western Australia, and for Tasmania to one of the laboratories in Victoria. Additional data for 1999 were received directly from South Australia at the time of compilation of this report; these have been added to existing 1999 data provided through LabVISE from South Australia so duplication of data may have occurred. Although viral isolation remains the gold standard for influenza diagnosis and surveillance most reports have relied on the detection of viral antigen and serological markers. Nucleic acid detection by Polymerase Chain Reaction (PCR) is now in limited use and some notifications, especially from Western Australia and Victoria, have been based on this.

Criteria for a laboratory diagnosis of influenza are direct detection of viral antigen, or isolation of virus, or serological markers of recent infection. When performed, detection of viral RNA by PCR may also be used to indicate influenza infection.

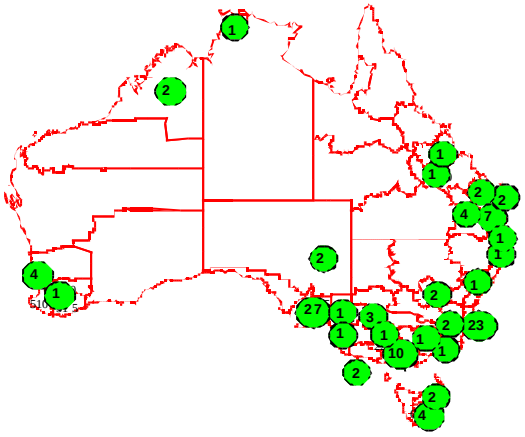
Sentinel general practitioner surveillance

Sentinel general practitioner surveillance schemes mostly detect and record clinical diagnoses of influenza-like illness. At a national (or multi-state) level there is ASPREN (Australian Sentinel Practice Research Network); and at a State or Territory level there are the New South Wales Sentinel General Practice Scheme, the Victorian Sentinel General Practice Scheme and the Northern Territory Tropical Influenza Surveillance Scheme. The New South Wales and Victorian schemes report cases of influenza-like illness from the beginning of May to September each year. ASPREN and the Northern Territory schemes report throughout the year. The ASPREN scheme is the only sentinel surveillance scheme that reports on cases of influenza-like illness from sentinel general practices located throughout Australia (Figure 1).

Most participating sentinel general practices are located in metropolitan areas and on the southeast coast. In all sentinel general practice surveillance systems, participation is voluntary; in 1999 the number of contributing practices

Contents, *continued*

Figure 1. Geographic distribution of ASPREN sentinel sites, by number of sites and location



varied from 30-62 per reporting period for the ASPREN scheme, 14-33 for the New South Wales scheme, 6-18 for the Victorian scheme and 6-18 for the Tropical Influenza Surveillance Scheme from the Northern Territory.

The case definition for a clinical diagnosis of an influenza-like illness varies between the different sentinel general practice surveillance schemes.

The case definition for ASPREN, and the Victorian and Northern Territory schemes is:

- viral culture or serological evidence of influenza virus infection; or
- influenza epidemic, plus four criteria listed below; or
- six of the following clinical criteria
  - sudden onset (within 12 hours),
  - cough,
  - rigours or chills,
  - fever,
  - prostration and weakness,
  - myalgia, widespread aches and pains,
  - no significant respiratory physical signs other than redness of nasal mucous membranes and throat,
  - influenza in close contacts.

An alternative, used by the New South Wales scheme, is :

- cough; and
- myalgia; and
- no abnormal respiratory physical signs other than redness of nasal mucous membranes and throat; and
- two of the following
  - sudden onset,
  - rigours or chills or fever,
  - prostration or weakness,
  - influenza in close contacts.

Absenteeism surveillance

Australia Post provided de-identified sick leave absenteeism data during 1999. Absenteeism was defined as an absence due to sickness for at least 3 consecutive days. This definition was used to increase the specificity for absenteeism related to influenza infection. Absenteeism was reported as the rate per 100 employees and rates were calculated on a weekly basis.

WHO Collaborating Centre for Reference and Research on Influenza

The WHO Collaborating Centre for Reference and Research on Influenza contributes reports on the subtypes and antigenic analysis of influenza viruses isolated throughout the year in Australia. This information is used to monitor the nature of influenza strains present in Australia and the rest of the world, assess suitability of the current vaccine (level of matching between circulating strains and the current vaccine) and determine the composition of the following year's vaccine.

Independent influenza surveillance program

A number of pharmaceutical and laboratory companies conducted surveillance programs in Australia from April to September 1999. The largest involved sentinel general practitioner sites selected from New South Wales, Queensland, Victoria, South Australia and Western Australia. Approximately 1,000 nose and throat swabs were collected from individuals fitting the ASPREN clinical case definitions for influenza-like illness (some States used different definitions) and these were processed for virus isolation. The laboratories involved in this program were the Institute of Clinical Pathology and Medical Research (New South Wales and Queensland), Victorian Infectious Diseases Reference Laboratory (Victoria), Institute of Medical and Veterinary Science (South Australia) and PathCentre (Western Australia).

Results

Laboratory surveillance (LabVISE)

A total of 3,247 reports was received with 2,861 for influenza A and 386 for influenza B. The ratio of influenza A to B was 7.4:1.

Total influenza reports showed a low grumbling baseline until May, a small peak of 85 reports per week in June (week 23), a second higher and broader peak from late June to early September (weeks 26 to 37) with a maximum of 212 reports per week (week 29) and then a decline to a low grumbling baseline that continued from mid-October until the end of December (weeks 41-53, Figure 2). This primarily reflected the pattern for influenza A which predominated with peaks of 78 reports per week in early June (week 23), 200 reports per week in mid-July (week 29) and 186 reports per week in early August (week 32). The pattern for influenza B was a low grumbling baseline with a delayed peak from late August to early September (33-37 weeks) with a maximum of 29 reports per week (35 and 37 weeks) (Figure 2).

The overall pattern of total influenza reports for 1998 and 1999 was similar, with a slightly delayed and lower and flatter peak of 803 reports in July 1999 compared with 980 reports in July 1998 (Figure 3).

Figure 2. Laboratory reports of influenza, Australia, 1999, by influenza type and week of specimen collection

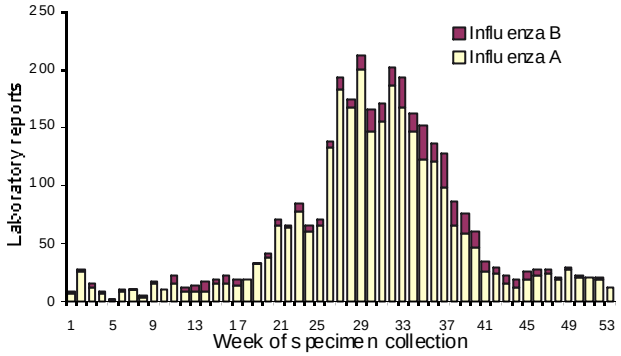


Figure 3. Laboratory reports of influenza, Australia, 1998 to 1999, by month of specimen collection

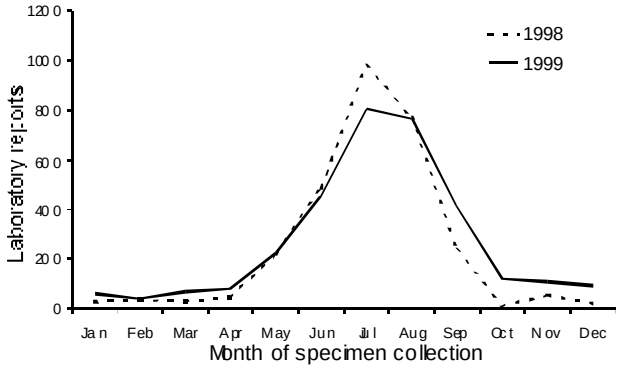
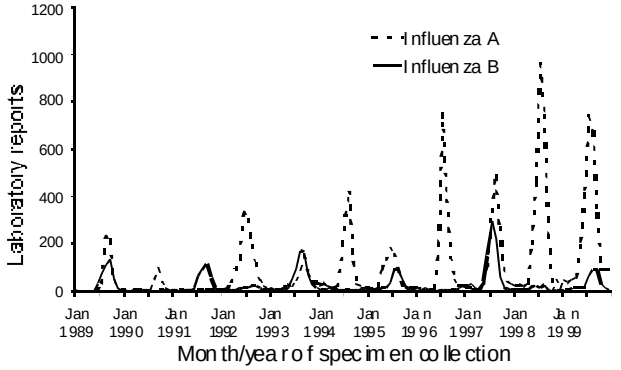


Figure 4. Influenza A and B laboratory reports, Australia, 1989 to 1999, by month/year of specimen collection



The pattern of influenza A and B reports was examined over a 10-year period from 1989 to 1999. Over this period annual peaks of influenza A occurred mostly in July with the highest peaks in 1998 (980 reports in July) followed by 1996 (758 reports in July) and then 1999 (740 reports in July). Peaks of influenza B mostly occurred biennially. The expected peak in 1999 was delayed and small (91 reports in August and 91 reports in September) in contrast with the previous highest peak of 301 reports in July 1997 (Figure 4).

The 15-44 year age range yielded the largest number of laboratory reports although a similar but slightly lower level of laboratory reports was reported for children aged 0-4 years. Overall there were similar numbers of reports for males and females (1,683:1,541 ie. 1.1:1). Figure 5 shows some variation in the ratio of males to females by age group.

The State or Territory of origin of laboratory influenza reports was based on the State or Territory of residence of the patient (Figure 6a). In 39% of reports (1,273/3,247) postcode of residence was unknown. For comparison a further figure has been provided using the State or Territory of the reporting influenza laboratory (Figure 6b). This comparison shows a similar pattern except that, using the State or Territory of residence of the patient, a small number of reports from the Northern Territory, Australian Capital Territory and Tasmania were seen and these were not seen using the State or Territory of the laboratory. No reports were seen from South Australia using the State or Territory of residence of the patient but reports from South Australia were seen when the State or Territory of the laboratory was used. Laboratory staff in South Australia confirmed that the reporting system that existed in 1999 did not include information on the State or Territory of residence of the patient (Dr Geoff Higgins, Institute of Medical and Veterinary Science, South Australia; personal communication). As information on the State or Territory of residence of the patient more accurately reflected the distribution of influenza activity, subsequent comments on the pattern of influenza relate to Figure 6a.

Of the 2,861 reports of influenza A, information on the State or Territory of residence of the patient was unknown for 38% (1,102). As South Australia did not provide information on the State or Territory of residence of the patient, all data from South Australia were included as part of the unknown

Figure 5. Laboratory reports of influenza, Australia, 1999, by age and sex

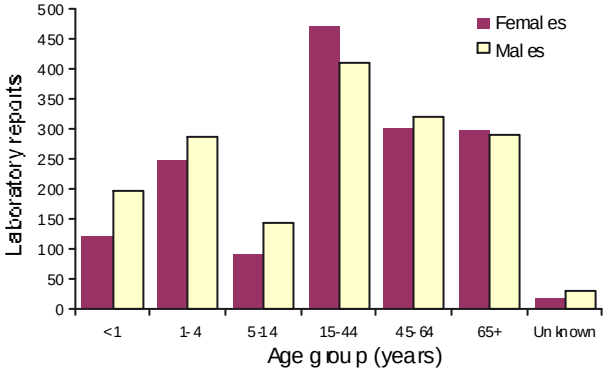


Figure 6a. Laboratory reports of influenza, Australia, 1999, by influenza type and State/Territory of residence

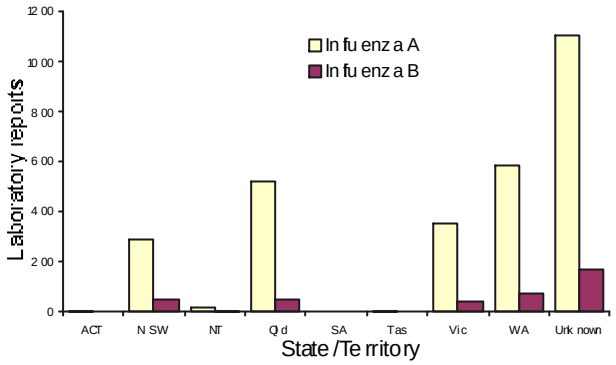


Figure 6b. Laboratory reports of influenza, Australia, 1999, by influenza type and State/Territory of laboratory

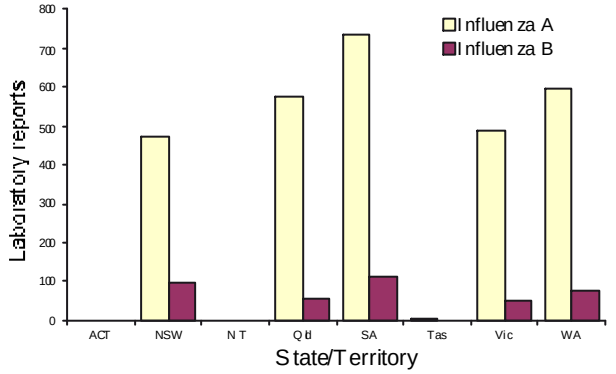
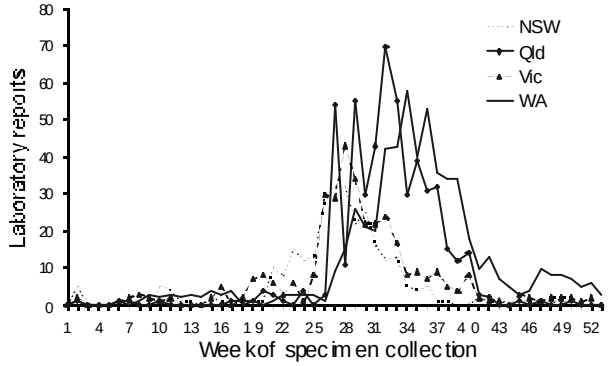


Figure 7. Influenza A laboratory reports, Australia, 1999, by week and State



group. Of the remaining reports with information on the State or Territory of residence of the patient (1,759) the greatest number was from Western Australia (582), followed by Queensland (521), Victoria (351), New South Wales (286), and the Northern Territory (16).

A smaller number of influenza B reports (386) was received from all States and Territories. The postcode of patient residence was unknown for 44% of reports (171/386). South Australia did not provide information on the State or Territory of residence of the patient, so all South Australian data were included in the unknown group. Of the remaining 215 reports with information on the State or Territory of residence of the patient, the greatest number was from Western Australia (73), followed by Queensland (50), New South Wales (47), Victoria (43) and the Northern Territory (2) (Figure 6a).

A breakdown of weekly reports for influenza A by State or Territory showed that the peak in reports occurred first in New South Wales (32 reports per week for weeks 27 and 28) followed by Victoria (43 reports per week for week 28) at the beginning of July, then Queensland (70 reports per week for week 32) in early August and later in Western Australia (58 reports per week for week 34) in mid-August (Figure 7). A low baseline of reports was received from the Northern Territory (maximum 4 reports per week), 2 reports from Tasmania, and one report from the Australian Capital Territory. South Australian data were included in the unknown group.

Breakdown of influenza B weekly reports by State and Territory showed that the appearance of influenza B was delayed and the pattern differed from that seen for influenza A. A peak in influenza B reports was first seen in New South Wales in July (5 reports per week for weeks 27, 30, 34 and 36), followed by Victoria in mid-August to early September (4 reports per week for weeks 33 and 37), Queensland from late August to mid-September (9 reports per week for week 35 and 8 reports per week for week 38) and last in Western Australia in mid-September (8 reports per week for week 37). The Northern Territory had a low baseline of 1 report per week. There were no reports from Tasmania nor the Australian Capital Territory. South Australian data were included in the unknown group.

Sentinel general practitioner (GP) surveillance

Both ASPREN and the New South Wales Sentinel General Practice Scheme showed a wide peak of GP attendances for influenza-like illness from mid-May to September 1999. The Victorian Sentinel General Practice Scheme showed a smaller peak in July. The Northern Territory Tropical Influenza Surveillance Scheme showed a clear bimodal pattern with peaks in March to April and July to September (Figure 8).

GP attendances for influenza-like illness peaked in the 15-44 year age group. Overall there was a similar rate amongst males and females, although males predominated in those younger than 14 years and in the peak age group of 15-44 years; and females predominated in those older than 45 years (Figure 9).

Comparison of ASPREN and LabVISE (Figure 10) showed a similar pattern and level of activity with the trend in ASPREN data followed about two weeks later by the trend in LabVISE data.

Figure 8. Sentinel general practitioner consultation rates, influenza-like illness, 1999, by week and sentinel surveillance scheme

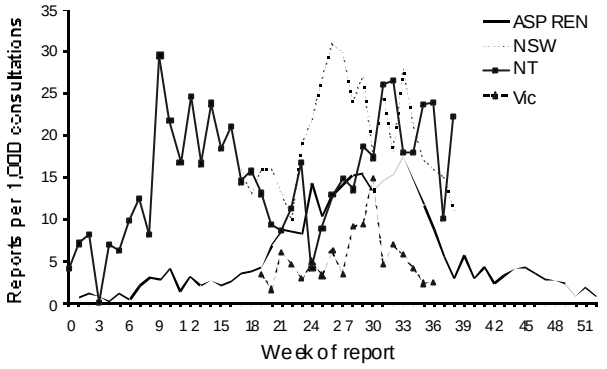


Figure 9. ASPREN reports of influenza-like illness, Australia, 1999, by age and sex

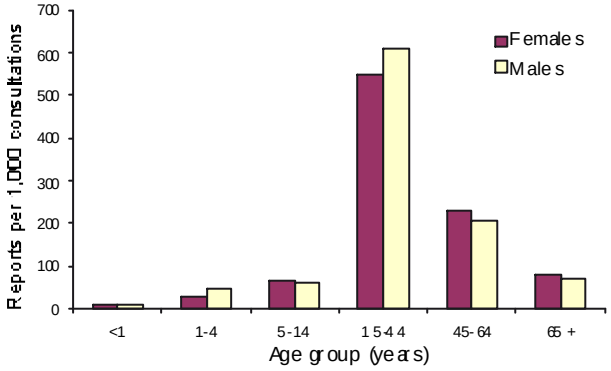
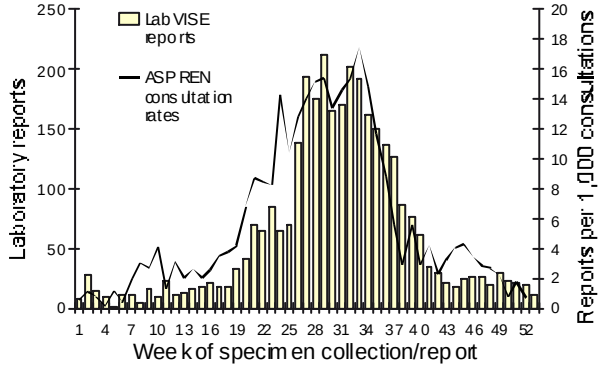


Figure 10. Comparison of LabVISE influenza reports and ASPREN influenza-like illness consultation rates, Australia, 1999, by week of specimen collection/report



Absenteeism surveillance

The national rate of absenteeism of three days per week reported by Australia Post was between 0.2% and 1% in the period from the end of March to September 1999 with the highest level of absence in August and September (Figure 11).

Independently conducted influenza surveillance program

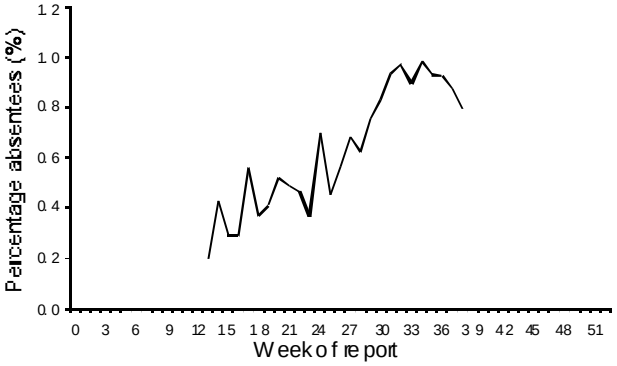
Influenza virus isolation and influenza-like illness rates peaked earlier on the east coast than the west coast, with maximal activity in June for Victoria (week 25) and South Australia (week 26), in July for New South Wales and Queensland (week 28) and in August for Western Australia (week 33). The overall ratio of influenza A to B was approximately 5:1, and rates were similar to 1998. Influenza B activity occurred later in the season. Most isolates were A Sydney 5/97 H3N2-like viruses

WHO Collaborating Centre for Reference and Research on Influenza

Influenza Virus Isolates

A total of 813 viable isolates of influenza was received from Australian laboratories for antigenic analysis, less than the number of viable isolates received in the two previous years (1,127 in 1998 and 1,178 in 1997) and representing 25% (813/3,247) of total influenza reports through LabVISE compared with 38% (1,127/2,943) for 1998. Of these 813 isolates, 683 were influenza A and 130 influenza B, a ratio of 5.3:1. The majority of influenza A strains were H3N2 subtype with only three H1N1 subtype isolates. Antigenic and genetic analysis indicated that the influenza A H3N2 subtype viruses were closely related to the A/Sydney/5/97 vaccine strain. As in the previous season there was some antigenic heterogeneity among these isolates, with approximately 20% reacting more strongly with antisera against a recent isolate A/Moscow/10/99 than the A/Sydney/5/97 vaccine strain. However, overall there was no evidence of substantial antigenic drift among the influenza A (H3N2) isolates. The three A (H1N1) isolates did demonstrate significant antigenic changes from the vaccine strain A/Beijing/262/95 and were closely related to a new

Figure 11. Percentage absenteeism in Australian Post, Australia, 1999, by week of report



variant A/New Caledonia/20/99. Influenza B isolates remained closely related to the vaccine reference strain B/Beijing/184/93.

World Trends

The pattern of influenza in Australia was similar to that seen in most parts of the world during 1999: influenza A/Sydney-like H3N2 subtype viruses predominated, influenza B/Beijing/184/93-like strains were present in smaller numbers and influenza A H1N1 subtypes mostly occurred sporadically. A/Sydney/5/97-like viruses were first identified in Australia (June 1997) and since then have spread around the world becoming the predominant strain in most regions.

The level of influenza activity in Australia was lower than many regions in which more severe outbreaks occurred. In particular, in the Pacific region there was significantly increased influenza activity reported in New Zealand and New Caledonia. In New Zealand the level of influenza activity (as measured by reports of influenza-like illness and laboratory-confirmed cases) was greater than in the two preceding years, although in 1998 influenza activity in New Zealand occurred at a low to moderate activity. New Caledonia recorded two significant outbreaks of influenza: the first was due to influenza A H3N2 A/Sydney-like viruses in February and March 1999; and the second was due to influenza A H1N1 viruses in May and June. Of importance, influenza A H1N1 viruses from the second outbreak in New Caledonia showed significant antigenic-drift from the A/Beijing/262/95 vaccine reference strain. Subsequently this new influenza A (H1N1) variant (A/New Caledonia-like strains) became prominent in many parts of Asia during the latter part of 1999.

Discussion

Overall there was consistency in the pattern of virological findings from LabVISE, the WHO Centre and the pharmaceutical surveillance program. This is reassuring, as virological isolation is the recognised gold standard.[7]

The 1999 influenza season in Australia had the third highest level of laboratory diagnosed influenza A for the last 10 years. The predicted biennial influenza B peak was small and delayed. Overall the timing of influenza activity in 1999 was slightly delayed compared with 1998, with a flatter and slightly lower peak from May to August. The overall pattern primarily reflected the pattern for influenza A, with a peak in influenza A reports from May to August, first in New South Wales and then Victoria in July, followed by Queensland in early August and then Western Australia in mid-August. Influenza B weekly reports peaked from July to September, first in New South Wales in early July, followed by Victoria and then Queensland in August and Western Australia again in September. These numbers and patterns need to be interpreted carefully as referral patterns could contribute to the observed pattern. South Australia did not provide information on the State or Territory of residence of the patient, so all data from South Australia were included in the unknown group.

The results from the pharmaceutical surveillance program were similar to the LabVISE results with influenza A predominating. There was also some similarity with respect to the timing and location of the peak for influenza overall, as well as for influenza A and influenza B individually. Although such surveillance programs are very useful and are used by pharmaceutical companies to trigger enrolment in clinical trials, they are transitory in their funding. One pharmaceutical company has discontinued its virus isolation based surveillance program, although another is continuing a similar program in 2000. A number of companies are undertaking influenza surveillance based on rapid point of care (or bedside) influenza tests, but their role in surveillance is as yet unknown, particularly in the absence of confirmatory virus isolations. As there is a risk that the plethora of surveillance schemes will lead to confusion and competition for sentinel practices, it is preferable that efforts and funding are directed towards a nationally consistent scheme as advocated in the Australian Influenza Pandemic Preparedness Plan.[5] Such a scheme could then act as a single source of information for a variety of purposes. It would also allow the proper evaluation of the new point-of-care tests and their role in surveillance.

Viable influenza isolates received by the WHO Centre for 1999 were less than the numbers received for each of the last 2 years. Most circulating influenza A strains were the H3N2 subtype with only three H1N1 subtype isolates. Of note there was evidence of significant antigenic drift amongst the H1N1 subtype isolates from the vaccine strain A/Beijing/262/95 to strains closely related to a new variant A/New Caledonia/20/99.

In general the trend of the GP surveillance schemes mirrored the pattern seen for laboratory reports from LabVISE, with a peak from May to September. As seen in 1998 there was a delay of two weeks between the timing of the peak for GP surveillance schemes and the LabVISE surveillance scheme. The lag may reflect a delay in laboratory testing and reporting. As in previous years, the Northern Territory Tropical Influenza Surveillance Scheme showed a bimodal pattern with an early peak in March to April heralding the beginning of the influenza season.

The expected predominance amongst the elderly of influenza reports or consultations was not seen in the collected surveillance data. The predominant age groups varied between the GP surveillance scheme and LabVISE. Most cases of influenza-like illness from GP surveillance were reported from the 15-44 year age range. In contrast the main groups affected by influenza according to LabVISE were children aged 0-4 years and adults aged 15-44 years. The laboratory reporting pattern may reflect selection bias in that samples for testing were more likely to be taken from children presenting to hospitals with respiratory symptoms. The GP consultation pattern may also reflect selection bias in that this age group largely reflects working adults who may require a GP consultation for the purpose of certification for work or symptom management. Laboratory-based surveillance in Western Australia has noted that direct detection data is skewed towards children who go to hospital and adults who go to surveillance sites. However, serological data which reflects more significant adult disease, particularly lower respiratory tract infection, showed a peak in the elderly for 1999 (Dr David Smith, PathCentre, Western Australia; personal communication).

The predominant age groups seen in 1999 were as seen in 1998. Australian Bureau of Statistics mortality data now available for 1998 show that peak mortality occurred in the older age groups. Overall, mortality data from 1998 showed a decline from 1997 levels, but the decline in mortality due to pneumonia was smaller. Similar mortality rates were seen

for males and females with overall mortality most frequent in those aged greater than 79 years and pneumonia-related mortality greatest in those aged greater than 74 years.

The number of influenza-like illness consultations per 1,000 consultations per week in 1999 peaked at 30, an increase from 1998 which peaked at 25, but less than 1997 which peaked at 35 and similar to 1995 and 1996.[6] In contrast, laboratory reports from LabVISE decreased in 1999 compared with 1998,[6] which may reflect less laboratory testing of suspected influenza cases or may reflect a different age distribution affected by influenza with consequent differences in testing.

National absenteeism rates reported by Australia Post showed a broad peak from July to September consistent with information from the other surveillance systems. This may reflect the maturing of this surveillance system as well as the high incidence of influenza.

The frequent development of antigenic variants through antigenic drift is the virological basis for seasonal epidemics and the reason for the incorporation of one or more new strains in each year's influenza vaccine.[3] The composition of the influenza vaccine for 2000 was determined on the basis of the strains in circulation during 1999 in Australia and the rest of the world. The Australian Influenza Vaccine Committee (AIVC), in conjunction with the WHO recommendations, decided that the influenza vaccine composition for the year 2000 season should be as follows:[8]

- H1N1 A/New Caledonia/20/99-like strain
- H3N2 A/Sydney/5/97-like strain
- B B/Beijing/184/93-like strain

This differs from the 1999 vaccine by the replacement of the A/Beijing/262/95-like strain with the A/New Caledonia/ 20/99-like strain due to the detected antigenic drift.

Influenza vaccination is the primary method for preventing or attenuating influenza infection and its more severe complications. Vaccination is offered in the autumn and is primarily targeted at people aged 65 years and over, and people under 65 years with chronic underlying medical conditions.[2,3] More recently, additional groups for whom influenza vaccination is suggested include those with respiratory disease and pregnant women who will be in their second and third trimester during the influenza season.

Awareness among health care providers of current influenza activity and circulating strains is necessary for reducing the impact of influenza and related complications. As an integral part of control of influenza the National Influenza Surveillance Scheme will continue conducting surveillance in the winter of 2000.[2]

Acknowledgements

We would like to thank all contributors and their staff for the collection of these data. They include: the Australian Sentinel Practice Research Network; *Communicable Diseases Intelligence* Virology and Serology Laboratory Reporting Scheme reporting laboratories (LabVISE); New South Wales Department of Health; Territory Health Services; Victorian Department of Human Services; Australia Post; and the World Health Organization (WHO) Collaborating Centre for Influenza Reference and Research. In addition we would like to also thank those contributing to the independently conducted influenza surveillance program.

Special thanks to Dominic Dwyer (Virology, ICPMR, New South Wales), David Smith (PathCentre, West Australia), Heath Kelly (VIDRL, Victoria), Geoff Higgins (IMVS, South Australia) and Ian Wilson for their comments and assistance in the preparation of this report.

Thanks also to staff of the Surveillance and Management Section, in particular Linda Halliday and Alison Milton.

References

1. Department of Communicable Disease Surveillance and Control, World Health Organization. Influenza Pandemic Preparedness Plan: The role of WHO and guidelines for national or regional planning. Geneva:Switzerland,1999.
2. National Health and Medical Research Council. The Australian Immunisation Handbook. 7th ed. Canberra: Australian Government Publishing Services, 2000.
3. Advisory Committee on Immunization Practices. Prevention and control of influenza: Recommendations of the Advisory Committee on Immunization Practices. *MMWR Morb Mortal Wkly Rep* 2000;49[No.RR-3]:1-38.
4. Curran M, Hampson A. National influenza surveillance, 1997. *Commun Dis Intell* 1998:22:69-74.
5. Influenza Pandemic Planning Committee for the Communicable Diseases Network - Australia and New Zealand. A framework for an Australian influenza pandemic plan: version 1. *Commun Dis Intell* Technical Report Series No. 4 1999:1-179.
6. Halliday L, Roberts L, Hampson A. Annual report of the National Influenza Surveillance Scheme 1998. *Commun Dis Intell* 1999;22:69-74.
7. Quenel P, Dab W, Hannoun C, Cohen JM. Sensitivity, specificity and predictive values of health service based indicators for the surveillance of influenza A epidemics. *Int J Epidemiol* 1994;23:849-855.
8. Australian Influenza Vaccine Committee. Australian recommendations for the influenza vaccine composition for the year 2000 season. *Commun Dis Intell* 1999;23:323.

Review of leptospirosis notifications in Queensland and Australia: January 1998 – June 1999

Lee Smythe,[1] Michael Dohnt,[1] Meegan Symonds,[1] Leonie Barnett,[1] Michael Moore,[1] Dianne Brookes,[2] Mary Vallanjon[3]

Abstract

The World Health Organization/Food and Agricultural Organization Collaborating Centre for Reference and Research on Leptospirosis, Western Pacific Region, accredited since 1958, is part of Queensland Health Scientific Services, which provide tertiary level support in epidemiology, surveillance, training and diagnosis for hospitals and pathology laboratories across the State. Databases for leptospirosis on a global, Australian and State-wide basis are maintained on site and support public health authorities in Australia, WHO and the International Leptospirosis Society. Queensland data collated and analysed from leptospirosis questionnaires, and a brief overview of Australian data based on questionnaire responses for notified cases from 1998 to June 1999, are summarised. The increase in leptospirosis notifications (77%) during 1998 possibly signalled greater awareness of the disease by clinicians. There was a significant increase in leptospirosis notifications for children and students and a high rate of hospitalisation of cases. An outbreak in North Queensland during the first half of 1999 resulted in 184 notifications with over 50% of cases hospitalised. Polymorphic presentation of the disease with severe pulmonary haemorrhage is associated in particular with the serovar australis. Serovar zanoni continues to be a major cause of severe clinical leptospirosis. Several cases were diagnosed in tourists. One of these cases presented with severe respiratory distress and required 14 days in hospital. *Commun Dis Intell* 2000;24:153-157.

Keywords: leptospirosis, surveillance, clinical, occupation, activity, rat, dog, cattle

Introduction

Leptospirosis was first recognised in Australia in 1934 among cane-cutters of Ingham, North Queensland.[1] It is an acute febrile disease occurring in humans and animals worldwide.[2] There are more than 250 pathogenic serovars of *Leptospira interrogans*. The disease is potentially lethal, with involvement of the hepatic, renal and central nervous systems. The source of infection is water or soil that has been contaminated with the infected urine of wild, feral or domestic animals.[3]

Leptospirosis occurs in all parts of Australia with the highest incidence of the disease in Queensland and Victoria.[4] Leptospirosis is a notifiable disease in all States and Territories of Australia. A laboratory based notification system was introduced in Queensland from 1988, requiring all laboratory diagnoses to be reported to the Communicable Diseases Branch of Queensland Health. Prior to 1988 all notifications were practitioner initiated.

Methods

The World Health Organisation/Food and Agricultural Organization Collaborating Centre for Reference and Research on Leptospirosis has sought information on incidence of leptospirosis from 1985 to date in Australia through notifications and a mail questionnaire. The response rate of clinicians, laboratory staff and public health units to the questionnaire in Queensland was exceptional at over 99%. Response rates are steadily increasing for the Australia-wide aspect of the program.

Since 1988 notifications of leptospirosis for Queensland have been based on isolation of the organism, or positive serology. The latter was defined as a four-fold or greater change in Leptospira Microscopic Agglutination Test (MAT) titre, or a single raised MAT titre equal to or greater than 400. This may be supported through demonstration of elevated leptospiral IgM using an Enzyme Linked Immunosorbent Assay (ELISA). The National Health and Medical Research Council (NHMRC) notification criteria for leptospirosis are similar to, but vary from, those of Queensland Health. The NHMRC criterion of requiring a 'single raised agglutination titre with clinically compatible illness' may not accurately represent all leptospirosis cases due to the non-specific nature of symptoms of leptospirosis in humans and unspecified determination of a raised agglutination titre value. The NHMRC notification criteria also do not specify the MAT as the serological test of preference. The MAT is the internationally recognised method for the diagnosis of leptospirosis and is most commonly constructed with a panel of serovars representative for a region.[5] Other serological test methods cannot provide a determination of the infecting serovar or serogroup.

1. WHO/FAO Collaborating Centre for Reference and Research on Leptospirosis, 39 Kessels Road, Coopers Plains, Queensland, Australia 4108
2. Tropical Public Health Unit, Cairns, Queensland, Australia 4870
3. World Health Organization, Geneva, Switzerland

Corresponding author: Lee Smythe, Queensland Health Scientific Services, PO Box 594, Archerfield, Queensland, Australia 4108. Fax: (07) 3274 9175. E-mail: Lee_Smythe@health.qld.gov.au

The reference laboratory receives either tertiary specimens for confirmation by MAT, or primary specimens on which an ELISA and/or the MAT are performed. Specimens are tested against a panel of 21 serovars. This panel of 21 serovars is representative of all the known serogroups in the Western Pacific Region. Due to known cross-reactions at the serogroup level, these 21 serovars provide the optimum regional panel for testing local and overseas sera. This panel includes representatives previously recovered from humans in Australia.

In 1996 the laboratory commenced a program of placing Ellingshausen-McCullough-Johnson-Harris (EMJH) agar (specific isolation medium for *Leptospira*) in Queensland Public Health Hospitals in areas where leptospirosis cases were relatively common. It was requested that all suspect leptospirosis cases and cases with pyrexia of unknown origin have blood collected and cultured for attempted isolation of *Leptospira*. A specimen collection chart to indicate the ideal times for collection of samples for culture and serology was produced, and copies forwarded to clients.

The questionnaire component of the study commenced in 1991. It sought information on occupation, animal contacts, outdoor recreational activities, travel, age, gender, symptoms and location of residence prior to onset of symptoms. Questionnaires were sent to the referring doctors of those patients whose leptospiral serology was positive or from whom a leptospire was isolated.

Results

Queensland

For the year 1998 there was a total of 108 notifications in Queensland - a 77% increase in cases compared with 1997 when 61 cases were reported. The 108 notifications represent an incidence rate of 3.2 per 100,000 population, compared with a rate of 2.2 per 100,000 for 1997. The Queensland notifications represent 60% of all Australian cases for 1998. From 1 January 1999 to 30 June 1999 a total of 184 notifications was reported to Queensland Health. The average age at diagnosis for Queensland was 35.0 years for males and 35.7 years for females, with an average of 35.1 years for all notifications.

The most common case symptoms reported for Queensland and Australia for 1998 to the end of June 1999 are shown in Table 1. These symptoms resemble those of many other pyrexia of unknown origin, and frequently leptospirosis may not be considered in the diagnosis by the clinician.

Hospitalisation information has only recently been collected through the questionnaires. Of 216 respondents, 128 (59.3%) had been hospitalised for an average of 5 days. The maximum time of hospitalisation was 27 days and the minimum one day. Serovars australis and zanoni accounted for the majority of hospitalisations. It is hoped that previous notifications can be followed up and hospitalisation information collated for 1998 for Australia as a whole.

Of the 292 Queensland notifications, all reported contact with animals. Significant exposures were to rats at 170 (58.2%), dogs at 132 (45.2%) and cattle at 110 (37.7%) (Figure 1). Figure 1 also compares reported animal contacts for Australian notifications. In many instances exposure could be related to activity e.g. rats, feral pigs and mice with the banana and related fruit industries, and cattle with abattoirs and meat processing industries. The banana industry accounted for the majority of notifications at 74 (25.3%) (Table 2). Clinicians need to be aware of occupations and activities not traditionally implicated in the disease since 31.9% of the notifications were associated with these.

In occupations based on the animal industry, serovars hardjo, pomona and zanoni accounted for the majority of cases (Figure 2). Serovars zanoni and australis were implicated in most of the cases in the agricultural industries. Overall, the serovars hardjo, zanoni and australis accounted for 182 (62%) of the notifications (Figure 3).

In the review period, 78 *Leptospira* isolates were recovered from 539 isolation attempts (14.5% recovery) (Table 3). There were 38 isolations from patients without follow up convalescent serology. Without these cultures, 13.0% (38 of 292) of notifications would have been missed.

Figure 1. Notifications of leptospirosis, Queensland and Australia, 1 January 1998 to 30 June 1999, by frequency of animal contacts

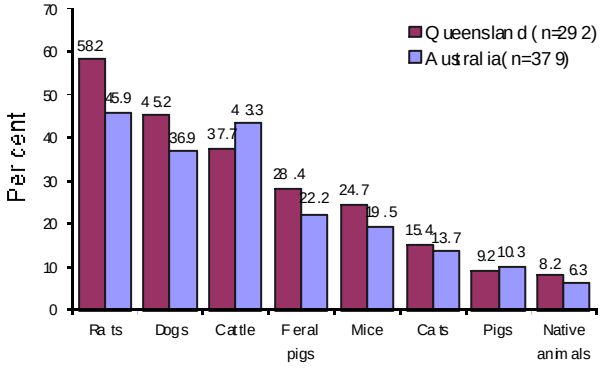


Figure 2. Notifications of *Leptospira*, Queensland, 1 January 1998 to 30 June 1999, by serovar in animal industry-based occupations (n=76)

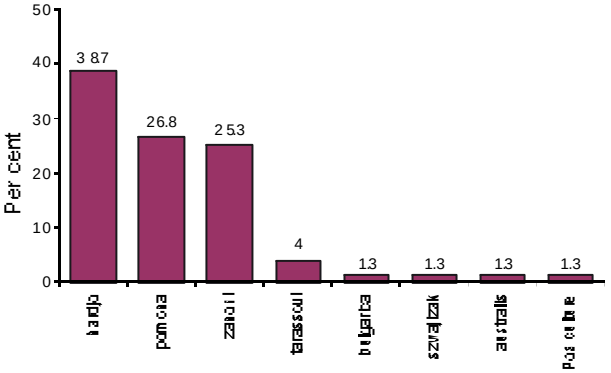


Table 1. Notifications of leptospirosis in Queensland and Australia, 1 January 1998 to 30 June 1999, by frequency of reported symptoms

	Notifications for Queensland (n = 292)		Notifications for Australia (n = 381)	
Symptom	Number	%	Number	%
Headache	225	77.1	280	73.5
Severe fever	217	74.3	254	66.7
Chills	208	71.2	253	66.4
Sweats	205	70.2	253	66.4
Myalgia	201	68.8	259	68.0
Nausea/vomiting	179	61.3	213	55.9
Arthralgia	150	51.4	183	48.0
Back pain	102	34.9	127	33.3
Conjunctival suffusion	71	24.4	80	21.0
Mild fever	63	21.6	92	24.1
Renal involvement	48	16.4	60	15.7
Respiratory symptoms	48	16.4	59	15.5
Vision disturbance	35	12.0	37	9.7
Diarrhoea	22	7.5	25	6.6
Rash	18	6.2	23	6.0
Pulmonary haemorrhage	13	4.5	15	3.9

Table 2. Notifications of leptospirosis, Queensland and Australia, 1 January 1998 to 30 June 1999, by occupation or activity

	Notifications for Queensland		Notifications for Australia	
Occupation or activity	Number	%	Number	%
Banana farmers[1]	74	25.3	74	19.4
Meatworkers[1]	36	12.3	67	17.6
Dairy farmers[1]	25	8.6	43	11.3
Children/students	20	6.8	20	5.2
Farmers[1]	15	5.1	20	5.2
Agricultural/rural workers[1]	10	3.4	10	2.6
Cane farmers[1]	9	3.1	9	2.4
Graziers[1]	9	3.1	11	2.9
Building labourers	8	2.7	8	2.1
Transport workers	7	2.4	7	1.8
Station hands[1]	6	2.1	9	2.4
Home duties	4	1.4	4	1.0
Nursery worker/landscaper	4	1.4	4	1.0
Tradesperson	4	1.4	5	1.3
White water rafting guides	4	1.4	4	1.0
Unemployed	4	1.4	5	1.3
Mechanic	3	1.0	4	1.0
Clerical duties	3	1.0	3	0.8
Retired	2	0.7	3	0.8
Tourist	2	0.7	4	1.0
Other	28	9.6	32	8.4
Unknown	15	5.1	35	9.2
Total	292	100	381	99.7

1. Occupations traditionally associated with leptospirosis

Figure 3. Notifications of *Leptospira*, Queensland, 1 January 1998 to 30 June 1999, by serovar as percentages (n = 292)

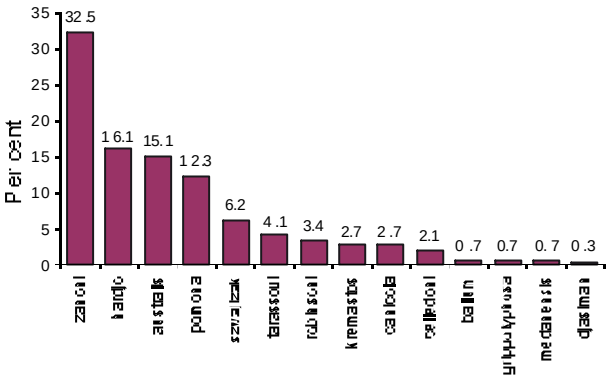
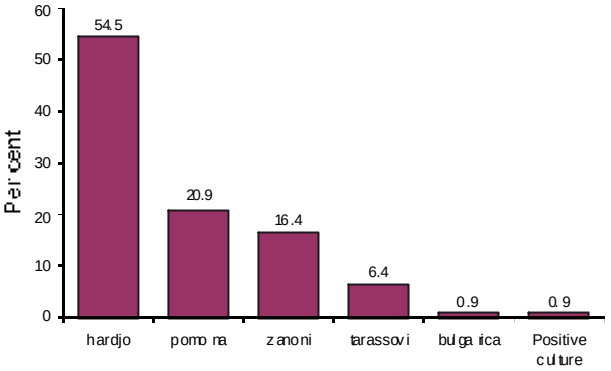


Figure 4. Notifications of leptospirosis, in Australia, 1 January 1998 to 30 June 1999, by serovars as a percentage for dairy and meatworker industries (n = 110)



The outbreak in North Queensland during the first six months of 1999 resulted in 184 notifications, compared with 47 for the same period in 1998. This increase was possibly due to heavy, prolonged rainfall and flooding in the region and an increased rodent population. Isolation and/or serology detected at least 14 different serovars of *Leptospira* for this period, australis and zanoni accounting for 92 (31.6%) of the notifications.

Australia as a whole

The Australian leptospirosis surveillance program is well under way and there has been a significant increase in the questionnaire response rate with an 82% return rate on the reported notifications for the year. This is, however, biased by the large number of notifications recorded for Queensland with a questionnaire return rate of 99%.

A comparison of notifications by State or Territory for 1998 is shown in Table 4. (Personal communication, Communicable Diseases Network Australia New Zealand – National Notifiable Diseases Surveillance System).

Table 3. *Leptospira* in Queensland, 1 January 1998 to 30 June 1999, byserovar isolated from patients

Serovar	Serovar isolations	
	Number	%
zanoni	37	47.4
australis	14	19.9
robinsoni	6	7.7
tarassovi	5	6.4
hardjo	4	5.1
szwajizak	3	3.8
kremastos	3	3.8
pomona	3	3.8
celledoni	2	2.6
medanensis	1	1.3
Total	75	100

The average age of leptospirosis cases notified was 34.7 years for males and 33.0 years for females. The average age of all notifications in Australia was 34.7 years.

The most common symptoms for the disease reported through the questionnaires are shown in Table 1. Again, the polymorphic nature of the disease could easily result in clinicians discounting leptospirosis in their differential diagnoses.

Animal related industries accounted for 28.5% of the national notifications (meatworkers 17.2%, dairy workers 11.3%) while the banana industry accounted for 19.5% (Table 2). Students and children accounted for 5.2% of the notifications. The data clearly demonstrate the broad range of occupations or activities associated with leptospirosis.

The leptospiral serovars most commonly reported were hardjo, zanoni, pomona and australis. In the dairy and meat working industries, serovars hardjo and pomona accounted for the majority of notifications with 60 (55.6%) and 22 (20.3%) respectively (Figure 4). Two overseas cases (from Malaysia and Indonesia) were both diagnosed with serovar djasiman.

Only two of the 381 Australian cases reported no contact with animals. The most frequent animal contact was with rats at 174 (45.9%) and cattle at 164 (43.3%) (Figure 1). Of

Table 4. Notifications of leptospirosis, 1998, by State or Territory

State or Territory	Number of notifications
ACT	0
NSW	48
NT	3
Qld	109
SA	1
Tas	1
Vic	25
WA	10
Total (Australia)	197

interest is the high proportion of contacts with dogs at 140 (36.9%). The total number of reported contacts with mice was 74 (19.5%). Of the 140 (36.9%) dog contacts reported, 113 (79%) also had rat and/or mice contact, and of the 52 (13.7%) cat contacts, 36 (69.2%) also had rat and/or mice contact.

Discussion

Leptospirosis continues to be increasingly responsible for ill health, affecting people employed in a wide range of occupations and activities, some of which are not traditionally associated with the disease in Australia. The large increase in notifications in Queensland during this period could be explained by high and consistent rainfall and reported increases in rodent numbers. Leptospirosis is still under-reported with greater awareness of the disease needed in children at the initial clinical assessment. Children accounted for at least 5% of leptospirosis notifications in Australia. The agricultural industry continues to emerge as the major national source of notifications along with the dairy and meatworking industries. While rats and mice are recognised sources of infection worldwide, the real public health risk associated with dogs has yet to be fully determined. To date the reference laboratory has not been able to recover leptospires from the urine of dogs, with the only isolation of *Leptospira* being from blood. It is possible that dogs in Australia do not have a role as maintenance hosts, or that local serovars are not yet canine-adapted. More detailed investigations into clinical aspects of each case and its sources of infection will further enhance our understanding of the health impact of the disease, indicate improvements to management of the patient in our hospital and medical systems, and assist in the implementation of effective control and preventative measures.

Acknowledgments

We would like to thank all the contributors for their time and assistance in sending out the questionnaires and collection of these data. Some of these are the Queensland Public Health Units, Monash University (Victoria), Westmead Hospital (New South Wales), PathCentre (Western Australia), Sullivan Nicolaides Pathology, and Queensland Medical Laboratory.

References

1. Emanuel ML, Mackerras I M, Smith DJW. The epidemiology of leptospirosis in North Queensland; general survey of animal hosts. *J Hyg (Camb)* 1964;62:451-484.
2. Faine S. *Leptospira* and leptospirosis . Boca Raton: CRC Press, 1994.
3. Faine S ed. Guidelines for the control of leptospirosis. Offset Publication No 67. Geneva: World Health Organization, 1982.
4. Herceg A, Oliver G, Myint Het al. Annual report of the National Notifiable Diseases Surveillance System. *Commun Dis Intell* 1996;20:440-464.
5. Minutes of the Meeting, 6-10 August 1982, Boston, Massachusetts, International Committee on Systematic Bacteriology, Subcommittee on the Taxonomy of *Leptospira. Int J Syst Bacteriol* 1984;34:258-259.

Infection control in the health care setting - guidelines for the prevention of transmission of infectious diseases

Public consultation draft July 2000

Prepared by Communicable Diseases Network of Australia and New Zealand (CDNANZ) - Infection Control Guidelines Review Steering Committee

For the Commonwealth Department of Health and Aged Care

The Commonwealth Department of Health and Aged Care is currently revising the National Health and Medical Research Council (NHMRC) document *Infection Control in the Health Care Setting - Guidelines for the Prevention of Transmission of Infectious Diseases*.

The draft document will be available for consultation from 22 July 2000 to 1 September 2000.

This document contains amalgamated amendments and revisions of two documents:

1. *Infection Control in the Health Care Setting - Guidelines for the Prevention of Transmission of Infectious Diseases*, NHMRC, 1996; and,
2. *Creutzfeldt-Jakob Disease and Other Human Transmissible Spongiform Encephalopathies: Guidelines on patient management and infection control*, NHMRC, 1995.

The outcome of the process will be a revised version of these guidelines which is intended to be applicable in a broad range of health care settings. They will be evidence-based best practice guidelines, which encompass principles of risk communication and management.

The web address for a copy of the draft and details for submissions is:

http://www.health.gov.au/pubhlth/strateg/communic/review/

The telephone number for inquiries will be available on the web site.

A cluster of leptospirosis among abattoir workers

Janet Terry, [1] *Marianne Trent, Mark Bartlett*
Northern Rivers Public Health Unit, Institute of Health and Research, Northern Rivers Area Health S ervice, Lismore, New South Wales.

Abstract

In early December 1998, the Northern Rivers Public Health Unit (north-eastern New South Wales) was alerted to a possible cluster of leptospirosis cases by the supervising scientist of the Western Pacific Region World Health Organization/Food and Agricultural Organization Collaborating Centre for Reference and Research on Leptospirosis. Investigation revealed a cluster of eight leptospirosis cases diagnosed during Octob er and November 1998. All were employees of a local meat works. *Leptospira* serovars isolated included pomona and hardjo. Symptoms included headache, fever, muscle pain, sore eyes, abdominal pain, vomiting, jaundice, and rash. Five of the eight cases were hospitalised. The infection could not be traced to any particular source. Unfo rtunately, records of stock killed during the exposure periods were not available. All cases reported exposure to large volumes of animal urine during the course of their work. Protective clothing provided included an apron, gloves, and rubber boots. All of the patients said they wore rubber boots and seven of the eight wore the apron provided. Only two patients reported wearing gloves, the remainder thought these were too difficult to work in. *Commun Dis Intell* 2000;24:158-160.

Keywords: leptospirosis, surveillance, occupation, abattoir, urine, cattle

Introduction

Leptospirosis is a zoonosis with worldwide distribution resulting from infection with the spirochaete *Leptospira*. In excess of 200 leptospira serotypes are currently known to be pathogenic. The illness usually develops about 7 to 12 days (range 2 to 20) following exposure to the urine of infected animals. [1] Exposure may be direct or indirect via water, moist soil, and vegetation. [2] The clinical presentation and course of the illness is variable. Subclinical infection, although rare, has been shown to occur in occupational settings. [3] The illness is usually characterised by a sudden onset of fever, headache, and myalgia. Some patients suffer a more severe illness that progresses to include hepatic, renal, and vascular dysfunction.[3]

Leptospirosis is a notifiable disease in all Australian States and Territories, occurring throughout the country with the highest reported incidence in Victoria and Queensland. [4] In 1998 there were 50 cases of leptospirosis reported for New South Wales (NSW) compared with 34 in 1997. (Notifiable Diseases Database, New South Wales Health Department; personal communication).

Leptospirosis serology is carried out by a number of pathology services within the north coast area of New South Wales. Confirmatory serology and *Leptospira* serovar identification is done either by the Institute of Clinical Pathology and Medical Research (ICPMR), at Westmead Hospital, or by the Queensland Health Scientific Services, World Health Organization/Food and Agricultural Organization (WHO/FAO) Collaborating Centre for Reference and Research on Leptospirosis.

Methods

Sporadic leptospirosis cases are not usually followed up by the Northern Rivers Public Health Unit (NRPHU). However in early December 1998 the notifying laboratory scientist alerted the Unit, located in north-eastern New South Wales, to an increase in leptospirosis among employees of an abattoir within the Northern Rivers Area. An investigation of this suspected cluster was implemented in consultation with the Occupational Health and Safety Officer at the abattoir, with Worksafe, and with local general practitioners. In all, eight cases of leptospirosis had been confirmed during October and November 1998, compared with six cases for the 8 months prior to October.

A questionnaire was developed and administered by telephone to all cases identified as abattoir employees with illness onset dates after 25 August 1998. Interviews were conducted by the Public Health Nurse and the Public Health Officer over three days from 8-10 December. The following information was sought:(1) information relating to previous history of leptospirosis, (2) job particulars within the abattoir during the incubation period (7-20 days prior to onset of illness) including exposure to animal urine, (3) details of protective apparel worn at work, (4) details relating to symptoms and hospitalisation, and (5) contact with possible sources of infection outside the workplace.

Additional cases were sought by examination of the abattoir absentee lists. All employees with a history of an influenza-like illness lasting longer than two days, unconfirmed leptospirosis, or confirmed leptospirosis with an onset date later than 25 August 1998 were identified for follow up. A fact sheet was distributed to abattoir employees which included information relating to symptoms and a

1. Corresponding author: Janet Terry, 31 Uralba St, Lismore, New South Wales, Australia 2480. Fax: (02) 6622 2151. E-mail: jterr@doh.health.nsw.gov.au

request to seek medical advice in the event of recognition of symptoms.

General practitioners in the town were informed of the cluster by mail and asked to notify the NRPHU on suspicion of further cases. Active surveillance was continued for all cases of leptospirosis notified to the NRPHU for the remainder of 1998.

The Occupational Health and Safety Officer assisted in an inspection of the work-site; this was undertaken after the day's kill had been completed. Information relating to details of stock processed during the illness incubation period was also sought.

Results

Overall there were 50 cases of leptospirosis reported in New South Wales during 1998, an increase from 34 and 33 respectively during the previous two years. Of these 50 cases, 17 (34%) were notified to the NRPHU - the highest number of cases reported to this Unit since 1992, when six cases were reported (Table1) (Notifiable Diseases Database, New South Wales Health Department; personal communication).

Eight (47%) of the case reports received during 1998 were reported during October and November. All of these cases were employees of the local abattoir and when contacted all agreed to participate in a telephone interview. No additional cases were identified by active case finding within the abattoir, or by local general practitioners.

The cases ranged in age from 16 to 53 years; all were males employed in the slaughtering section of the abattoir. The *Leptospira* serovars identified were hardjo (4 cases) and pomona (4 cases). All eight patients reported fever, headache and muscle pains. Other symptoms reported included sore eyes (4 cases), abdominal pain (6 cases), jaundice (5 cases) and rash (4 cases). Five of the eight cases were hospitalised and all cases were ill and away from work for more than two weeks. Four of the cases had not recovered at the time of the interview.

The work areas where the patients were employed included the killing area, known as the 'stick hole', the beef slaughter floor, the condemned room, and the small stock slaughter floor. All the cases reported exposure to animal urine during the course of their work. Table 2 shows illness onset dates, infecting serovar, and work area for each of the cases involved in this investigation.

Protective clothing supplied included gumboots, plastic knee length aprons, and gloves. All of the individual cases reported wearing gumboots, seven of the eight wore aprons, and two of the eight wore gloves while working. Each work station had individual access to hand-washing facilities.

A majority (6) of the cases involved in this cluster had been employed at the meat works for at least three years. One case commenced work 12 months prior to his illness. For one, information relating to length of employment was not obtained. Two of the men reported a previous diagnosis of leptospirosis. Three reported contact with farm animals away from work.

Table1. Leptospirosis cases reported to the Northern Rivers Public Health Unit, 1990 to 1998[1]

Year	Cases reported to NRPHU	Cases reported: NSW	% NSW cases reported to NRPHU
1990	4	42	9.5
1991	3	28	10.7
1992	6	19	31.6
1993	3	18	16.7
1994	5	14	35.7
1995	2	6	25.0
1996	5	33	15.1
1997	3	34	8.8
1998	17	50	34.0
1999	28	56	50.0

1. Source: Notifiable Diseases Database, New South Wales Health Department

Table 2. Frequency of leptospirosis among abattoir workers reported to the Northern Rivers Public Health Unit, October and November 1998, by onset date, work area, and serovar

	Case incidence and work area			
Date of onset	Beef slaughter floor	Small stock slaughter floor	Condemned area	Serovar
14/09	1			hardjo
18/09	1			hardjo
21/09	1			pomona
9/10		1		hardjo
26/10	1			pomona
4/11	1			pomona
8/11			1	pomona
16/11		1		hardjo

Discussion

The abattoir supplies the export market and is only involved in processing cattle. Pigs had previously been slaughtered, but this had ceased some years previously. The cattle are purchased from a wide geographical area and records of the origin of the cattle killed during the exposure periods were not available to assist in this investigation. This made it impossible to trace possible sources of infected cattle. Anecdotal evidence supplied during case interviews suggests a large number of cattle from flooded areas of Queensland had recently been killed. This may have resulted in the processing of a significant number of infected cattle. The fact that there were no further cases of leptospirosis identified among the workers at this abattoir for

the remainder of 1998 may lend some support to this possibility.

The increase in notifications continued into1999 for both the Northern Rivers Area (n=28) and NSW (n=56). No further clusters were identified during 1999 and cases were distributed throughout the northern rivers area. The increase in case notifications may be as a result of increased rainfall, and higher mean temperatures over the northern half of New South Wales and southern Queensland during 1998-99.[5] There may also have been an increased awareness of this disease by clinicians.

The most commonly reported symptoms were similar to those described for the Queensland cases reported in 1997.[4] However, the symptoms reported by the cases are suggestive of moderately severe leptospirosis illness, since five reported jaundice which is indicative of a more severe form of the disease.[3] There was no apparent difference in the severity of disease caused by the two serovars.

The serovars isolated from this cluster were among those leptospires most commonly associated with humans in Queensland during 1997. Those most commonly infected with these serovars were also meat workers.[4]

It is noteworthy that all the cases involved in this cluster reported significant exposure to animal urine during their work and, although gloves were supplied, only two of the eight cases wore them. One of the cases - who worked in the condemned area - told the interviewer he was often exposed to large amounts of urine as a result of cattle bladders bursting as they hit the bottom of a chute in the conveying system of the abattoir.

The severity of the illness, and the associated compensation and health costs associated with this cluster, highlight a need for continued efforts at prevention. The use of protective clothing for abattoir workers should be encouraged. General preventative measures involve vaccination of cattle and other domestic animals - but this is not compulsory in Australia and it is thought that beef cattle are generally not vaccinated. (Lee Smythe, WHO/FAO Collaborating Centre for Research on Leptospirosis, Western Pacific Region, Queensland Health Scientific Services; personal communication).

Acknowledgments

The authors wish to thank the following for assistance in this investigation and in the development of this paper; Lee Smythe (supervising scientist, WHO/FAO Collaborating Centre for Research on Leptospirosis, Western Pacific Region, Queensland Health Scientific Services), Rick Standing (Occupational Health and Safety Officer, Northern Co-operative Meat Company) and staff of Communicable Diseases Network Australia New Zealand, National Notifiable Diseases Surveillance System.

References

1. Farrer, E. 1995. Leptospira Species (Leptospirosis). In: G L Mandell, J E Bennett, R Dolin, eds. Mandell, Douglas and Bennett's principles and practice of infectious disease. 4th ed. New York: Churchill Livingston,1995.
2. Centers for Disease Control and Prevention. Update: leptospirosis and unexplained acute febrile illnesses among athletes participating in triathlons – Illinois and Wisconsin, 1998.*JAMA* 1998;280:1474-1475.
3. Farr WA. Leptosporosis. *Clin Infect Dis* 1995;21:1-6.
4. Smythe L, Norris M, Symonds M, Dohnt M. Annual Leptospirosis Surveillance Report Number 1 (Queensland) January-December 1997. Brisbane: Document Management, Queensland Health, 1999.
5. Department of the Environment and Heritage. Appendix 7. The weather of 1998-99. Bureau of Meteorology Annual Report 1998-99. Canberra: AusInfo, 2000.

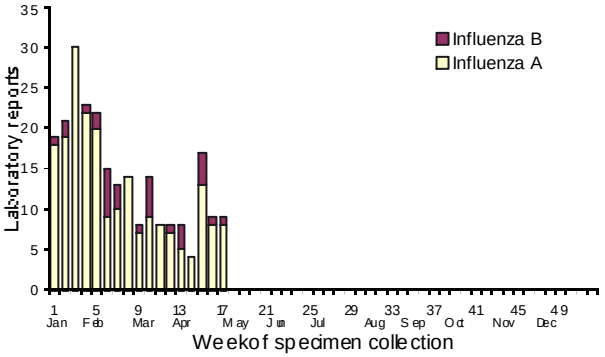
Erratum

The graph published in issue 24(5):137, in the Communicable Diseases Surveillance report: National Influenza Surveillance, 2000, as:

Figure 10. Laboratory reports of influenza, 2000, by type and week of specimen collection

is incorrect. It is to be replaced by the following graph:

Figure 10. Laboratory reports of influenza, 2000, by influenza type and week of specimen collection



A re-evaluation of immunisation coverage estimates from the Australian Childhood Immunisation Register

Brynley P Hull,[1,2] Peter B McIntyre[2]

Abstract

Immunisation coverage reporting using data from the Australian Childhood Immunisation Register is likely to underestimate immunisation uptake. Since 1997, several initiatives have been introduced to improve both immunisation uptake and notification of immunisation encounters. These initiatives seemed likely to have changed previous coverage estimates. Re-calculation of immunisation coverage estimates for the previously reported cohorts was undertaken. This used current Australian Childhood Immunisation Register data - especially the immunisation history form and the impact of catch-up immunisations - to evaluate delayed reporting. Previous coverage estimates published in *Communicable Diseases Intelligence* were shown to be at least 2% to 4% below estimates based on data now held by the Australian Childhood Immunisation Register, with greater differences observed in particular jurisdictions. *Commun Dis Intell* 2000;24:161-164.

Keywords: immunisation, surveillance, history, vaccine, childhood

Introduction

The Australian Childhood Immunisation Register (ACIR) was implemented in 1996, with the first coverage estimates published in *Communicable Diseases Intelligence (CDI)* in March 1998.[1] Subsequent reports have shown a progressive increase in coverage, especially in jurisdictions such as Western Australia and the Northern Territory where initial estimates were disproportionately low. Coverage appears likely to still be underestimated, as a result of non-reporting or delayed reporting.[2] Since 1997, and as part of the Immunise Australia program, several initiatives have been introduced to improve both immunisation uptake and notification of immunisation encounters to the Health Insurance Commission (HIC). These initiatives include the Commonwealth Childcare Rebate scheme (with the accompanying Immunisation History Form), the Childcare Assistance scheme and the General Practice Immunisation Incentives scheme. The Immunisation History Form and improvements in data transmission to the ACIR seemed likely to have changed previous coverage estimates. The cohort method of reporting immunisation coverage does not allow for assessment of 'catch-up' immunisation occurring after the assessment age of 12 months.[1] Re-analysis of earlier immunisation coverage estimates, using current ACIR data, was undertaken to evaluate changes in coverage and to measure the impact of 'catch-up' immunisations.

Methods

Coverage estimates for the first milestone vaccines at 12 months of age for three separate 3-month birth cohorts born early 1996, early 1997 and late 1997 were taken from *CDI* reports in 1998[1,3] and 1999.[4] Coverage estimates for these birth cohorts were re-calculated by the same algorithm originally applied but using ACIR data as of 30 June 1999. This allowed measurement of changes in coverage due to late notifications, with the proportion of the change due to Immunisation History Form notifications (related to the Child Care Rebate scheme) identified separately. To evaluate catch-up immunisation, coverage estimates for these birth cohorts were re-calculated, assessing at the age of 24 months rather than 12 months. This allowed any first milestone immunisations given up to two years of age to be included. Full immunisation against pertussis was defined as receipt of a total of three pertussis-containing vaccines by 24 months. A similar analysis was also undertaken for receipt of Measles-Mumps-Rubella (MMR) vaccine by 24 months and 36 months.

As the differences in coverage estimates are presumed to be due to late notifications, the mean and median notification lag time was also examined. Lag-time was calculated as the number of days between the date of the immunisation encounter and the date of processing at the HIC. The lag time was calculated for the first three doses of Diphtheria-Tetanus-Pertussis (DTP) for two separate cohorts. To evaluate possible factors influencing the proportion of late notifications, differences in lag times were examined by method of notification, state or territory and provider type between the two time periods.

Results

For the first cohort (born 1 Jan 1996 to 31 March 1996) coverage estimates at 12 months, after including late notifications, increased in absolute terms by 3.9% for Australia as a whole (Table 1). Much of this increase

1. Family Medicine Research Centre, University of Sydney, Wentworthville, New South Wales.
2. National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, Royal Alexandra Hospital for Children, Westmead, New South Wales.

Corresponding author: Brynley Hull, Family Medicine Research Centre, University of Sydney, PO Box 533, Wentworthville, New South Wales, Australia 2145;Fax: +61 2 9845 8155; E-mail: brynleyh@genprac.wsahs.nsw.gov.au

Table 1. Re-calculation of immunisation coverage estimates – first three doses of DTP for birth cohort 1 (1 January 1996 to 31 March 1996); assessment date 31 March 1997.

	State or Territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Initial coverage: *CDI* report 30 November 1997	80.3	74.8	64.8	80.7	79.1	77.6	82.7	66.5	77.4
NCIRS report: 30 June 1999[1]	84.4	80.5	66.0	83.2	83.1	80.8	85.3	73.4	81.3
Absolute change in coverage due to late notifications	4.1	5.7	1.2	2.5	4.0	3.2	2.6	6.9	3.9
% due to history form[2]	2.6	4.3	2.7	1.7	2.7	2.4	2.2	4.0	3.0
NCIRS report: 30 June 1999[1] assessed at 24 months[3]	86.9	83.3	70.8	87.0	86.4	85.3	88.2	76.7	84.5
Change in coverage due to late encounters	2.5	2.8	4.8	3.8	3.3	4.5	2.9	3.3	3.2

1. Coverage estimates allowing for late notifications.
2. % Coverage calculated at 30 June1999 due to Immunisation History Form notifications .
3. Coverage estimates allowing for late notifications and late encounters.

Table 2. Re-calculation of immunisation coverage estimates – first three doses of DTP for birth cohort 5 (1 January 1997 to 31 March 1997); assessment date 31 March 1998.

	State or Territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Initial coverage: *CDI* report 31 August 1998	84.7	80.9	64.3	85.1	82.7	84.0	84.5	78.7	82.4
NCIRS report: 30 June 1999[1]	89.5	83.3	76.3	87.6	85.3	85.9	86.4	81.8	84.8
Absolute change in coverage due to late notifications	4.8	2.4	12.0	2.5	2.6	1.9	1.9	3.1	2.4
% due to history form[2]	1.7	2.3	1.0	1.1	1.7	2.0	1.9	2.2	1.9
NCIRS report: 30 June 1999[1] assessed at 24 months[3]	90.7	85.6	81.4	90.5	88.3	89.1	89.0	84.7	87.5
Change in coverage due to late encounters	1.2	2.3	5.1	2.9	3.0	3.2	2.6	2.9	2.7

1. Coverage estimates allowing for late notifications.
2. % Coverage calculated at 30 June 1999 due to Immunisation History Form notifications.
3. Coverage estimates allowing for late notifications and late encounters.

Table 3. Re-calculation of immunisation coverage estimates – first three doses of DTP for birth cohort 8 (1 October 1997 to 31 December 1997); assessment date 31 December 1998.

	State or Territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Initial coverage: *CDI* report 31 March 1999	88.1	84.2	80.2	88.6	88.2	87.8	87.2	85.5	86.3
NCIRS report: 30 June 1999[1]	90.3	84.9	81.3	89.2	88.8	89.4	87.7	85.8	86.7
Absolute change in coverage due to late notifications	2.2	0.7	1.1	0.6	0.6	1.6	0.5	0.3	0.4
% due to history form[2]	1.1	1.4	0.2	0.6	1.6	0.9	1.2	1.8	1.2

1. Coverage estimates allowing for late notifications.
2. % Coverage calculated at 30 June 1999 due to Immunisation History Form notifications.

appears to be due to late notifications resulting from Immunisation History Forms, which are used for documentation of immunisation required by the Child Care Rebate scheme. A further 3.2% absolute increase in coverage occurred after allowing for immunisation encounters occurring after 12 months of age. There were differences by jurisdiction, with Immunisation History Forms having a greater effect in some States, most notably New South Wales and Western Australia. Similar patterns were observed in later cohorts, but the absolute changes in coverage estimates were fewer (Tables 2 and 3). Similar changes were also observed in coverage for MMR (Tables 4 and 5).

Mean and median notification lag time varied by method of notification, jurisdiction and provider type and decreased substantially in the period between the two cohorts. In most cases the median lag time was substantially lower than the mean lag time suggesting there were a number of very late notifications. For the 1996 cohort, notification by manual form was the only method associated with a low lag time between encounter and processing (Table 6). However, for the 1997 cohort, all methods of transmission had lower lag times except for Internet transmissions, which had a very substantial mean and median lag time. However, Internet notifications comprised a very small proportion of notifications to the HIC. Notifications from the Northern Territory and Queensland have the longest processing time at the HIC but are entered locally before transmission to the HIC. The lag period for all jurisdictions has also improved over time (Table 7). General Practitioners have low notification lag times except in Queensland where lag times for General Practitioners are substantially higher (Table 8). Immunisations given by Aboriginal health services, community health services and flying doctors take the greatest time to be received by the HIC. Notifications from private hospitals comprised an insignificant proportion of all notifications to the HIC.

Table 4. Re-calculation of first-dose MMR immunisation coverage estimates for birth cohort 1 (1 January 1996 to 31 March 1996); assessment date 31 March 1998 at 24 months.

	State or Territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Initial coverage: *CDI* report 31 August 1998	86.4	80.2	70.5	86.6	82.3	84.4	85.2	76.3	82.5
NCIRS report: 30 June 1999[1]	89.2	83.7	71.4	88.6	85.7	87.2	87.1	80.3	85.1
Absolute change in coverage due to late notifications	2.8	3.5	0.9	2.0	3.4	2.8	1.9	4.0	2.6
% due to history form[2]	2.9	4.6	3.3	1.8	3.3	2.3	2.9	3.7	3.4
NCIRS report: 30 June 1999[1] assessed at 36 months[3]	89.7	85.3	72.9	90.1	87.0	88.6	88.7	82.3	86.7
Change in coverage due to late encounters	0.5	1.6	1.5	1.5	1.3	1.4	1.6	2.0	1.6

1. Coverage estimates allowing for late notifications.
2. % Coverage calculated at 30 June 1999 due to Immunisation History Form notifications.
3. Coverage estimates allowing for late notifications and late encounters.

Table 5. Re-calculation of first-dose MMR immunisation coverage estimates for birth cohort 3 (1 July 1996 to 30 September 1996); assessment date 30 September 1998.

	State or Territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Initial coverage: *CDI* report 21 January 1999	85.9	83.0	77.4	89.5	83.9	84.8	86.9	80.6	85.0
NCIRS report: 30 June 1999[1]	89.3	84.7	76.5	91.0	86.3	87.5	88.2	82.9	86.5
Absolute change in coverage due to late notifications	3.4	1.7	-0.9	1.5	2.4	2.7	1.3	2.3	1.5
% due to history form[2]	3.2	3.4	2.0	1.5	2.8	2.4	2.5	3.1	2.7

1. Coverage estimates allowing for late notifications.
2. % Coverage calculated at 30 June 1999 due to Immunisation History Form notifications.

Table 6. Mean and median notification lag time by method of notification (number of days)[1]

Method of notification	Birth cohort: 1 January to 31 March 1996		Birth cohort: 1 January to 31 March 1997	
	Mean	Median	Mean	Median
Manual voucher	52	20	88	24
Disk	134	36	88	27
Scanned voucher	176	70	52	21
Electronic transmission	181	93	107	47
Internet	na[2]	na	534	512

1. NB: history form notifications and notifications later then 2 years excluded. This is because the number of extremely delayed notifications will be greater for the earlier cohort as more time is available from when an encounter occurs to when it is processed at HIC.
2. Not applicable.

Table 7. Mean and median notification lag time by jurisdiction (number of days)[1]

State	Birth cohort: 1 January to 31 March 1996		Birth cohort: 1 January to 31 March 1997	
	Mean	Median	Mean	Median
ACT	92	31	73	23
NSW	63	21	56	22
NT	356	387	266	224
Qld	202	94	99	36
SA	56	20	54	21
Tas	60	20	49	20
Vic	62	28	74	28
WA	112	25	73	22

1. NB: history form notifications and notifications later then 2 years excluded. This is because the number of extremely delayed notifications will be greater for the earlier cohort as more time is available from when an encounter occurs to when it is processed at HIC.

Table 8. Mean notification lag time by provider type (number of days)[1]

Provider type	Birth cohort: 1 January to 31 March 1996		Birth cohort: 1 January to 31 March 1997	
	Mean	Median	Mean	Median
General Practitioner	61	22	57	22
Council	69	29	81	30
State Health Dept.	111	57	56	20
Public Hospital	150	27	89	26
Aboriginal Health Service	176	78	148	51
Private Hospital	197	175	83	35
General Practitioner, Qld	202	95	95	35
Community Health Service	215	104	120	33
Flying doctor	362	423	187	136

1. NB history form notifications and notifications later then 2 years excluded. This is because the number of extremely delayed notifications will be greater for the earlier cohort as more time is available from when an encounter occurs to when it is processed at HIC.

Discussion

This analysis demonstrates that the immunisation coverage estimates originally reported by the ACIR in *CDI* require modification. The increases in coverage found are largely due to Immunisation History Form notifications arising from requirements of the Childcare Rebate scheme introduced in April 1998. The other initiatives introduced during 1998 may also have contributed to the increases in coverage, as not all the increase was due to history forms. Previous coverage estimates published in *CDI* underestimated the 'true' level of immunisation coverage in Australia by at least 2-4%, with greater differences observed in particular jurisdictions. Greater underestimation occurred in earlier estimates, with an overall increase of 3.9% in DTP coverage in the first cohort, declining to 2.4% in the fifth cohort and 0.4% in a 3-month period, or a maximum of 1.6% over 12 months, for the eighth cohort. Similarly, the change in MMR coverage estimates has declined over time. This trend correlates with the reduction in notification lag times shown in Tables 6-8. The data on lag times should be treated with caution as the processing date may not be the date the HIC first received the notification. The processing date is the date the record was last amended by a data entry operator. If there was a problem with the notification it may have been amended a number of times, so jurisdictions - or provider types - that have more problems with 'incorrect' notifications will have artificially greater lag times. The longer lag times for Queensland General Practitioners are likely to be due to transmission delays following local data entry rather than truly delayed notification.

This analysis also gives some indication of the amount of catch-up immunisation occurring. This is not included in routine coverage reports but does impact on the coverage estimates used for the General Practice Immunisation Incentives (GPII) program.[2] Overall, an additional 2.7-3.2% of children had received a third dose of DTP vaccine by 24 months. Although some of these children would still not have been classified as fully immunised at the two year milestone, three doses constitutes full pertussis immunisation in many countries and receipt of three or more doses is an important public health target. Similarly, an additional 1.6 % of children had received a dose of MMR vaccine between 24 and 36 months of age. Although every effort should be made to promote timely immunisation, these data represent catch-up immunisation of some 1,700 children Australia-wide. This is an important indicator of immunisation activity.

References

1. O'Brien ED, Sam GA, Mead C. Methodology for measuring Australia's childhood immunisation coverage. *Commun Dis Intell* 1998;22:36-37.
2. Hull BP, McIntyre PB, Heath TC, Sayer GP. Measuring immunisation coverage in Australia. A review of the Australian Childhood Immunisation Register. *Aust Fam Physician* 1999;28:55-60.
3. National Centre for Disease Control. Childhood immunisation coverage. *Commun Dis Intell* 1998;22:233.
4. National Centre for Disease Control. Childhood immunisation coverage. *Commun Dis Intell* 1999;23:110.

Diphtheria in Australia, recent trends and future prevention strategies

Heather F Gidding,[1] Margaret A Burgess,[1] Gwendolyn L Gilbert[1, 2]

Keywords: diphtheria, outbreak, adult vaccination

Introduction

Diphtheria has become rare in Australia. There has not been a reported case or death due to this disease since 1992, in stark contrast to the first half of the 20th Century (Figure 1). At the height of the 1921 epidemic, there were 23,199 notifications (annual notification rate 426 per 100,000 population)[1] and, in the decade between 1926 and 1935, there were 4,043 deaths from diphtheria.[2]

Although diphtheria is no longer evident in Australia, a recent case in neighbouring New Zealand[3] and an extensive outbreak in the Newly Independent States (NIS) of the former Soviet Union[4] highlight the potential for diphtheria to re-emerge. This article focuses on the recent epidemiology of diphtheria and ways of preventing its recurrence in Australia.

Diphtheria in Australia (1991-1998)

Australia's National Notifiable Diseases Surveillance System (NNDSS) was established in 1991 and uses the following case definition for notification of diphtheria: isolation of toxigenic *Corynebacterium diphtheriae* and either (a) pharyngitis and/or laryngitis (with or without membrane), or (b) toxic (cardiac or neurological) symptoms.[5] Since the establishment of the NNDSS, there have been 23 notified cases of diphtheria, including one fatality. These cases occurred in 1991 (eight cases) and 1992 (15 cases); 12 were male (male:female ratio 1.1:1). Of the 23 patients, 16 (14 Aborigines) resided in the Northern Territory. Most (64%) of these recent cases were aged at least 15 years (range 1-78 years), in contrast with the pre-vaccine era when less than 30% of cases were aged 15 years and older.[4,6] Even though the numbers of notifications reported here are small, they probably overestimate the true incidence of diphtheria as defined by the notification criteria since only six toxigenic isolates of *C. diphtheriae* from the Northern Territory were identified in 1991-2 (Dr Jan Lanser, Institute of Medical and Veterinary Science, Adelaide; personal communication), compared with 16 notifications for the same period. The remaining 10 notified cases were probably cutaneous infections and/or infections with non-toxigenic strains.

Comparisons with other countries

Despite the potential limitations of Australia's diphtheria notification data, the recent picture is similar to that reported for many industrialised nations with long standing vaccination programs, in that:

- the incidence of diphtheria has declined – now being rare in many European countries,[7] the United Kingdom,[8,9] and the United States;[10]
- recent outbreaks have been concentrated in poorly immunised disadvantaged groups living in crowded conditions, with high notification rates amongst indigenous peoples;[6,11]
- there has been an increase in the proportion of adult cases.

Why are adults at risk of diphtheria?

Diphtheria is now more commonly seen in adults than in children in industrialised countries for several reasons. Firstly, improved living conditions impacted on the incidence of childhood diphtheria even before vaccines became available.[6] Smaller families and less overcrowding meant that preschool children were not exposed to the same intensity of infection as previously. As a result, many reached adulthood without having been exposed to diphtheria. Secondly, the implementation of mass childhood vaccination programs further reduced both the incidence of diphtheria and the circulation of toxigenic *C. diphtheriae*, so there was less opportunity to acquire natural immunity or to boost waning vaccine-induced immunity.

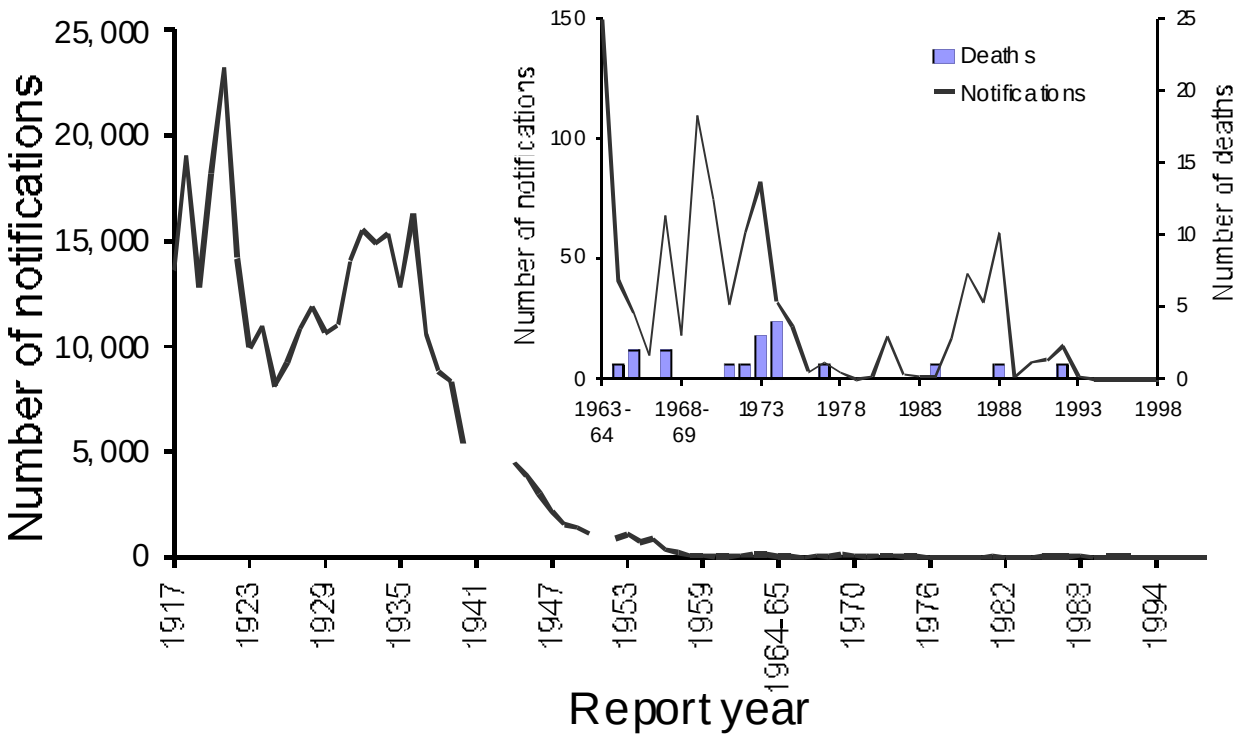
Are Australian adults at risk?

Low levels of adult immunity have been identified in two Australian studies. In 1972, a serosurvey in Victoria showed that only 40% of adults aged 40-49 years were immune.[12] Since this survey, however, there have been changes to the vaccination schedule. Booster doses of diphtheria toxoid vaccine for children aged 5-6 years and young adults aged 15 years were recommended in 1975 and 1982 respectively, while in 1984 the combined adult diphtheria and tetanus toxoid vaccine (Td, 'ADT') replaced tetanus toxoid vaccine for adult booster vaccinations at 10 year intervals.[13-15] In 1998, a study in Sydney of 548 adults aged between 19 and 70 years identified a higher level of immunity (74%) than that found in the Victorian study.[16] However, the proportion of susceptible adults was still high; one-quarter of the study population had no detectable diphtheria antibodies. The results of an Australia-wide serosurvey conducted by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) will be available soon, and should provide a national picture of age-specific immunity to diphtheria.

As adults often suffer a severe form of illness, existence of a pool of susceptible adults in Australia would be of concern.

1. National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, Royal Alexandra Hospital for Children, Westmead, New South Wales, 2145
2. Centre for Infectious Diseases and Microbiology, Institute of Clinical Pathology and Medical Research, Westmead Hospital, Westmead, New South Wales, 2145

Figure 1. Diphtheria notifications (1917-1999) and deaths (1963-1998) for Australia[1]



1. Data sources:
Historical notification data: Hall R. Notifiable disease surveillance, 1917 to 1991. *Commun Dis Intell* 1993;17:226-36.
Notification data 1992-1999: the National Notifiable Diseases Surveillance System
Death data: the Australian Bureau of Statistics Causes of Death Collection.

However, ongoing transmission has not occurred in most countries with documented evidence of low adult immunity; for diphtheria to spread extensively, a pool of susceptible children, acting as the reservoir for infection, is also required.[4] Thus large scale outbreaks are unlikely at present, since Australian children are estimated to have high coverage with the primary course of diphtheria vaccinations.[17]

The epidemic of diphtheria in the Newly Independent States (NIS)

During the 1990s in the NIS, there were both low levels of adult immunity and poor childhood vaccination coverage. These circumstances contributed to the largest outbreak of diphtheria in an industrialised country since the epidemic of over one million cases during World War II. Between 1990 and 1997 there were 140,000 cases of diphtheria and over 4,000 deaths in the NIS.[4] An important feature of this outbreak was the high proportion of adult cases. In the early and late phase of the epidemic about 70% of cases were aged at least 15 years.[6]

The following factors contributed to the outbreak in the NIS.

- An increase in adult susceptibility as a result of vaccination programs implemented prior to the break-up of the Soviet Union – those born around the time when the diphtheria vaccine was introduced (1940s and 1950s)[4] were most at risk, as they may not have been reached by newly implemented programs, and, as circulation of toxigenic *C. diphtheriae* had already declined, had not had their immunity boosted by natural reinfection.
- A reduction in childhood vaccination coverage to 70% during the 1980s[4] - this occurred mainly because of political and economic problems associated with the break-up of the Soviet Union, but also because of an increase in the number of accepted contraindications to vaccination.[4]
- Changes to childhood vaccination practices, which resulted in a reduction in immunity - during the 1980s, childhood vaccines with a lower antigenic content (the adult formulation) were used, and from 1986 children did not receive a booster dose at school entry.[4]
- Mass population movements, and a reduction in living standards associated with the break-up of the Soviet Union, made conditions favourable for the transmission of diphtheria to susceptible populations.
- Changes to the predominant circulating strain of *C. diphtheriae*.

Although microbial factors distinguishing epidemic and non-epidemic strains have not been identified, the emergence of a clone of *C. diphtheriae* during the NIS epidemic supports a role for the agent in the development of this epidemic, although the source of the strains responsible is unknown. However, high carriage rates were identified in

the military, and diphtheria had never been eradicated from central Asian countries.[4] Another possibility is that, as reservoirs of infection have also been identified in the United States,[8] South East Asia[18] and central Australia,[19] a highly transmissible strain of *C. diphtheriae* had been imported.

Implications for Australia

A case of respiratory diphtheria in New Zealand in 1998 (the first to be reported in 19 years) indicates that, as reservoirs of toxigenic *C. diphtheriae* still exist, diphtheria could re-emerge in Australia if immunity were not to be maintained, This patient, an unimmunised 32 month old child, was probably infected by his father who had returned from Bali with an infected skin abrasion.[3] Most recent cases in the United Kingdom,[20] the USA,[8] and countries bordering the NIS,[7] have also been associated with imported infections. To reduce the risk of imported cases of diphtheria in Australia, travellers need to be up-to-date with their vaccinations.

To prevent sporadic cases and transmission in Australia, such as occurred in the NIS, high levels of immunity are required in all age groups. To improve immunity in adults, the NHMRC has revised its recommendations for diphtheria vaccination. Adults who have been fully vaccinated in the past should receive a booster dose of adult tetanus-diphtheria vaccine (Td, 'ADT') at the age of 50 years unless either a booster has been documented in the previous ten years or five doses have been completed as an adolescent or adult. For adults who have no history of vaccination, primary vaccination with three doses of Td or monovalent adult diphtheria vaccine (d) - each two months apart - is required, followed by two boosters at ten year intervals.[21] To increase coverage with five doses of a diphtheria-containing vaccine, Td can be administered for the prophylaxis of tetanus-prone wounds.

Although there has not been a case of diphtheria notified in Australia since 1992, the potential for sporadic cases and outbreaks exists. Clinicians, laboratory workers and the public need to be aware of this. To reduce the risk of diphtheria re-emerging, vaccination coverage should be high in all age groups.

Acknowledgments

Thank you to Dr Jan Lanser for the provision of data about *C. diphtheriae* isolates from the Northern Territory received at the Institute of Medical and Veterinary Science, Adelaide.

References

1. Hall R. Notifiable diseases surveillance, 1917 to 1991. *Commun Dis Intell* 1993;17:226-236.
2. Feery B. One hundred years of vaccination. *NSW Public Health Bull* 1997;8:61-63.
3. Baker M, Taylor P, Wilson E, Jones N, Short P. A case of diphtheria in Auckland–implications for disease control. *N Z Public Health Rep* 1998;5:73-76.
4. Vitek CR, Wharton M. Diphtheria in the former Soviet Union: reemergence of a pandemic disease. *Emerg Infect Dis* 1998;4:539-550.
5. Public Health Committee, National Health and Medical Research Council. Surveillance case definitions. Canberra: Australian Government Publishing Service; 1994.
6. Galazka A, Tomaszunas-Blaszczyk J. Why do adults contract diphtheria? *Eurosurveillance* 1997;2:60-63.
7. Anonymous. Diphtheria cases notified in the European Union. *Eurosurveillance* 1997;2:63-64.
8. Welty T, La Fromboise C, Dixon J, *et al*. Toxigenic *Corynebacterium diphtheriae* – Northern Plains Indian Community, August-October 1996. *MMWR Morb Mortal Wkly Rep* 1997;46:506-510.
9. Begg N, Balraj V. Diphtheria: are we ready for it?*Arch Dis Child* 1995;73:568-572.
10. Bisgard KM, Hardy IR, Popovic T, *et al*. Respiratory diphtheria in the United States, 1980 through 1995. *Am J Public Health* 1998;88:787-791.
11. Mortimer EA, Jr, Wharton M. Diphtheria Toxoid. In: Plotkin SA, Orenstein WA, editors. Vaccines. Philadelphia: W B Saunders Company; 1999:104-157.
12. Forsell P. Diphtheria immunity in Victoria. *Med J Aust* 1972;1:1023-1026.
13. National Health and Medical Research Council. Immunisation procedures. 1st ed. Canberra: Australian Government Publishing Service; 1975.
14. National Health and Medical Research Council. Immunisation procedures. 2nd ed. Canberra: Australian Government Publishing Service; 1982.
15. Kilham H, Benn R. Diphtheria – the Australian perspective [editorial]. *Commun Dis Intell* 1997;21:164-165.
16. Turnbull FM, Heath TC, Jalaludin BB, Burgess MA, Ramalho AC. A randomised trial of two acellular pertussis vaccines (dTpa and pa) and a licensed diphtheria-tetanus vaccine (Td) in adults. *Vaccine* 2000. In press.
17. National Centre for Disease Control. Childhood immunisation coverage. *Commun Dis Intell* 2000;24:42.
18. Centers for Disease Control and Prevention. Diphtheria outbreak - Saraburi Province, Thailand, 1994. *MMWR Morb Mortal Wkly Rep* 1996;45:271-273.
19. Patel M, Morey F, Butcher A, Moore C, Brennan R, Mollison L. The frequent isolation of toxigenic and non-toxigenic *Corynebacterium diphtheriae* at Alice Springs Hospital. *Commun Dis Intell* 1994;18:310-311.
20. Communicable Disease Surveillance Centre. Toxigenic *Corynebacterium diphtheriae* acquired in England. *Commun Dis Rep CDR Wkly* 1996;6:231.
21. National Health and Medical Research Council. The Australian Immunisation Handbook. 7th ed. Canberra: Australian Government Publishing Services; 2000.

The NCIRS was established by the National Centre for Disease Control, Commonwealth Department of Health and Aged Care. The Centre analyses, interprets, and evaluates national surveillance data on immunisation coverage and vaccine preventable diseases. NCIRS also identifies research priorities, and initiates and coordinates research on immunisation issues and the epidemiology of vaccine preventable diseases in Australia.

Communicable Diseases Surveillance

Presentation of NNDSS data

In the March 2000 issue an additional summary table was introduced. Table 1 presents 'date of notification' data, which is a composite of three components: (i) the true onset date from a clinician, if available, (ii) the date the laboratory test was ordered, or (iii) the date reported to the public health unit. Table 2 presents data by report date for information only. In Table 2 the report date is the date the public health unit received the report.

Table 1 now includes the following summary columns: total current month 2000 data; the totals for p revious month 2000 and corresponding month 1999; a 5 year mean which is calculated using previous, corresponding and following month data for the previous 5 years (MMWR Weekly Feb 25, 2000:49(07);139-146); year to date (YTD) figures; the mean for the year to date figures for the previous 5 years; and the ratio of the current month to the mean of the last 5 years.

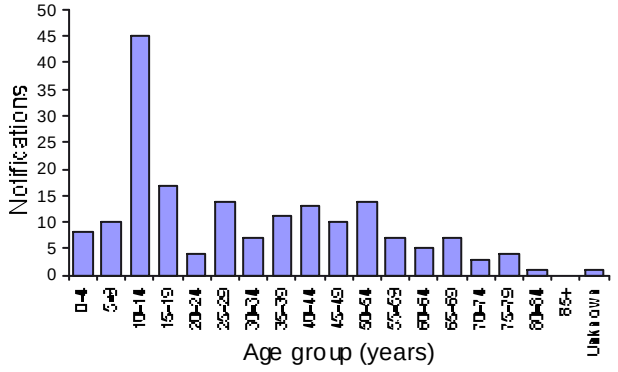
Highlights

Communicable Diseases Surveillance consists of data from various sources. The National Notifiable Diseases Surveillance System (NNDSS) is conducted under the auspices of the Communicable Diseases Network Australia New Zealand. The *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme. The Australian Sentinel Practice Research Network (ASPREN) is a general practitioner-based sentinel surveillance scheme. In this report, data from the NNDSS are referred to as 'notifications' or 'cases', whereas those from ASPREN are referred to as 'consultations' or 'encounters' while data from the LabVISE scheme are referred to as 'laboratory reports'.

Vaccine Preventable Diseases (VPD)

Overall, 219 notifications of VPDs with an onset date in May 2000 were received. Pertussis continued to be the most frequently notified VPD (Figure 1). There were no notifications of *Haemophilus influenzae* type b, diphtheria, tetanus or poliomyelitis.

Figure 1. Notifications of pertussis, Australia, May 2000, by age group and sex



There were five cases of measles in this period compared to 17 in April 2000. Four cases were from Queensland and the other case was from South Australia. Of the Queensland cases, one was imported and the other cases occurred in a family. Eighty per cent (4/5) of cases were female and the overall male to female ratio was 0.3:1. The immunisation status of all cases was recorded as unknown. Two of the cases were aged 2 and 5 years. Additional information identified the imported case as immunised.

There were 16 notifications of rubella in this period, similar to the number of notifications reported for April 2000 (17). Most cases were reported from Queensland (5/16, 31%) and Victoria (6/16, 38%). Cases occurred in a range of age groups with the maximum number of cases in the 25-29 year age group. Three cases were aged less than 1 year and one case was aged 6 years. Amongst those aged 0 to 29 years, all except one case were male; in contrast all those aged 30 years and above were female. The overall male to female ratio was 2.2:1. Immunisation status was recorded as unknown for 88% (14/16) of cases and two were recorded as not immunised.

The number of mumps notifications remained stable over this period with 17 notifications compared with 14 notifications for April 2000. Most cases (11/17, 65%) were from New South Wales with two reports from the Australian Capital Territory and four reports from Victoria. Mumps cases occurred in those aged less than 24 years with most (11/17, 65%) occurring in the 15-24 year age range. Three cases were in children aged 6 years and under (4, 5 and 6 years). The overall male to female ratio was 1.1:1. Immunisation status was reported as unknown in 76% of cases (13/17) and as not immunised in 6% (1/17). Two cases (2/17, 12%) were reported as partially immunised and one case (1/17, 6%) as fully immunised. There was no information on the immunisation status of the cases aged 4 and 5 years.

Pertussis cases in this period had decreased (181) compared with April 2000 (193) and the mean of the last five years (325). Pertussis notifications remained most frequent in New South Wales (71/181, 39%), Victoria (51/181, 28%) and Queensland (28/181, 15%). Cases of pertussis occurred in all age groups but peaked in those aged 10-14 years. Ten cases were in children aged 6 years and

under, with six children aged less than 1 year, two children aged 4 years and one child each aged 5 and 6 years. There was a slight female predominance, with an overall male to female ratio of 0.9:1. Immunisation status was reported as unknown or not immunised in 93% of cases. The nine cases reported as partially immunised were aged 4 to 14 years. Of the ten cases aged 6 years and under, one 4 year old was reported as partially immunised and the remainder were reported as unknown immunisation status.

Bloodborne diseases

There were 1,546 notifications of hepatitis C in May 2000. This was a decrease from May last year (1,770), but an increase from April 2000 (1,520) and the mean of the last five years (1,334). Overall, 9,157 notifications of hepatitis C have been received for the year to date 2000. This was an increase from the year to date mean of the last five years (6,617). Of the notifications for May 2000, 14 were reported as hepatitis C incident cases. Thirty-six per cent of incident case notifications were in the 25-29 year age group. The male to female ratio was 1:1.2.

Gastrointestinal diseases

There were 584 notifications of salmonellosis in May 2000. This was an increase from April 2000 (525), from May last year (497) and from the mean of the last five years (521). Thirty-eight per cent of cases (220) were in the 0-4 year age group (Figure 2). The overall male to female ratio of cases was 1:1.2.

There were five notifications of typhoid in May 2000 (one case in New South Wales was confirmed by telecommunication as paratyphoid) and the ages ranged from 15 to 42 years. All four confirmed typhoid cases had a history of having been overseas prior to onset (two in Indonesia, one in the Philippines and Indonesia, and one in India).

Four States currently report SLTEC/VTEC. There was one case reported in May 2000 from South Australia.

Quarantinable diseases

There were no cases of cholera, plague, rabies, yellow fever or viral haemorrhagic fever in May 2000.

Sexually transmissible diseases

There were 1,241 notifications of chlamydial infection in May 2000, which was an increase from April 2000 (1,142), from May last year (1,163) and from the mean of the last five years (828). Overall, 6,337 notifications of chlamydial infection have been received for the year to date 2000, which was a 52% increase from the year to date mean of the last five years (4,158). Most cases of chlamydial infection were reported from Queensland (35%) and Victoria (23%). Eighty-five per cent of the cases were aged 15 to 34 years. The overall male to female ratio was 1:1.6. The trend of the January 1991 to May 2000 monthly notifications demonstrates that the number of reports of chlamydial infections is increasing steadily (Figure 3). The increase was partially due to inclusion of New South Wales reporting of chlamydial infection (commenced in September 1998. *Commun Dis Intell* 1999;23:290) and the introduction of nucleic acid detection tests for chlamydia since 1995; the latter have improved the sensitivity of laboratory tests and made specimen collection easier, and this may have impacted on the notification rates (Halliday L, Petersen M. Communicable Diseases in the ACT 1993-1997. Health Series Number 20. Canberra: Australian Capital Territory Department of Health and Community Care,1998).

There were 477 notifications of gonococcal infection in May 2000, a decrease from April 2000 (480) and May last year (510), but an increase from the mean of the last five years (415). Most cases were reported from the Northern Territory (29%), then Queensland (21%), Victoria (18%) and Western Australian (17%). The ages of cases ranged from 0 to 59 years, with 84% of gonococcal notifications aged 15 to 39 years. The male to female ratio was 1.9:1.

Overall 139 syphilis notifications were received in May 2000, a decrease from both May last year (183) and the mean of the last five years (142), but an increase from April 2000 (121). The year to date 2000 figure (701) was close to the year to date mean of the last five years (729). Most of the notifications were reported from Queensland (58%), followed by New South Wales (22%) and the Northern Territory (15%). Syphilis cases were aged 14 to 85 years with peaks in the 20 to 24 (34%), 30 to 39 (19%) and 50 to 59 (17%) year age ranges. The male to female ratio was 1:1.1.

Figure 2. Notifications of salmonellosis, Australia, May 2000, by age group and sex

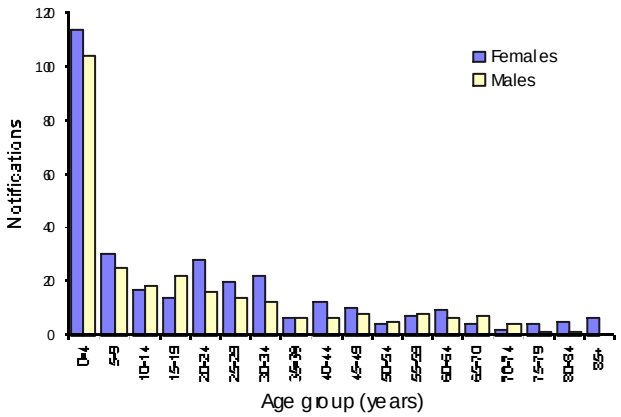
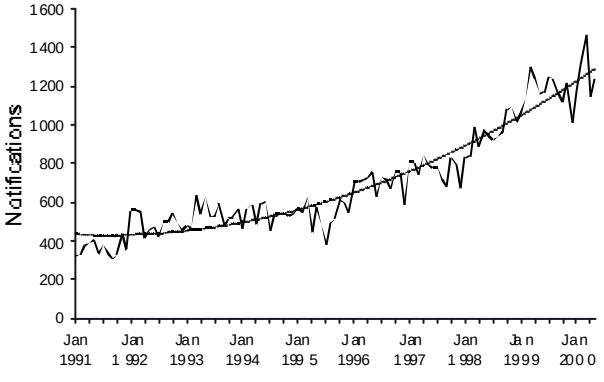


Figure 3. Notifications of *Chlamydia* infection, Australia, January 1991 to May 2000, by month



Vectorborne diseases

There were only three notifications for dengue in May 2000, which was a decrease from both April 2000 (18) and the mean of the last five years (11), but similar to May last year (4). There were two imported cases reported from the balance of the Northern Territory, i.e. outside Darwin, and one from Far North Queensland. A total of 175 notifications of dengue was received for the year to date 2000. This was an increase from the year to date mean of the last five years (96).

There were 476 notifications of Ross River virus infection in May 2000, which was a decrease from April 2000 (568), from May last year (495) and from the mean of the last five years (524). Most notifications were still from Queensland (31%); however New South Wales reported 131 cases in this period compared with 85 cases in April 2000, while Western Australia reported 118 cases in this period compared with 90 cases in April 2000. The age of cases ranged from 3 to 99 years old with a mean of 41 years of age (Median 40 years; Mode 37 years). The overall male to female ratio was 1.2:1.

There were 71 notifications of Barmah Forest virus infection in May 2000, an increase from April 2000 (50), but a decrease from both May last year (81) and the mean of the last five years (74). New South Wales reported 29 cases in this period compared with nine cases in April 2000, and the Mid North Coast of New South Wales was the most common residential region of notifications (18). More cases occurred in the 40 to 49 year age range (22; 31%) with a male to female ratio of 1.6:1.

There were eight notifications of arbovirus infection (NEC) in May 2000, fewer than for April 2000 (17) (Figure 4) but more than for both May last year (1) and the mean of the last 5 years (4). Six cases were reported from Western Australia (Kimberley, two cases; Central, two cases; Pilbara, one case and Perth, one case) and Victoria (Wimmera, two cases). The cases were aged from 22 to 69 years, with five male and three female notifications. Two of the cases of arbovirus infection (NEC) were notified as Murray Valley Encephalitis with onset dates in the month of May and were reported in *CDI* 2000;24:127.

There were 110 notifications of malaria in May 2000, which almost doubled the number from April 2000 (67), from May last year (51) and from the mean of the last 5 years (58). The notifications for the year to date 2000 totalled 449, an increase from the year to date mean of the last five years (367). Most malaria cases were reported from Queensland (53), New South Wales (22) and Victoria (20), and it can be assumed that all were imported. The cases were due to *P. vivax* (78), *P. falciparum* (12), *P. ovale* (2), and *P. falciparum/P. vivax* (1). Fifty-six per cent of notifications were aged 20 to 34 years. The male to female ratio was 4.2:1.

Other diseases

There were 35 notifications of legionellosis in May 2000, a decline from the peak in April 2000 (152), but still above the level for May last year (25) and the mean of the last five years (18) (Figure 5). Victoria is still the top reporting State (40%), followed by Queensland (17%), South Australia (17%), and New South Wales (14%). There was one male case under 1 year of age; the remaining notifications were mostly evenly distributed between the 20 to 85 year age groups, with a male to female ratio of 2.2:1. Of these notifications, 18 (51%) were due to *L. pneumophila*, 13 (37%) *L. longbeachae*, and the rest unknown/other.

Thirty four reports of meningococcal infection were received with an onset date in May 2000, less than the number of notifications from April 2000 (38) and the same as the mean of the last five years (34). Most meningococcal cases were from New South Wales (13/34, 38%), Victoria (7/34, 21%) and Western Australia (9/34, 26%). Meningococcal notifications were most frequent in those aged under 30 years, predominantly in those aged 0-4 and 15-24 years. The overall male to female ratio was 0.8:1. Serogroup information was provided for 32% (11/34) of cases; of these 72% (8/11) were serogroup B, and there was one case each of serogroup C, serogroup W and serogroup Y.

Figure 4. Notifications of arbovirus virus infections, Australia, January 1995 to May 2000, by month

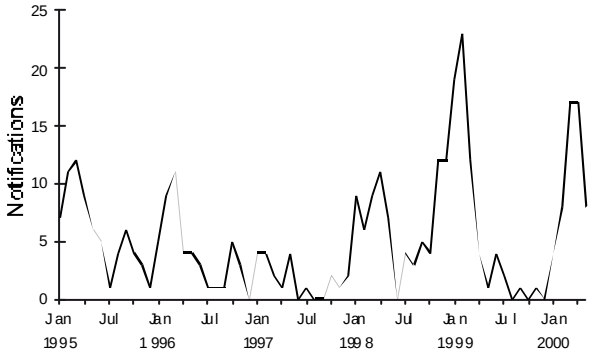
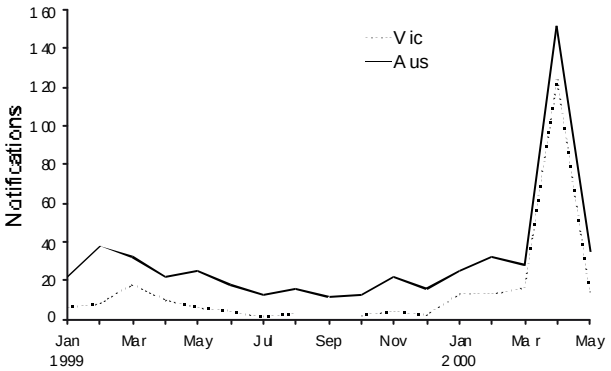


Figure 5. Notifications of legionellosis, Victoria and Australia, January 1999 to May 2000, by month



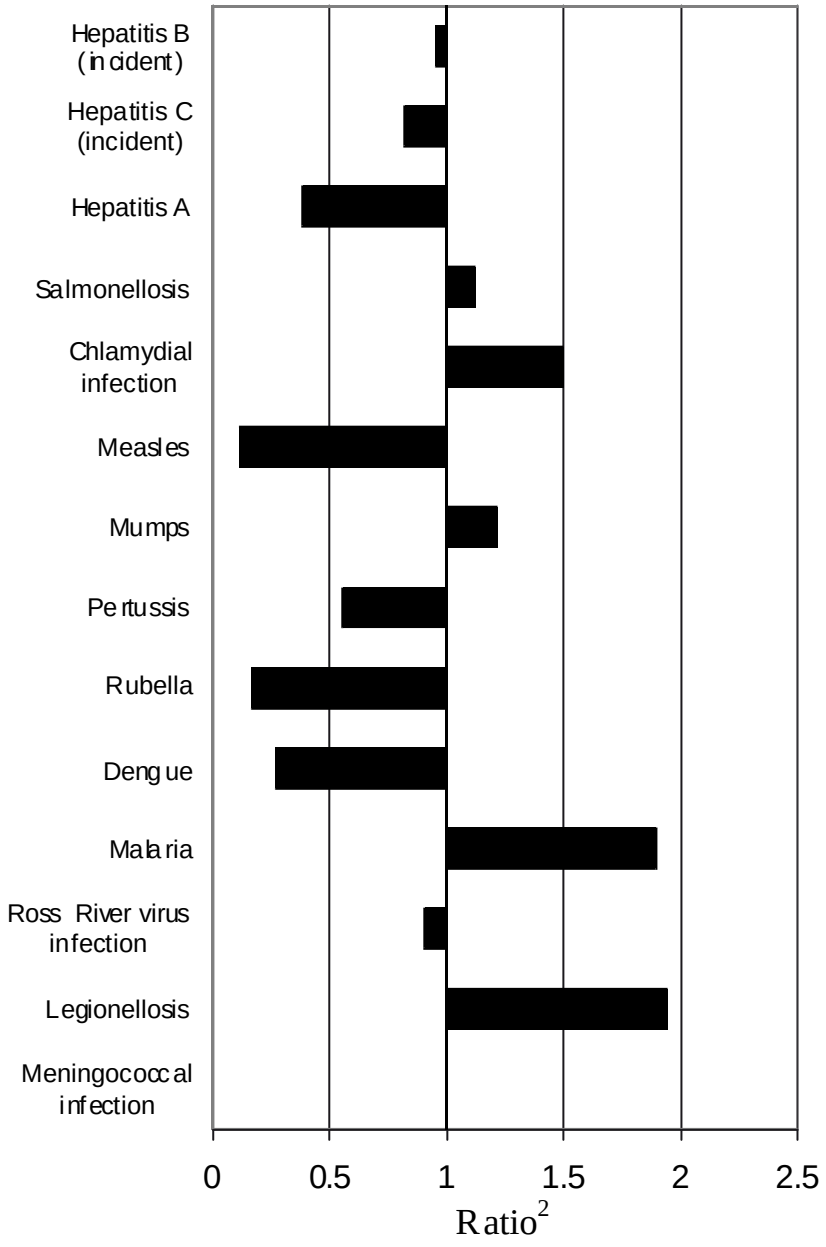
Tables

There were 6,899 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date in May 2000 (Table 1). Data by date of report for weeks 18 to 21, ending 28 May 2000, are included in this issue of *CDI* (Table 2). The number of reports for selected diseases[1] have been compared with a 5 year mean, calculated using April to June data for the previous five years (Figure 6).

There were 2,083 reports received by the *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 to 31 May 2000 (Tables 3 and 4).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 18 to 21, ending 28 May 2000, are included in this issue of *CDI* (Table 5).

Figure 6. Selected[1] diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 to 31 May 2000 with historical data[2]



1. Selected diseases are chosen each calendar month according to current activity
2. Ratio of current month total to mean of the April to June data for the previous five years

Table 1. Notifications of diseases received by State and Territory health authorities in the period 1 to 31 May 2000, by date of notification[#]

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total May 2000[1]	Total April 2000[1]	Total May 1999[1]	Last 5 years mean	Year to date 2000	Last 5 years YTD mean	Ratio*
Bloodborne															
Hepatitis B (incident)	0	3	0	6	3	0	4	6	22	20	35	23	122	124	1.0
Hepatitis B (unspecified)[2]	4	178	0	79	0	5	217	81	564	589	630	586	3,301	2,912	1.0
Hepatitis C (incident)	0	3	0	0	4	3	1	3	14	24	29	17	139	82	0.8
Hepatitis C (unspecified)[2]	17	457	6	311	52	27	527	135	1,532	1,496	1,741	1,317	9,018	6,535	1.2
Hepatitis D	0	0	0	0	0	0	0	0	0	3	2	2	7	6	0.0
Gastrointestinal															
Botulism	0	0	0	0	0	0	0	0	0	0	0	*	0	0	0.0
Campylobacterosis[3]	33	-	14	359	106	27	447	140	1,126	937	994	869	5,263	4,720	1.3
Haemolytic uraemic syndrome	NN	0	0	0	0	0	NN	0	0	1	2	2	6	4	0.0
Hepatitis A	0	11	3	13	4	1	23	12	67	82	117	173	459	1,094	0.4
Hepatitis E	0	0	0	0	0	0	0	0	0	0	0	2	0	2	0.0
Listeriosis	0	0	0	3	0	0	0	1	4	6	5	4	36	30	1.0
Salmonellosis	8	121	24	210	42	10	93	76	584	525	497	521	3,191	3,679	1.1
Shigellosis[3]	2	-	11	10	7	0	8	14	52	49	40	56	230	327	0.9
SLTEC,VTEC[4]	NN	0	0	NN	1	0	0	0	1	3	1	2	17	6	0.5
Typhoid	0	2	0	1	1	0	0	1	5	7	9	5	34	40	1.0
Yersiniosis[3]	0	-	0	5	0	0	2	0	7	4	8	16	40	110	0.4
Quarantinable															
Cholera	0	0	0	0	0	0	0	0	0	0	0	*	1	2	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Rabies	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
YellowFever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Sexually transmissible															
Chancroid	0	0	0	0	0	0	0	0	0	0	0	*	0	1	0.0
Chlamydial infection[5]	18	139	108	132	60	35	289	160	1,241	1,142	1,163	828	5,337	4,158	1.5
Donovanosis	0	0	1	0	NN	0	0	0	1	1	1	3	8	21	0.3
Gonococcal infection[6]	5	55	138	100	11	3	85	80	477	480	510	415	2,521	2,039	1.1
Lymphogranuloma venereum	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Syphilis[7]	1	31	21	80	0	0	0	6	139	121	183	142	701	729	1.0

Table 1 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 to 31 May 2000, by date of notification#

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total May 2000[1]	Total April 2000[1]	Total May 1999[1]	Last 5 years mean	Year to date 2000	Last 5 years YTD mean	Ratio*
Vaccine preventable															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Haemophilus influenzae type b	0	0	0	0	0	0	0	0	0	0	4	5	3	21	0.0
Measles	0	0	0	4	1	0	0	0	5	17	17	42	53	262	0.1
Mumps	2	11	0	0	0	0	4	0	17	14	18	14	76	68	1.2
Pertussis	3	71	0	28	14	11	51	3	181	199	258	325	1,289	2,028	0.6
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Rubella[8]	0	3	0	5	1	1	6	0	16	17	33	95	79	600	0.2
Tetanus	0	0	0	0	0	0	0	0	0	0	0	-	3	3	0.0
Vectorborne															
Arbovirus infection NEC	0	0	0	0	0	0	2	6	8	17	1	4	54	39	2.0
Barmah Forest virus infection	0	29	2	36	0	0	1	3	71	50	61	74	302	446	1.0
Dengue	0	0	2	1	0	0	0	0	3	18	4	1	175	96	0.3
Malaria	0	22	6	53	5	1	20	3	110	67	51	58	449	367	1.9
Ross River virus infection	1	131	10	147	25	3	41	118	476	568	495	524	3,063	4,038	0.9
Zoonoses															
Brucellosis	0	0	0	1	0	0	0	0	1	0	4	2	6	13	0.5
Hydatid infection	0	NN	0	0	1	0	1	2	4	1	4	3	18	13	1.3
Leptospirosis	0	12	0	18	0	0	3	0	33	19	50	18	120	97	1.8
Ornithosis	0	NN	0	NN	0	0	4	0	4	8	8	7	34	34	0.6
Q Fever	0	6	0	23	0	1	2	0	32	42	40	48	214	216	0.7
Other															
Legionellosis	1	5	0	6	6	2	14	1	35	152	25	18	272	98	1.9
Leprosy	0	0	0	0	0	0	0	0	0	0	0	-	0	3	0.0
Meningococcal infection	0	13	2	1	1	1	7	9	34	38	41	34	165	125	1.0
Tuberculosis	0	8	0	1	0	1	18	5	33	42	79	77	317	418	0.4
Total	95	1,311	348	1,933	345	132	1,870	865	6,699	6,753	7,180	6,337	33,123	35,616	

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. Unspecified numbers should be interpreted with some caution as the magnitude may be a reflection of the numbers of tests being carried out.
3. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
4. Infections with Shiga-like toxin (verotoxin) producing *E. coli* (SLTEC/VTEC).
5. WA: genital only.
6. NT, Qld, SA, Vic and WA: includes gonococcal neonatal ophthalmia.
7. Includes congenital syphilis.
8. Includes congenital rubella.

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

NN Not Notifiable.

NEC Not Elsewhere Classified.

\- Elsewhere Classified.

na Not applicable.

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

Table 2. Notifications of diseases received by State and Territory health authorities for weeks 18 to 21, by date of report,* May 2000

Week number	18	19	20	21	Year to
Week ending on	7 May 2000	14 May 2000	21 May 2000	28 May 2000	date total
Disease[1]					
Bloodborne					
Hepatitis B (incident)	1	5	8	6	105
Hepatitis B (unspecified)[2]	109	212	159	140	2,826
Hepatitis C (incident)	1	5	6	3	134
Hepatitis C (unspecified)[2]	350	439	444	365	7,688
Hepatitis D	1	0	0	0	6
Gastrointestinal					
Botulism	0	0	0	0	0
Campylobacterosis[3]	240	229	311	257	4,167
Haemolytic uraemic syndrome	1	0	0	0	5
Hepatitis A	25	17	26	16	395
Hepatitis E	0	0	0	0	0
Listeriosis	2	1	1	2	31
Salmonellosis	142	162	189	150	2,620
Shigellosis[3]	13	22	12	18	164
SLTEC,VTEC[4]	1	2	0	0	17
Typhoid	1	1	2	1	32
Yersiniosis[3]	3	0	1	1	33
Quarantinable					
Cholera	0	0	0	0	1
Plague	0	0	0	0	0
Rabies	0	0	0	0	0
Viral haemorrhagic fever	0	0	0	0	0
Yellow Fever	0	0	0	0	0
Sexually transmissible					
Chancroid	0	0	0	0	0
Chlamydial infection[5]	289	291	359	293	5,118
Donovanosis	0	0	0	0	8
Gonococcal infection[6]	98	152	105	177	2,001
Lymphogranuloma venereum	0	0	0	0	0
Syphilis[7]	57	29	32	50	588
Vaccine preventable					
Diphtheria	0	0	0	0	0
Haemophilus influenzae type b	0	0	0	0	5
Measles	4	0	0	6	46
Mumps	2	5	5	7	59
Pertussis	70	61	73	60	1,232
Poliomyelitis	0	0	0	0	0
Rubella[8]	2	6	7	1	64
Tetanus	0	0	0	0	4
Vectorborne					
Arbovirus infection NEC	2	10	5	3	35
Barmah Forest virus infection	8	24	32	11	232
Dengue	8	8	1	3	174
Malaria	33	30	22	22	328
Ross River virus infection	185	179	178	113	2,512

Table 2 (continued). Notifications of diseases received by State and Territory health authorities for weeks 18 to 21, by date of report*, May 2000

Week number	18	19	20	21	Year to
Week ending on	7 May 2000	14 May 2000	21 May 2000	28 May 2000	date total
Disease[1]					
Zoonoses					
Brucellosis	0	0	1	0	6
Hydatid infection	0	1	3	1	13
Leptospirosis	2	3	9	17	86
Ornithosis	0	2	10	0	31
Q Fever	11	3	16	7	193
Other					
Legionellosis	57	30	19	6	158
Leprosy	0	0	0	0	0
Meningococcal infection	6	6	11	7	137
Tuberculosis	14	18	17	15	330
Total	1,738	1,953	2,064	1,758	31,584

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. Unspecified numbers should be interpreted with some caution as the magnitude may be a reflection of the numbers of tests being carried out.
3. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
4. Infections with Shiga-like toxin (verotoxin) producing *E coli* (SLTEC/VTEC).
5. WA: genital only.
6. NT, Qld, SA , Vic and WA: includes gonococcal neonatal ophthalmia.
7. Includes congenital syphilis.
8. Includes congenital rubella.

* Date of report is the date the public health unit received the report.

NN Not Notifiable.

NEC Not Elsewhere Classified.

\- Elsewhere Classified.

Table 3. Virology and serology laboratory reports by contributing laboratories for the reporting period 1 to 31 May 2000[1]

State or Territory	Laboratory	This period	Total this period[2]
Australian Capital Territory	The Canberra Hospital	23	39
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	137	143
	New Children's Hospital, Westmead	100	112
New South Wales	Repatriation General Hospital, Concord	0	0
	Royal Prince Alfred Hospital, Camperdown	36	84
	South West Area Pathology Service, Liverpool	0	0
Queensland	Queensland Medical Laboratory, West End	407	421
	Townsville General Hospital	10	17
South Australia	Institute of Medical and Veterinary Science, Adelaide	451	329
Tasmania	Northern Tasmanian Pathology Service, Launceston	4	0
	Royal Hobart Hospital, Hobart	0	0
Victoria	Monash Medical Centre, Melbourne	0	6
	Royal Children's Hospital, Melbourne	122	105
	Victorian Infectious Diseases Reference Laboratory, Fairfield	140	229
Western Australia	PathCentre Virology, Perth	587	1,463
	Princess Margaret Hospital, Perth	49	62
	Western Diagnostic Pathology	17	28
Total		2,083	3,038

1. The complete list of laboratories reporting for the 12 months, January to December 2000, will appear in every report from January 2000 regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.
2. Total reports include both reports for the current period and outstanding reports to date.

Table 4. Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 to 31 May 2000, and total reports for the year[2]

	State or Territory[1]								This period 2000	This period 1999	Year to date 2000[3]	Year to date 1999
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	0	0	0	0	2	0	1	0	3	10	21	127
Mumps virus	0	0	0	0	0	0	1	2	3	6	29	27
Rubella virus	0	0	0	2	0	0	0	1	3	10	19	39
Hepatitis viruses												
Hepatitis A virus	0	0	0	4	5	0	0	8	17	27	85	173
Arboviruses												
Ross River virus	0	10	5	34	37	1	5	104	196	180	988	973
Barmah Forest virus	0	1	2	7	0	0	0	2	12	33	95	108
Dengue not typed	0	0	2	0	0	1	0	3	6	7	161	31
Murray Valley encephalitis virus	0	0	1	0	0	0	0	11	12	1	19	2
Kunjin virus	0	0	0	0	0	0	0	3	3	2	4	4
Flavivirus (unspecified)	0	0	0	1	0	0	0	0	1	0	35	16
Adenoviruses												
Adenovirus type 3	0	0	0	0	0	0	2	0	2	4	12	16
Adenovirus type 40	0	0	0	0	0	0	0	15	15	2	59	22
Adenovirus not typed/pending	0	7	2	0	32	0	4	40	85	86	449	441
Herpes viruses												
Herpes virus type 6	0	0	0	0	0	0	0	2	2	1	4	4
Cytomegalovirus	5	17	0	21	43	1	25	15	127	97	529	508
Varicella-zoster virus	1	8	0	28	11	0	30	42	120	147	643	706
Epstein-Barr virus	0	7	1	70	106	0	8	25	217	273	928	1,071
Other DNA viruses												
Papovavirus group	0	0	0	0	0	0	0	1	1	4	5	7
Molluscum contagiosum	0	0	0	0	0	0	0	2	2	0	8	6
Contagious pustular dermatitis (Orf virus)	0	0	0	0	0	0	0	3	3	0	6	6
Parvovirus	0	0	0	0	5	1	4	18	28	38	140	169
Picorna virus family												
Coxsackievirus A9	0	1	0	0	0	0	0	0	1	0	4	5
Echovirus type 7	0	2	0	0	1	0	0	0	3	0	16	1
Echovirus type 22	0	1	0	0	0	0	0	0	1	0	2	11
Echovirus type 30	0	1	0	0	0	0	3	0	4	0	95	6
Echovirus type 33	0	1	0	0	0	0	0	0	1	0	2	0
Rhinovirus (all types)	0	22	0	0	0	0	2	17	41	37	166	144
Enterovirus not typed/pending	1	5	0	2	1	0	41	16	66	39	515	297
Ortho/paramyxoviruses												
Influenza A virus	0	5	1	0	20	0	2	9	37	147	264	307
Influenza B virus	0	1	0	0	3	0	0	8	12	12	50	55
Parainfluenza virus type 1	0	0	0	3	13	0	4	18	38	5	148	18
Parainfluenza virus type 2	0	1	0	0	2	0	1	2	6	19	16	55
Parainfluenza virus type 3	0	0	0	0	5	0	0	6	11	19	97	170
Respiratory syncytial virus	7	121	0	17	16	0	53	88	302	222	702	550
Other RNA viruses												
Rotavirus	5	26	0	0	26	0	3	3	63	124	208	338
Norwalk agent	0	0	0	0	0	0	2	0	2	12	4	29

Table 4 (continued). Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 to 31 May 2000, and total reports for the year[2]

	State or Territory[1]								This period 2000	This period 1999	Year to date 2000[3]	Year to date 1999
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other												
Chlamydia trachomatis not typed	5	50	59	83	45	4	13	106	365	263	1,496	1,259
Chlamydia psittaci	0	0	0	0	0	0	8	1	9	7	40	35
Chlamydia species	0	2	0	0	0	0	0	0	2	2	5	9
Mycoplasma pneumoniae	0	3	2	15	9	0	8	8	45	85	231	453
Coxiella burnetii (Q fever)	0	0	0	0	1	0	0	3	4	24	31	78
Streptococcus group A	0	3	3	18	0	0	0	0	24	26	160	28
Bordetella pertussis	1	1	0	7	3	0	34	1	47	79	225	276
Legionella pneumophila	0	0	0	0	7	0	0	3	10	2	13	14
Legionella longbeachae	2	1	0	0	2	0	0	2	7	3	29	17
Cryptococcus species	0	1	0	0	2	0	0	0	3	0	5	6
Leptospira species	0	3	0	3	1	0	0	0	7	9	21	9
Treponema pallidum	0	3	20	30	50	0	0	1	104	45	275	53
Toxoplasma gondii	0	0	0	0	1	0	1	0	2	1	6	5
Echinococcus granulosus	0	0	0	0	4	0	0	4	8	0	13	0
Total	27	304	98	345	453	8	255	593	2,083	2,110	9,078	8,684

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. Australian Sentinel Practice Research Network reports, weeks 18 to 21, 2000

Week number	18		19		20		21	
Week ending on	7 May 2000		14 May 2000		21 May 2000		28 May 2000	
Doctors reporting	66		71		68		69	
Total encounters	8,339		9,048		7,946		8,596	
Condition	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	40	4.8	39	4.3	49	6.2	62	7.2
Chickenpox	9	1.1	13	1.4	13	1.6	12	1.4
Gastroenteritis	61	7.3	60	6.6	66	8.3	59	6.9
Gastroenteritis with stool culture	12	1.4	17	1.9	13	1.6	17	2.0
ADT immunisations	52	6.2	53	5.9	46	5.8	47	5.5

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia New Zealand. The system coordinates the national surveillance of more than 40 communicable diseases or disease groups endorsed by the National Health and Medical Research Council (NHMRC). Notifications of these diseases are made to State and Territory health authorities under the provisions of their respective public health legislations. De-identified core unit data are supplied fortnightly for collation, analysis and dissemination. For further information, see CDI *2000;24:6-7.*

LabVISE is a sentinel reporting scheme. Currently 17 laboratories contribute data on the laboratory identification of viruses and other organisms. This number may change throughout the year. Data are collated and published in Communicable Diseases Intelligence *every four weeks. These data should be interpreted with caution as the number and type of reports received is subject to a number of biases. For further information, see* CDI *2000;24:10.*

ASPREN currently comprises about 120 general practitioners from throughout the country. Between 7,000 and 8,000 consultations are reported each week, with special attention to 14 conditions chosen for sentinel surveillance in 2000. CDI *reports the consultation rates for five of these. For further information, including case definitions, see* CDI *2000;24:7-8.*

Additional Reports

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. Telephone: (02) 9332 4648; Facsimile: (02) 9332 1837; http://www.med.unsw.edu.au/nchecr.

HIV and AIDS diagnoses and deaths following AIDS reported for 1 to 31 January 2000, as reported to 30 April 2000, are included in this issue of CDI *(Tables 6 and 7).*

Table 6. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 31 January 2000, by sex and State or Territory of diagnosis

										Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2000	This period 1999	Year to date 2000	Year to date 1999
HIV diagnoses	Female	0	3	0	2	0	0	2	2	9	3	9	3
	Male	3	34	1	11	1	0	15	4	69	47	69	47
	Sex not reported	0	1	0	0	0	0	0	0	1	0	1	0
	Total[1]	3	38	1	13	1	0	18	6	80	50	80	50
AIDS diagnoses	Female	1	2	0	0	0	0	1	0	4	0	4	0
	Male	0	9	0	3	0	0	6	1	19	10	19	10
	Total[1]	1	11	0	3	0	0	7	1	23	10	23	10
AIDS deaths	Female	1	0	0	1	0	0	1	0	3	0	3	0
	Male	0	1	0	0	0	0	1	1	3	21	3	21
	Total[1]	1	1	0	1	0	0	2	1	6	22	6	22

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

Table 7. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 31 January 2000, by sex and State or Territory

		State or Territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	26	603	11	150	61	5	212	115	1,183
	Male	224	10,837	109	1,976	673	79	3,887	912	18,697
	Sex not reported	0	254	0	0	0	0	24	0	278
	Total [1]	250	11,713	120	2,133	734	84	4,137	1,031	20,202
AIDS diagnoses	Female	9	186	0	47	25	3	69	26	365
	Male	86	4,641	36	821	345	44	1,612	349	7,934
	Total [1]	95	4,839	36	870	370	47	1,688	377	8,322
AIDS deaths	Female	4	113	0	32	15	2	49	16	231
	Male	65	3,168	24	564	230	29	1,268	248	5,596
	Total [1]	69	3,289	24	598	245	31	1,323	265	5,844

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

Update on the outbreak of cholera affecting Pohnpei State in the Federated States of Micronesia

Adapted from a report forwarded from the Pacific Public Health Surveillance Network[1]

The outbreak was first recognised on 17 April 2000. As of 26 June 1,596 cases of watery diarrhoea have been reported – a figure based on inpatient and outpatient records from the Pohnpei Hospital together with data provided by community-based dispensaries. Nine deaths have been associated with the outbreak. *Vibrio cholerae* has been isolated by the Pohnpei Hospital and confirmed as the Ogawa serotype. Of these suspected cholera cases, 954 meet the World Health Organization case definition for cholera.[2] The epidemic is consistent with an initial point-source outbreak at a large funeral at Kitti, followed by a mixture of person-to-person transmission and additional point source outbreaks in a clockwise direction around the island.

Up to 27 June 2000, 368 (92%) of the 399 admissions to hospital, and 625 (80%) of 777 outpatient and emergency department presentations with acute watery diarrhoea, met the WHO case definition for cholera. Of the hospitalised cases, 40% were male, with adults more commonly infected. There appeared to be a bimodal distribution, with highest rates in males in the 65-74 year age group, and in females 20-24 years or over 75 years old.

Of the 777 non-hospitalised suspected cholera patients, 50% were male and 119 were less than five years of age. Due to possible multiple outpatient visits by the same patient, together with recording and data entry errors, the figures for non-hospitalised cases need to be treated with caution.

1. Information provided by the Federated States of Micronesia Department of Health via the Pacific Public Health Surveillance Network, 30 June 2000.
2. For surveillance purposes, in an epidemic situation, the case definition for suspected cholera is aperson five years of age or greater developing acute watery diarrhoea.

Overseas briefs

Source: World Health Organization (WHO)
This material has been summarised from information on the WHO Internet site. A link to this site can be found under 'Other Australian and international communicable diseases sites' on the CDI homepage.

Accidental exposure to smallpox vaccine in the Russian Federation

The recent report of illness amongst eight young children in Vladivostock who had played with discarded ampoules of smallpox vaccine, has now been confirmed by the Ministry of Health of the Russian Federation. Laboratory confirmation of the illness in the children is being sought. The report has evoked much public concern. In some of the reports, there were misconceptions about the components of the vaccine used to prevent smallpox, and about why any country might still be retaining stocks of smallpox vaccine. This note aims to clarify these issues.

1) Smallpox vaccine is not made from smallpox virus.

The vaccine which was used for centuries to vaccinate against smallpox was not made from smallpox, but from vaccinia virus. Vaccinia is a different virus from the virus that causes smallpox. However, it is a member of the same family of viruses to which the smallpox virus belongs. The smallpox virus is also known as variola virus. Mass vaccinations with smallpox vaccine made from vaccinia virus led to the eradication of smallpox announced by WHO in 1980. People vaccinated with smallpox vaccine (vaccinia) develop reactions to it which range from mild and transient to severe and, very rarely, fatal.

2) Two countries still keep smallpox virus (variola) stocks.

Although smallpox disease has been eradicated, two laboratories still hold stocks of smallpox virus (variola). These are the WHO Collaborating Centres in Atlanta (USA) and Koltsovo (Russian Federation).

3) Many countries still hold smallpox vaccine (vaccinia) stocks.

WHO recommends that countries which still have stocks of smallpox vaccine (vaccinia) maintain these stocks. This recommendation has been made for two reasons. Firstly, small amounts of vaccine are still needed to vaccinate laboratory personnel handling vaccinia virus and other members of this virus family. Some of these viruses are found in nature and cause illness among animals, and some are used in research to make new, safer vaccines against a variety of infectious diseases. Secondly, smallpox vaccine (vaccinia) will also be needed in case of a deliberate or accidental release of smallpox virus (variola), which is a very unlikely event but currently of great concern to some countries.

4) Disposal of biological materials and pharmaceuticals

All biological materials and pharmaceuticals such as vaccines, drugs and diagnostic specimens should be disposed of safely. Some may require inactivation before disposal. This can be accomplished by autoclaving or incineration.

Acute haemorrhagic fever syndrome in Afghanistan

An outbreak of acute haemorrhagic fever syndrome has been reported from an isolated village in Gulran district, Afghanistan. Disease symptoms are compatible with Crimean-Congo haemorrhagic fever (CCHF). An international team, coordinated by WHO, arrived in the affected area on 16 June to control and investigate this outbreak. The team comprises experts from the WHO Collaborating Centre at the National Institute of Virology (NIV - South Africa), Epicentre (France) and WHO. Preliminary findings indicate that cases began at the beginning of May and are continuing to occur. Twenty-five suspect cases, including 15 deaths, were identified by the team. Samples were collected and transported to NIV where diagnostic laboratory tests will be performed to establish the aetiology of the outbreak.

The team has provided basic protective materials (gloves, masks), disinfectants (chlorine bleach) and instructions for their use in caring for patients with bleeding symptoms. An isolation area has been identified in the hospital in Herat to deal with haemorrhagic patients and will be equipped by WHO. Training in barrier nursing will be provided to medical and nursing staff.

E. coli O157 in Canada

The Public Health Unit for the town of Walkerton, Ontario (population 5,000) has reported an outbreak of *E. coli* O157. As at 30 May, 5 people had died and 27 were hospitalised. Exposure to *E. coli* is believed to have occurred between 12-15 May. Tests have confirmed *E. coli* contamination in the Walkerton water supply system. On 21 May, the Public Health Unit issued a "Boil Water Order" to the residents of Walkerton which is still in effect. Investigations are under way by provincial and federal health and environmental authorities.

Subscriptions

CanPrint, PO Box 7456, Canberra Mail Centre, ACT, 2610;
Fax: +61 2 6295 4888 (Overseas) or (02) 6295 4888 (Australia).

Website

http://www.health.gov.au/pubhlth/cdi/cdihtml.htm

Contributions

Contributions covering any aspects of communicable diseases are invited. All contributions are subject to the normal refereeing process. **Instructions to authors can be found in *CDI* 2000;24:5-6.**

Contacts other than subscriptions

CDI is produced every four weeks by the National Centre for Disease Control, Department of Health and Aged Care, GPO Box 9848, Canberra, ACT, 2601; Fax: (02) 6289 7791, Phone: (02) 6289 8245; email: cdi.editor@health.gov.au.

This journal is indexed by *Index Medicus* and Medline.