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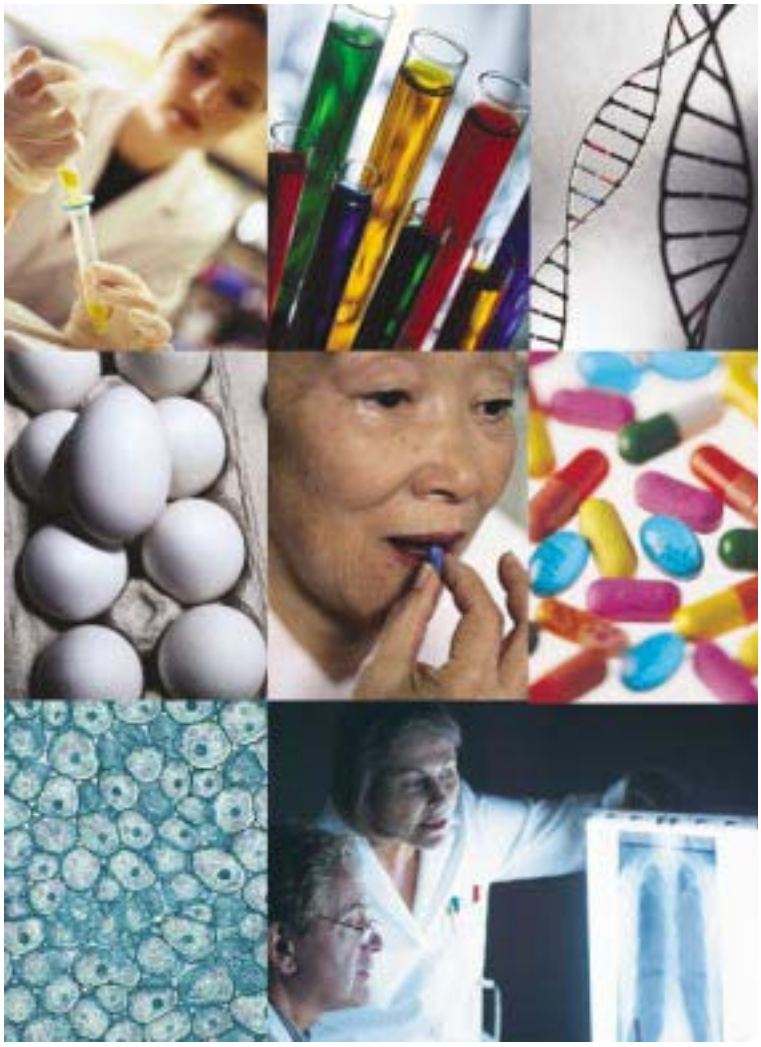

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Editorial: Polio eradication in Australia and the world

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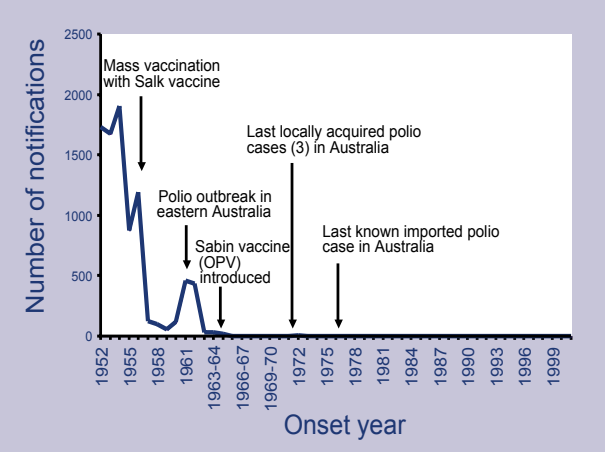
Vaccination prevents an estimated 650,000 cases of paralytic polio in each annual global birth cohort.[1] In October 2000, the World Health Organization (WHO) declared the Western Pacific region, including Australia to be polio-free.[2] This marks an important achievement for childhood health and is a true vaccination 'success story.' Since the creation of the Global Polio Eradication Initiative by the World Health Assembly in 1988, the estimated number of polio cases has fallen from 350,000 to less than 3,500, a decrease of more than 99 per cent.[3]

Mass vaccination against polio in Australia began in 1956 with the Salk inactivated polio vaccine (IPV) in a large publicly funded program.[4] The impact on the incidence of polio was dramatic (Figure).[5] The last laboratory-confirmed case of poliomyelitis in Australia was in 1967 and there were three clinically compatible cases notified in 1972.[6] The last known imported case of poliomyelitis was in 1977 (Figure). All cases notified since have been investigated and classified as cases of vaccine-associated poliomyelitis (VAPP). This includes a case in 1986, originally reported as wild polio, but recently re-classified as VAPP.[7]

Figure. Notifications of poliomyelitis, Australia, 1952 to 2001, by year of report



Certification of the eradication of polio required the documentation of the absence of circulating wild poliovirus (by surveillance for clinical polio and screening enteroviruses in laboratory specimens) as well as the monitoring of acute flaccid paralysis and vaccine-associated paralytic poliomyelitis. These surveillance activities and the continued vaccination of children against polio need to be sustained until global polio eradication is achieved. The paper by D'Souza in this issue[8] describes polio surveillance activities in Australia up to 2000. This editorial will discuss five areas of importance to polio eradication and highlight issues raised by the D'Souza's report.

Maintaining vaccine coverage

More than 90 per cent of all Australian children have received 3 doses of oral polio vaccine (OPV) by their 1st birthday and nearly 94 per cent of children by their 2nd birthday. (ACIR data: September 2001). However, antibody responses to poliovirus may be lower in some groups of vaccinated children within Australia, who may therefore be vulnerable to the imported virus.[9] Maintaining a high level of vaccine coverage is essential until global eradication of polio is achieved.

In 2001, 3 cases of paralytic polio occurred in Bulgaria among unvaccinated Roma children, 10 years after the last reported case of polio in that country. All 3 cases were identified as wild type and were genetically identical to wild-type virus from Northern India.[10] The importation of the virus into Europe struck the most vulnerable group with the lowest immunity.

Surveillance of acute flaccid paralysis

An important component of surveillance for polio is the continued monitoring of acute flaccid paralysis (AFP) in children under 15 years of age. Table 1 indicates the level of reporting in 2000 in Australia against 6 criteria for AFP surveillance. Acute flaccid paralysis surveillance data from the National Polio Reference laboratory at the Victorian Infectious Diseases Reference Laboratory,[11] suggests that Australia is meeting WHO standards in all but 2 criteria (Table 1).

It is estimated that AFP incidence in the absence of polio should be approximately 1 per 100,000 in this population and this is the minimum rate that an effective surveillance system should be reporting. While this surveillance target is being met nationally, low AFP detection rates in Tasmania and the Northern Territory as reported in this issue, suggest that AFP surveillance is sub-optimal in these jurisdictions. Moreover, the review of hospital records described in the article shows that a number of AFP cases go unreported. For each case of AFP, reporting and investigations should be instigated within 48 hours and 2 faecal samples collected 24 hours apart within 14 days of the onset of paralysis to detect poliovirus. The major un-met criteria are the timely investigation of AFP cases and the repeat stool testing to detect poliovirus. It should be noted that reporting of AFP is not routine in all industrialised countries and a number of countries are not meeting WHO surveillance standards.[12] The recent experience in Australia is that increased awareness among paediatricians of the importance of AFP surveillance and the centralising of clinical and virological surveillance has improved performance against WHO targets.[11]

Laboratory surveillance of enteroviruses

Laboratory surveillance of enteroviruses from faecal samples is important to measure the circulation of polioviruses in the environment. Concern has been expressed that poliovirus may persist in the environment because of faecal shedding from children receiving the OPV. This route is known to be responsible for the infection of household members. Indeed one of the rationales for using the live OPV is the ability to build herd immunity rapidly by such indirect effects. Faecal shedding appears to be limited in healthy children to 2–3 months after receiving the vaccine, although case reports of long term faecal shedding of poliovirus from children with inherited immuno-deficiencies have been documented.[13]

Environmental sampling of sewerage samples in Israel and the Palestinian Authority after polio was eliminated identified wild-type polio in 17 of 2,294 samples collected between 1989 and 1997.[14] These samples were clustered in four 'silent outbreaks' (that is they were not associated with cases of polio) and occurred at times when population immunity as assessed by serological surveys was high. Most of the isolates were identified in communities with poor sanitary conditions. One of the 'silent outbreaks' coincided with an influx of Palestinians into the Gaza Strip from countries in which poliovirus was endemic and where vaccine coverage was low.

Table 1. AFP surveillance in Australia, 2000[10]

WHO surveillance target	Indicator	AFP surveillance performance in 2000
Non-polio AFP cases per 100,000 population aged less than 15 years	1/100,000 (minimum 40 cases per annum)	48 cases (1.2/100,000) 43 cases with follow-up data
Percentage of routine surveillance sites that provide routine reports (including zero reports) on time	>80%	98% of reports provided to the Australian Paediatric Surveillance Unit each month
Percentage of AFP cases that are investigated	>80%	88% completed first and second questionnaires and/or collected 2 faecal samples
Percentage of AFP cases that are investigated within 48 hours of notification	>80%	48% investigated for clinical details and stool collection within 48 hours of notification
Percentage of AFP cases with a follow up examination for residual paralysis at 60 days after the onset of paralysis	>80%	88%
Percentage of AFP cases with 2 adequate stool samples	>80%	31%

While the study demonstrated the potential for polioviruses to circulate in sewerage, the lack of association with clinical cases leaves the significance of the findings for polio eradication uncertain. It should be noted that infection with the poliovirus is asymptomatic in 95 per cent of cases. Given the costs and complexity of such surveillance it is unlikely that this kind of surveillance can be instituted in many countries.

Most countries, like Australia, use opportunistic screening of faecal samples for polioviruses. The Virology and Serology Laboratory Reporting Scheme (LabVISE) has been used as the basis of surveillance in Australia. This scheme, which has been operating in its present form since 1991, reports only positive isolations and has reported 877 isolates of poliovirus in the 10 years, 1991 to 2000. These comprised 7 per cent of the 12,148 enterovirus isolated. LabVISE data are drawn from 15 to 20 laboratories, which include most major public hospital laboratories, but the numbers and types of poliovirus identified may not be fully representative of the national prevalence. Further, the proportion of enteroviruses that have been fully identified has been declining in recent years, making the value of enterovirus surveillance through LabVISE more uncertain.

There has been no circulating wild poliovirus in Australia for the last 30 years but laboratory stocks of poliovirus or material infected with poliovirus are potential sources of infection. Such laboratory material must be destroyed or contained as part of the global eradication program.[12] The Commonwealth Department of Health and Ageing is coordinating laboratory containment activities with the WHO. To date more than 70% of 2,200 organisations have responded to surveys and the process will be completed by June 2002.

Circulation of vaccine-derived polioviruses

Concerns about environmental contamination with vaccine-derived poliovirus (VDPV) and the possibility of viral reversion to neurovirulence, prompted the WHO to commission a study on the transmission and persistence of poliovirus. The authors concluded that 'OPV viruses could persist under various plausible circumstances, and that this potential should be a major consideration when planning the cessation of OPV vaccination'.[15]

Concerns about viral reversion to neurovirulence have been bolstered recently by three separate reports. An outbreak of 21 cases of polio in the Dominican Republic and Haiti which began in October 2000,[16] has been shown to be associated with a vaccine-derived poliovirus type 1, which had recovered the capacity to cause paralytic disease. A retrospective study of polioviruses circulating in Egypt between 1982 and 1993 demonstrated that a vaccine derived poliovirus type 2 was associated with 32 cases of polio.[17] A third outbreak of paralytic disease associated with vaccine derived poliovirus occurred in October 2001 in the Philippines, where 3 children were infected with a poliovirus type 1 variant.[18] The occurrence of variant neurovirulent polioviruses in populations with low vaccination rates in three different geographic areas raise concerns that these could be more widely spread.

The significance of these events for global eradication of polio remains to be evaluated.[19] Increased vigilance may have uncovered what have been infrequent events occurring for some years. A review of more than 2,000 isolates from AFP cases globally has not revealed any additional variant vaccine-derived poliovirus strains.[18]

Stopping polio vaccination in Australia?

One of the main rationales for the polio eradication initiative was that an end to polio would mean financial savings for developing countries, by allowing the cessation of vaccination programs. Globally, these savings were calculated to be US$1.5 billion per annum.[3] However, even before the advent of variant vaccine derived viruses causing polio disease, experts were divided on the vaccine strategies that should be implemented post-eradication.[13]

Some of the advantages and disadvantages of some of the proposed post-eradication vaccination strategies are shown in Table 2. Discussions have revolved around whether it would be 'safe' to discontinue polio vaccination entirely or whether vaccination should continue, either with the cheap live OPV or with the inactivated and more expensive IPV. The advantages of IPV over OPV are that live vaccine viruses would not be released into the environment and the development of variant viruses would be halted. In addition, IPV has not been shown to cause vaccine associated paralytic polio (VAPP) which affects approximately one in 2.4 million OPV recipients.[20]

However, the IPV vaccine is more expensive and must be delivered by injection. Serological studies in developing countries have shown that IPV is much less effective in developing protective

immunity.[3] These disadvantages were important in the initial choice of OPV over IPV in the global polio eradication initiative. New polio vaccines are a distant possibility for the post eradication world. More research is needed to assess the relative value of each strategy. However, recent events indicate that polio vaccination should continue, probably with OPV in use through the developing world and IPV being increasingly used in the wealthier nations.

Alternative polio vaccination strategies need to be considered including the 'pulsed vaccination' of smaller cohorts of children in place of universal vaccination.[21] The Commonwealth Government is currently considering proposals to replace OPV with IPV in the Australian standard vaccination schedule. New vaccines in which IPV is combined with childhood vaccines such as diphtheria, tetanus and pertussis are an option.

Global polio eradication is an issue that will continue to affect Australian polio vaccination policies and surveillance activities. The recent emergence of neurovirulent vaccine derived polioviruses show that complacency about polio is not an option.

Table 2. Some advantages and disadvantages of proposed strategies for future polio vaccination

Strategy	Advantages	Disadvantages	Comments
Coordinated discontinuation of OPV use worldwide after certification of global polio eradication	• Cessation of vaccination estimated to save US$1.5 billion per annum • Cease vaccination when world immunity is maximal, perhaps after global immunisation days	• Potential for transmission of vaccine-derived polioviruses causing disease in susceptible newborns • Need to retain capacity for vaccine production and stockpile vaccine in case of epidemics	• Ethical issues if some countries switch to IPV since developing countries may not be able to afford IPV vaccination
Replacement of OPV with IPV	• Preserve individual immunity • Low risk of VAPP • Eliminate environmental contamination with vaccine derived polio and attendant risks of polio reversion to wild type characteristics of transmission and neurovirulence	• Costly option requiring injection and changes to child vaccination schedule • Seroconversion rates induced by IPV low in developing countries • No financial benefit from polio eradication: indeed a new financial burden for developing countries	• IPV vaccine manufacturers would need to greatly boost production to meet demand • IPV vaccine manufacturers would have bio-security concerns as the last repository of live polioviruses • Increased risks of bloodborne viruses due to increased injections
Development of a new poliovirus vaccine	• Development of new live virus with low risk of causing VAPP	• Major hurdles to regulatory approval • Very large field trials required to prove efficacy in a world where polio is very rare	• Basic research required to identify candidate vaccines • Little financial incentive for vaccine manufacturers to develop new vaccines

Adapted from Wood (2000)[12] and Technical Consultative Group (2002).

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Australia's notifiable diseases status, 2000

Annual report of the National Notifiable Diseases Surveillance System

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With contributions from:

National organisations

Communicable Diseases Network Australia

Australian Childhood Immunisation Register

Australian National Creutzfeldt-Jakob Disease Registry

Australian Gonococcal Surveillance Programme

Australian Meningococcal Surveillance Programme

Australian Sentinel Practice Research Network

Australian Quarantine and Inspection Service

National Centre in HIV Epidemiology and Clinical Research

National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

National Enteric Pathogens Surveillance Scheme

National Rotavirus Research Centre

Sentinel Chicken Surveillance Programme

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Abstract

In 2000, there were 89,740 notifications of communicable diseases in Australia collected by the National Notifiable Diseases Surveillance System (NNDSS). The number of notifications in 2000 was an increase of 5.9 per cent over those reported in 1999 (84,743) and the largest reporting year since the NNDSS commenced in 1991. Notifications in 2000 consisted of 28,341 bloodborne infections (32% of total), 24,319 sexually transmitted infections (27%), 21,303 gastrointestinal infections (24%), 6,617 vaccine preventable infections (7%), 6,069 vectorborne infections (7%), 2,121 other bacterial infections (legionellosis, meningococcal infection, leprosy and tuberculosis) (2%), 969 zoonotic infections (1%) and only one case of a quarantinable infection. Steep declines in some childhood vaccine preventable diseases such as *Haemophilus influenzae* type b, measles, mumps and rubella, continued in 2000. In contrast, notifications of pertussis and legionellosis increased sharply in the year. Notifications of bloodborne viral diseases (particularly hepatitis B and hepatitis C) and some sexually transmitted infections such as chlamydia, continue to increase in Australia. This report also summarises data on communicable diseases from other surveillance systems including the Laboratory Virology and Serology Surveillance Scheme (LabVISE) and sentinel general practitioner schemes. In addition this report comments on other important developments in communicable disease control in Australia in 2000. *Commun Dis Intell* 2002;26:118-203.

Keywords: Surveillance, communicable diseases, epidemiology

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Abbreviations used in this report

ABS	Australian Bureau of Statistics
ACT	Australian Capital Territory
ACIR	Australian Childhood Immunisation Register
ADF	Australian Defence Forces
ADT	Adult diphtheria tetanus vaccine
AFP	Acute flaccid paralysis
AGSP	Australian Gonococcal Surveillance Programme
AIDS	Acquired immune deficiency syndrome
AIHW	Australian Institute of Health and Welfare
Ag	Antigen
AHMC	Australian Health Minister's conference
ASPREN	Australian Sentinel Practice Research Network
ATAGI	Australian Technical Advisory Group on Immunisation
BBV	Bloodborne viruses
CDC	Centres for Disease Control and Prevention, Atlanta, Georgia
CDI	Communicable Diseases Intelligence
CDNA	Communicable Diseases Network Australia
CIJIG	Commonwealth Inter-departmental JETACAR Implementation Group
CJD	Creutzfeldt-Jakob Disease
CSF	Cerebrospinal fluid
DoHA	Department of Health and Ageing
DTP	Diphtheria, Tetanus, Pertussis (vaccine)
EAGAR	Expert Advisory Group on Antimicrobial Resistance
ELISA	Enzyme-linked Immunosorbant assay
GBS	Guillain Barre Syndrome
FAO	Food and Agriculture Organization of the United Nations
HBsAg	Hepatitis B surface antigen
HBV	Hepatitis B virus
HCV	Hepatitis C virus
HDV	Hepatitis D virus
HEV	Hepatitis E virus
Hib	*Haemophilus influenzae* type b
HIV	Human immunodeficiency virus
HUS	Haemolytic uraemic syndrome
ICD–10	International Classification of Diseases, version 10
IFA	Immunofluorescence assay
IgG	Immunoglobulin G
IgM	Immunoglobulin M
JE	Japanese encephalitis
JETACAR	Joint Expert Technical Advisory Committee on Antibiotic Resistance
LabVISE	Laboratory Virology and Serology Reporting Scheme
MMR	Measles-mumps-rubella (vaccine)
MVE	Murray Valley encephalitis
NNDSS	National Notifiable Diseases Surveillance System

NCHECR	National Centre in HIV Epidemiology and Clinical Research
NEC	Not elsewhere classified
NEPSS	National Enteric Pathogen Surveillance Scheme
NHMRC	National Health and Medical Research Council
NMSS	National Mycobacterial Surveillance System
NN	Not notifiable
NSW	New South Wales
NT	Northern Territory
OPV	Oral polio vaccine
PCR	Polymerase chain reaction
Qld	Queensland
SA	South Australia
SD	Statistical Division
SLTEC	Shiga-like toxin producing *Escherichia coli*
STI	Sexually transmitted infection
Tas	Tasmania
TB	Tuberculosis
UK	United Kingdom
USA	United States of America
vCJD	Variant Creutzfeldt-Jakob disease
Vic	Victoria
VPD	Vaccine preventable diseases
VTEC	Verotoxin-producing *Escherichia coli*
WA	Western Australia
WHO	World Health Organization
WPR	Western Pacific Region

2000: The year in review

In 2000, there were continuing challenges to and advances in communicable disease control in Australia. Important initiatives were taken which will have impacts on communicable diseases surveillance and control well into the future.

In September 2000, Sydney hosted the Olympic games. This event drew around 300,000 domestic and international visitors as well as 15,000 athletes and officials from 200 countries. Media attention was intense with around 15,000 media personnel attending the games. The size of this event necessitated active health surveillance covering notifiable diseases as well as surveillance of presentations to emergency departments and medical centres, and environmental and food safety inspections.[1] Data from all these sources were entered into a special database and reviewed daily by medical epidemiologists. High priority diseases for surveillance included foodborne diseases, pneumonia, influenza, pertussis, meningitis, measles and hepatitis. The Games were completed without any major public health incidents.

In April 2000, a large outbreak of legionellosis occurred in Melbourne, with 113 cases notified in Victoria and another 12 cases elsewhere in Australia and New Zealand. The outbreak was associated with visits to the Melbourne aquarium and resulted in 4 deaths. A contaminated water-cooling tower was implicated.

The deployment of 5,500 Australian Defence Forces (ADF) to East Timor in late 1999 resulted in increased exposure to malaria and dengue. Two hundred and sixty-seven ADF personnel contracted malaria, with 64 developing clinical symptoms in East Timor and 212 being diagnosed on return to Australia. A further 26 ADF personnel contracted dengue.

A milestone in communicable disease control was passed in October 2000, when Australia, along with all other countries in the Western Pacific Region (WPR) was declared polio-free by the World Health Organization (WHO). Australia's last case of polio was reported in 1977 and all cases since then have been vaccine associated.[2]

A special issue of the *Medical Journal of Australia* in October 2000 focussed attention on the burden of pneumococcal disease in Australia and future use of pneumococcal vaccines. The incidence of pneumococcal disease among Aboriginal children in central Australia is among the highest in the world.[3] To date, vaccines composed of pneumococcal polysaccharides were the only available and these were not effective in preventing infections in children. In 2000, the first efficacy trial of a new multivalent conjugate vaccine against *Streptococcus pneumoniae* showed a very high protective efficacy against invasive pneumococcal disease in children.[4] The vaccine was licensed for use in Australia in December 2000 and recommendations for a vaccination program in Australian children were published in March 2001. It is hoped that this vaccination program, focussing on groups of children at highest risk of disease, will have a major impact on pneumococcal disease in Australia.

An important new initiative in the control of foodborne disease in Australia was launched in 2000. The Commonwealth Department of Health and Ageing (DoHA) established and funded a collaborative network, called 'OzFoodNet' to enhance the existing surveillance mechanisms for foodborne disease across Australia. OzFoodNet aims to estimate the incidence of foodborne disease in Australia, to learn more about causes and determinants of foodborne disease, to identify risky practices associated with food handling and preparation and to train foodborne disease epidemiologists.[5] Specific studies include a national survey of diarrhoeal disease prevalence, case control studies on risk factors for infections with *Campylobacter*, *Salmonella* Enteriditis, *Listeria* and Shiga-like toxin producing *Escherichia coli* (SLTEC) and developing a register to record foodborne disease outbreaks.

In August 2000, the Commonwealth published an implementation plan in response to the report by the Joint Expert Advisory Committee on Antibiotic Resistance (JETACAR) *The use of antibiotics in food-producing animals: antibiotic resistant bacteria in animals and humans.* An inter-departmental implementation group guided by an expert advisory committee is working on developing surveillance systems to monitor the prevalence of antibiotic resistance and other measures to control the prevalence of antibiotic resistance in Australia.

As international concern over variant Creutzfeldt-Jakob disease (vCJD) increased in 2000, Australian Health Ministers implemented a blood donor deferral policy from people who had resided in the United Kingdom (UK) for 6 months or more during the period 1980 to 1996. No cases of bovine spongiform encephalopathy have been found in Australian cattle nor have there been any cases of vCJD in Australia to date.

Since 1991, national communicable disease notification data has been collected and collated under the National Notifiable Diseases Surveillance System (NNDSS). In 2000, Australian States and Territories agreed with the Commonwealth to collect more comprehensive data on each case notified to the NNDSS and began planning for enhanced surveillance for a number of key diseases. The new data set will provide more detail on the causative organism and the vaccination status of the case, and provide more comprehensive epidemiological data. This will allow more sophisticated analyses of the national communicable diseases data set. Improvements in the electronic transfer of data from States and Territories to NNDSS continued in 2000. Development of a new data acquisition system was commenced and discussion around appropriate data collection for enhanced tuberculosis (TB) surveillance was initiated. This enhanced surveillance was an initiative of the National TB Advisory Committee and will improve national monitoring of TB in Australia by recording complete clinical data, including antibiotic susceptibility, and outcomes of treatment on all notified cases.

In summary, communicable disease control in Australia in 2000 was advanced by the certification of polio eradication and the introduction of new vaccine initiatives for pneumococcal disease. Improvements to understanding the epidemiology of foodborne disease in Australia through the OzFoodNet initiative and the prevalence of antibiotic resistance will have long-term benefits for disease control. Improvements to data quality and information systems will further enhance the national surveillance system and communicable disease control.

Introduction

It is of critical importance to collect, analyse and report surveillance data on potential communicable diseases. This action is essential to the success of public health efforts. Surveillance allows the detection of disease outbreaks prompting the appropriate investigation and control measures to be instigated. Surveillance also allows for the monitoring of trends in disease prevalence and considers the impact and effectiveness of interventions to control the spread of diseases. Surveillance systems exist at national, state and local levels. State and local surveillance systems are crucial to the timely and effective detection and management of outbreaks and in assisting in the effective implementation of national policies. The national surveillance system combines some of the data collected from state and territory-based systems to provide an overview at a national level. Specific functions of the national surveillance system include: detection and management of outbreaks affecting more than one jurisdiction; monitoring of the need for and impact of national control programs; guidance of national policy development; resource allocation and description of the epidemiology of rare diseases for which there are only a few notifications in each jurisdiction. National surveillance also assists with quarantine activities and facilitates agreed international collaborations such as reporting to the WHO.

The National Notifiable Diseases Surveillance System was established in its current form in 1991, under the auspices of the Communicable Diseases Network Australia (CDNA, formally the Communicable Diseases Network of Australia and New Zealand). The CDNA monitors the notification/reporting of an agreed list of communicable diseases in Australia. Data are regularly published in the *Communicable Diseases Intelligence (CDI)* journal and on the Communicable Diseases — Australia Website. This is achieved through the national collation of notifications of these diseases received by health authorities in the States and Territories. In 2000, 50 diseases or disease categories were included, largely as recommended by the National Health and Medical Research Council (NHMRC).[6] In years since 2000 the list of notifiable diseases and categories has undergone review and revision. Information collected on notifiable diseases has been published in the Annual Report of the NNDSS since 1991.[7,8,9,10,11,12,13,14,15]

In 2000, 50 diseases or disease categories were nationally notifiable in Australia (Table 1) and the national case definitions used in this year are listed in Appendix 1a–1h.

Table 1. Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2000

Disease group	Disease	Comments
Bloodborne diseases	Hepatitis B (incident)	All jurisdictions
	Hepatitis B (unspecified)	All jurisdictions except NT
	Hepatitis C (incident)	All jurisdictions except NT, Qld*
	Hepatitis C (unspecified)	All jurisdictions
	Hepatitis D	All jurisdictions except WA
	Hepatitis (NEC)	All jurisdictions except WA
Gastrointestinal diseases	Botulism	All jurisdictions except WA
	Campylobacteriosis	All jurisdictions except NSW
	Haemolytic uraemic syndrome	All jurisdictions
	Hepatitis A	All jurisdictions
	Hepatitis E	All jurisdictions except WA
	Listeriosis	All jurisdictions
	Salmonellosis	All jurisdictions
	Shigellosis	All jurisdictions except NSW
	SLTEC,VTEC	All jurisdictions except Qld, WA
	Typhoid	All jurisdictions
	Yersiniosis	All jurisdictions except NSW
Quarantinable diseases	Cholera	All jurisdictions
	Plague	All jurisdictions
	Rabies	All jurisdictions
	Viral haemorrhagic fever	All jurisdictions
	Yellow fever	All jurisdictions
Sexually transmitted infections	Chancroid	All jurisdictions
	Chlamydial infections	All jurisdictions
	Donovanosis	All jurisdictions except NSW, SA
	Gonococcal infection	All jurisdictions
	Lymphogranuloma venereum	All jurisdictions except WA
	Syphilis	All jurisdictions
Vaccine preventable diseases	Diphtheria	All jurisdictions
	Haemophilus influenzae type B	All jurisdictions
	Measles	All jurisdictions
	Mumps	All jurisdictions except Qld†
	Pertussis	All jurisdictions
	Poliomyelitis	All jurisdictions
	Rubella	All jurisdictions
	Tetanus	All jurisdictions
Vectorborne diseases	Arbovirus infection (NEC)	All jurisdictions
	Barmah Forest virus infection	All jurisdictions
	Dengue	All jurisdictions
	Malaria	All jurisdictions
	Ross River virus infection	All jurisdictions
Zoonoses	Brucellosis	All jurisdictions
	Hydatid disease	All jurisdictions except NSW
	Leptospirosis	All jurisdictions
	Ornithosis	All jurisdictions except NSW and Qld
	Q fever	All jurisdictions
Other bacterial infections	Legionellosis	All jurisdictions
	Leprosy	All jurisdictions
	Meningococcal infection	All jurisdictions
	Tuberculosis	All jurisdictions

* Notifications of hepatitis C (incident) were reported under hepatitis C (unspecified) in the Northern Territory and Queensland.

† Notification of mumps was removed from the notification list in Queensland from 2 July 1999 and the entire year of 2000.

NEC: not elsewhere classified

Methods

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

In 2000, data were transmitted from States and Territories to DoHA, fortnightly. Summaries of the data were published on the Communicable Diseases — Australia Website fortnightly and in the *CDI* monthly. The Commonwealth received final data sets from the States and Territories of cases reported in 2000 by August 2001. Missing data and apparent errors together with any queries arising from the data were returned to jurisdictions for review, correction of errors and ascertainment of completeness of case information for the year.

For each case the national data set included fields for a unique record reference number; jurisdiction of notification; disease code; age; sex; Indigenous status; postcode of residence; the date of onset of the disease and date of report to the State or Territory health authority. Analysis of the data by Indigenous status was not possible because of the incomplete reporting of this information. Additional information was available on the species and serogroups isolated in cases of legionellosis, meningococcal disease and malaria, and on the vaccination status in cases of childhood vaccine preventable diseases. Additional information was obtained from States and Territories concerning mortality and specific health risk factors of some diseases.

Analyses in this report are based on date of disease onset, unless specified. For analysis of seasonal trends, notifications were reported by month of onset. Population notification rates were calculated using 2000 mid-year estimates of the resident population supplied by the Australian Bureau of Statistics (ABS). An adjusted rate was calculated where a disease was not notifiable in a State or Territory, using a denominator which excluded that population.

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

Notes on interpretation

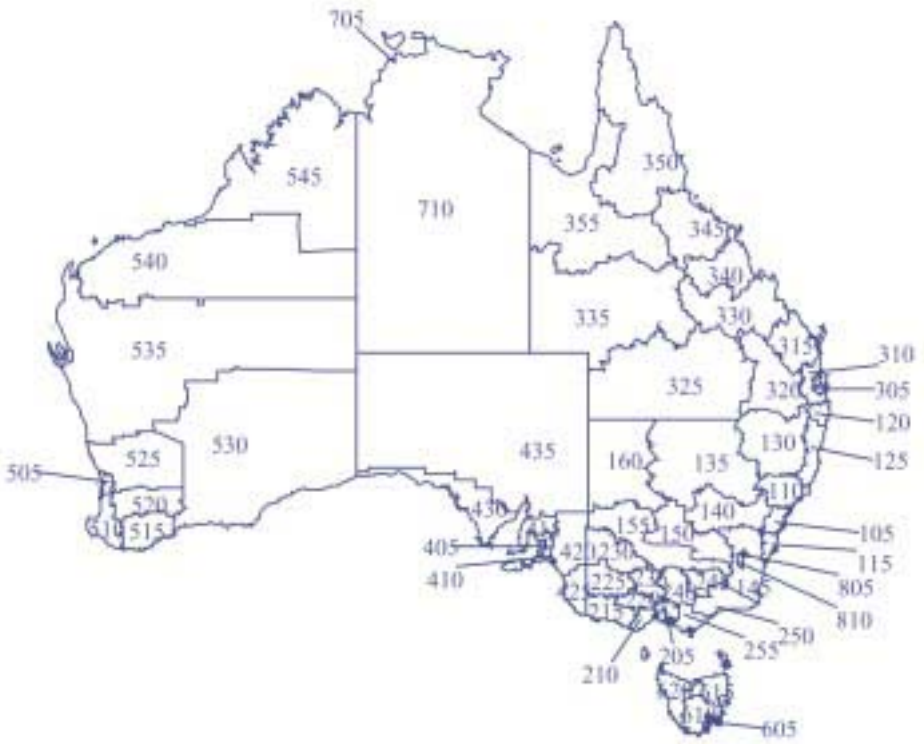
The notifications compiled by the NNDSS may be influenced by a number of factors that should be considered when interpreting the data. Due to under-reporting, notified cases are likely to only represent a proportion of the total number of cases that occurred (Figure 1). This proportion (the 'notified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

Figure 1. Communicable disease surveillance pyramid

Adopted from CDC Website.
(http://www.cdc.gov/foodnet/Surveys.htm#whatpyr)

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

Map 1. Australian Bureau of Statistics Statistical Divisions



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

Statistical Division	Population
Queensland continued	
320 Darling Downs	202,352
325 South West	25,597
330 Fitzroy	181,206,215
335 Central West	12,135
340 Mackay	127,531
345 Northern	200,174
350 Far North	225,522
355 North West	35,760
South Australia	
405 Adelaide	1,096,102
410 Outer Adelaide	110,663
415 Yorke & Lower North	44,225
420 Murray Lands	68,497
425 South East	62,794
430 Eyre	33,493
435 Northern	81,860
Tasmania	
605 Greater Hobart	194,228
610 Southern	34,832
615 Northern	133,080
620 Mersey-Lyell	108,236

Statistical Division	Population
Victoria	
205 Melbourne	3,466,025
210 Barwon	249,067
215 Western District	99,477
220 Central Highlands	138,229
225 Wimmera	50,838
230 Mallee	88,372
235 Loddon-Campaspe	162,031
240 Goulburn	188,124
245 Ovens-Murray	90,102
250 East Gippsland	79,849
255 Gippsland	154,034
Western Australia	
505 Perth	1,381,127
510 South West	187,862
515 Lower Great Southern	52,128
520 Upper Great Southern	19,610
525 Midlands	52,304
530 South Eastern	58,926
535 Central	60,300
540 Pilbara	40,429
545 Kimberley	30,539
Total Australia	**19,157,037**

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

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

As no personal identifiers are collected in records, duplication in reporting may occur if patients moved from one jurisdiction to another and were notified in both. Data from those Statistical Divisions with small populations (Map 1) may result in high notification rates even with small numbers of cases.

The completeness of data in this report is summarised in Appendix 2. The patient's sex was missing in 0.5 per cent of notifications (n = 420) and patient's age missing in 0.4 per cent of notifications (n = 340). The patient's Indigenous status was reported for 28,552 notifications (31.8%) nationally. The proportion of reports with missing data in these fields varied by State or Territory, and also by disease.

Data were analysed by date of disease onset, unless specified. The date of disease onset is uncertain for some communicable diseases and is often equivalent to the date of presentation to a medical practitioner or date of specimen collection at a laboratory. Analysis by disease onset is an attempt to estimate disease activity within a reporting period. Analysis by date of onset should be interpreted with caution, particularly for chronic diseases such as hepatitis B and C, as considerable time may have elapsed between onset and report date for these diseases. To overcome this problem, analysis was performed by report date for hepatitis B (unspecified) and hepatitis C (unspecified).

Rates per 100,000 population were calculated using State/Territory and national population estimates for mid-year 2000 (Appendix 3) supplied by the Australian Bureau of Statistics. Mortality statistics for 2000 were available from the ABS in 2001. The Australian Institute of Health and Welfare (AIHW) supplied hospital admission data for the financial year 1999/2000.

Between May and August every year, the NNDSS receives a final annual dataset from all jurisdictions to update its system. This yearly operation only updates the notifications reported to the NNDSS during the last calendar year. States and Territories continue to revise totals from previous years as duplicates are removed and other data corrected. However, the NNDSS had not revised its historical notifications since 1991. As a result, there was considerable difference in the number of notifications held in the NNDSS and the State and Territory records. Providing high quality and precise information that is consistent with State and Territory records is a vital part of maintaining good surveillance information. In 2001, the CDNA approved the revising of the NNDSS records with jurisdictions' 1991 to 1999 historical notifications. During November to December 2001, all jurisdictions except Victoria resent notifications collected between 1991 and 1999 to the NNDSS. Victoria confirmed that records held at the Commonwealth level were accurate. Comparative historical data for 1991 to 1999 used in this report represents more accurate information and may vary from previous reports.

During 2000, data were analysed monthly and the result and commentary published in *CDI*. In contrast, this report is based on 'finalised' annual data from each jurisdiction, from which duplicates or erroneous records have been removed. For this reason, totals in this report may vary from the cumulative totals of the numbers reported in the monthly *CDI* reports. This report is informed by the discussions and comments of members of the CDNA, who met fortnightly by teleconference to discuss developments in communicable disease in their jurisdiction. The CDNA data managers also met through 2000 and their contribution to accurate data in this report is gratefully acknowledged.

Results

Summary of 2000 data

There were a total of 89,740 communicable disease notifications for 2000 (Table 2). Notification rates per 100,000 population for each disease by State or Territory are described in Table 3.

The number of notifications in 2000 was an increase of 5.9 per cent on notifications in 1999 (84,743) and the largest number of reports since the NNDSS commenced in 1991 (Figure 2). Nationally in 2000, bloodborne infections remained the most frequently notified disease group (28,341 cases; 32% of total), followed by 24,319 sexually transmitted infections (27%), 21,303 gastrointestinal infections (24%), 6,617 vaccine preventable diseases (7%), 6,069 vectorborne diseases (7%), 2,121 other bacterial infections (2%), 969 zoonotic infections (1%) and only one case of a quarantinable disease (Figure 3).

Figure 2. Trends in notification rates of communicable diseases, Australia, 1991 to 2000

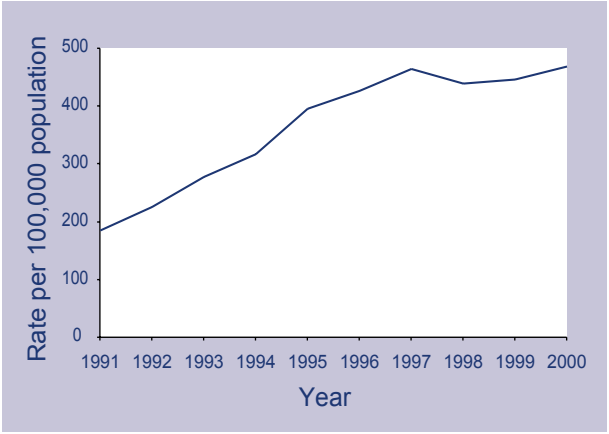
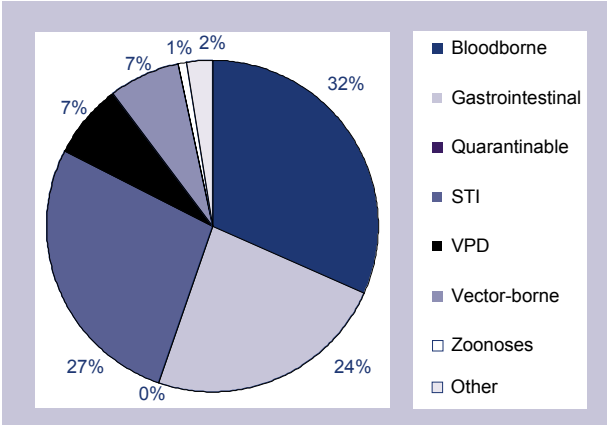
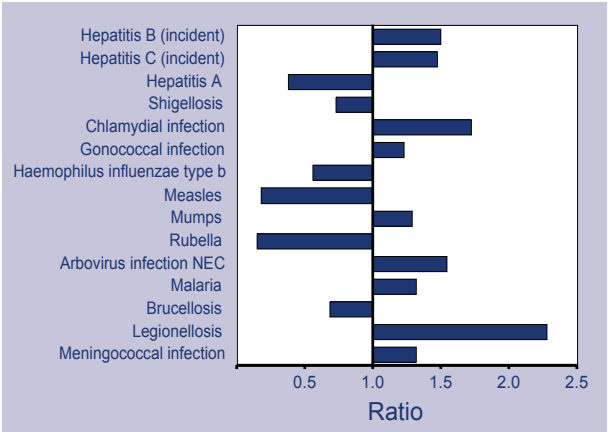


Figure 3. Breakdown of communicable disease notifications by disease category



The major changes in notifications in 2000 are shown in Figure 4. as a ratio of notifications received in the year compared with the mean of the preceding 5 years. Only diseases with major changes in numbers of notifications in 2000 are shown. There were major increases in notifications of legionellosis and dengue. Increases were also noted in the reporting of hepatitis B (both incident and unspecified), hepatitis C (incident), gonococcal infection, mumps, malaria and meningococcal infection. Measles notifications fell by more than 50 per cent compared with 1999. Declines in *Haemophilus influenzae* type b (Hib) infections and rubella were also noted.

Figure 4. Selected diseases from National Notifiable Diseases Surveillance System, comparison of totals for 2000 with previous 5 year mean



In 2000, infectious diseases accounted for 3.6 per cent of all deaths in Australia (4,582 deaths, 23.9 deaths per 100,000 population). Pneumonia and influenza remained as the major cause of mortality, accounting for more 50 per cent of deaths from infectious diseases (2,937 deaths, 15.3 deaths per 100,000 population). Death rates from pneumonic and influenza generally increased with age and were greater for males than females aged 60 years and over. There was a total of 12,859 infectious disease related hospitalisations during the 1999/2000 financial year. (Source: National Hospital Morbidity Database, 1990–2000: AIHW) Among these hospitalisations, influenza/pneumonia was the most common cause for admission, accounting for 20.1 per cent of the total hospitalisations (2,591 admissions). It should be noted that a range of causative agents are included in the broad ICD–10 coding group of 'influenza/pneumonia'.

Table 2. Notifications of communicable diseases, Australia, 2000, by State or Territory*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis B (incident)	3	96	6	56	30	18	114	72	**395**
Hepatitis B (unspecified)†‡	48	3,893	NN	896	257	48	1,964	802	**7,908**
Hepatitis C (incident)	20	139	–	–	89	31	87	75	**441**
Hepatitis C (unspecified)†‡§	212	7,265	183	3,395	788	335	5,730	1,661	**19,569**
Hepatitis D	0	10	0	5	0	0	12	NN	**27**
Hepatitis (NEC)	0	1	0	0	0	0	0	NN	**1**
Gastrointestinal diseases									
Botulism	0	0	1	0	0	0	1	NN	**2**
Campylobacteriosis‖	333	–	182	3,675	1,883	510	5,037	1,975	**13,595**
Haemolytic uraemic syndrome	0	9	0	2	1	0	2	1	**15**
Hepatitis A	5	200	44	133	54	3	193	180	**812**
Hepatitis E	0	9	0	0	0	1	0	NN	**10**
Listeriosis	0	18	3	13	7	3	10	13	**67**
Salmonellosis	105	1,409	304	1,827	450	131	1,021	904	**6,151**
Shigellosis‖	7	–	114	108	30	2	120	106	**487**
SLTEC,VTEC¶	0	0	0	NN	33	0	0	NN	**33**
Typhoid	0	27	0	2	3	0	14	12	**58**
Yersiniosis‖	3	–	2	59	0	0	8	1	**73**
Quarantinable diseases									
Cholera	0	0	0	0	1	0	0	0	**1**
Plague	0	0	0	0	0	0	0	0	**0**
Rabies	0	0	0	0	0	0	0	0	**0**
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	**0**
Yellow fever	0	0	0	0	0	0	0	0	**0**
Sexually transmissible diseases									
Chancroid	0	0	0	0	0	0	0	0	**0**
Chlamydial infection	243	3,482	959	4,931	1,023	332	3,336	2,560	**16,866**
Donovanosis	0	NN	5	6	NN	0	0	1	**12**
Gonococcal infection**	14	1,060	1,128	1,137	270	17	742	1,318	**5,686**
Lymphogranuloma venereum	0	0	0	0	0	0	0	NN	**0**
Syphilis††	13	541	175	887	13	9	8	109	**1,755**

Table 2. Notifications of communicable diseases, Australia, 2000, by State or Territory,* continued

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Vaccine preventable diseases									
Diphtheria	0	0	0	0	0	0	0	0	**0**
Haemophilus influenzae type b	0	8	2	12	2	0	3	1	**28**
Measles	3	35	0	26	11	1	21	10	**107**
Mumps	17	92	4	NN	15	2	43	39	**212**
Pertussis	208	3,683	5	525	588	143	699	91	**5,942**
Poliomyelitis	0	0	0	0	0	0	0	0	**0**
Rubella‡‡	4	191	0	46	7	1	67	6	**322**
Tetanus	0	2	0	0	3	0	1	0	**6**
Vectorborne diseases									
Arbovirus infection NEC	0	12	9	10	0	1	26	11	**69**
Barmah Forest virus infection	0	190	9	346	12	0	19	58	**634**
Dengue	1	21	93	84	6	0	2	8	**215**
Malaria	18	231	76	409	41	7	115	54	**951**
Ross River virus infection	16	746	128	1,477	415	8	326	1,084	**4,200**
Zoonoses									
Brucellosis	0	1	0	26	0	0	0	0	**27**
Hydatid infection	0	NN	0	8	0	0	13	5	**26**
Leptospirosis	1	53	8	134	8	0	35	4	**243**
Ornithosis	0	NN	1	NN	6	6	85	2	**100**
Q fever	0	130	0	390	11	1	27	14	**573**
Other diseases									
Legionellosis	5	41	1	47	89	4	250	35	**472**
Leprosy	0	2	0	1	1	0	0	0	**4**
Meningococcal infection	5	253	9	60	32	15	162	85	**621**
Tuberculosis	18	438	43	89	58	10	284	84	**1,024**
Total	**1,302**	**24,288**	**3,494**	**20,822**	**6,237**	**1,639**	**20,577**	**11,381**	**89,740**

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

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

‡ The analysis was performed by report date.

§ Includes hepatitis C (incident) cases in Northern Territory and Queensland.

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

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

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

†† Includes congenital syphilis.

‡‡ Includes congenital rubella.

NN Not notifiable.

NEC Not Elsewhere Classified.

– Elsewhere classified.

Table 3. Notification rates of communicable diseases, Australia, 2000, by State or Territory (rate per 100,000 population)*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis B (incident)	1.0	1.5	3.1	1.6	2.0	3.8	2.4	3.8	**2.1**
Hepatitis B (unspecified)†‡	15.3	60.2	NN	25.1	17.2	10.2	41.2	42.6	**41.7**
Hepatitis C (incident)	6.4	2.2	–	–	5.9	6.6	1.8	4.0	**2.9**
Hepatitis C (unspecified)†‡§	67.5	112.4	93.6	95.2	52.6	71.2	120.2	88.2	**102.2**
Hepatitis D	0.0	0.2	0.0	0.1	0.0	0.0	0.3	NN	**0.2**
Hepatitis (NEC)	0.0	< 0.1	0.0	0.0	0.0	0.0	0.0	NN	**< 0.1**
Gastrointestinal diseases									
Botulism	0.0	0.0	0.5	0.0	0.0	0.0	< 0.1	NN	**< 0.1**
Campylobacteriosis‖	106.0	–	93.1	103.0	125.7	108.4	105.7	104.8	**107.1**
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.1	0.1	0.0	< 0.1	0.1	**0.1**
Hepatitis A	1.6	3.1	22.5	3.7	3.6	0.6	4.0	9.6	**4.2**
Hepatitis E	0.0	0.1	0.0	0.0	0.0	0.2	0.0	NN	**0.1**
Listeriosis	0.0	0.3	1.5	0.4	0.5	0.6	0.2	0.7	**0.3**
Salmonellosis	33.4	21.8	155.5	51.2	30.0	27.9	21.4	48.0	**32.1**
Shigellosis‖	2.2	–	58.3	3.0	2.0	0.4	2.5	5.6	**3.8**
SLTEC,VTEC¶	0.0	0.0	0.0	NN	2.2	0.0	0.0	NN	**0.2**
Typhoid	0.0	0.4	0.0	0.1	0.2	0.0	0.3	0.6	**0.3**
Yersiniosis‖	1.0	–	1.0	1.7	0.0	0.0	0.2	0.1	**0.6**
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	**< 0.1**
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Sexually transmissible diseases									
Chancroid	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Chlamydial infection	77.4	53.9	490.6	138.3	68.3	70.6	70.0	135.9	**88.0**
Donovanosis	0.0	NN	2.6	0.2	NN	0.0	0.0	0.1	**0.1**
Gonococcal infection**	4.5	16.4	577.1	31.9	18.0	3.6	15.6	70.0	**29.7**
Lymphogranuloma venereum	0.0	0.0	0.0	0.0	0.0	0.0	0.0	NN	**0.0**
Syphilis††	4.1	8.4	89.5	24.9	0.9	1.9	0.2	5.8	**9.2**

Table 3. Notification rates of communicable diseases, Australia, 2000, by State or Territory (rate per 100,000 population)*, continued

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Haemophilus influenzae type b	0.0	0.1	1.0	0.3	0.1	0.0	0.1	0.1	**0.1**
Measles	1.0	0.5	0.0	0.7	0.7	0.2	0.4	0.5	**0.6**
Mumps	5.4	1.4	2.0	NN	1.0	0.4	0.9	2.1	**1.4**
Pertussis	66.2	57.0	2.6	14.7	39.3	30.4	14.7	4.8	**31.0**
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Rubella‡‡	1.3	3.0	0.0	1.3	0.5	0.2	1.4	0.3	**1.7**
Tetanus	0.0	< 0.1	0.0	0.0	0.2	0.0	< 0.1	0.0	**< 0.1**
Vectorborne diseases									
Arbovirus infection NEC	0.0	0.2	4.6	0.3	0.0	0.2	0.5	0.6	**0.4**
Barmah Forest virus infection	0.0	2.9	4.6	9.7	0.8	0.0	0.4	3.1	**3.3**
Dengue	0.3	0.3	47.6	2.4	0.4	0.0	< 0.1	0.4	**1.1**
Malaria	5.7	3.6	38.9	11.5	2.7	1.5	2.4	2.9	**5.0**
Ross River virus infection	5.1	11.5	65.5	41.4	27.7	1.7	6.8	57.5	**21.9**
Zoonoses									
Brucellosis	0.0	< 0.1	0.0	0.7	0.0	0.0	0.0	0.0	**0.1**
Hydatid infection	0.0	NN	0.0	0.2	0.0	0.0	0.3	0.3	**0.2**
Leptospirosis	0.3	0.8	4.1	3.8	0.5	0.0	0.7	0.2	**1.3**
Ornithosis	0.0	NN	0.5	NN	0.4	1.3	1.8	0.1	**1.1**
Q fever	0.0	2.0	0.0	10.9	0.7	0.2	0.6	0.7	**3.0**
Other diseases									
Legionellosis	1.6	0.6	0.5	1.3	5.9	0.9	5.2	1.9	**2.5**
Leprosy	0.0	< 0.1	0.0	< 0.1	0.1	0.0	0.0	0.0	**< 0.1**
Meningococcal infection	1.6	3.9	4.6	1.7	2.1	3.2	3.4	4.5	**3.2**
Tuberculosis	5.7	6.8	22.0	2.5	3.9	2.1	6.0	4.5	**5.3**

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

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

‡ The analysis was performed by report date.

§ Includes hepatitis C (incident) cases in Northern Territory and Queensland.

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

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

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

†† Includes congenital syphilis.

‡‡ Includes congenital rubella.

NN Not notifiable.

NEC Not elsewhere classified.

– Elsewhere classified.

Bloodborne diseases

Introduction

In 2000, bloodborne viruses (BBV) reported to the NNDSS included hepatitis B, C, D and hepatitis 'not elsewhere classified' (NEC). Newly acquired hepatitis C virus (HCV) and hepatitis B virus (HBV) infections (incident) were differentiated from those where the timing of disease acquisition is unknown (unspecified). HIV and AIDS diagnoses are reported directly to the National Centre in HIV Epidemiology and Clinical Research (NCHECR). Information on national HIV/AIDS surveillance can be obtained through the NCHECR Website at www.med.unsw.edu.au/nchecr.

As considerable time may have elapsed between onset and report date for chronic hepatitis infections, the analysis of unspecified HBV and unspecified HCV infections in the following sections is by report date, rather than by onset date.

In 2000, bloodborne virus infections accounted for 28,341 notifications to the NNDSS, which was 31.6 per cent of the total notified cases.

The overall trends in the number of notifications and rates for bloodborne viruses reported to the NNDSS since 1991 are shown in Tables 4 and 5. Hepatitis C remains the most commonly notified BBV in Australia. While most of the BBV show an increase in the total number of notifications across this reporting period, the increases are likely to reflect changes in surveillance practices rather than a true change in disease activity. Changes in surveillance are discussed on a disease by disease basis in the following sections. Only the reporting of hepatitis NEC has decreased over time, probably due to improved classification into the other hepatitis groups

Table 4. Trends in notifications of bloodborne viruses, Australia, 1991 to 2000*†

Disease	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Hepatitis B (incident)	–	–	–	283	271	212	269	265	303	395
Hepatitis B (unspecified)	3,469	4,847	5,282	5,394	4,434	5,580	6,542	6,562	7,164	7,908
Hepatitis C (incident)	–	–	25	26	77	71	154	350	396	441
Hepatitis C (unspecified)	–	–	–	–	17,154	17,674	17,290	18,075	18,655	19,569
Hepatitis D	–	–	–	–	–	–	–	–	19	27
Hepatitis (NEC)	253	34	33	23	12	15	6	4	0	1

* Notifications of hepatitis B (unspecified) and hepatitis C (unspecified) were analysed by report date.

† All jurisdictions reported for all years with the following exceptions:

Hepatitis B (incident) not reported from the Australian Capital Territory (1994)

Hepatitis B (unspecified) not reported from the Northern Territory (1991 to 2000)

Hepatitis C (incident) not separated from hepatitis C (unspecified) in Queensland or the Northern Territory (1991 to 2000)

Hepatitis D not reported from Western Australia

Hepatitis (NEC) not reported from Western Australia

Table 5. Trends in notification rates of bloodborne viruses, Australia, 1991 to 2000*† (rate per 100,000 population)

Disease	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Hepatitis B (incident)	–	–	–	1.6	1.5	1.2	1.5	1.4	1.6	2.1
Hepatitis B (unspecified)	20.3	28.0	30.2	30.5	24.8	30.8	35.7	35.4	38.2	41.7
Hepatitis C (incident)	–	–	0.2	0.2	0.5	0.5	1.0	2.3	2.6	2.9
Hepatitis C (unspecified)	–	–	–	–	94.9	96.5	93.4	96.5	98.4	102.2
Hepatitis D	–	–	–	–	–	–	–	–	0.1	0.2
Hepatitis (NEC)	1.6	0.2	0.2	0.1	0.1	0.1	< 0.1	< 0.1	0.0	< 0.1

* Notifications of hepatitis B (unspecified) and hepatitis C (unspecified) were analysed by report date.

† All jurisdictions reported for all years with the following exceptions:

Hepatitis B (incident) not reported from the Australian Capital Territory (1994).

Hepatitis B (unspecified) not reported from the Northern Territory (1991 to 2000).

Hepatitis C (incident) not separated from hepatitis C (unspecified) in Queensland or the Northern Territory (1991 to 2000).

Hepatitis D not reported from Western Australia.

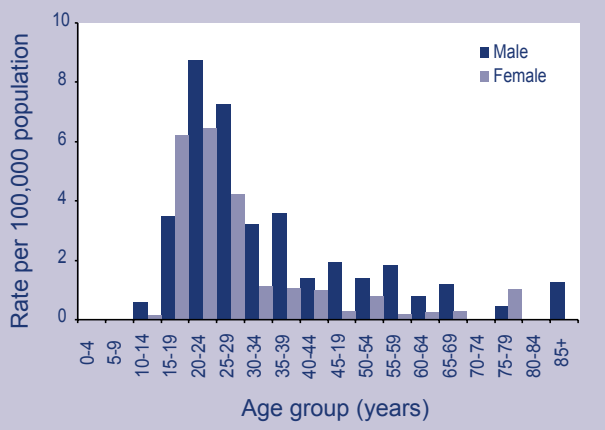
Hepatitis (NEC) not reported from Western Australia.

Hepatitis B

In the early 1990s incident and unspecified hepatitis B notifications were not reported separately by most jurisdictions. Since 1994, all jurisdictions have reported incident cases of hepatitis B to the NNDSS. The overall trend in incident HBV notification rates between 1994 and 2000 shows a relatively stable reporting rate, between 1–2 cases per 100,000 population.

In total, 395 incident cases of hepatitis B were reported to the NNDSS with an onset date in 2000, giving a national notification rate of 2.1 cases per 100,000 population for this year. This represents an increase from the 303 incident cases reported in 1999 (1.6 cases per 100,000 population), with the most notable increases in the number of notifications from Western Australia, Tasmania and New South Wales. In 2000, the highest rates were reported from Western Australia (3.8 cases per 100,000 population), Tasmania (3.8 cases per 100,000 population) and the Northern Territory (3.1 cases per 100,000 population). The majority of incident hepatitis B notifications were in the 20–24 year age group (Figure 5). Overall, infections in males exceeded those in females (male to female ratio of 1.6:1).

Figure 5. Notification rates of incident hepatitis B infections, Australia, 2000 by age and sex



Risk factor information for incident HBV infection was available from four jurisdictions, the Australian Capital Territory, South Australia, Tasmania and Victoria and is summarised in Table 6. The following analyses refer only to incident HBV cases reported in these jurisdictions in 2000, thus the jurisdictional totals reported below may vary from the analysis by onset date.

Table 6. Risk factors identified in notifications of incident hepatitis B virus infections, 2000, Australia, by reporting State or Territory

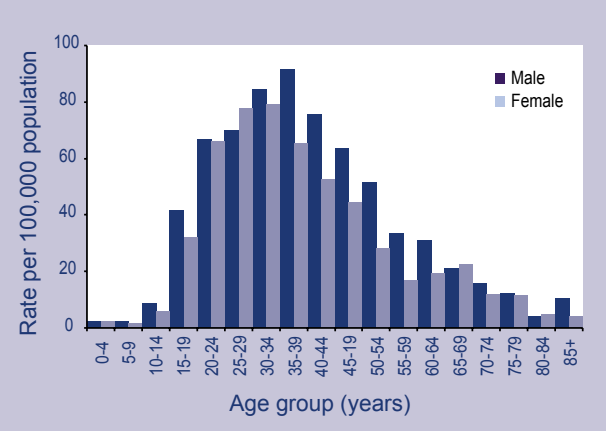
Risk factor	Australian Capital Territory		South Australia		Tasmania		Victoria	
	n	%	n	%	n	%	n	%
Injecting drug user*	3	100	15	50	11	61	65	57
Sexual contact with HBV case	0	0	5	17	0	0	31	27
Household/other contact	0	0	2	6	1	5	1	1
Overseas travel	0	0	3	10	0	0	0	0
Occupational	0	0	0	0	0	0	1	1
Other	0	0	0	0	3	17	0	0
None identified	0	0	5	17	3	17	16	14
Total	**3**	**100**	**30**	**100**	**18**	**100**	**114**	**100**

* Injecting drug users may have multiple risk factors for HBV infection

Unspecified hepatitis B notifications have been reported to the NNDSS by all jurisdictions except the Northern Territory since 1997. The notification rate has remained stable between 1997 and 2000, at around 40 cases per 100,000 population (Table 5). In 2000 there were 7,908 unspecified HBV cases notified, at a rate of 41.7 cases per 100,000 population (Tables 4 and 5). This rate is consistent with that recorded in 1999 (38.2 cases per 100,000 population). The male to female ratio for unspecified HBV cases reported in 2000 was 1.2:1. By jurisdiction, the highest rates of notification were in New South Wales (60.2 cases per 100,000 population), Western Australia (42.6 cases per 100,000 population) and Victoria (41.2 cases per 100,000 population). The highest rates were in the 35–39 year age group for men (91.4 cases per 100,000 population) and in the 30–34 year age group for women (79.1 cases per 100,000 population, Figure 6).

Figure 6 indicates a small number of unspecified HBV cases reported in the 0–4 age group. Some of these cases may be perinatally acquired (particularly in 0–1 year olds), and could be reported as incident cases if the timing of infection is known to be at birth.

Figure 6. Notification rates of unspecified hepatitis B infections, Australia, 2000, by age and sex



While free universal neonatal vaccination was introduced in the Northern Territory in 1990, prior to 1997 most other jurisdictions only vaccinated infants from ethnic groups with a high hepatitis B carriage rate, or those born to known HBV positive mothers. In 1997 there was an interim recommendation that universal vaccination of infants at birth be introduced, and in 2000, with the availability of combination vaccines, DTPa–hepB vaccine was included in the childhood immunisation schedule. Continued surveillance is essential to measure the impact of this vaccination program.

Hepatitis C

Hepatitis C infection has been notifiable in most Australian jurisdictions since 1991 (Table 7). The total number of unspecified hepatitis C notifications has remained stable since 1994 at around 15,000–20,000 cases per annum.

In 2000, there were 19,569 unspecified hepatitis C infections reported to the NNDSS, a notification rate of 102.2 cases per 100,000 population, slightly higher than the 98.4 cases per 100,000 population reported in 1999. Of the total notifications of unspecified hepatitis C, 37 per cent of the notifications were from New South Wales. The highest notification rates were from Victoria (120.2 cases per 100,000 population) and New South Wales (112.4 cases per 100,000 population). The male to female ratio was 1.8:1. The highest notification rates were in the 25–29 year age group for males (279.7 cases per 100,000 population) and in the 20–24 year age group for females (159.9 cases per 100,000 population, Figure 7).

Similar to HBV, there were a number of HCV notifications in the 0-4 age group (Table 8) which could be classified as incident if perinatally acquired.

Incident cases of hepatitis C have been separately notifiable since 1997 in all jurisdictions except the Northern Territory and Queensland (Table 9). It is recognised that the number of notifications vastly underestimates the true incidence of hepatitis C in Australia. The increase in incident hepatitis C notifications to the NNDSS should not necessarily be interpreted as evidence of increasing transmission in the Australian community. Instead the increase in the number of incident HCV notifications is largely a product of improved surveillance, increased awareness, and more widespread testing.

The numbers of incident cases detected are likely to be affected by the surveillance methods.[16] In the larger jurisdictions classification of incident cases is determined by passive reporting. In smaller jurisdictions, where all (or the majority) of hepatitis C notifications were actively investigated to determine if they were incident or prevalent during this time period, a much higher proportion of incident cases was reported.

Figure 7. Notification rates of unspecified hepatitis C infections, Australia, 2000, by age and sex

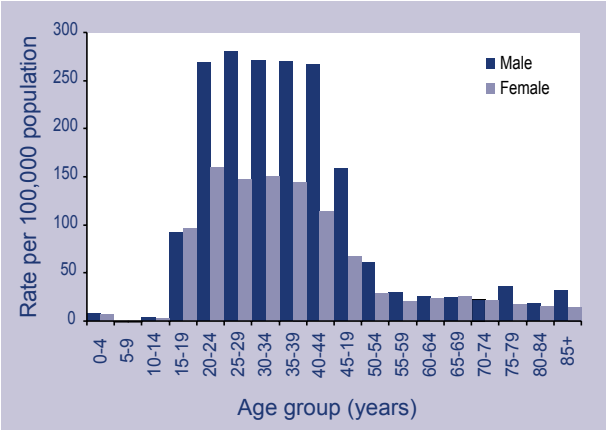


Table 7. Trends in notifications of unspecified hepatitis C virus infections, Australia 1991 to 2000, by State or Territory and date of report

Year	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
1991	59	657	10	1,491	NR	NR	1,667	NR	**3,884**
1992	110	3,761	93	2,702	NR	NR	1,262	NR	**7,928**
1993	244	5,640	212	2,670	NR	NR	2,659	1,106	**12,531**
1994	308	7,564	301	2,990	NR	NR	3,523	1,305	**15,991**
1995	330	6,782	301	2,808	1,026	274	4,506	1,127	**17,154**
1996	267	7,318	216	2,796	1,075	291	4,597	1,114	**17,674**
1997	315	6,775	295	2,843	835	234	4,940	1,053	**17,290**
1998	290	6,759	233	2,921	795	275	5,681	1,121	**18,075**
1999	282	6,780	191	3,046	854	310	6,165	1,027	**18,655**
2000	212	7,265	183	3,395	788	335	5,730	1,661	**19,569**

NR not reported

Table 8. Trends in notifications of hepatitis C virus infections in the 0–4 age group, Australia, 1997 to 2000

Year	Incident HCV infections*	Unspecified HCV infections†	Total
1997	0	167	167
1998	5	573	578
1999	1	105	106
2000	1	97	98

* By date of onset.

† By date of report.

Table 9. Trends in notifications of incident hepatitis C virus infections, by State or Territory, 1993 to 2000

Year	ACT	NSW	SA	Tas	Vic	WA	Aust
1993	NR*	23	NR	NR	NR	2	**25**
1994	6	20	NR	NR	NR	0	**26**
1995	8	33	33	2	NR	1	**77**
1996	8	19	28	4	NR	12	**71**
1997	3	19	48	2	9	73	**154**
1998	8	110	67	18	21	126	**350**
1999	20	100	80	18	70	108	**396**
2000	20	139	89	31	87	75	**441**

NR not reported

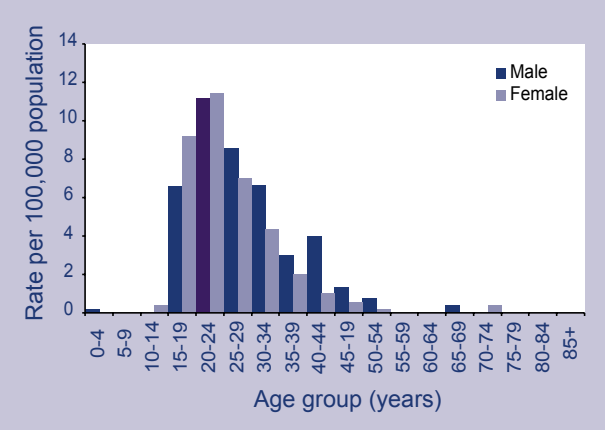
Table 10. Demographics of incident hepatitis C cases reported in the Australian Capital Territory, the Northern Territory, South Australia, Tasmania and Victoria, 2000

	ACT (n=20)	NT (n=5*)	SA (n=88)	Tasmania (n=27)	Victoria (n=77)
Median age in years (range)					
Males	24 (22–39)	32 (28–45)	26 (18–45)	26 (18–48)	23 (15–42)
Females	28 (17–28)	37 (27–47)	24 (15–50)	28 (17–36)	22 (14–39)

* Since enhanced surveillance commenced in July 2000.

In total there were 441 incident cases of hepatitis C reported with an onset date in 2000, giving a rate of 2.9 cases per 100,000 population. The proportion of all HCV notifications that were known to be incident cases was 2.2 per cent in 2000, similar to the proportion in 1999 (2.1%), but reflecting the upward trend in this proportion since 1993. The highest rates of incident hepatitis C infection were reported from Tasmania (6.6 cases per 100,000 population), the Australian Capital Territory (6.4 cases per 100,000 population) and South Australia (5.9 cases per 100,000 population). The majority of incident hepatitis C notifications were in the 20–24 year age group (Figure 8).

Figure 8. Notification rates of incident hepatitis C infections, Australia, 2000, by age and sex



In 2000, additional data were collected on incident hepatitis C infections in the Australian Capital Territory, the Northern Territory, South Australia, Tasmania and Victoria (M. Robotin, NCHECR, personal communication). The following analyses refer only to incident hepatitis C cases reported in these jurisdictions in 2000, thus the jurisdictional totals reported below may vary from the analysis by onset date.

Demographic profile of incident hepatitis C cases

The age and sex of incident hepatitis C cases notified in 2000 are summarised in Table 10, according to the State or Territory of diagnosis.

Method of diagnosis of incident hepatitis C

The basis of diagnosis was seroconversion in 72 per cent, clinical hepatitis in 23 per cent or a combination in 4 per cent of cases.

Reason for testing or reporting source

The reason for testing or the reporting source was recorded in three jurisdictions (the Northern Territory, Tasmania and Victoria). While no direct comparison can be made, as varying reasons were investigated in each jurisdiction, drug and alcohol screening was the major reason for testing in Tasmania (accounting for 44% of cases) and Victoria (27% of cases), while the investigation of symptoms was the major reason for testing in the Northern Territory (accounting for 60% of cases).

Exposure assessment for incident hepatitis C infections

Information on exposure assessment was available from four jurisdictions (the Australian Capital Territory, the Northern Territory, Tasmania and Victoria). Injecting drug use was the most common mode of transmission, accounting for 60 per cent of cases in the Northern Territory, 70 per cent of cases in the Australian Capital Territory, 74 per cent of cases in Tasmania and 86 per cent of cases in Victoria. Less common modes of transmission (for example, via tattoos, sexual exposure, iatrogenic exposure) were documented, although multiple exposures were not always recorded in each jurisdiction. In jurisdictions where multiple exposures were recorded, the majority were associated with injecting drug use.

Hepatitis D

Hepatitis D is an unusual virus as it uses the hepatitis B surface antigen in its own replication, and therefore requires co-infection with HBV.[17] Infection can occur concurrently with HBV, or can occur as a superinfection, providing the individual is a HBV carrier.

There were 27 notifications of hepatitis D to the NNDSS in 2000 at a notification rate of 0.2 cases per 100,000 population. Of the 27 notifications, 12 were reported from Victoria, 10 from New South Wales and 5 from Queensland. The majority (85%) of notifications were from males, with the highest rate reported in 30–34 year age group (0.9 cases per 100,000 population).

Gastrointestinal disease

Introduction

Gastrointestinal and foodborne diseases are a major cause of illness in Australia. In 2000, gastrointestinal illness accounted for 21,303 notifications to the NNDSS, which was 23.7 per cent of the total notified diseases. Notifications of foodborne diseases to the NNDSS and notification rates for foodborne diseases in Australia are shown in Tables 11 and 12.

The true prevalence of gastrointestinal disease is not easy to quantify. There is a significant under-reporting in surveillance data especially of milder gastrointestinal disease. Mead et al[18] have estimated that the notified fraction of foodborne disease in the United States of American (USA) varied from 2 per cent to 50 per cent depending on the severity of the disease. In the UK, it was estimated that for every case of infectious intestinal disease notified there were on average 136 cases in the community.[19] This under-reporting varied depending on the pathogen concerned. In addition there are multiple modes of transmission for the organisms that cause gastrointestinal disease (i.e. some pathogens are also transmitted via other routes). This complicates our ability to estimate what proportion of infections are actually transmitted by food. Again, this may vary by disease. The estimated proportion of gastrointestinal disease which is attributable to food ranges from 5 per cent for hepatitis A to 99 per cent for *Listeria*.[18] Surveillance data may also be biased by different levels of reporting of gastrointestinal disease in different age groups, with children and the elderly more likely to be seen by a medical practitioner.

Differences in laboratory testing practices and surveillance methods in States and Territories may also account for the difference in observed notification rates. This is particularly true for diseases such as Shiga-like toxin producing /Verotoxin producing *E. coli* (SLTEC/VTEC), where laboratory diagnosis is difficult. States and Territories also have different reporting requirements for doctors and laboratories, which can make national comparison difficult. In 2000, all Australian States and Territories supplied data to the NNDSS on hepatitis A, haemolytic uraemic syndrome, listeriosis, salmonellosis and typhoid. Data on botulism, campylobacteriosis, hepatitis E, shigellosis, Shiga-like toxin producing /Verotoxin producing *E. coli* (SLTEC/VTEC) and yersiniosis were available from most but not all jurisdictions (Table 1). To overcome some of these difficulties, the CDNA agreed to standardise reportable conditions in each jurisdiction from 1 January 2001.

The National Enteric Pathogen Surveillance Scheme (NEPSS) maintained by the Microbiological Diagnostic Unit, Department of Microbiology and Immunology at the University of Melbourne, provide important surveillance data on bacterial enteric pathogens. NEPSS collects, analysis and disseminates data on human enteric bacterial infections diagnosed in Australia. These pathogens include *Salmonella, Shigella, E. coli, Vibrio, Yersinia, Plesiomonas, Aeromonas and Campylobacter* spp. NEPSS holds more than 140,000 records of human infections and 78,000 records of isolates from non-human sources such as food and animals. NEPSS monitors trends in the epidemiology of human enteric bacterial infections, identifies outbreaks (particularly when geographically and/or temporally dispersed), identifies potential sources of pathogens causing human disease and monitors antibiotic resistance among bacterial enteric pathogens.

Botulism

There have been no cases of foodborne botulism reported to the NNDSS since the inception of the system in 1991. There were 2 cases of infant botulism reported in 2000, one case each from Victoria and the Northern Territory, both in children aged less than one year.

Infant (or intestinal) botulism cases arise from ingestion of *Clostridium botulinum* spores, which germinate in the intestine. Spores are widespread and are found in soil and dust as well as in foods such as honey. Symptoms include acute flaccid paralysis (AFP) thus botulism is often identified and reported in the differential diagnosis of AFP, which is an important part of polio surveillance in Australia.

Table 11. Trends in notifications of foodborne disease, Australia, 1991 to 2000*

Disease	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Botulism	0	0	0	0	0	0	0	1	0	2
Campylobacteriosis	8,813	9,221	8,070	10,069	11,240	12,109	11,752	13,433	12,657	13,595
Haemolytic uraemic syndrome	–	–	–	–	–	–	–	–	23	15
Hepatitis A	2,234	2,118	1,951	1,912	1,621	2,104	3,044	2,497	1,554	812
Hepatitis E	–	–	–	–	–	–	–	–	9	10
Listeriosis	49	45	49	36	59	70	73	55	64	67
Salmonellosis	5,496	4,416	4,505	5,199	5,873	5,786	7,054	7,613	7,147	6,151
Shigellosis	913	716	691	740	731	680	795	599	547	487
SLTEC,VTEC	–	–	–	–	–	–	–	–	47	33
Typhoid	93	41	80	66	70	80	79	60	68	58
Yersiniosis	329	352	370	311	207	215	207	160	125	73

* All jurisdictions reported for all years with the following exceptions

Botulism not reported from Western Australia.

Campylobacteriosis not reported from New South Wales.

Hepatitis E not reported from Western Australia.

Listeriosis not reported from South Australia (1991) or Northern Territory (1991 to 1993).

Shigellosis not reported from New South Wales.

SLTEC/VTEC not reported from Queensland or Western Australia.

Yersiniosis not reported from New South Wales (1991 to 2000) or Australian Capital Territory (1991 to 1992).

Table 12. Trends in notification rates of foodborne disease, Australia, 1991 to 2000* (rate per 100,000 population)

Disease	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	< 0.1	0.0	< 0.1
Campylobacteriosis	77.4	80.0	69.2	85.4	94.1	100.1	95.9	108.4	100.8	107.1
Haemolytic uraemic syndrome	–	–	–	–	–	–	–	–	0.1	0.1
Hepatitis A	12.9	12.1	11.0	10.7	9.0	11.5	16.4	13.3	8.2	4.2
Hepatitis E	–	–	–	–	–	–	–	–	0.1	0.1
Listeriosis	0.3	0.3	0.3	0.2	0.3	0.4	0.4	0.3	0.3	0.3
Salmonellosis	31.8	25.2	25.5	29.1	32.5	31.6	38.1	40.7	37.7	32.1
Shigellosis	8.0	6.2	5.9	6.3	6.1	5.6	6.5	4.8	4.4	3.8
SLTEC,VTEC	–	–	–	–	–	–	–	–	0.3	0.2
Typhoid	0.5	0.2	0.5	0.4	0.4	0.4	0.4	0.3	0.4	0.3
Yersiniosis	3.0	3.1	3.2	2.6	1.7	1.8	1.7	1.3	1.0	0.6

* All jurisdictions reported for all years with the following exceptions

Botulism not reported from Western Australia.

Campylobacteriosis not reported from New South Wales.

Hepatitis E not reported from Western Australia.

Listeriosis not reported from South Australia (1991) or Northern Territory (1991 to 1993).

Shigellosis not reported from New South Wales.

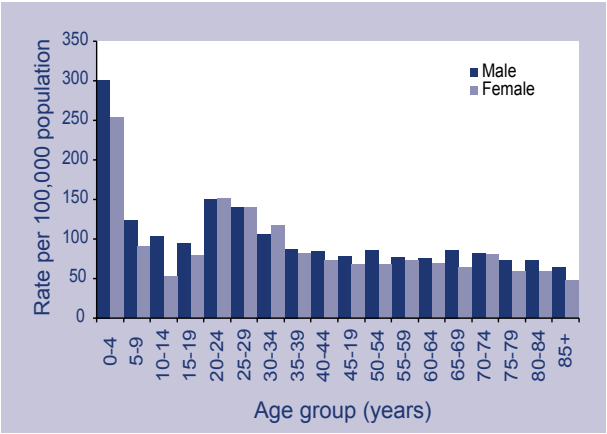
SLTEC/VTEC not reported from Queensland or Western Australia.

Yersiniosis not reported from New South Wales (1991 to 2000) or Australian Capital Territory (1991 to 1992).

Campylobacteriosis

The rate of campylobacteriosis has steadily increased in Australia since reporting to the NNDSS began in 1991. At 107.1 cases per 100,000 population in 2000, campylobacteriosis is reported more than 3 times as frequently as salmonellosis. Campylobacteriosis is now the most common cause of bacterial gastroenteritis in many industrialised countries.[20] The apparent increase in *Campylobacter* in recent decades reflects the easier laboratory identification due to the development of selective media in the 1980s and polymerase chain reaction (PCR) for *Campylobacter* in the 1990s. Researchers believe that chicken accounts for between 50 and 70 per cent of *Campylobacter* infections and it is now recognised that chicken flocks are almost universally infected. Under cooking of chicken or contamination of other foods with juices from uncooked chicken may be the major routes of infection. Consumption of other kinds of foods and contact with animals are also recognised transmission routes. A joint Food and Agriculture Organization of the United Nations (FAO) and WHO expert consultation on risk assessment of microbiological hazards in foods is currently assessing hazard identification and characterisation and exposure to *Campylobacter* spp. from broiler chickens.[21] *Campylobacter* infections cause an acute self-limiting gastroenteritis, although a significant proportion of infections may be asymptomatic. *C. jejuni* infection appears to be an important risk factor in the development of Guillain-Barré syndrome (GBS). The risk of developing GBS is 100-fold higher following a symptomatic episode of *C. jejuni* infection.[22]

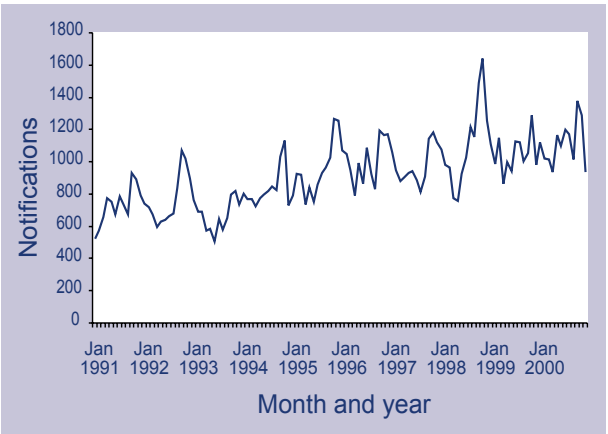
Figure 9. Notification rates of campylobacteriosis, Australia, 2000, by age and sex



There were 13,595 cases of campylobacteriosis notified to the NNDSS with symptom onset in 2000, which was an increase of 7.4 per cent from the 12,657 cases notified in 1999. Reports were received from every jurisdiction except New South Wales where cases are included in the categories 'foodborne disease' or 'gastroenteritis in an institution'. *Campylobacter* species are the most common cause of gastrointestinal disease notified to the NNDSS. Despite this there are very few outbreaks detected due to the lack of a robust typing method. During 2000, Tasmania reported a small outbreak affecting 3 students in a student residential setting, but no food source was identified.[23] Another cluster of cases in South Australia identified an association between consumption of raw milk and *Campylobacter* infection.[24] Overall, the highest age-specific rate of campylobacteriosis was 281 cases per 100,000 population in 0–4 year-old children (Figure 9). The male to female ratio was 1.2:1.

The highest notification rate was in South Australia (125.7 cases per 100,000 population) and the lowest rate was in the Northern Territory (93.1 cases per 100,000 population). Reports of campylobacteriosis were more frequent in Spring and Summer (Figure 10).

Figure 10. Trends in notifications of campylobacteriosis, Australia, 1991 to 2000, by month of onset



Hepatitis A

Overall hepatitis A in Australia has declined significantly over the past 30 years although levels in Indigenous communities have remained high. National notifications of hepatitis A have declined considerably since the peak rate recorded in the NNDSS in 1997 (16.4 cases per 100,000 population). The impact of control measures, including vaccination of susceptible populations and improvements in hygiene, have clearly had an impact on the incidence of hepatitis A in Australia (Tables 11 and 12).

In Australia, three patterns of hepatitis A epidemiology are recognised.[25] Firstly, there are large, slowly evolving community outbreaks, occurring at intervals of 5 years. Community outbreaks affect groups of people prone to infection, who are susceptible to intense levels of transmission within their groups. Infected individuals are also a potential source for infection for the wider community. Settings for community outbreaks include child care centres and pre-schools, communities of men who have sex with men, schools and residential facilities for the intellectually disabled and communities of injecting drug users. Secondly, sporadic cases of hepatitis A may arise in people without obvious risk factors although some may be associated with overseas travel or travel to Indigenous communities. Thirdly, point-source outbreaks of hepatitis A may arise from contaminated food or water or an infected food-handler. These are relatively rare in Australia. The last major point-source outbreak of hepatitis A arose from contaminated oysters in New South Wales in 1997.[26]

Vaccination against hepatitis A in Australia is recommended for travellers to endemic areas, visitors to remote Indigenous communities, childcare and pre-school personnel, the intellectually disabled and their carers, healthcare workers, sewerage workers, men who have sex with men, injecting drug users, patients with chronic liver disease (or with hepatitis C), haemophiliacs who may have received pooled plasma concentrates, and food handlers.[25]

There were 812 cases of hepatitis A notified to the NNDSS with symptom onset in 2000, which was a decrease of 48 per cent from the 1,554 cases notified in 1999 (Figure 11). The highest age-specific rate was in the 25–29 year age group (8.4 cases per 100,000 population) (Figure 12) and the male to female ratio was 1.5:1.

Figure 11. Trends in notification rates of hepatitis A, Australia, 1994 to 2000, by year of onset

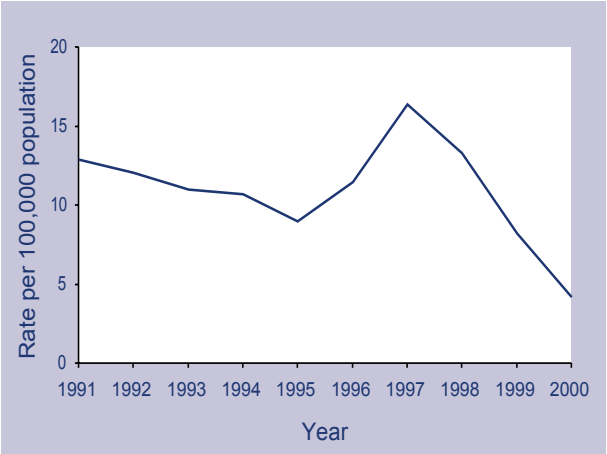
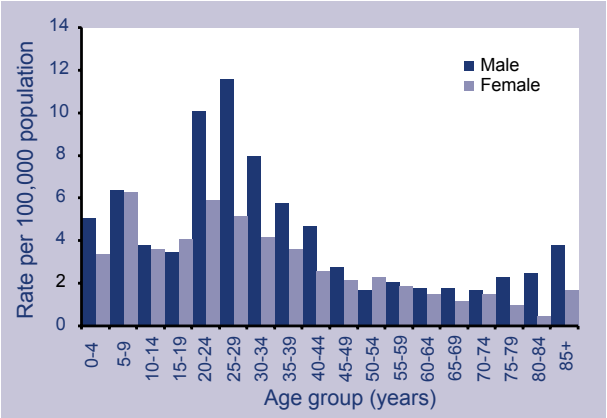


Figure 12. Notification rates of hepatitis A, Australia, 2000, by age and sex



The highest notification rate was in the Northern Territory (22.5 cases per 100,000 population) and the lowest rate was in Tasmania (0.6 cases per 100,000 population). There has been a marked decline in notifications of hepatitis A throughout north Queensland since hepatitis A vaccination was introduced for Indigenous children in the region in early 1999 (J. Hanna, personal communication).

Hepatitis E

Hepatitis E is endemic in many countries of Asia but is rarely reported in Australia. Women in the third trimester of pregnancy are susceptible to fulminant hepatitis E disease, with a case fatality rate as high as 20 per cent.[27] Outbreaks in South Asia pose a risk to Australian travellers to these regions. There were 10 cases of hepatitis E notified to the NNDSS in 2000, 9 from New South Wales and one from Tasmania. Of the 10 cases, one was associated with travel to India. There was no travel history available for the remaining cases.

Listeriosis

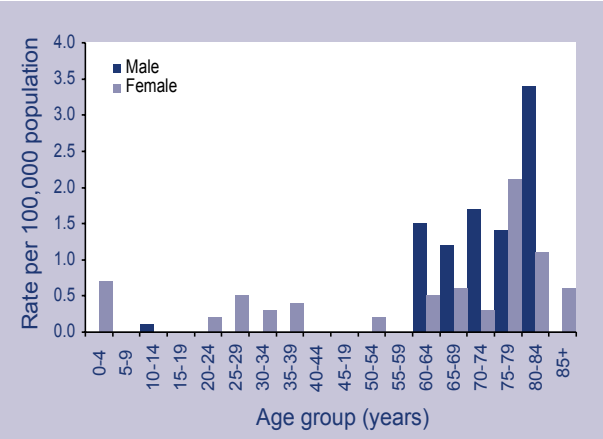
Listeriosis is a serious but relatively rare foodborne disease to which neonates, pregnant women, the immuno-compromised and the elderly are particularly susceptible. In pregnant women the infection can be passed to the foetus. Infants may be stillborn, born with septicaemia or develop meningitis in the neonatal period. Clusters of cases of listeriosis have been noted in hospitals, nurseries and aged care facilities.[27]

The notification rate of listeriosis in Australia has remained steady over the past 10 years (Tables 11 and 12). As food preparation practices change a variety of products have been found to be vehicles for *Listeria* spp. The Australian Quarantine Inspection Service (AQIS) and the Australia New Zealand Food Authority are responsible for the laboratory testing of food imported into Australia. Between 1995 and 1998, *Listeria* contamination of foods such as smoked fish and soft cheeses, constituted the most frequent findings.[28]

Listeriosis was notifiable in all Australian jurisdictions in 2000; however practices varied as to whether a materno-fetal case constituted one or two cases. There were 67 cases of listeriosis reported to the NNDSS in 2000, which was a similar number to previous years (Table 11). The highest age-specific rate of listeriosis was 2.0 cases per 100,000 population in the 80–84 year age group (Figure 13) and the male to female ratio was 0.7:1.

The highest notification rate was in the Northern Territory (1.5 cases per 100,000 population) There were no cases reported from the Australian Capital Territory. There was no clustering of cases of listeriosis. Five materno-foetal pairs were reported which resulted in three foetal deaths.

Figure 13. Notification rates of listeriosis, Australia, 2000, by age and sex

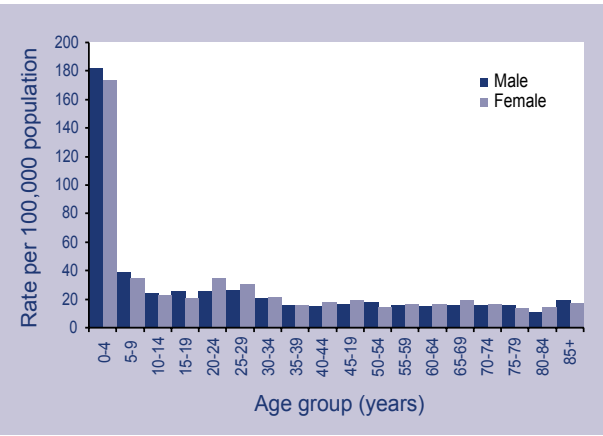


Salmonellosis (excluding typhoid)

Salmonellosis remains the second most common cause of gastroenteritis in Australia and the most important cause of bacterial foodborne disease outbreaks. In 2000, rates of *Salmonella* notifications fell for the second year running, to 32.1 cases per 100,000 population.

There were 6,151 cases of salmonellosis reported to the NNDSS with symptom onset in 2000, which was a decrease of 13.9 per cent from the 7,147 cases reported in 1999 (Table 11). The highest age-specific rate was 179.2 cases per 100,000 population in 0–4 year-old children (Figure 14) and the male to female ratio was 1:1. The highest notification rate was in the Northern Territory (155.5 cases per 100,000 population) and the lowest rate was reported from Victoria (21.4 cases per 100,000 population).

Figure 14. Notification rates of salmonellosis, Australia, 2000, by age and sex



The Kimberley Statistical Division in Western Australia had the highest rate of salmonellosis, in excess of 399 cases per 100,000 population, which was comparable to previous years (Map 2). Reports of salmonellosis were greatest in the months of January to March (Figure 15).

Map 2. Notification rates of salmonellosis, Australia, 2000, by Statistical Division of residence

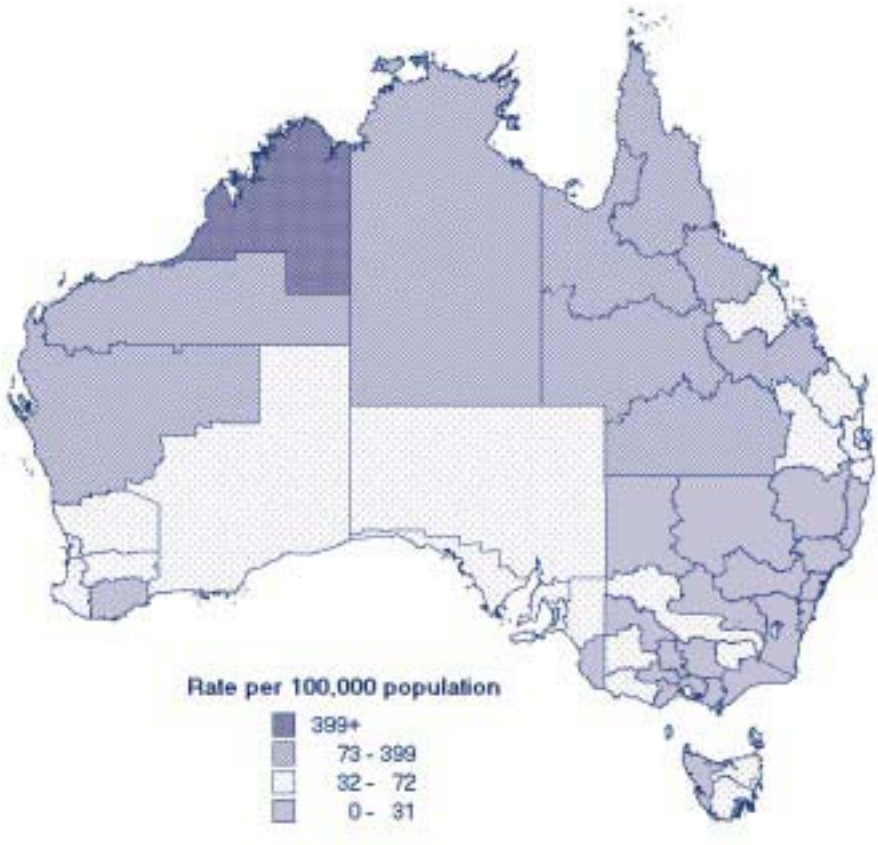
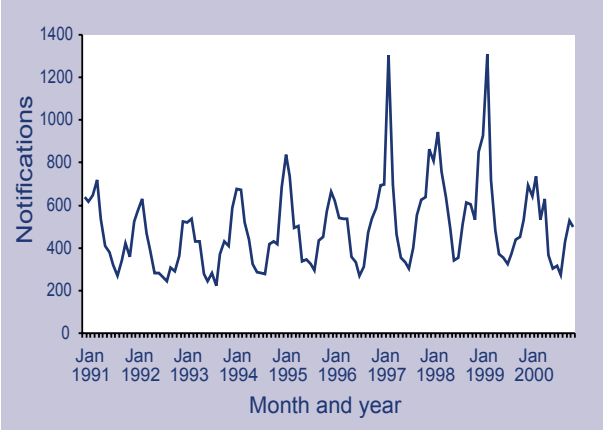


Figure 15. Trends in notifications of salmonellosis, Australia, 1991 to 2000, by month of onset



The NEPSS reported 6,121 cases of *Salmonella* in 2000.[29] The top 10 *Salmonella* infections reported to NEPSS are shown in Table 13.

NEPSS recorded 19 outbreaks of *Salmonella* in Australia in 2000. One outbreak, which was S. Paratyphi B bv Java RDNC/AUS2 associated with fish tanks, was Australia-wide while all others were confined to a single jurisdiction. Six outbreaks were recorded in South Australia, three each in New South Wales and Western Australia, two each in Queensland and Victoria and one each in the Australian Capital Territory and the Northern Territory. No *Salmonella* outbreaks were reported from Tasmania. The largest outbreak was of *Salmonella* Typhimurium PT135 in Western Australia. This outbreak continued through the year with peaks of reports in February, April and May. All age groups were affected but no food vehicle or common source was identified.

South Australia reported an outbreak of *Salmonella* Typhimurium PT44 in October 2000.[30] Ten cases were associated with eating at an Adelaide restaurant and although an investigation found lapses in hygienic practices, no food source for this outbreak was identified.

An outbreak of *Salmonella* Mgulani involving 42 laboratory-confirmed cases in December 1999 to January 2000 occurred in New South Wales.[31] No environmental or food source was identified and DNA 'fingerprinting' suggested the strains had been circulating in Australia for some years.

Salmonella Enteriditis was the most common salmonella infection among travellers returning from overseas in 2000. Of 142 cases, there were 85 cases of S. Enteriditis phage type 4.

Table 13. Top 10 isolates of *Salmonella*, Australia, 2000 (data from the National Enteric Pathogen Surveillance Scheme)*

Isolate	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust	% of total
S. Typhimurium PT 135†	10	148	2	143	6	6	69	221	**605**	**9.9**
S. Typhimurium PT 9†	32	187	3	57	24	22	178	47	**550**	**9.0**
S. Virchow†	8	56	4	256	16	2	110	6	**458**	**7.5**
S. Saintpaul†	6	39	20	186	26	2	15	47	**341**	**5.6**
S. Enteriditis	5	55	5	72	14	9	32	56	**248**	**4.0**
S. Typhimurium PT 64	1	101	1	19	16	1	77	10	**226**	**3.7**
S. Birkenhead†	1	77	0	100	4	1	13	0	**196**	**3.2**
S. Muenchen†	2	21	10	40	11	0	7	29	**120**	**2.0**
S. Chester	0	13	17	38	18	0	5	17	**108**	**1.8**
S. Bovismorbificans	0	45	2	13	7	1	27	10	**105**	**1.7**
Others	0	0	0	0	0	0	0	0	**3,164**	**51.6**
Total	**65**	**742**	**64**	**924**	**142**	**44**	**533**	**443**	**2,957**	**48.3**

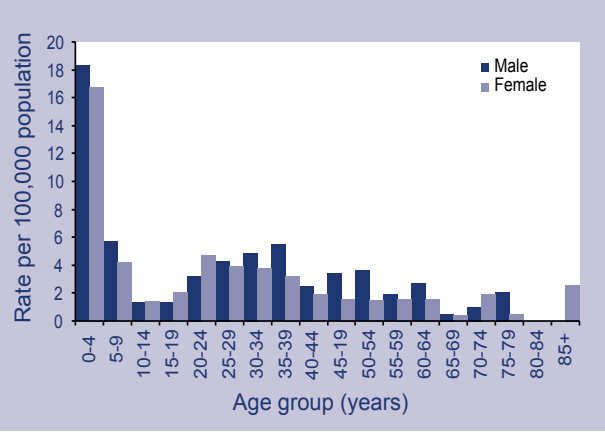
* Adapted from NEPSS annual report, 2000

† Associated with an identified outbreak

Shigellosis

In 2000, the NNDSS notification rate of shigellosis fell for the third year running, and was the lowest rate recorded since the surveillance system began. There were 487 cases of shigellosis reported to the NNDSS with onset of symptoms in 2000, which was an 11 per cent decrease from 547 cases reported in 1999. The highest age-specific rate was 18 cases per 100,000 population in 0–4 year-old children (Figure 16) and the male to female ratio was 1.2:1.

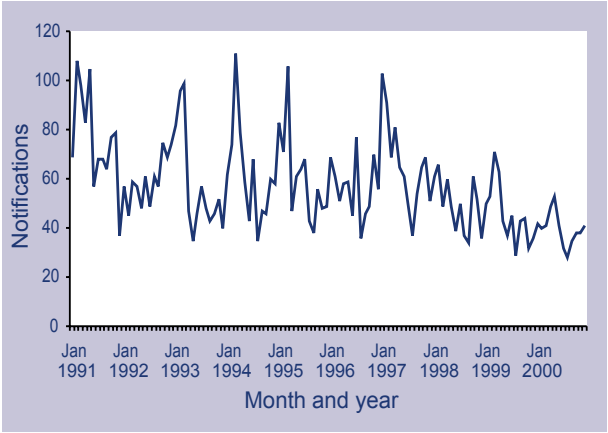
Figure 16. Notification rates of shigellosis, Australia, 2000, by age and sex



Reports were received from every jurisdiction except New South Wales where cases are included in the categories 'foodborne disease' or 'gastroenteritis in an institution'. The highest notification rate was in the Northern Territory (58.3 cases per 100,000 population) and the lowest rate was reported from Tasmania (0.4 cases per 100,000 population). Cases were more commonly notified during the months of January to April (Figure 17).

Reports of *Shigella* to the NEPSS identified 147 cases of *S. sonnei* biotype g among gay men in inner city Sydney. This outbreak was associated with casual sex at sex-on-premises-venues.[32] (O'Sullivan et al Communicable Diseases Control Conference 2001, Abstract No.31)

Figure 17. Trends in notifications of shigellosis, Australia, 1991 to 2000, by month of onset



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

There were 33 cases of STEC/VTEC reported to the NNDSS with symptom onset in 2000, which was a 24 per cent decrease from 43 cases reported in 1999. SLTEC/VTEC was a notifiable disease in 2000 in all jurisdictions except Queensland and Western Australia. In 2000, all of the 33 cases of SLTEC/VTEC were reported from South Australia. This reflects the practice in South Australia of screening faecal specimens from all cases of bloody diarrhoea for toxin genes, by PCR.

The highest age-specific rate was 0.9 cases per 100,000 population in the 80–84 year age group and the male to female ratio was 1.8:1.

Haemolytic uraemic syndrome

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

There were 15 cases of HUS notified to the NNDSS with symptom onset in 2000 (Table 11). There was no evidence of clustering among HUS cases.

The highest age-specific rate was 0.6 cases per 100,000 population in 0–4 year-old children and the male to female ratio was 1.1:1.

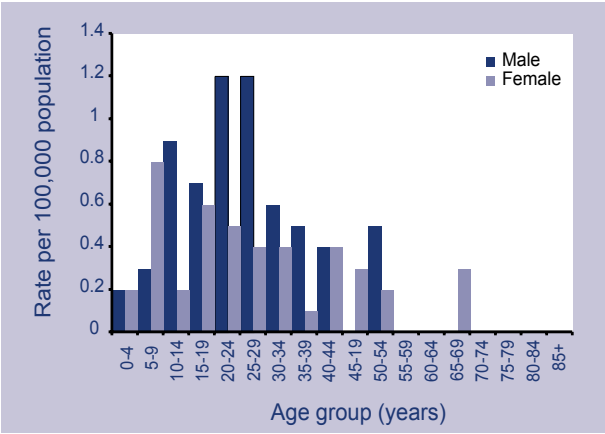
Typhoid

Typhoid notifications in Australia are strongly associated with overseas travel. Since the majority of cases are imported, the education of overseas travellers, especially young people travelling in Asia, is the most important public health action to control typhoid in Australia.

There were 58 cases of typhoid reported to the NNDSS with symptom onset in 2000, a reduction of nearly 15 per cent compared with the 68 cases reported in 1999. Of the 56 isolations of S. Typhi by NEPSS in 2000, all but 2 cases had a history of travel (mostly in Asia) prior to onset.

The highest age-specific rate of typhoid was 0.7 cases per 100,000 population in the 20–29 year age group (Figure 18) and the male to female ratio was 1.5:1. The highest notification rate was in Western Australia (0.6 cases per 100,000 population).

Figure 18. Notification rates of typhoid, Australia, 2000, by age and sex



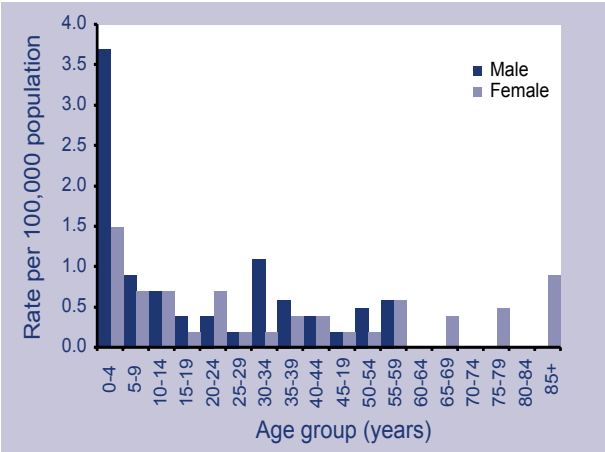
Yersiniosis

The notification rate for yersiniosis has fallen over the 10-year period from 1991 to 2000. These declines are a worldwide phenomenon and may relate to changes in laboratory testing of faeces and improvements in animal slaughtering practice (M. Barton, personal communication). The steady decline in the incidence of this disease and a lack of outbreaks lead the CDNA to remove yersiniosis from the national notifiable diseases list in January 2001.

Yersinia enterocolitica, the causative organism of yersiniosis, causes both sporadic cases and disease outbreaks, with pork a frequently incriminated food.[27] Person-to-person transmission has been documented in outbreak settings and direct transmission from dogs to humans has been postulated.

There were 73 cases of yersiniosis reported to the NNDSS with dates of symptom onset in 2000, which was a 42 per cent decrease from the 125 cases reported in 1999. The highest age-specific rate was 2.6 cases per 100,000 population in 0–4 year old children (Figure 19) and the male to female ratio was 1.5:1. Reports were received from every jurisdiction except New South Wales where cases are included in the categories 'foodborne disease' or 'gastroenteritis in an institution'. The highest notification rates were in Queensland (1.7 cases per 100,000 population) and the Northern Territory (1.0 cases per 100,000 population).

Figure 19. Notification rates of yersiniosis, Australia, 2000, by age and sex



Policy initiatives in foodborne disease surveillance and control in 2000

A Food Policy Unit was formed within the Commonwealth Department of Health and Ageing in 2000. The Unit aims to coordinate policy development with a focus on food safety; strengthening the evidence base for decision making; fostering collaboration between government, consumers and the food industry; and promoting nationally consistent policy regulation and action.[34]

In the latter part of 2000, the Commonwealth Department of Health and Ageing established and funded a collaborative network called 'OzFoodNet' to enhance surveillance mechanisms for foodborne disease across Australia. The aims of OzFoodNet are to:

- estimate the incidence of foodborne disease in Australia;
- learn more about the causes and determinants of foodborne disease;
- identify risky practices associated with food preparation and handling;
- train foodborne disease epidemiologists.

The work of OzFoodNet will improve surveillance of foodborne disease across Australia. Collaborators of OzFoodNet include State and Territory health authorities, the National Centre for Epidemiology and Population Health, the Public Health Laboratory Network and national government agencies.[35]

Quarantinable diseases

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

The only case of quarantinable disease reported in Australia in 2000 was a case of cholera (*V. cholerae 0139*). The disease was acquired in Bali, Indonesia. The continued reporting of cholera in travellers returning from foreign countries demonstrates the importance of travellers consuming safe food and drink in areas where cholera is known to occur, including many Asian and South Pacific countries.

Although no cases of rabies or yellow fever were reported in Australia, worldwide these two diseases continue to cause fatalities and travellers should be aware of measures they can take to prevent infection with these viruses. Travellers intending to visit central Africa or central South America are encouraged to receive the yellow fever vaccine from an approved Australian vaccination centre. Information on quarantinable diseases can be found on the DoHA Website at: http://www.health.gov.au/pubhlth/strateg/quaranti/index.htm.

Sexually transmitted infections

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

In 2000, STI reports accounted for 24,319 notifications to the NNDSS, which was 27 per cent of all notifications.

STIs reported to the NNDSS in 2000 included chancroid, chlamydial infection, donovanosis, gonococcal infection, lymphogranuloma venereum and syphilis. Laboratory diagnoses of chlamydia and syphilis were also reported via LabVISE. Other STIs not subject to national surveillance through the NNDSS or via LabVISE include genital herpes (herpes simplex virus type I and II), genital warts (human papilloma virus, several types), trichomoniasis and parasitic infestations such as pubic lice and scabies.

The trends in the number and rates of STI notifications reported to the NNDSS between 1991 and 2000 are shown in Tables 14 and 15. Notification rates for chancroid, lymphogranuloma venereum and syphilis remained relatively stable over the decade. The number of donovanosis notifications decreased over time, while increased numbers of chlamydia and gonococcal infections were reported. Some of the increases may be due to higher levels of infections. Changes in surveillance methods and laboratory tests (particularly the use of nucleic acid testing) may also account for some of the observed increases.

Table 14. Trends in notifications of sexually transmitted infections, Australia, 1991 to 2000*

Disease	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Chancroid	1	4	0	0	2	1	1	1	0	0
Chlamydial infection	–	–	–	6,153	6,407	8,366	9,239	10,927	14,045	16,866
Donovanosis	72	80	71	121	87	51	49	31	17	12
Gonococcal infection	2,705	2,889	2,811	2,968	3,308	4,144	4,684	5,469	5,644	5,686
Lymphogranuloma venereum	0	3	1	2	1	0	0	0	0	0
Syphilis	1,974	2,683	2,260	2,275	1,735	1,449	1,296	1,683	1,844	1,755

* All jurisdictions reported for all years with the following exceptions:
Chlamydial infection not reported from New South Wales (1994 to 1998).
Donovanosis not reported from New South Wales or South Australia (all years) or Tasmania (1991 to 1992).
Lymphogranuloma venereum not reported from Western Australia.

Table 15. Trends in notification rates of sexually transmitted infections, Australia, 1991 to 2000* (rate per 100,000 population)

Disease	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Chancroid	< 0.1	< 0.1	0.0	0.0	< 0.1	< 0.1	< 0.1	< 0.1	0.0	0.0
Chlamydial infection	–	–	–	52.2	53.7	69.1	75.4	88.2	74.1	88.0
Donovanosis	0.8	0.8	0.7	1.2	0.8	0.5	0.5	0.3	0.2	0.1
Gonococcal infection	15.7	16.5	15.9	16.6	18.3	22.6	25.3	29.2	29.8	29.7
Lymphogranuloma venereum	0.0	< 0.1	< 0.1	< 0.1	< 0.1	0.0	0.0	0.0	0.0	0.0
Syphilis	11.4	15.3	12.8	12.7	9.6	7.9	7.0	9.0	9.7	9.2

* All jurisdictions reported for all years with the following exceptions:
Chlamydial infection not reported from New South Wales (1994 to 1998).
Donovanosis not reported from New South Wales or South Australia (all years) or Tasmania (1991 to 1992).
Lymphogranuloma venereum not reported from Western Australia.

Chancroid

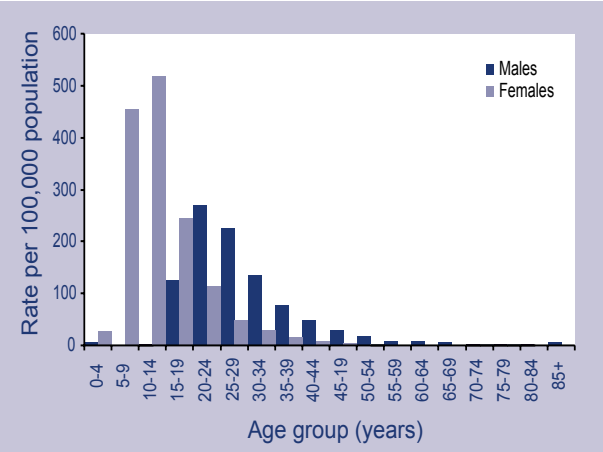
Cases of chancroid (a bacterial infection causing genital ulcers) have rarely been reported to the NNDSS since 1991. No cases of chancroid were reported in Australia in 2000, and in 2001 this disease was removed from the list of nationally notifiable diseases.

Chlamydial infection

Chlamydial infections were the most commonly reported STI and the second most commonly reported notifiable disease in Australia in 2000. In this year 16,866 notifications of chlamydial infection were reported, an increase on the 14,045 cases reported in 1999 (Table 14). There were 81 cases reported in children aged less than 10 years. Notifications reported in young children may be cases of chlamydial conjunctivitis. In 2000, Queensland had a campaign of screening for chlamydial infections, including PCR testing of samples from young women and Indigenous people.

The notification rate for chlamydial infections in 2000 was 88 cases per 100,000 population, while in 1999 the rate was 74.1 cases per 100,000 population. This reflects an upward trend in the number of syphilis notifications reported to the NNDSS since 1997. In 2000, the male to female ratio was 0.7:1. In both males and females the highest rate of disease was recorded for the 20–24 year age group (Figure 20). High rates of notification were reported from northern Australia (including the Northern Territory, Western Australia and Queensland), with rates over 490 cases per 100,000 population in the Northern Territory in 2000 (Map 3).

Figure 20. Notification rates of chlamydia, Australia, 2000, by age and sex



Some important surveillance issues must be taken into account when analysing the trends in chlamydia notification rates. Firstly, in New South Wales, reporting of genital chlamydial infection commenced in September 1998, so that the reporting of this infection was national for the first time in 1999. Secondly, chlamydial infections may be under-reported because of the high proportion of asymptomatic infections, particularly among women.[27] The introduction of screening programs can have a marked effect on notification rates over time. Thirdly, the use of nucleic acid tests for chlamydia may also explain increases in notifications.

Based on NNDSS data, the NCHECR reported rates of chlamydial disease in Indigenous Australians.[36] Using data from the Northern Territory, South Australia and Western Australia (the only jurisdictions to report Indigenous status in more than half of notifications), the estimated crude rate of chlamydial infection among Indigenous Australians in 2000 was 1,207 cases per 100,000 population compared with a rate of 95 cases per 100,000 population in non-Indigenous Australians. For these jurisdictions, 831 of notifications did not have Indigenous status reported. Trends in notification rates of chlamydia in Indigenous and non-Indigenous Australians between 1993 and 2000 are shown in Figure 21.

Map 3. Notification rates of chlamydial infection, Australia, 2000, by Statistical Division of residence

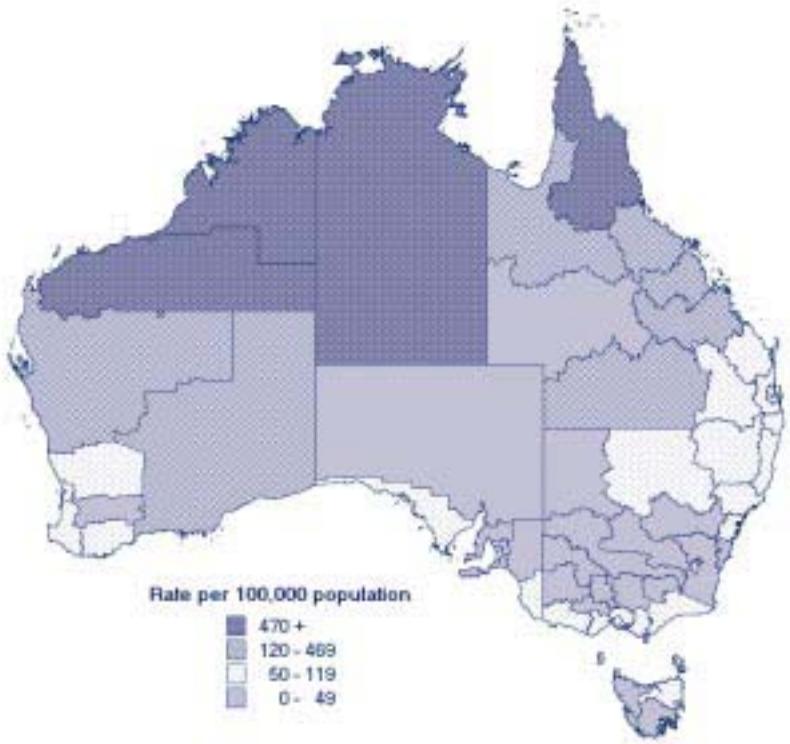
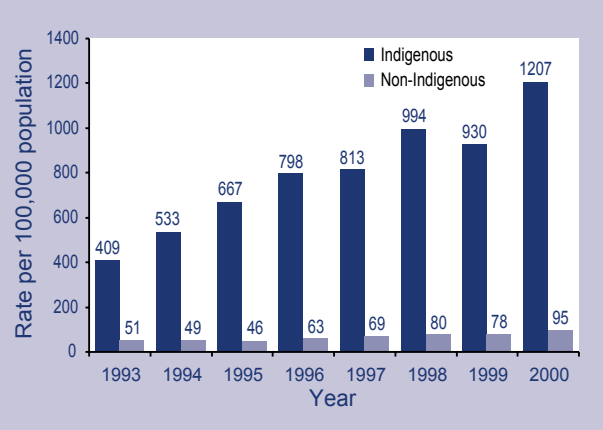


Figure 21. Trends in notification rates of chlamydia, the Northern Territory, South Australia and Western Australia, 1993 to 2000, by Indigenous status



Lymphogranuloma venereum

Lymphogranuloma venereum is a sexually acquired chlamydial infection caused by certain serovars of *Chlamydia trachomatis*. There were no cases of lymphogranuloma venereum reported from any State or Territory in 2000. In Australia, there have only been 7 reports of lymphogranuloma venereum to the NNDSS since 1991 and none since 1995. In 2001 lymphogranuloma venereum was removed from the list of nationally notifiable diseases in Australia.

Donovanosis

Donovanosis is a relatively uncommon STI, characterised by genital ulceration which may develop into a chronic ulcerative disease if untreated. Lesions may be extensive and extra-genital in some cases, and may be associated with secondary bacterial infection. Donovanosis is generally found in tropical countries, and in Australia occurs mostly in Indigenous people in rural and remote communities. The causative organism, formerly known as *Calymmatobacterium granulomatis*, has been redesignated *Klebsiella granulomatis.*

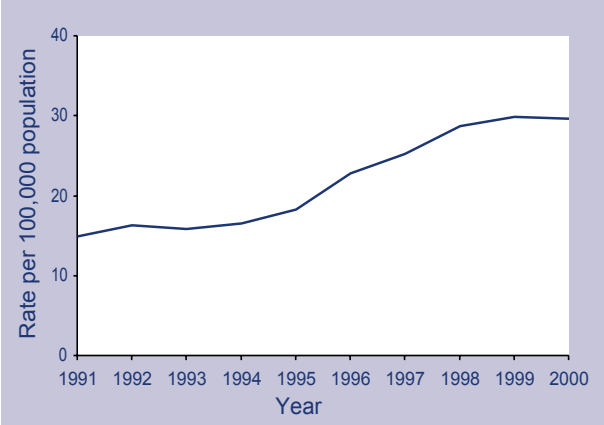
Donovanosis is a notifiable disease in all jurisdictions except New South Wales and South Australia. Notifications of donovanosis have fallen over the past 10 years, and particularly since 1994. Eradication of donovanosis was proposed as part of the 1997 National Indigenous Australians' Sexual Health Strategy, and since then significant advances have been made in the control of this disease in Indigenous Australians.[37] The decreases in notifications are due to earlier diagnosis and treatment (including the introduction of more sensitive and acceptable testing methods and more effective treatment with azithromycin), better education strategies, with a partnership approach encompassing Aboriginal and Torres Strait Islander people.

A total of 12 notifications of donovanosis were received in 2000, including five from the Northern Territory, six from Queensland and one from Western Australia. This represents a decrease from 1999, when 17 notifications were received nationally. In 2000, the highest rate of notifications was in the 25–34 year age range. The male to female ratio was 1:1, a change from 1999, when the male to female ratio was 1:7.

Gonococcal infection

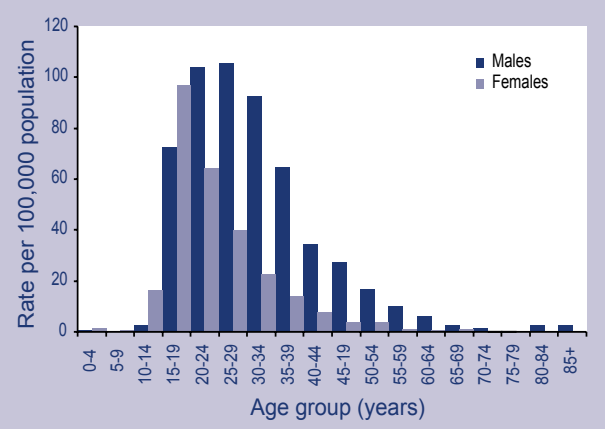
The number of notifications of gonococcal infection in Australia has increased over the past decade. In 2000, a total of 5,686 notifications of gonococcal infection were received nationally (Table 14), similar to the 5,644 received in 1999. The notification rate of gonococcal infection has increased steadily from around 16 cases per 100,000 population in 1993 to around 30 cases per 100,000 population in 1998 to 2000 (Figure 22). This rate remains far below the very high rates recorded in the 1970s and early 1980s, which peaked at 84.4 cases per 100,000 population in 1982.[38] In 2000, Queensland and the Northern Territory had screening programs for gonococcal infection in Indigenous communities.

Figure 22. Trends in notification rates of gonococcal infections, Australia, 1991 to 2000



In 2000, the male to female ratio for gonococcal notifications was 2:1, similar to the ratio in previous years (in 1999 the ratio was 2.2:1). Peak notification rates for females (97 cases per 100,000 population) occurred in the 15–19 year age group. For males the corresponding group was the 25–29 year age group, where the notification rate was 105 cases per 100,000 population (Figure 23). There was a wide geographical variation in the rate of notification of gonococcal infection (Map 4). The highest rates of notification were from the Northern Territory (577 cases per 100,000 population) and from northern Statistical Divisions in Western Australia (Map 4).

Figure 23. Notification rates of gonococcal infection, Australia, 2000, by age and sex

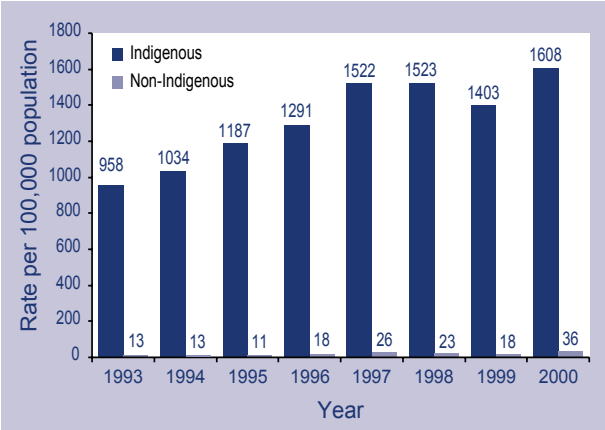


Division of residence

The increase in the number of gonococcal notifications is due in part to an ongoing outbreak of gonorrhoea among men who have sex with men.[39] The proportion of male cases of gonococcal infection associated with homosexual contact was reported in four jurisdictions and varied from 28 per cent in metropolitan Western Australia to 75 per cent in Tasmania. Increased acceptance of non-invasive sample collection for nucleic acid testing may also increase testing rates and encourage opportunistic screening, leading to increased diagnoses. Increased testing may also result from the introduction of sexual health programs and other health promotion activities.

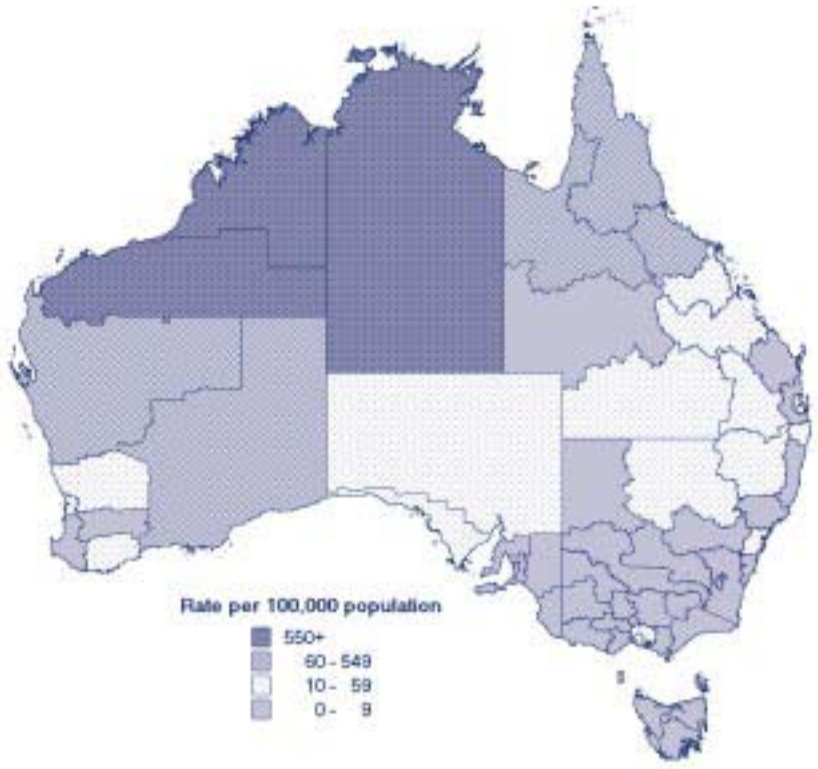
The NCHECR reported rates of gonococcal disease in Indigenous Australians, based on the NNDSS data.[36] These analyses are based on data from the Northern Territory, South Australia and Western Australia, which were the only jurisdictions to report Indigenous status in more than half of notifications. From these three jurisdictions, 11 per cent of notifications did not have Indigenous status recorded. It is estimated that in 2000 the rate of gonococcal infections among Indigenous Australians was 1,608 per 100,000 population, compared with a rate of 36 per 100,000 population in non-Indigenous Australians, largely explaining the geographic variation in notifications of gonococcal infection. This represents an increase in gonococcal notification rates since 1993 for Indigenous Australians (Figure 24). Small increases were also observed in non-Indigenous Australians.

Figure 24. Trends in notification rates of gonococcal infections, the Northern Territory, South Australia and Western Australia, 1993 to 2000, by Indigenous status



The Australian Gonococcal Surveillance Programme is the national laboratory-based surveillance system that documents the antibiotic sensitivity of gonococcal isolates. The program is undertaken by a network of reference laboratories in each state and territory, using agreed standard methodology to quantitatively determine the susceptibility of gonococci to a core group of antibiotics. Surveillance of antibiotic resistance in gonococci is important, as resistance rates can be quite volatile, and it is recommended that a particular treatment regime be discontinued once 5 per cent of isolates are resistant to that agent.

Map 4. Notification rates of gonococcal infections, Australia, 2000, by Statistical Division of residence



A survey of the antibiotic susceptibility of *Neisseria gonorrhoeae* by the AGSP in 2000, has been published.[40] The proportion of *Neisseria gonorrhoea* isolates with antibiotic resistance in the WHO Western Pacific Region for 2000 have been compared.[41] Table 16 shows the trends in antibiotic resistance in Australia between 1998 and 2000. As in previous years, antibiotic susceptibility patterns in 2000 varied significantly between regions. Generally, rates of resistance to penicillin and quinolone groups of antibiotics were higher in urban than in rural areas. Quinolone resistance became more widespread in 2000, with increases in Queensland, Western Australia and South Australia. A high rate of quinolone resistant gonococci isolated from homosexually active men was observed in 1999 in New South Wales and Victoria. High rates were again seen in 2000 in Victoria, but not in New South Wales.

Syphilis

In 2000, all jurisdictions reported syphilis (including primary, secondary and latent syphilis) and congenital syphilis to the NNDSS. A total of 1,755 notifications of syphilis were received in 2000 (Tables 14 and 15) with a rate of 9.2 cases per 100,000 population, consistent the rate in 1999 (1,844 notifications, a rate of 9.7 cases per 100,000 population). The peak notification rate occurred in 1992. Rates have since decreased and been relatively stable since 1998 (Table 15).

In 2000, there was wide geographical variation in the notification rate for syphilis (Table 14, Map 5). The highest rate was described in the Northern Territory (89.5 cases per 100,000 population). The male to female ratio for syphilis notifications was 1.2:1. Notification rates were higher among females in the 25–29 year age group (21.8 cases per 100,000 population). In comparison, the corresponding peak age group for males was the 50–54 year age group, where the rate was 17.4 cases per 100,000 population, although the reporting rates in all age groups for adult males is generally quite similar (Figure 25).

Table 16. Proportion of gonococcal isolates showing antibiotic resistance, Australia, 1998 to 2000

Year	Penicillin (% resistant)		Quinolone resistance (% resistant)	High level tetracycline resistance (% resistant)
	Plasmid mediated resistance	Chromosomally mediated resistance		
1998	5.3	21.8	5.2	NR
1999	7.4	14.3	17.2	7.9
2000	8.7	10.6	17.8	9.1

Map 5. Notification rates of syphilis, Australia, 2000, by Statistical Division of residence

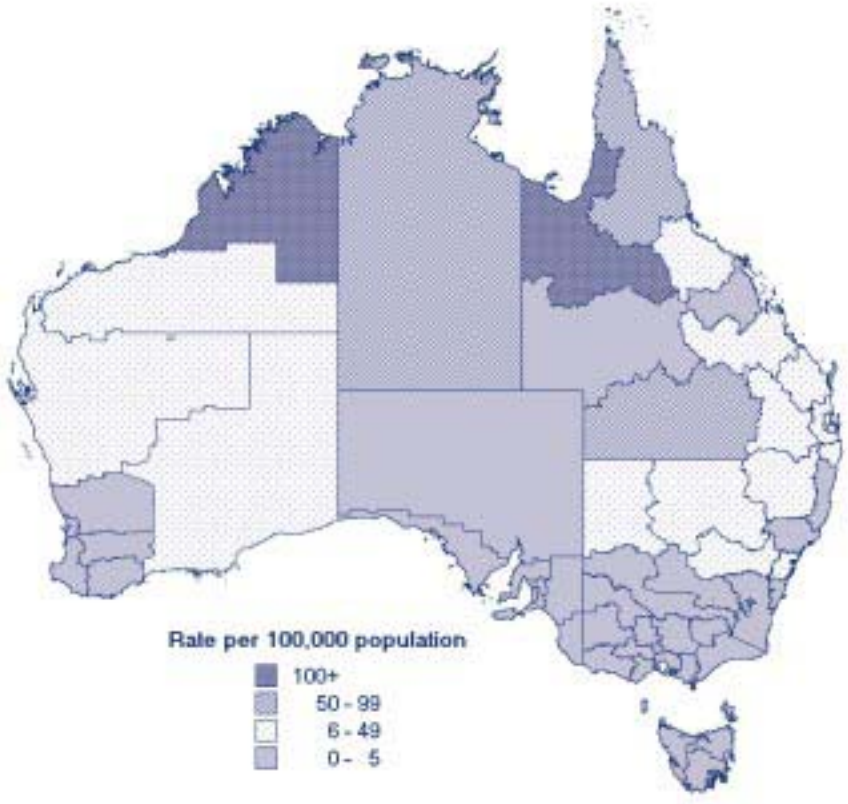
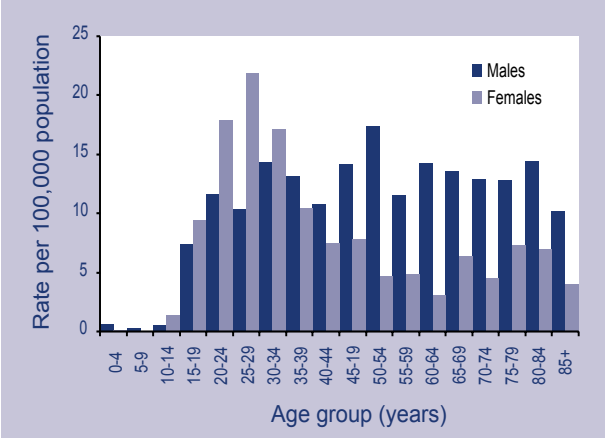


Figure 25. Notification rates of syphilis, Australia, 2000, by age and sex

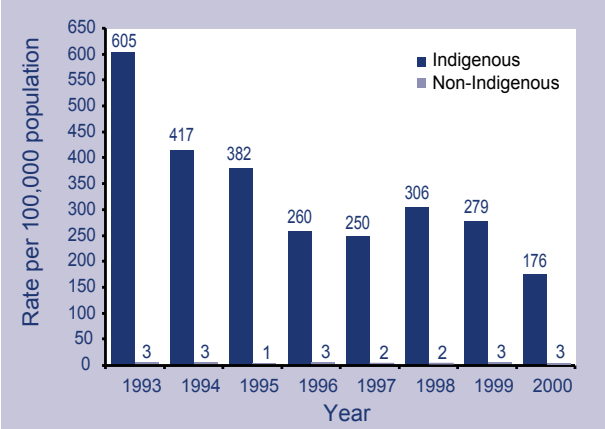


In 2000, there were 5 cases of syphilis reported in the 0–4 year age group, and 2 cases in 5–9 year age group. Of these, 2 cases (one each in New South Wales and Queensland) were confirmed as congenital syphilis.

The NCHECR has reported rates of syphilis in Indigenous Australians based on NNDSS data.[36] These estimates are based on data from the Northern Territory, South Australia and Western Australia, which were the only jurisdictions to report Indigenous status in more than half of notifications. Of the reports from these jurisdictions, only 4 per cent did not have Indigenous status identified. The estimated rate of syphilis among Indigenous Australians in 2000 was 176 per 100,000 population compared with a rate of 2.8 per 100,000 population in non-Indigenous Australians.

Trends in notification rates of syphilis in Indigenous and non-Indigenous Australians from these states and territories between 1993 and 2000 are shown in Figure 26.

Figure 26. Notification rates of syphilis, the Northern Territory, South Australia and Western Australia, 1993 to 2000, by Indigenous status



Vaccine preventable diseases

Introduction

This section summarises the national notification data for diseases targeted by the Australian Standard Childhood Vaccination Schedule in 2000. This includes diphtheria, *Haemophilus influenzae* type b infection, measles, mumps, pertussis, poliomyelitis, rubella and tetanus.

There were 6,617 notifications of vaccine preventable diseases (VPDs) in 2000; 7.4 per cent of the total notifications. Pertussis was by far the most common accounting for 5,942 notifications or 89.8 per cent of all VPD notifications. Notifications of vaccine preventable diseases to the NNDSS and notification rates for vaccine preventable diseases in Australia are shown in Tables 17 and 18.

Table 17. Trends in notifications of vaccine preventable diseases, Australia, 1991 to 2000*

Disease	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Diphtheria	1	12	0	0	0	0	0	0	0	0
Haemophilus influenzae type b	533	465	370	163	77	49	51	35	40	28
Measles	1,438	1,452	4,693	4,805	1,185	481	838	288	238	107
Mumps	–	–	–	–	156	125	191	182	172	212
Pertussis	343	795	4,413	5,441	4,230	4,545	10,825	5,791	4,417	5,942
Polio	0	0	0	0	0	0	0	0	0	0
Rubella	–	–	4,006	3,488	5,751	2,933	1,387	753	377	322
Tetanus	13	13	10	13	7	3	7	8	2	6

* All jurisdictions reported for all years with the following exceptions:

Haemophilus influenzae type b not reported from Western Australia (1991 to 1993).

Mumps not reported from Queensland (1995,1996, 1999 and 2000).

Rubella not reported from Tasmania (1993 to 1994).

Tetanus not reported from Queensland (1991 to 1993).

Table 18. Trends in notification rates of vaccine preventable diseases, Australia, 1991 to 2000* (rate per 100,000 population)

Disease	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Diphtheria	< 0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	3.4	2.9	2.3	0.9	0.4	0.3	0.3	0.2	0.2	0.1
Measles	8.3	8.3	26.6	26.9	6.6	2.6	4.5	1.5	1.3	0.6
Mumps	–	–	–	–	1.1	0.8	1.0	1.0	1.1	1.4
Pertussis	2.0	4.5	25.0	30.5	23.4	24.8	58.4	30.9	23.3	31.0
Polio	0	0	0	0	0	0	0	0	0	0
Rubella	–	–	23.3	20.1	31.8	16.0	7.5	4.0	2.0	1.7
Tetanus	0.1	0.1	0.1	0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1

* All jurisdictions reported for all years with the following exceptions:

Haemophilus influenzae type b not reported from Western Australia (1991 to 1993).

Mumps not reported from Queensland (1995,1996, 1999 and 2000).

Rubella not reported from Tasmania (1993 to 1994).

Tetanus not reported from Queensland (1991 to 1993).

In 2000, the following changes to the childhood immunisation schedule25 occurred:

New vaccines

New combination vaccines for:

- diphtheria-tetanus-acellular pertussis-hepatitis B (DTPa-hepB); and
- *Haemophilus influenzae* type b — hepatitis B (Hib (PRP-OMP)-hep B),

for all three doses in the primary vaccination schedule. This allowed the introduction of universal hepatitis B vaccination (commencing at birth) without requiring an extra injection.

New schedule

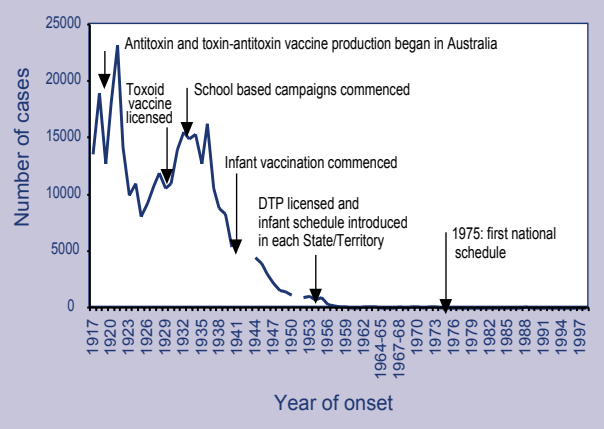
- Two alternative schedules depending on which of the above combination vaccines is used and differing only in the timing of the 4th dose of these vaccines.
- All Australian children recommended to receive the same Hib vaccine (PRP-OMP), which reduces the number of injections and the complexity of the schedule.
- Introduction of universal vaccination for hepatitis B beginning at birth. Infants born to hepatitis B carrier mothers receive hepatitis B immunoglobulin and vaccine at birth. Preadolescent hepatitis B vaccination now recommended at 10–13 years. Booster doses of hepatitis B vaccine no longer recommended.
- Second booster of DTPa now recommended at 4 years, instead of 4–5 years.
- Second dose of MMR now given at 4 years instead of 10–16 years.
- Tetanus and diphtheria boosters no longer recommended every 10 years. A tetanus booster at age 50 is recommended if no boosters have been given within the last 10 years.
- Inactivated poliomyelitis vaccine is an acceptable alternative to live, oral poliomyelitis vaccine (OPV) in the primary vaccination schedule. However, OPV will remain the publicly funded vaccine.
- Influenza vaccine recommended for children with cystic fibrosis, people with severe asthma and pregnant women in the second or third trimester of pregnancy during the influenza season.

The annual report of vaccination coverage estimates for children aged 12 months and the second annual report for children aged 24 months (using data extracted from the Australian Childhood Immunisation Register-ACIR) are also included in this section. A full description of the methodology used for calculating these estimates have been described previously.[42]

Diphtheria

There were no cases of diphtheria notified in 2000. The last known case occurred in 1992 and was notified in 1993. There has been a dramatic decline in the incidence of diphtheria in Australia since the first half of the 20th century (Figure 27).

Figure 27. Trends in notifications of diphtheria, Australia, 1917 to 1998

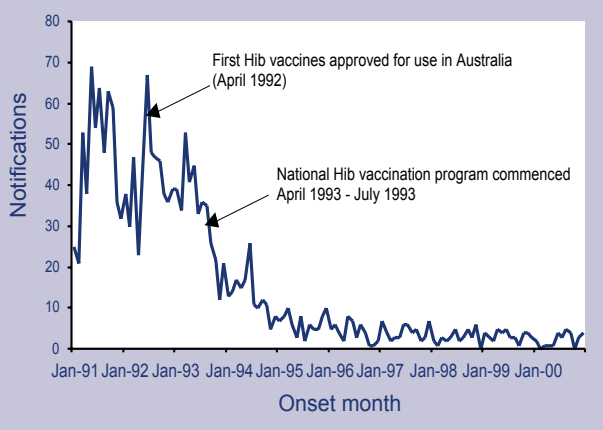


At the height of the 1921 diphtheria outbreak in Australia, there were 23,199 notifications giving a notification rate of 426 cases per 100,000 population.[43] Although diphtheria hasn't been found in Australia since 1992, a recent case in New Zealand[44] and the extensive outbreak in the former states of the Soviet Union in the 1990s[45] highlight the potential for diphtheria to re-emerge.

Haemophilus influenzae type b disease

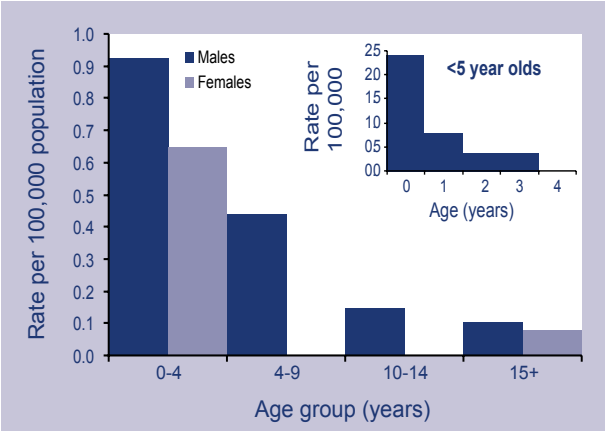
Notifications of *Haemophilus influenzae* type b (Hib) have fallen more than 30-fold since 1991 due to the impact of Hib conjugate vaccines (Figure 28). An assessment of the impact of conjugate vaccines on the global incidence of Hib disease concluded that few vaccines have induced such dramatic declines in disease incidence in such a short time. The prevention of nasopharyngeal colonisation by Hib in vaccinated individuals under most circumstances may explain the dramatic impact on Hib disease.[46]

Figure 28. Trends in notifications of *Haemophilus influenzae* type b infection, Australia, 1991 to 2000



There were 28 notifications of Hib disease in 2000, a rate of 0.1 cases per 100,000 population. This is 30 per cent less than in 1999, and the lowest number of notifications recorded since national surveillance began in 1991. As in previous years most notified cases (10, 36%) were less than 5 years of age. However the number and proportion of all cases in this age group has been declining. The most dramatic decreases have been in those aged less than two years. Infants aged less than 1 year, however, continued to have the highest rate in 2000 (2.4 cases per 100,000 population) (Figure 29). There were more males than females (male:female ratio 1.8:1) notified with Hib disease in 2000.

Figure 29. Notification rates of *Haemophilus influenzae* type b infection, Australia, 2000, by age and sex



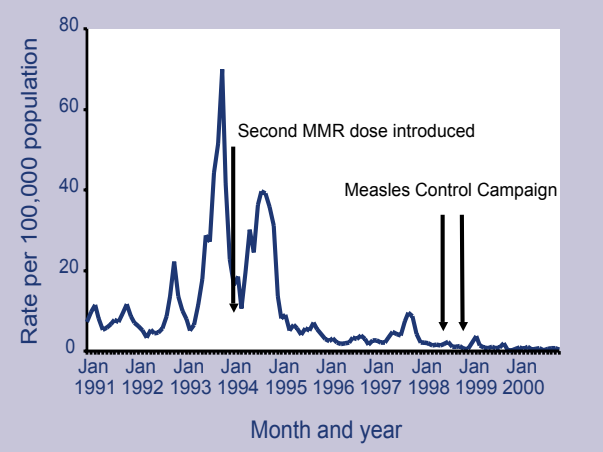
The Northern Territory had the highest notification rate (1 case per 100,000 population, 2 cases) although most cases (12/28) were from Queensland. Two cases occurred in fully vaccinated individuals, seven in partially vaccinated and five in unvaccinated individuals. The vaccination status for the other 14 cases was unknown.

Measles

Measles is the most important cause of vaccine preventable death in the world. In 1998, there were an estimated 30 million measles cases and 880,000 measles-associated deaths worldwide with 85 per cent of deaths occurring in Africa and South East Asia.[47] In recent years there has been a dramatic reduction in measles incidence and endemic measles transmission has been eliminated in a number of countries using a variety of vaccination strategies.[48]

In Australia, measles reports to the NNDSS are at the lowest levels ever recorded (Figure 30). This is the result of a series of successful vaccination initiatives over the past few years. One such initiative was the Australian Measles Control Campaign (August to November 1998) which involved vaccinating 1.7 million primary school children with the Measles-Mumps-Rubella vaccine (MMR) regardless of their past vaccination history. As a result, immunity to measles among these children increased from 84 per cent to 94 per cent.[49]

Figure 30. Trends in notification rates of measles, Australia, 1991 to 2000, by month of onset

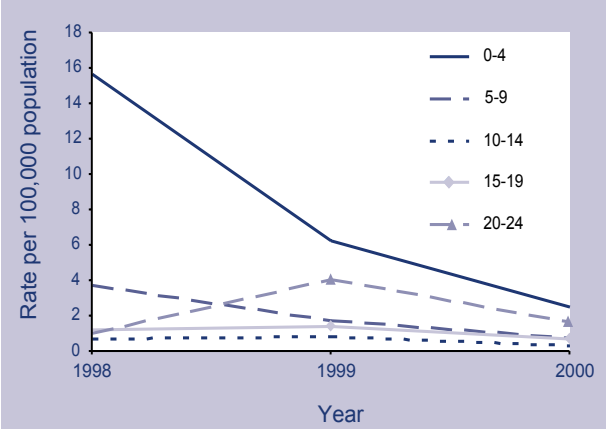


There is evidence that endemic transmission of measles in some parts of Australia is being interrupted. All measles cases in Western Australia in 1999/2000 were imported from overseas or epidemiologically linked to imported cases. (Dowse, Communicable Diseases Control Conference, April 2001, Abstract 58). Using measles virus genotyping, Lambert and colleagues have shown that endemic measles virus strains are no longer circulating in Victoria. Instead, sporadic introduction of imported strains is responsible for limited focal spread. (Lambert, Communicable Diseases Conference, April 2001, Abstract 60). If one accepts that measles elimination should be defined as a situation in which endemic transmission has stopped, sustained transmission cannot occur (because the proportion of susceptible people is sufficiently low), and secondary spread from importations will end naturally without intervention,[50] then Australia may have already achieved measles elimination.

There were 107 cases of measles notified in 2000, a national rate of 0.6 cases per 100,000 population. This is less than half the number reported in 1999 and is the lowest annual rate for Australia since national surveillance began in 1991. In 2000, Western Australia and the Australian Capital Territory began laboratory testing of all notified cases and initiated improved contact tracing. The highest rates of notification were in the Australian Capital Territory (1 case per 100,000 population; 3 cases), Queensland (0.7 cases per 100,000 population; 26 cases) and South Australia (0.7 cases per 100,000 population; 11 cases) (Tables 2 and 3). Twenty-two cases were documented as acquired overseas and 21 cases resulted from seven identified outbreaks in which the index case had acquired measles outside Australia. The source of infection for the remaining 85 cases was not recorded.

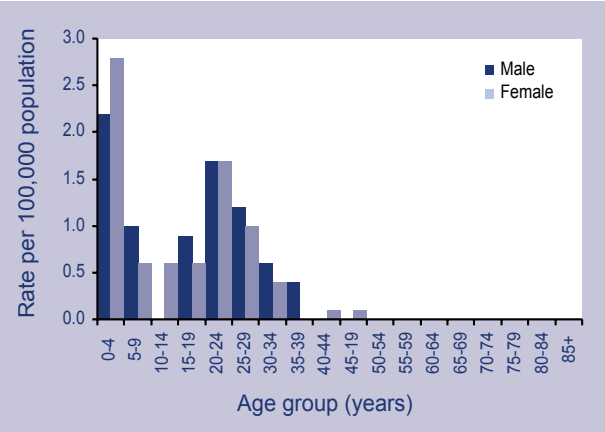
As in recent years, age-specific notification rates were highest for the 0–4 year age group (2.5 cases per 100,000 population) especially those aged less than one year (3.6 cases per 100,000 population) and one year of age (5.2 cases per 100,000 population). Rates for this age group were, however, considerably lower than in the past (Figure 31). Rates for the 5–9 year age group (0.8 cases per 100,000 population) were also the lowest on record.

Figure 31. Trends in notification rates of measles, Australia, 1998 to 2000, by age group



The 20–24 year age group had the second highest age-specific rate (1.7 cases per 100,000 population) and accounted for 21 per cent (23/107) of the reported cases in 2000 (similar to the 20% of cases this age group contributed in 1999). This age group is a 'missed middle' of young adults born in the second half of the 1970s, who have neither been vaccinated nor exposed to the wild measles virus. In the past few years, Australia has recorded measles outbreaks among young adults, often associated with an index case who has travelled to countries with high endemic levels of measles.[51,52,53] As in past years there were similar numbers of males and females with measles reported in 2000 (male:female ratio 1.1:1, Figure 32).

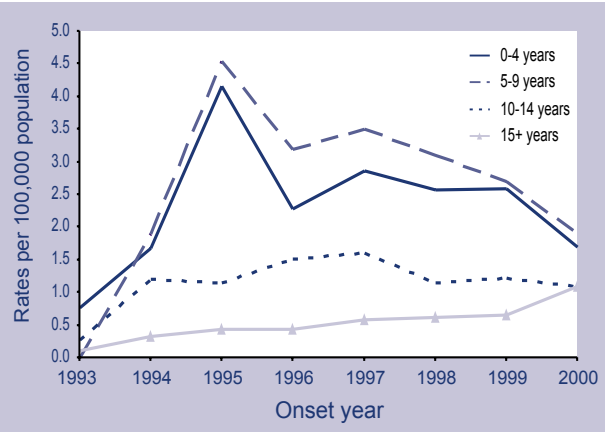
Figure 32. Notification rates of measles, Australia, 2000, by age and sex



Mumps

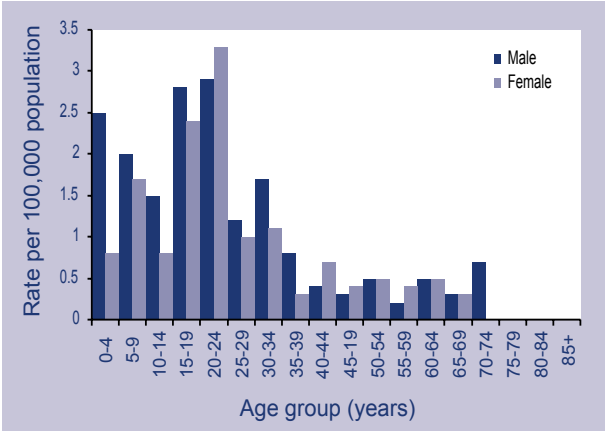
Mumps notification rates in Australia have been close to 1.0 cases per 100,000 population since 1997 (Table 18). Increased coverage of the Australian population with the MMR vaccine has not had the dramatic impact on mumps incidence that has been seen for measles and rubella. Moreover, in recent years the notification rates for mumps have increased in older age groups, in whom mumps morbidity is more severe (Figure 33). Increased use of the MMR vaccine in adolescents and adults over the next few years and ongoing surveillance are essential for mumps control and elimination in Australia. (Gidding, Communicable Diseases Control Conference, April 2001, Abstract 57)

Figure 33. Trends in notification rates of mumps, Australia, 1993 to 2000, by age group



In 2000, there were 212 notifications of mumps, a rate of 1.4 cases per 100,000 population. This is above the WHO elimination target of <1 case per 100,000 population and is a 23% increase on the 172 cases reported in 1999. There were cases in most age groups with the majority (151, 71%) aged 15 years or more (Figure 34). In contrast with previous years the highest notification rates were in the 20–24 year age group (3.1 cases per 100,000 population) and the 15–19 year age group (2.6 cases per 100,000 population). This pattern was apparent even in New South Wales where only laboratory-confirmed cases are notifiable. Overall, there was a slight preponderance of mumps notifications from males (male:female ratio 1.3:1). Mumps was not notifiable in Queensland from July 1999 to December 2000.

Figure 34. Notification rates of mumps, Australia, 2000, by age and sex



Pertussis

Pertussis continues to be the most common vaccine preventable illness in Australia, with periodic epidemics occurring at intervals of 3 to 5 years (Figure 35).[54] As a result of infant immunisation against pertussis in Australia (five doses given at 2, 4, 6, 18 and 48 months) the peak notification rate is now found among young adolescents (aged 10–14) (Figure 36).

Figure 35. Trends in notification rates of pertussis, Australia, 1991 to 2000, by month of onset

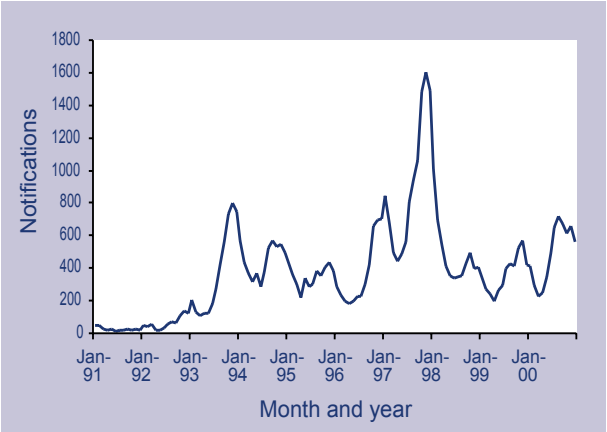
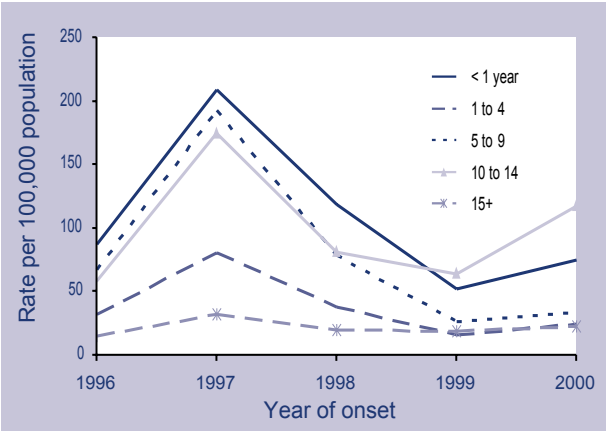


Figure 36. Trends in notification rates of pertussis, Australia, 1996 to 2000, by age group



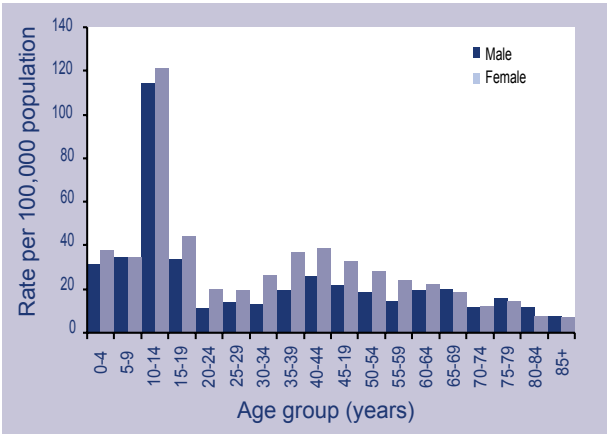
Despite high levels of vaccination, pertussis has increased in a number of countries since 1997. This has prompted investigations into the evolution of variants of *Bordetella pertussis*. Mooi and colleagues have observed antigenic divergence between vaccine strains and clinical isolates of *Bordetella pertussis* specifically in the surface-associated protein pertactin and the pertussis toxin.[55] Replacement of vaccine with non-vaccine strains as a result of herd immunity has not yet had any measurable effect on pertussis vaccine efficacy, but surveillance of variant strains of the bacteria may be important for the control of pertussis in the future.

Several recent studies have examined the importance of pertussis as a cause of prolonged coughing in adults and adolescents. A recent study in Canada suggests that up to 20 per cent of prolonged coughs are associated with laboratory evidence of pertussis infection.[56] The nature of that evidence is controversial, however, as only 2.3 per cent of symptomatic cases were confirmed by culture, PCR or a fourfold increase in pertussis antibody. The remainder were diagnosed on the basis of a single high pertussis antibody titre.

Since it is well established that adolescents and adults are frequently the source of pertussis infection for infants and children, and adolescents now have the highest rates of disease, vaccination of adolescents with acellular pertussis vaccines has been instituted in France, Germany and Canada. It remains to be seen how this will impact on the epidemiology of pertussis in these countries. Implementation of an adolescent vaccination program in Australia is currently being considered by a working party of the Australian Technical Advisory Group on Immunisation (ATAGI).

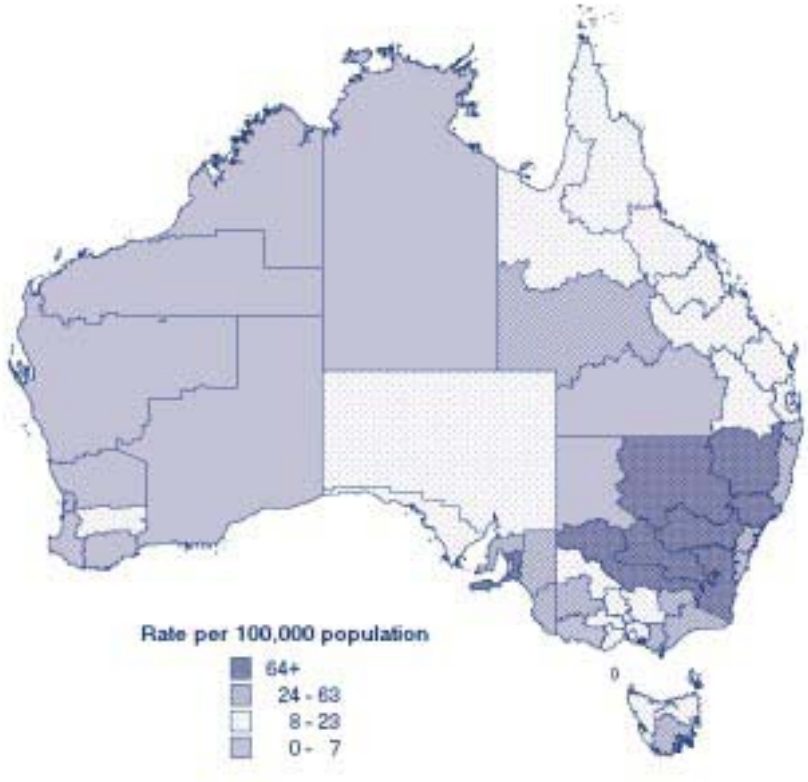
There were 5,942 notified cases of pertussis in 2000, 1,525 more than in 1999. The annual notification rate was 31.0 cases per 100,000 population. Pertussis notifications peaked in August, when 721 cases were notified. As in 1999, the 10–14 year age group had the highest notification rate of pertussis (117.7 cases per 100,000 population) (Figure 37).

Figure 37. Notification rates of pertussis, Australia, 2000, by age and sex



Notification rates of pertussis varied considerably by geographic location (Map 6). At the State/Territory level, rates were highest in the Australian Capital Territory (66.2 cases per 100,000 population) and lowest in the Northern Territory (2.6 cases per 100,000 population), where only 5 cases were notified. In 2000, South Australia included pertussis cases diagnosed by PCR for the first time.

Map 6. Notification rates of pertussis, Australia, 2000, by Statistical Division of residence



Poliomyelitis

No cases of poliomyelitis were reported in Australia in 2000. It is difficult to determine exactly when the last case of locally acquired poliomyelitis occurred in Australia. However, the last laboratory confirmed case was in 1967 and there were three clinically compatible cases notified in 1972 with no additional information currently available.[57] All cases notified since 1972 have been investigated further and this has led them to be re-classified as cases of vaccine-associated poliomyelitis. The last known imported case of poliomyelitis was due to wild poliovirus type 1 in 1977.

On 29 October 2000, the WHO certified the Western Pacific Region polio-free.[58] The last recorded case in the region was reported in Cambodia in 1997.

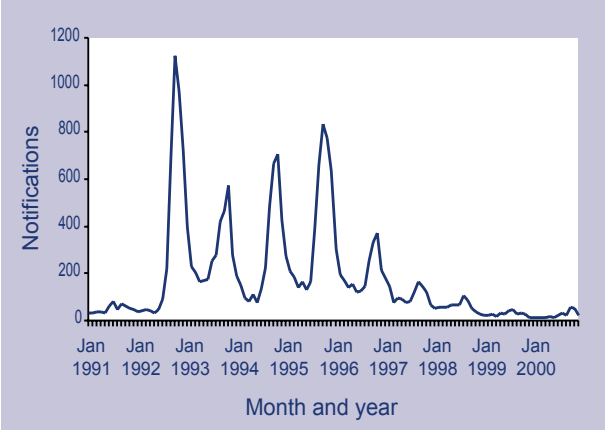
Since the live oral polio vaccine has the potential to cause vaccine associated disease, the USA has recently replaced this vaccine with an inactivated polio vaccine. This issue is under consideration in Australia by ATAGI

A report on the Australian National Polio Reference Laboratory is given later in this report (p188).

Rubella

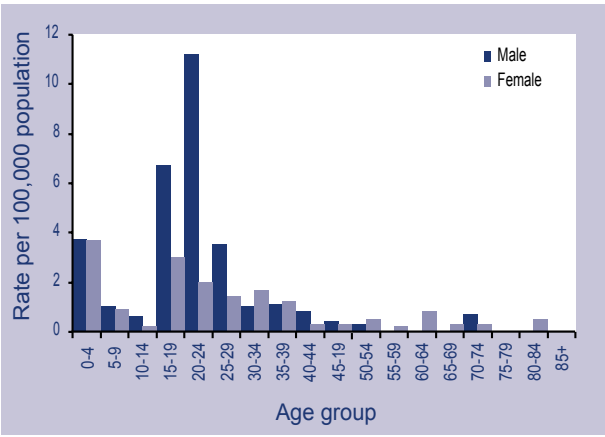
Since 1995, annual numbers of rubella notifications have been declining (Figure 38). This decrease has occurred at the same time as MMR vaccine usage has been increasing. In 2000, there were 322 notifications, a notification rate of 1.7 cases per 100,000 population. This is the lowest on record both nationally and in each State or Territory. As in previous years, the highest number of notified cases occurred in October, reflecting the expected seasonal increase in Spring months. The highest notification rate was from New South Wales (3.0 cases per 100,000 population), where all cases were laboratory-confirmed.

Figure 38. Trends in notification rates of rubella, Australia, 1991 to 2000, by month of onset



In 2000, the notification rate of rubella was highest in males in the 20–24 year age group (11.2 cases per 100,000 population, Figure 39). However, rates for this group have been decreasing in recent years due to the replacement of the schoolgirl rubella program with adolescent vaccination of both males and females between 1994 and 1998. Overall, there were more males than females notified with rubella (male:female ratio 2.0:1) in 2000.

Figure 39. Notification rates of rubella, Australia, 2000, by age and sex



There were 68 notifications of rubella from women of childbearing age (15–49 years) in 2000, a rate of 1.4 cases per 100,000 population. No notifications of congenital rubella were received in 2000 (Annual Report of the Australian Paediatric Surveillance Unit). Only 6 cases of congenital rubella have been reported since 1995, with the last case notified in 1999.

Tetanus

In 2000, there were 6 cases of tetanus notified to the NNDSS. Five of these were in adults aged 50 years or greater and one was in an infant. Of the 6 cases, 1 was partially vaccinated, 2 were unvaccinated (including the 2-year-old infant) and the vaccination status of the other three was unknown. Five of the 6 cases were females.

Childhood vaccination coverage reports

Estimates of vaccination coverage for both 'fully vaccinated' and individual vaccines for children at 12 months of age continued to improve in 2000 (Table 19). This trend was also evident in each State and Territory. Vaccination coverage at 12 months of age for Australia as a whole has now surpassed the Immunise Australia Program target of 90 per cent coverage for the first milestone vaccines.

Vaccination coverage at 2 years of age was first reported in 1998. Coverage estimates for individual vaccines recommended at 12 months and 18 months of age were higher in 2000 compared with the previous year, as were the estimates for being 'fully vaccinated' at 2 years of age (Table 20). 'Fully vaccinated' coverage estimates were reported to be considerably lower than estimates for individual vaccines. One likely factor is poor identification of children on immunisation encounter forms, which leads to difficulties matching new and existing vaccination records on the ACIR. Further, in their regular parent surveys, the Health Insurance Commission have found some parents have an objection to particular vaccines, although not always the same vaccines. It is important to note that in countries such as the United Kingdom, three doses of diphtheria-tetanus-poliomeylitis vaccine (DTP) and Hib vaccine constitute full vaccination with these vaccines at 2 years of age.

Table 19. Percentage of Australian children born in 1999 vaccinated at one year of age for four consecutive birth cohorts assessed during 2000 using the Australian Childhood Immunisation Register

Vaccine group	% vaccinated in birth cohort			
	1 Jan to 31 Mar 1999	1 Apr to 30 Jun 1999	1 Jul to 30 Sep 1999	1 Oct to 31 Dec 1999
DTP	89.8	89.8	91.8	91.5
OPV	89.8	90.2	91.8	91.4
Hib	89.3	90.3	91.7	94.6
Fully vaccinated	**88.4**	**89.0**	**91.3**	**91.2**

Table 20. Percentage of Australian children born in 1998 vaccinated at 2 years of age for four consecutive birth cohorts, assessed during 2000 using the Australian Childhood Immunisation Register

Vaccine group	% vaccinated in birth cohort			
	1 Jan to 31 Mar 1998	1 Apr to 30 Jun 1998	1 Jul to 30 Sep 1998	1 Oct to 31 Dec 1998
DTP	87.5	88.9	89.6	88.3
OPV	91.9	92.2	92.7	93.1
Hib	87.2	89.2	89.6	94.7
MMR	91.0	91.3	92.3	92.4
Fully vaccinated	**88.4**	**89.0**	**91.3**	**91.2**

Vectorborne diseases

Vectorborne diseases under surveillance in Australia in 2000 included arboviruses (arthropod borne viruses) and malaria. In this year the NNDSS collected information on 2 alpha viruses (Barmah Forest virus and Ross River virus), and one flavivirus (dengue) as well as malaria. Other arboviruses not including Barmah Forest, Ross River and dengue viruses were designated 'arbovirus not elsewhere classified (NEC)'. This category included infections with the flaviviruses Murray Valley encephalitis (MVE) virus, Kunjin virus, Japanese encephalitis (JE) virus, Kokobera virus and Stratford virus, as well as the alphavirus Sindbis. In 2000, there were 6,069 notifications of vectorborne diseases to the NNDSS (6.8% of total notifications).

Surveillance of human infection with MVE and Kunjin viruses is supplemented by sentinel chicken surveillance. Animal surveillance measuring seroconversions to JE in pigs is also used to complement surveillance in humans. Vector data, virus isolations and meteorological data are used to predict conditions suitable for an outbreak of arbovirus disease, and complement animal and human surveillance mechanisms.

Trends in the reporting of arboviruses over the period 1991 to 2000 are shown in Tables 21 and 22. The number of notifications classified as arbovirus (NEC) has decreased since 1995, when Barmah Forest virus became notified separately. Since then, notification rates for Barmah Forrest virus have remained stable. In comparison, dengue and Ross River virus notification rates have showed periods of increased disease activity over this time frame. The number of notifications of malaria have remained consistent over the decade. The notification rate of vectorborne disease depends on annual rainfall patterns, the mosquito populations and the exposure of humans to mosquitoes.

Control of mosquito populations and interception of exotic mosquito species, which may be disease vectors are important control strategies for vectorborne disease. Media warnings to residents during times of increased risk emphasise personal protection and risk reduction by reducing potential mosquito breeding sites.[59]

Table 21. Trends in number of notifications of arboviral infections, Australia, 1991 to 2000*

Disease	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Arbovirus infection NEC	196	64	173	31	43	12	19	88	62	69
Barmah Forest virus infection	–	–	–	–	762	876	691	529	638	634
Dengue	18	373	681	17	39	123	174	579	132	215
Malaria	787	731	669	706	618	853	749	660	732	951
Ross River virus infection	–	5,701	5,254	3,828	2,644	7,783	6,596	3,151	4,416	4,200

* All jurisdictions reported for all years with the following exception
Dengue not reported from Australian Capital Territory (1991 to 1992).

Table 22. Trends in notification rates of arboviral infections, Australia, 1991 to 2000* (rate per 100,000 population)

Disease	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Arbovirus infection NEC	1.1	0.4	1.0	0.2	0.2	0.1	0.1	0.5	0.3	0.4
Barmah Forest virus infection	–	–	–	–	4.2	4.8	3.7	2.8	3.4	3.3
Dengue	0.1	2.2	3.9	0.1	0.2	0.7	0.9	3.1	0.7	1.1
Malaria	4.6	4.2	3.8	4.0	3.4	4.7	4.0	3.5	3.9	5.0
Ross River virus infection	–	32.6	29.7	21.4	14.6	42.5	35.6	16.8	23.3	21.9

* All jurisdictions reported for all years with the following exception
Dengue not reported from Australian Capital Territory (1991 to 1992).

Alphavirus Infections

Barmah Forest virus infection

This virus was first isolated from mosquitoes trapped in the Barmah Forest in Victoria in 1974. Outbreaks of Barmah Forest disease have been described since the virus was first shown to cause human disease in 1988. Barmah Forest virus infection is characterised by polyarthritis, myalgia, rash, fever, lethargy and malaise and may cause a chronic disease in some patients.[60] *Aedes* and *Culex* mosquitoes are the major mosquito vectors, while marsupials are suspected vertebrate hosts.

In 2000, 634 notifications of Barmah Forest virus infection were reported, similar to the 638 cases reported in 1999. The highest rates were reported in the Northern Territory (4.6 cases per 100,000 population) and Queensland (9.7 cases per 100,000 population). Rates were very low in southern states; no cases were reported from the Australian Capital Territory and Tasmania (Map 7). The male to female ratio was 1.4:1. The highest rate of infection (6.6. cases per 100,000 population) was in those aged 50–54 years, although the notification rates across the 35–69 age range were similar (Figure 40). Peak notifications were in the period January to April and followed previously observed seasonal trends (Figure 41).

Map 7. Notification rates of Barmah Forest virus infection, Australia, 2000, by Statistical Division of residence

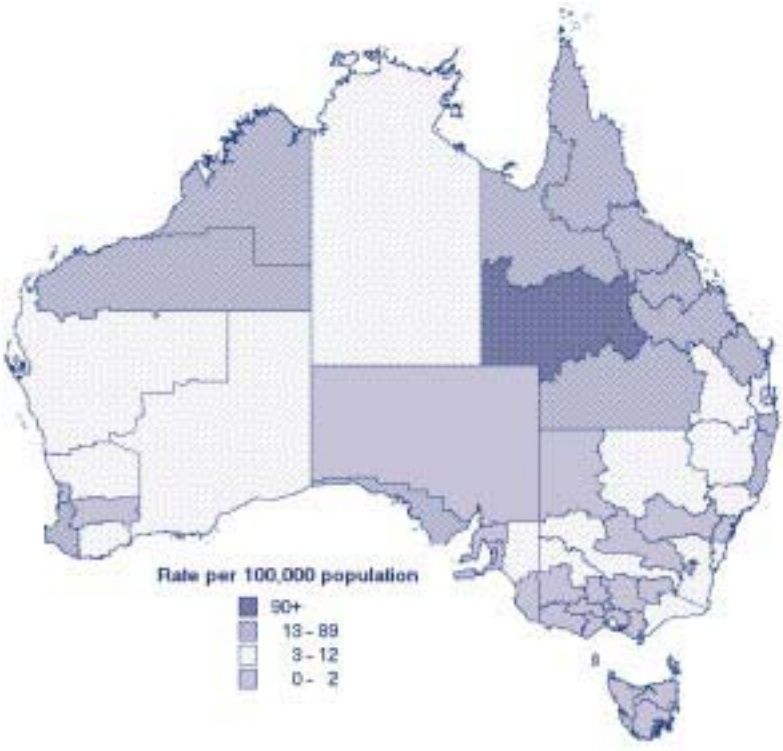


Figure 40. Notification rates of Barmah Forest virus infection, Australia, 2000, by age and sex

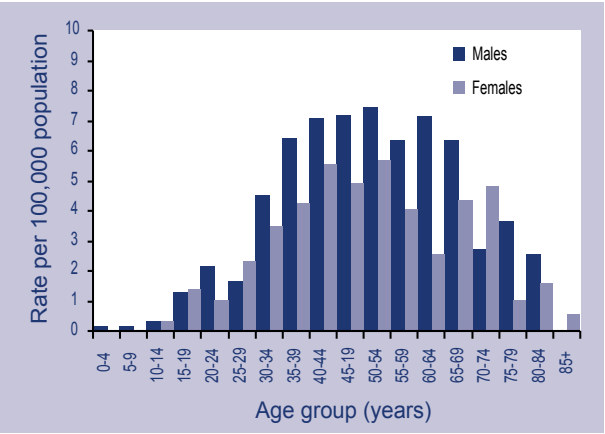
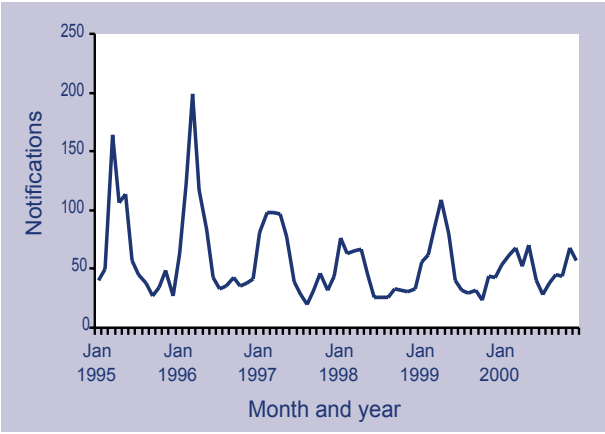


Figure 41. Trends in notification rates of Barmah Forest virus infection, Australia, 1995 to 2000, by month of onset



Ross River virus

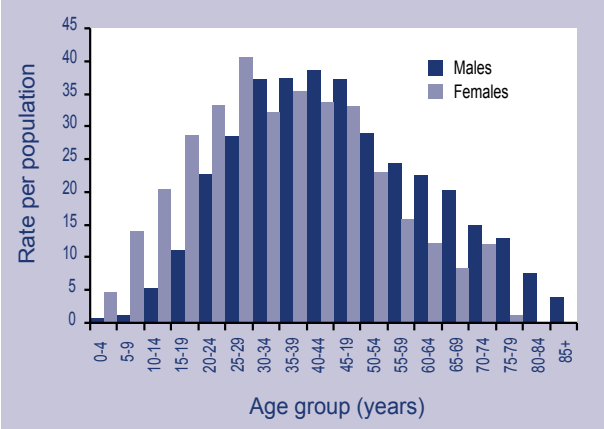
Ross River virus is the most common cause of arbovirus disease notified in Australia. While sporadic cases occur throughout Australia, epidemics occur in temperate regions and in tropical north-eastern Australia throughout the year. Epidemics in temperate regions are associated with heavy rainfall. Evidence indicates that the virus may persist in desiccation-resistant eggs of the *Aedes* spp mosquito, which would explain the rapid onset of cases after heavy rain and flooding. Marsupials and horses have been implicated as hosts for the virus and flying foxes may be responsible for the wide spread dispersal of different genetic types of the virus.[61]

Major outbreaks have been recorded in Western Australia (1991/1992 and 1995/1996), Victoria and South Australia (1993 and 1997), New South Wales (1996 and 1997) and Queensland (1996). Queensland has had the largest number of cases of Ross River virus infection for the past 3 years (1998 to 2000).

Clinical Ross River virus disease occurs most commonly in adults, marked by arthralgia and myalgia (joint and muscle pain). True arthritis occurs in over 40 per cent of patients, while about 50 per cent of patients have a fever or rash.[62]

There were 4,200 notifications of Ross River virus infections in 2000, giving a rate of 21.9 cases per 100,000 population, a slight decrease from the 23.3 cases per 100,000 population observed in 1999. Rates were highest in the Northern Territory (65.5 cases per 100,000 population), Western Australia (57.5 cases per 100,000 population) and Queensland (41.4 cases per 100,000 population) (Map 8). The male to female ratio was 1:1. The highest notification rate for females (40.6 cases per 100,000 population) was in the 35–39 year age group. The highest rate for men (38.5 cases per 100,000 population) was in the 40–44 year age group (Figure 42). Peak reporting was in the first and second quarters of the year (Figure 43).

Figure 42. Notification rates of Ross River virus infection, Australia, 2000, by age and sex



Map 8. Notification rates of Ross River virus infection, Australia, 2000, by Statistical Division of residence

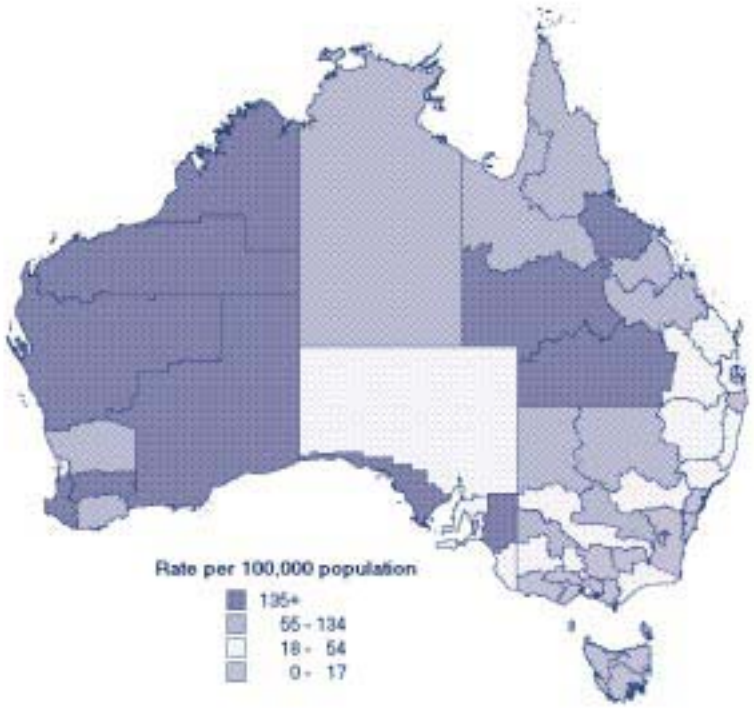
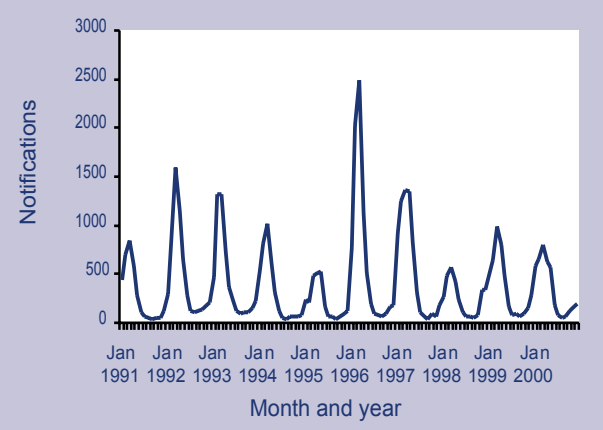


Figure 43. Trends in notification rates of Ross River virus infection, Australia, 1991 to 2000 by month of onset



Flavivirus infections

Dengue fever

Historical trends of dengue in Australia

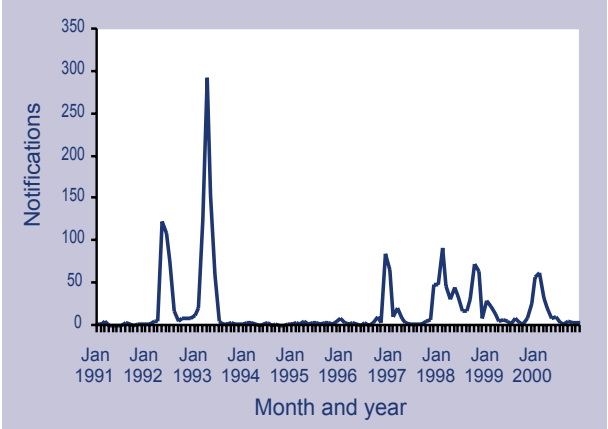
Dengue fever is an acute febrile illness characterised by sudden onset, fever, headache and rash. Dengue haemorrhagic fever is a major complication arising from secondary infection with heterologous serotypes of the dengue virus.[63] This complication has a high fatality rate. Two cases of dengue haemorrhagic fever have been reported in Australia, one in 1992 and another in 1997.[62] There is a concern that introduction of other dengue serotypes into northern Australia could increase the risk of dengue haemorrhagic fever.

Dengue virus is not endemic in Australia and the spread of dengue in Australia is limited to the range of the mosquito vector *Aedes aegypti* which spans the Torres Strait Islands and north Queensland.[62] An outbreak caused by dengue type 2 of more than 900 confirmed cases occurred in Townsville and Charters Towers in 1992 to 1993. In 1996/1997 another outbreak of dengue type 2 occurred in the Torres Strait. In 1997/1998 165 cases of dengue type 3 and 12 of dengue type 2 were reported from Cairns.[62]

Dengue occurrence in 2000

There were 215 notifications of dengue in 2000, a rate of 1.1 cases per 100,000 population, an increase on the 1999 rate of 0.7 cases per 100,000 population. The highest rates were found in the Northern Territory (47.6 cases per 100,000 population) and Queensland (2.4 cases per 100,000 population). The male to female ratio was 1.6:1. The highest notification rates among men were in the 35–39 and 50–54 year age groups (3.2 cases per 100,000 population). The highest notification rate for women, at 2.4 cases per 100,000 population, was in the 30–34 year age group. Notifications of dengue for 2000 peaked in Summer (first and fourth quarters of the year, Figure 44).

Figure 44. Trends in notification rates of dengue fever, Australia, 1991 to 2000, by month of onset



In all jurisdictions except Queensland, dengue cases were acquired overseas (n=131). In Queensland, 11 (13%) cases were identified as acquired within Australia, 22 (26%) acquired overseas and the source of infection was unknown in the remaining 51 cases.

The Western Pacific Region, which includes countries in East Asia and the Pacific, reported 45,603 cases of dengue in 2000. The number of cases in the region has decreased since 1998, when there was a pandemic of dengue across the region. In 2000, cases increased on 1999 figures in only 2 countries — Cambodia and Palau — and in both countries increases were seen in the numbers of all serotypes (WPR/WHO. Summary of the dengue situation in the Western Pacific Region an update:2001. http://www.wpro.who.int/document/DENGUE_SITUATION_IN_WPR_Aug01.doc).

Arbovirus: not elsewhere classified

In 2000, there were 69 notifications of arboviruses 'not elsewhere classified' reported to the NNDSS, giving a rate of 0.4 cases per 100,000 population. This rate was similar to that in 1999 (0.3 cases per 100,000 population). The jurisdiction reporting the largest number of cases of arbovirus infections NEC in 2000 was Victoria, notifying 26 (38%) of the 69 cases in that year. The male to female ratio was 1.4:1. The highest rate for women (0.8 cases per 100,000 population) was in the 50–54 year age group and for men the highest rate (1.2 cases per 100,000 population) was in the 65–69 year age group.

While cases of infection with Murray Valley encephalitis virus were only separately reported by Western Australia in 2000, information provided by the individual jurisdictions indicated there were 16 cases in the year (Table 23). Exceptional weather conditions in 2000 provided ideal conditions for mosquito breeding and MVE virus transmission. The activity in Western Australia was unusual as there was a new southerly extension into the Mid-west region.[64]

Data from sentinel chicken surveillance (Figure 45) provided an early warning of disease activity in 2000. In Western Australia, seroconversions in sentinel chickens preceded likely dates of exposure in human cases by 4–18 weeks in all but one case.[65]

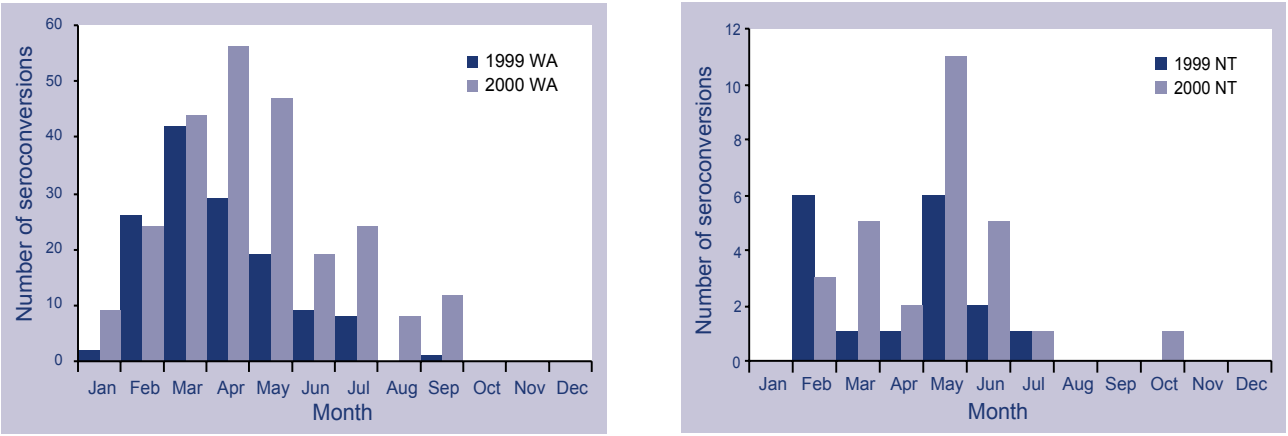
While not specifically identified in the NNDSS, there were 4 cases of Kunjin virus infection identified in 2000. No cases of Japanese encephalitis were reported in 2000. The last case of Japanese encephalitis in Australia was reported in 1998.

Table 23. Confirmed cases of Murray Valley encephalitis virus infection, Australia, 2000

Jurisdiction where diagnosis was made	Likely place of disease acquisition	Cases (Deaths)
Northern Territory	Darwin	1
	Alice Springs	3
	WA*	2(1)
	SA	1
Western Australia	Pilbara	2
	Mid-west	3
	Gascoyne	1
	Murchison	1
Total		**16**

* includes one case acquired in the Kimberley and a second case acquired in Mid-west/Kimberley region.

Figure 45. Seroconversions to Murray Valley encephalitis virus in sentinel chickens, Western Australia and Northern Territory, 1999 to 2000



Malaria

While Australia has been free of endemic malaria since 1983, sporadic cases are reported among travellers returning from malaria endemic countries. The three requirements for malaria transmission exist in Australia: infected humans carrying gametocytes in their blood, mosquito vectors and suitable climate. Thus, surveillance of human cases of malaria and the rapid entomological response to prevent infection of local *Anopheles* mosquitoes are important public health activities in northern Australia.[66]

In 2000, there were 951 cases of malaria reported to the NNDSS, giving a rate of 5.0 cases per 100,000 population. This represented an increase in the notification rate, compared with the 3.9 cases per 100,000 population reported in 1999. Among the jurisdictions, the highest rates were reported from the Northern Territory (38.9 cases per 100,000 population), Queensland (11.5 cases per 100,000 population) and the Australian Capital Territory (5.7 cases per 100,000 population). The male to female ratio was 3.7:1, an increase on the ratio for 1999 (2.6:1). The peak notification rates for men (29.2 cases per 100,000 population) and for women (4.5 cases per 100,000 population) were in the 20–24 year age group.

Malarial parasites were identified and reported in 943 (99%) of the 951 cases reported to the NNDSS. *Plasmodium vivax* was the most common isolate (717 cases, 75% of the total), followed by *P. falciparum* (194 cases, 20%).

Travel to malaria endemic countries was documented in all cases in New South Wales, the Northern Territory, South Australia, Tasmania, Victoria and Western Australia. The travel data were also recorded for 96 of 409 notifications in Queensland. The data were not collected in the Australian Capital Territory. Data on the use of anti-malaria prophylaxis were available from Victoria and the Northern Territory. In Victoria 50 per cent of cases had not taken prophylaxis and a majority of these were either newly arrived migrants or Australian residents visiting relatives in their country of birth. In the Northern Territory, 53 (70%) had received prophylaxis, 21 (28%) had not, while the status of the remaining 2 patients was unknown.

Malaria in the Australian Defence Forces returning to Australia from duty in East Timor in 2000 accounted for 267 cases. While all of the 5,500 troops were given prophylaxis with doxycycline, 64 developed symptoms of malaria during their 4–5 months in East Timor and a further 212 soldiers developed symptoms on return to Australia. Of soldiers developing malaria while in East Timor, two-thirds were infected with *P. falciparum*, all of which were successfully treated with mefloquine and doxycycline. By contrast all but two of the soldiers who developed malaria on return to Australia were infected with *P. vivax.* When these soldiers were treated with primaquine, 44 soldiers had relapses, which suggested that *P. vivax* in East Timor was primaquine tolerant.[67]

Other vectorborne disease surveillance

AQIS exotic mosquito interceptions in 2000

In 2000, the Australian Quarantine and Inspection Service reported 41 interceptions of mosquitoes on various imported goods. Of the 41 interceptions, 22 species were considered unknown to Australia, or of limited distribution, including 15 interceptions of *Aedes aegypti*, six of *Aedes albopictus* and one *Culex spathifurca*. Thus, in 2000 there remained a constant threat of importation of exotic mosquito species, some of which may be vectors of human disease.

Zoonoses

Zoonoses are diseases of humans acquired from an animal source. Although there are many recognised zoonoses in Australia, only five zoonotic infections were reported at the national level in 2000. These were brucellosis, hydatid infection, leptospirosis, ornithosis and Q fever. All notifiable zoonoses have epidemic potential and are often associated with particular occupations. Zoonotic infection may present with non-specific clinical symptoms and a definitive diagnosis depends on appropriate laboratory investigations. The trend in the number and rates of zoonoses disease notifications reported to the NNDSS between 1991 and 2000 are shown in Tables 24 and 25.

A total of 969 notifiable zoonotic infection cases were received by the NNDSS in 2000, which accounted for 1.1 per cent of all notifications. The number of notifications remained at almost the same level as in previous years.

Table 24. Trends in notifications of zoonotic disease, Australia, 1991 to 2000*

Disease	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Brucellosis	25	32	13	38	24	39	39	45	52	27
Hydatid infection	25	34	25	33	31	28	56	37	26	26
Leptospirosis	163	170	174	122	164	214	114	202	323	243
Ornithosis	136	110	83	87	185	86	35	64	84	100
Q fever	544	561	870	656	456	544	545	560	515	573

* All jurisdictions reported for all years with the following exceptions:
Hydatid infection not reported from New South Wales (1991–2000).
Ornithosis not reported from New South Wales (1991 to 2000) and only reported from Queensland during the period of 1992 to 1996.

Table 25. Trends in notification rates of zoonotic disease, Australia, 1991 to 2000* (rate per 100,000 population)

Disease	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Brucellosis	0.1	0.2	0.1	0.2	0.1	0.2	0.2	0.2	0.3	0.1
Hydatid infection	0.2	0.3	0.2	0.3	0.3	0.2	0.5	0.3	0.2	0.2
Leptospirosis	0.9	1.0	1.0	0.7	0.9	1.2	0.6	1.1	1.7	1.3
Ornithosis	1.6	1.0	0.7	0.7	1.5	0.7	0.4	0.7	0.9	1.1
Q fever	3.1	3.2	4.9	3.7	2.5	3.0	2.9	3.0	2.7	3.0

* All jurisdictions reported for all years with the following exceptions:
Hydatid infection not reported from New South Wales (1991–2000).
Ornithosis not reported from New South Wales (1991 to 2000) and only reported from Queensland during the period of 1992 to 1996.

All States and Territories reported brucellosis, leptospirosis and Q fever to the NNDSS in 2000. In New South Wales neither hydatid infection nor ornithosis were notifiable diseases in 2000 and ornithosis was not notifiable in Queensland. Zoonotic diseases are not found in all jurisdictions in Australia. The Northern Territory has not reported any cases of brucellosis or Q fever and only a single case of hydatid disease between 1991 and 2000, and Tasmania has not reported any cases of brucellosis during the same period.

The majority of zoonotic infections were reported from Queensland (558, 58%), followed by New South Wales (184, 19%). Queensland had the highest notification rate of Q fever (10.9 cases per 100,000 population), the Northern Territory had the highest notification rate of leptospirosis (4.1 cases per 100,000 population) while Victoria had the highest notification rate of ornithosis (1.8 cases per 100,000 population) (Table 25).

Brucellosis

Brucella are small aerobic gram-negative bacilli. Human brucellosis is caused by any of four species: *Brucella melitensis* (primarily from goats, sheep, and camels), *Brucella abortus* (from cattle), *Brucella suis* (from pigs) and *Brucella canis* (from dogs). *B. melitensis* and *B. abortus* are exotic to Australia and cases of *B. melitensis* are usually imported into Australia from overseas travellers who have consumed unpasteurised dairy products. *B. suis* is restricted to localised populations of feral pigs in Queensland.

Brucella remains one of the world's major zoonotic pathogens, and is responsible for enormous economic losses as well as considerable human morbidity in endemic areas, especially in developing areas of the Mediterranean Region, Middle East, western Asia and parts of Africa and Latin America. The *Brucella* organism is transmitted from *Brucella*-infected animals to humans by direct contact with blood, tissues and urine of infected animals. Infection is through breaks in the skin or through consumption of contaminated animal products, such as milk. Airborne transmission from animal to humans is also possible. The organism may also be transmitted from human to human via blood transfusion and bone marrow or organ transplantation, through the placenta, during breast-feeding, and during sexual activity.[68,69,70] The disease usually presents within weeks of exposure, but in some exceptional cases, the incubation period may be as long as several years.[71] The pathogen could also be a potential agent of biological terrorism, particularly *B. melitensis* and *B. suis*. The bacteria are highly infectious by aerosol and could be delivered as a slurry in bomblets which may survive for 6 weeks in dust and 10 weeks in soil or water.[72,73,74]

There were 27 notifications of brucellosis in 2000, a rate of 0.1 cases per 100,000 population, a decrease from 1999 (52 cases; 0.3 cases per 100,000 population). This is the lowest national notification rate on record since 1994.

The majority of notifications (20, 74%) occurred between August and December. The age-specific rate was highest in the 40–44 year age group at 0.5 cases per 100,000 population. Men were more often infected than women with the overall male to female ratio being 8:1.

Almost all the cases of brucellosis were reported from Queensland, except for one case which was reported from New South Wales. The highest rates of disease were reported in the Central West (24.7 cases per 100,000 population) and the South West (15.6 cases per 100,000 population) Statistical Divisions of Queensland. A previous study has suggested that there is a high frequency of *B. suis* infections in Queensland among men who hunt and slaughter feral pigs.[75]

Hydatid infection

Hydatid infection, caused by the larval stage of the tapeworm *Echinococcus granulosus*, is generally found in rural Australia. Disease typically occurs where humans become infected by the ingestion of eggs passed in the faeces of dogs, dingoes or foxes.[76] Wallabies, wombats, feral pigs, sheep and kangaroos are all intermediate hosts that act as reservoirs of the disease. Dogs and foxes, feeding off the offal or other remains of these animals become infected, and can carry the disease into rural communities, or to the periphery of urban settlements.[77]

Symptoms of hydatid disease usually occur only in the advanced stages of disease, and the infection may remain asymptomatic for many years. In the past, hydatid disease has been shown to be under-reported in Australia.[78]

In 2000, hydatid infection was notifiable in all States and Territories in Australia, except New South Wales. Following a successful elimination program in the period 1965 to 1996, Tasmania was declared free from hydatid disease in 1996 and has remained this status since. A total of 26 cases were notified during 2000. This is the same number as reported in 1999. The annual notification rate was 0.2 cases per 100,000 population. The highest number of cases (n=13) was reported in Victoria, followed by Queensland (n=8) and Western Australia (n=5). The highest rate of hydatid infection as reported in the Kimberley region of Western Australia (3.3 cases per 100,000 population).

Of the 26 hydatid cases notified, 10 were men, 13 were women and three were of unknown gender. The male to female ratio was 0.8:1. The highest age-specific rates were in women aged 65–74 years (0.9 cases per 100,000 population) and in men aged 75–79 years (1.4 cases per 100,000 population).

Information on the country of birth was obtained for 13 of 26 hydatid notifications. There were 6 cases among Australian born people (including an Indigenous Australian) and 7 cases among overseas born persons (4 cases from Greece; 1 case each from Bosnia, China and England).

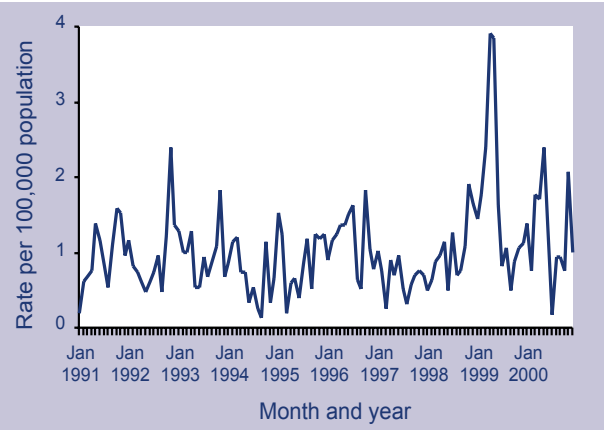
Leptospirosis

Leptospirosis is a zoonotic disease transmitted by wild and domestic animals. The causative organisms are the spirochetes of the *Leptospira* genus. The source of infection is often soil or water contaminated with the urine of domestic or wild animals. Farmers, veterinarians, abattoir workers and some recreational sporting athletes are recognised to be at high risk of infection.[27]

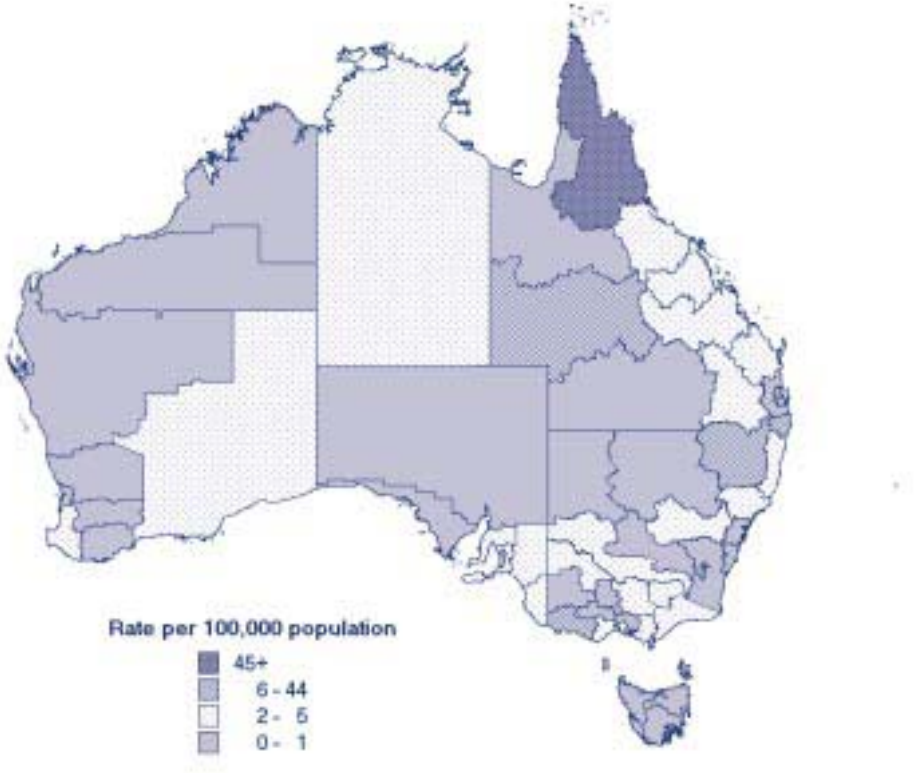
Leptospirosis was first recognised in Australia in 1934,[79] and occurs in all parts of Australia today. The disease may be asymptomatic, mild or severe and can cause death. The clinical manifestations of the disease include a variety of symptoms, but the most common presentations include fever, myalgia, meningitis, rash, haemolytic anaemia and jaundice.[27]

Leptospirosis is a notifiable disease in all States and Territories of Australia. There were 243 notifications of leptospirosis reported to the NNDSS in 2000, with an annual notification rate of 1.3 cases per 100,000 population (Figure 46). This represents a decrease in the number of notifications relative to 1999 (323 cases, 1.7 per 100,000 population). The majority of notifications (55%) were reported in Queensland, followed by New South Wales (22%) and Victoria (14%). The highest rates of disease were localised to the Far North Statistical Division of Queensland (43.9 cases per 100,000 population) and the Western District of Victoria (13.2 cases per 100,000 population) (Map 9).

Figure 46. Trends in notification rates of leptospirosis, Australia, 1991 to 2000, by month of onset

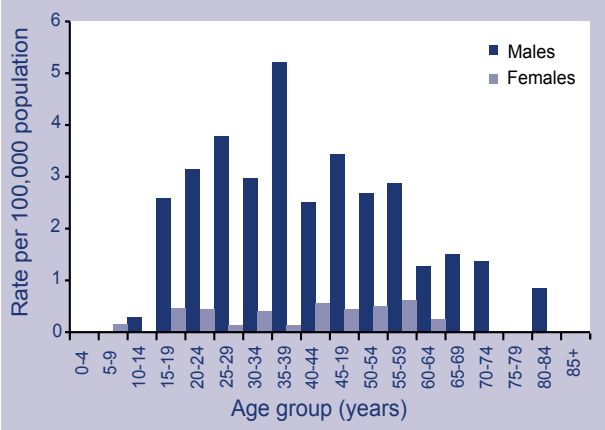


Map 9. Notification rates of leptospirosis, Australia, 2000, by Statistical Division of residence



The seasonal trends show two distinct peaks of leptospirosis in 2000. The first peak of 38 notifications occurred in May and the second peak of 33 notifications was in November (Figure 46). The majority of notifications were male, with a male to female ratio of 8.3:1. The highest age-specific rate for men was in those in the 35–39 year age group (5.2 cases per 100,000 population) (Figure 47).

Figure 47. Notification rates of leptospirosis, Australia, 2000, by age and sex



In early September, the Centers for Disease Control and Prevention (CDC) in United States of America alerted the DoHA to an outbreak of leptospirosis among participants of the Eco Challenge Race that was held in Sabah, Malaysia from 20 August to 3 September 2000. The event attracted a total of 304 athletes from 27 countries, which included 12 Australian athletes from New South Wales, Queensland, Tasmania and Victoria. The Surveillance and Epidemiology Section, DoHA, cooperated with the directors and epidemiologists of the health authorities in these jurisdictions to conduct the outbreak investigation. All of the athletes (except one who resided in the USA at the time of the investigation) were contacted by jurisdictional health authorities. Public health officers conducted interviews using a structured questionnaire collecting information on symptoms and possible exposures. The questionnaire was sent to the CDC via the Surveillance and Epidemiology Section. Blood samples were collected from some of athletes and sent to the Collaborating Centre for Reference and Research on Leptospirosis, WHO/FAO Western Pacific Region in Queensland.

There was no case of leptospirosis identified among the Australian participants in the overall investigation. During the international investigation, 158 (52%) the participating athletes were contacted by the CDC. Of the 158 respondents, 109 reported illness, including chills, myalgias, headache, diarrhoea, dark urine and arthralgias; 68 (44%) had illness that met the case definition of leptospirosis of the CDC.[80] The age of cases ranged from 22 to 50 years (median: 34 years) and 73 per cent were male. The median duration of illness was 6 days (range: 1–19 days) and 25 (34%) case-patients were hospitalised for the illness.[80]

An outbreak of leptospirosis was reported in the Northern Territory in November and December 2000.[81] Six cases of leptospirosis were notified to the Northern Territory Department of Health and Community Services. Two men and 3 women were involved in hunting during the time they acquired the disease. The third male case lived on a rural block where there are many animals and reported regularly going barefoot. In response to the outbreak a media release was issued by the Northern Territory health department, to highlight the risk for contracting this potentially fatal infection.

Other leptospirosis surveillance

The Collaborating Centre for Reference and Research on Leptospirosis, WHO/FAO Western Pacific Region report for 2000

This report summarises the leptospirosis notification data for 2000. The information has been collated from questionnaires distributed to all human cases in Australia and collected by the collaborating centre. In total, 204 cases of leptospirosis were investigated in 2000.

There was an elevated number of cases during the first 6 months of the year but not at the level reported in 1999. The increased number of notifications in the early part of the year reflects an increasing level of awareness of the disease among clinicians and a higher than average rainfall. The latter resulted in optimal conditions for the survival of the organism in the environment and favourable conditions for an increase in rodent populations.

Of the 204 cases, 179 reported illness, and the most frequently reported symptoms included headache (68.7%), followed by myalgia (60.9%), severe fever (57.0%), sweats (56.4%), chills (53.1%) and arthralgia (49.7%). The hospitalisation rate for leptospirosis remained high (56.3%) in 2000, and the average hospital stay for the patients was 5 days.

Data on occupation were available for 169 of the 204 leptospirosis cases. Animal associated occupations (63/169; 37%) and agricultural based occupations (61/169; 36%) accounted for the majority of the notifications nationally. The most frequently reported animal contacts were cattle (44.4%) and rats (38%). In Queensland however, agricultural related occupations were reported more commonly among the 115 leptospirosis cases (45.2%), specifically, banana farms accounted for 35.7 per cent of the total leptospirosis cases in Queensland.

Serovar information was recorded for 203 of 204 leptospirosis cases in 2000. Commonly identified serovars were *L. hardjo* (67/203; 33.0%), followed by *L. zanoni* (44 cases; 21.6%) and *L. australis* (37 cases; 18%).

The full report *Leptospirosis: surveillance report for 2000* can be found on the Queensland Health Website at: http://www.health.qld.gov.au/qhpss/qhss/lepto_report2000.htm

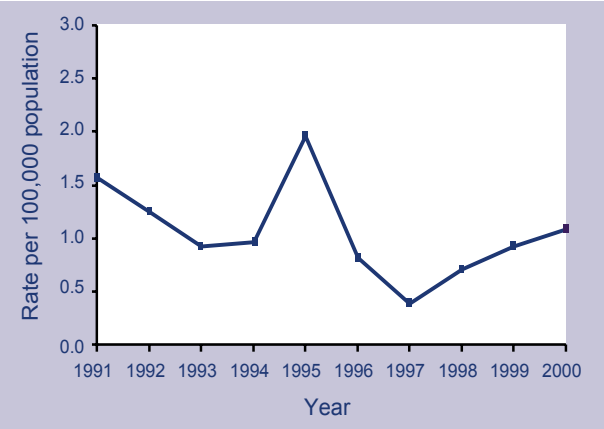
Ornithosis

Ornithosis, also known as psittacosis, is an acute generalised infection with *Chlamydia psittaci*. The disease in humans is commonly associated with exposure to birds, particularly parrots, although some studies showed an association with lawn mowing and gardening in areas with high numbers of native birds.[82] Shedding of *C. psittaci* into the environment by sick birds and subsequent inhalation of aerosolised dust and bird excreta may also lead to human infection.

In 2000, ornithosis was notifiable in all States and Territories in Australia, except New South Wales and Queensland. The NNDSS received 100 notifications of ornithosis in 2000, representing the third consecutive annual increase in the number of notifications since 1998 (Figure 48). The national notification rate was 1.1 cases per 100,000 population, and the majority (85%) of cases occurred in Victoria.

All but one case of ornithosis was linked to exposure to birds. The male to female ratio of disease was 2.6:1. The highest age-specific rates were reported in the 60–64 year age group for both men (7.0 cases per 100,000 population) and women (2.7 cases per 100,000 population). Reported rates of ornithosis are highest in the older age groups, which may reflect increased investigation and laboratory testing for atypical community acquired pneumonia in this group.[82]

Figure 48. Trends in notification rates of ornithosis, Australia, 1991 to 2000, by year of onset



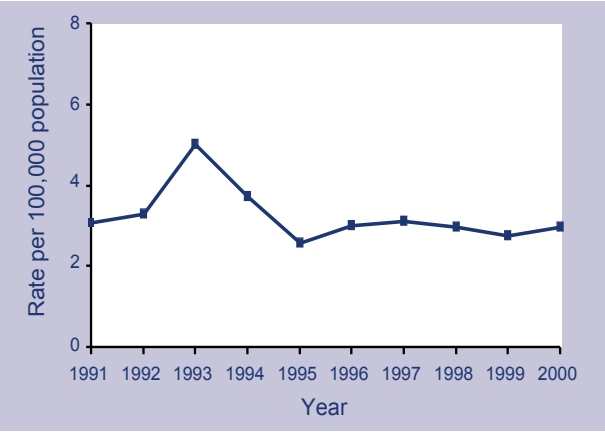
Q fever

Q fever is the most common zoonotic disease reported to the NNDSS in Australia. Q fever is a rickettsial illness caused by *Coxiella burnetii.* Livestock, such as sheep, cattle, goats, cats, dogs, some wild animals (bandicoots and many species of feral rodents), birds and ticks are natural reservoirs.[83] Risk occupations include stockyard workers, meat packing and rendering workers, abattoir and dairy workers, and medical and veterinary research facility workers.[84] An effective vaccine is available for Q fever in Australia[85] to protect the populations which are high at risk of this disease.

Transmission is usually through airborne dissemination of the organism in dust particles, through direct contact with contaminated material, ingestion of contaminated placentas or ingestion of milk. Ticks may also be involved in transmission of the organism. Cases have occurred in individuals with no direct contact with contaminated animals and their bodily fluids. These cases, however, have resided downwind from contaminated areas.[27]

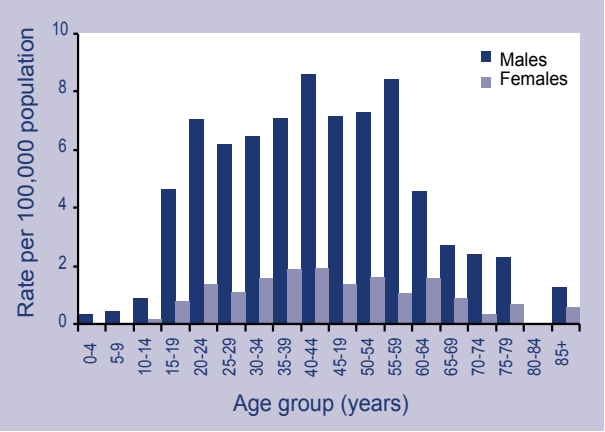
In 2000, 573 notifications of Q fever were reported to the NNDSS with an annual notification rate of 3.0 cases per 100,000 population, a slight increase from 2.7 cases per 100,000 population reported in 1999 (Figure 49). The majority of Q fever cases occurred in Queensland (68.1%), followed by New South Wales (22.7%). High notification rates were localised to the South West (250.0 cases per 100,000 population) and the Central West (164.8 cases per 100,000 population) of Queensland.

Figure 49. Trends in notification rates of Q fever, Australia, 1991 to 2000, by year of onset



The majority (92.8%) of the Q fever cases were adults in the 15–64 age range. The highest age-specific notification rates were in the 40–44 year age group for men (8.6 cases per 100,000 population) and in the 35-44 year age range for women (1.9 cases per 100,000 population) (Figure 50). The male to female ratio was 4.8:1. The true prevalence of the disease is likely to be under-estimated as the disease may be asymptomatic or self-limited.[86] Among 1,417 Australian abattoir staff tested for Q fever, 394 (27.8%) had serological evidence of exposure to Q fever.[87]

Figure 50. Notification rates of Q fever, Australia, 2000, by age and sex



Other bacterial infections

Legionellosis, leprosy, meningococcal infection and tuberculosis were notifiable in all States and Territories in 2000 and in the NNDSS are grouped as 'other bacterial infections'. A total of 2,121 notifications were classified as other bacterial infections in 2000, which accounted for 2.3 per cent of all notifications. Notifications of other bacterial infections reported to the NNDSS are shown in Tables 26 and 27.

Legionellosis

Legionellosis is an acute bacterial infection with two clinical manifestations: Legionnaires' disease and Pontiac fever. Legionellosis describes a group of diseases caused by various species of *Legionella* as well as the pneumonia of classical Legionnaires' disease caused by *Legionella pneumophila.*

L. pneumophila occurs in water sources, and can tolerate a wide range of temperatures, pH and dissolved oxygen contents. Depending on favourable temperatures, sediment accumulation and the presence of commensal microflora, the bacteria can proliferate in cooling towers and water systems, despite chlorination. Inhalation of aerosols containing the bacteria is the major mode of transmission. The risk of infection with *Legionella* is increased by age, chronic lung disease, immuno-suppression and cigarette smoking.[88]

L. longbeachae has been recognised for some years as a frequent cause of *Legionella* pneumonia in Australia.[89,90] A study found that 26 of 45 Australian potting soils were tested positive for *L. longbeachae*, suggesting this route of exposure may be important in the epidemiology of sporadic legionellosis in Australia.[91,92]

Legionellosis is notifiable in all the States and Territories in Australia, and includes notifications of infections caused by all *Legionella* species. There were 472 notifications of legionellosis in 2000 resulting in a notification rate of 2.5 cases per 100,000 population which has reached the highest level since 1991 (Figure 51). The seasonal trend showed a peak of 114 notifications in November 2000.

Table 26. Trends in notifications of other bacterial infections, Australia, 1991 to 2000*

Disease	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Legionellosis	119	208	170	178	161	208	157	262	249	472
Leprosy	14	20	17	10	10	7	12	3	6	4
Meningococcal infection	347	308	377	385	377	420	494	480	591	621
Tuberculosis	661	904	986	994	1093	978	989	960	1,143	1,024

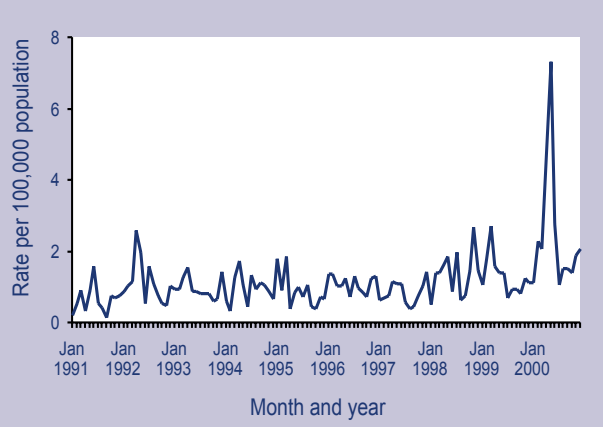
* All jurisdictions reputed for all years

Table 27. Trends in notification rates of other bacterial infections, Australia, 1991 to 2000 (rate per 100,000 population)*

Disease	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Legionellosis	0.7	1.2	1.0	1.0	0.9	1.1	0.8	1.4	1.3	2.5
Leprosy	0.1	0.1	0.1	0.1	0.1	< 0.1	0.1	< 0.1	< 0.1	< 0.1
Meningococcal infection	2.0	1.8	2.1	2.2	2.1	2.3	2.7	2.6	3.1	3.2
Tuberculosis	3.8	5.2	5.6	5.6	6.0	5.3	5.3	5.1	6.0	5.3

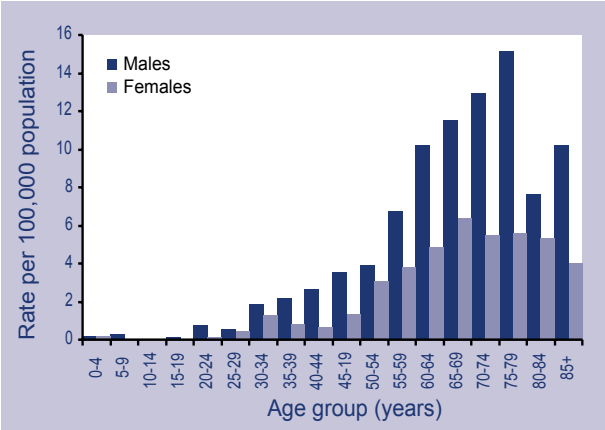
* All jurisdictions reputed for all years

Figure 51. Trends in notification rates of Legionellosis, Australia, 1991 to 2000, by month of onset



The reporting rates of legionellosis were highest in South Australia (5.9 cases per 100,000 population) and Victoria (5.2 cases per 100,000 population) (Tables 2 and 3). Men accounted for 65 per cent of reported cases. Cases occurred in almost all age groups, with a peak in the 75–79 year age group for men (15.1 cases per 100,000 population) and the 65–69 year age group for women (6.4 cases per 100,000 population) (Figure 52).

Figure 52. Notification rates of Legionellosis, Australia, 2000, by age and sex



Data on the causative species were available for 448 (95%) of the legionellosis cases. Of these, 311 (69%) cases were identified as *L. pneumophilia*, followed by *L. longbeachae* (131 cases, 29%), *L. micdadei* (4 cases) and *L. bozemannii* (2 cases).

In 2000, there was a total of 22 deaths as the result of legionellosis reported by States or Territories. Victoria reported 12 deaths (11 cases of *L. pneumophila* and 1 case of *L. micdadei*), South Australia reported 5 deaths (3 cases of *L. pneumophilia* and 2 cases of *L. longbeachae*), New South Wales reported 3 deaths (2 cases of *L. pneumophilia* and 1 case of *L. longbeachae*) and Western Australia reported 2 deaths (species data unavailable).

Six outbreaks of legionellosis were identified in 2000 and all occurred in Victoria.[93,94,95] Australia's largest outbreak of legionellosis to date occurred in Melbourne in April 2000, with a total of 125 confirmed cases (J. Greig, Victoria Department of Human Services, personal communication). The outbreak was linked to the newly opened Melbourne Aquarium. Of the 125 cases, 110 occurred in visitors to the aquarium between 11 and 27 April, and the remainder were in people who were within 500m of the building. During this time period 83,500 people visited the tourist attraction, giving a crude attack rate of 0.13 per cent.

The median age of cases was 64 years, and 57 per cent were male. Of the cases, 76 per cent were hospitalised for an average of 12.8 days, and 17 per cent of cases required admission to intensive care at some time during their hospital stay. The overall case fatality rate was 3.2 per cent, including 2 aquarium visitors and 2 people who were in the vicinity during the risk period. Most cases (83%) were diagnosed by urinary antigen test for *L. pneumophila* serogroup 1. Use of the urinary antigen test for early diagnosis of cases and rapid public health action probably contributed to relatively low morbidity and case fatality rates.

Of the remaining 5 outbreaks identified in Australia in 2000, four were in metropolitan Melbourne and one was in rural Victoria. A total of 28 cases were involved in these 5 outbreaks.[95]

Leprosy

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

There were 4 cases of leprosy notified nationally in 2000 compared with six in 1999. Two of the cases in 2000 occurred in New South Wales with one each in Queensland and South Australia. Of the 4 cases, one was male and three were female and the age range was 20–59 years. Information on country of birth was available for 3 cases, one was born in India, one in the Philippines and another in Viet Nam.

Invasive meningococcal disease

Neisseria meningitidis is the one cause of bacterial meningitis. Worldwide, invasive meningococcal disease accounts for at least 500,000 cases and 50,000 deaths per annum. Many of these occur in the sub-Saharan Africa 'meningitis belt'. Serogroup A is mainly associated with the pandemic of meningococcal disease in Africa.[96,97] Serogroups B and C are the major cause of both sporadic and epidemic meningococcal disease in industrialised countries, including Australia, while serogroups Y and W-135 are uncommon. New Zealand has experienced an on-going epidemic of meningococcal disease associated with serogroup B since 1991 which peaked at 16.9 cases per 100,000 population in 1997. The average rate for the period 1996 to 2000 was 13.9 cases per 100,000 population.[98]

Four distinct clinical situations are associated with meningococcal infection; asymptomatic nasopharyngeal colonisation, benign bacteremia, meningitis and meningococcemia. The organism is carried in the nose of up to 5–10 per cent of the population. A small minority of those colonised will progress to invasive disease. Meningococcal meningitis is the most common pathologic presentation, especially during epidemics. Meningococcal septicaemia is the most severe form of infection and has a high fatality rate.[99]

In Australia, there were 621 notifications of invasive meningococcal disease nationally in 2000. The annual notification rate of 3.2 cases per 100,000 population is the highest rate since 1991 (Figure 53). Of the total, 471 (75.8%) cases were culture-confirmed. Of these, 274 (58.2%) were serogroup B, 173 (36.7%) were serogroup C, 11 (2.3%) were serogroup W-135 and 13 (2.8%) were serogroup Y. Although serogroup B remains the predominant serogroup among the notifications, notifications of serogroup C have increased steadily during the period (Table 28).

Figure 53. Trends in notification rates of invasive meningococcal infection, Australia, 1991 to 2000, by month of onset

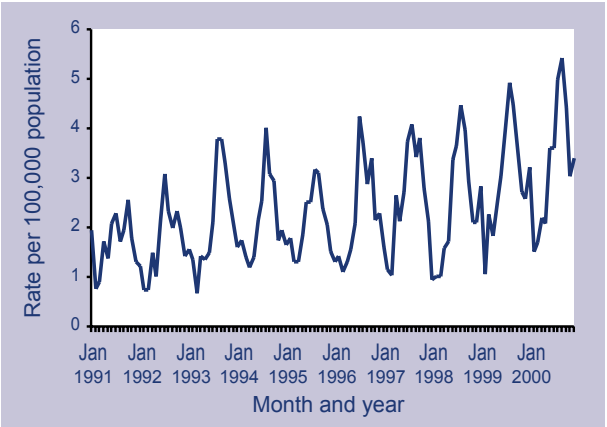
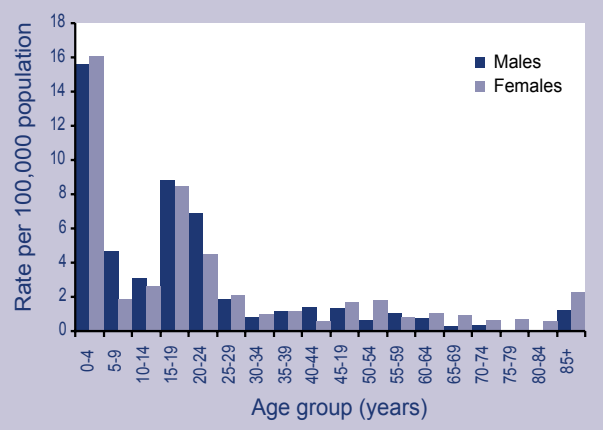


Table 28. Meningococcal notifications, Australia, 1995 to 2000, by serogroup

Year	B		C		Other or Unknown		Total
	n	%	n	%	n	%	
1995	31	8.1	15	4.0	335	87.9	**381**
1996	155	36.8	85	20.2	181	43.0	**421**
1997	96	19.8	57	11.8	331	68.4	**484**
1998	139	30.7	83	18.3	231	51.0	**453**
1999	212	37.3	143	25.2	213	37.5	**568**
2000	274	44.1	173	27.8	174	28.0	**621**

In 2000, the pattern of seasonal variation in meningococcal notifications continued, with the greatest number of cases occurring in late Winter or early Spring (Figure 53). The distribution of notifications by age shows the highest peak in children aged 0–4 years (15.8 cases per 100,000 population) and an additional peak in the 15–24 year age range (8.7 cases per 100,000 population) (Figure 54). The overall male to female ratio was 1.1:1.

Figure 54. Notification rates of invasive meningococcal infection, Australia, 2000, by age and sex



Forty-one deaths from meningococcal infections were reported in 2000, including 14 deaths in New South Wales, 13 deaths in Victoria, 6 deaths in Western Australia, 4 deaths in Queensland and 2 deaths in both South Australia and Tasmania. There were no major outbreaks of invasive meningococcal infection reported, and only three pairs of linked cases were identified.

The notification rate for meningococcal disease has been slowly, but consistently increasing over the past 10 years from 1.8 cases per 100,000 population in 1992 to 3.2 cases per 100,000 population in 2000 (Figure 53). It was suggested that the increase of meningococcal disease in Australia has been primarily due to the expansion of virulent phenotypes of serogroups B and C.[100,101,102,103] In addition, the case definition has been changed in some jurisdictions to include suspected cases and expanded laboratory diagnosis methods.[104] Despite rising public awareness and improvements in personal and environmental health measures, meningococcal disease remains the major life-threatening infection for children and adolescents in Australia.

Laboratory based meningococcal surveillance

The Australian Meningococcal Surveillance Programme annual report for 2000[105] summarised the phenotype and antibiotic susceptibility of *Neisseria meningitidis* from invasive cases of meningococcal disease. In 2000, a total of 388 isolates were examined by the National Neisseria Network laboratories, the highest number of isolates since the inception of the program in 1994.

Of the 388 isolates typed, serogroup B still predominated nationally (217 type B; 56% of total) and in all the jurisdictions, except Victoria. This was followed by serogroup C (143 isolates; 37% of total), serogroup Y (13 isolates; 3.2%) and serogroup W-135 (9 isolates; 2.3%). Serogroup C was the major serogroup in Victoria (58 isolates, 53.7% of total). Nationally the proportion of serogroup B of all strains was lower than in the previous 3 years. Phenotypes C:2a:P1.4(7),

C:2a:P1.2 and C:2a:P1.5 were first isolated in Australia in 1999. Phenotypes C:2a:P1.4(7) and C:2a:P1.2 were still commonly isolated in Victoria in 2000, but were occasionally encountered in other jurisdictions. Phenotype C:2a:P1.5 remained common in New South Wales. About two-thirds of all isolates showed decreased susceptibility to the penicillin group of antibiotics (MIC 0.06 to 0.5 mg/L). All isolates tested were susceptible to third generation cephalosporins and to the prophylactic agents rifampicin and ciprofloxacin.

In 2000, the number of non-culture diagnoses of invasive meningococcal disease increased to 147 cases from 92 cases in 1999. Of the 147 cases, 91 tested positive by PCR, 49 were positive by serology only and 7 tested positive by both PCR and serology.

Data on outcome (whether the patient survived or died) were available for 278 patients (71%). Of the 278, 25 (9%) patients died as a result of their infection. There were 13 deaths among cases with serogroup C infection, 9 deaths of serogroup B infection, 2 deaths of serogroup Y and 1 death of serogroup W-135.

Tuberculosis

There are three national surveillance systems for tuberculosis. The NNDSS provides the timeliest information on national TB notifications. The National Mycobacterial Surveillance System (NMSS), a surveillance system dedicated to tuberculosis and atypical mycobacterial infections, provides more detailed information on risk factors, diagnostic methods, drug therapy and relapse status.[106] The Australian Mycobacterial Reference Laboratory Network maintains national data on drug susceptibility profiles, site of disease, age, sex and laboratory method of diagnosis for all mycobacterial isolates. These data are published annually in conjunction with the NMSS surveillance report.[107]

In 2000, 1,024 TB notifications were received by the NNDSS, giving a reporting rate of 5.3 cases per 100,000 population. The highest rate was reported in the Northern Territory (22.0 cases per 100,000 population), followed by New South Wales (6.8 cases per 100,000 population) and Victoria (6.0 cases per 100,000 population).

There was little difference in notifications between the genders, with a male to female ratio of 1.1:1. While cases have occurred in all age groups, most cases occurred in the 20–24 year age group and older. The highest age-specific rates were in men in the 80–84 year age group (19.5 cases per 100,000 population) and in women in the 25–29 year age group (8.7 cases per 100,000 population).

Other communicable disease surveillance

Laboratory Virology and Serology Reporting Scheme

The Laboratory Virology and Serology Reporting Scheme is a passive surveillance scheme based on voluntary reports of infectious agents managed by the Commonwealth Department of Health and Ageing. LabVISE receives data from virology and serology laboratories around Australia. In 2000, reports from the scheme were analysed and published monthly in *Communicable Diseases Intelligence.*

LabVISE provides information on a number of viruses and other infectious agents (bacteria, parasites and fungi), and the demographic characteristics of persons they infect. The scheme records information on some infectious agents that are not reported by other surveillance systems. The database currently holds over 500,000 records collected since 1982.

LabVISE data interpretation is limited by uncertainties about the representativeness of the data, the lack of denominator data to calculate rates and variable reporting coverage over time. In addition, there are no consistent case definitions currently in use. For example, in 2000, there were 18 reports of Murray Valley encephalitis virus identification from Western Australia in LabVISE compared with 9 cases reported to the Western Australian health department. The LabVISE reports probably include positive screening test results from people without clinical disease, which falsely inflates the prevalence of clinical MVE disease.

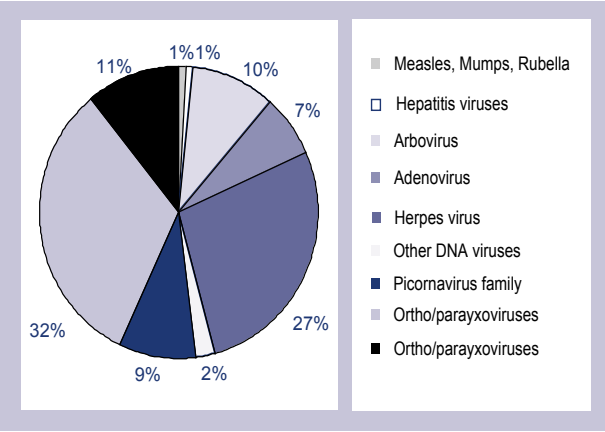
In 2000, 14 laboratories contributed 23,655 reports to LabVISE. This was a decrease of 10.6 per cent compared with the number of reports in 1999 (26,452). Although there were no contributing laboratories in either the Northern Territory or the Australian Capital Territory, samples from these jurisdictions were included in the reports from reference laboratories (Table 29).

The breakdown of LabVISE reports in 2000 is shown in Table 29. Of the 23,663 reports received, 17,337 (73%) were of viral infections and 6,326 (27%) were bacterial, spirochaetes, fungal, protozoan or helminthic infections. Ortho/paramyxoviruses (including influenza A and B, parainfluenza and respiratory syncytial virus) represented the most commonly reported group of viral infections, accounting for 32 per cent of viral reports. Reports of herpesviruses (including herpes type 6, cytomegalovirus, varicella-zoster and Epstein Barr virus) accounted for 27 per cent of viral reports (Figure 55). Chlamydia accounted for more than half (52%) of all reports of non-viral infections.

Table 29. Infectious agents reported to LabVISE, Australia, 2000

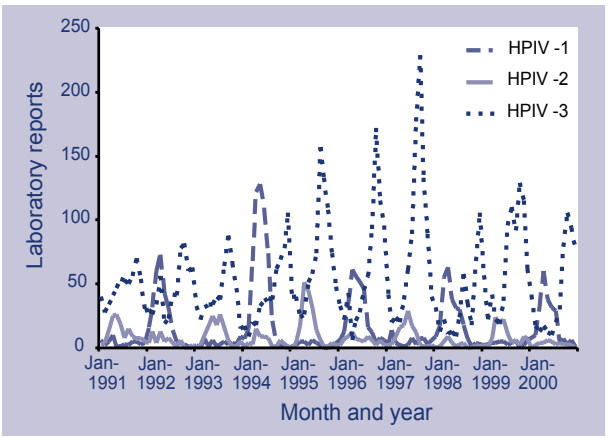
Organism	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Measles virus	3	4	0	0	8	0	19	10	**44**
Mumps virus	0	0	2	0	4	0	5	38	**49**
Rubella virus	1	6	3	16	7	0	10	8	**51**
Hepatitis A virus	2	4	11	28	29	0	3	69	**146**
Hepatitis D virus	0	1	0	3	0	0	4	1	**9**
Hepatitis E virus	0	0	1	0	0	2	0	1	**4**
Ross River virus	1	29	80	322	261	4	28	543	**1,268**
Barmah Forest virus	0	5	11	120	5	0	1	27	**169**
Dengue	1	4	62	1	0	1	0	112	**181**
Murray Valley encephalitis virus	0	0	2	0	0	0	0	18	**20**
Kunjin virus	0	0	1	0	0	0	0	3	**4**
Flavivirus (unspecified)	0	0	4	23	0	2	11	0	**40**
Adenovirus	8	162	9	15	378	6	192	435	**1,205**
Herpes virus	57	366	36	1,246	1,316	37	686	994	**4,738**
Other DNA viruses	9	6	5	78	37	2	79	198	**414**
Picornavirus family	12	369	10	26	30	7	582	491	**1,527**
Ortho/paramyxoviruses	89	1,272	12	239	1,234	48	761	1,949	**5,604**
Other RNA viruses	112	740	2	1	464	17	279	249	**1,864**
Chlamydia trachomatis	51	453	230	770	520	32	98	1,009	**3,163**
Chlamydia pneumoniae	30	6	0	0	0	0	0	0	**36**
Chlamydia psittaci	1	0	0	0	0	6	82	13	**102**
Mycoplasma species	3	49	13	203	128	4	207	87	**694**
Coxiella burnetii	1	8	0	34	11	0	24	23	**101**
Rickettsia species	0	0	1	0	0	6	4	11	**22**
Streptococcus group A	0	27	56	201	0	0	64	0	**348**
Yersinia enterocolitica	0	11	0	3	0	0	0	1	**15**
Brucella species	0	1	0	4	1	0	0	0	**6**
Bordetella pertussis	13	84	2	88	129	4	342	27	**689**
Legionella pneumophila	0	0	0	1	10	0	26	7	**44**
Legionella longbeachae	2	1	0	0	23	0	1	32	**59**
Legionella species	0	1	0	0	0	0	4	0	**5**
Cryptococcus species	0	6	0	0	11	0	1	0	**18**
Leptospira species	0	3	1	41	15	0	0	3	**63**
Treponema pallidum	0	68	222	262	331	0	0	27	**910**
Protozoa	1	2	0	5	5	0	13	7	**33**
Echinococcus granulosus	0	1	0	0	7	1	0	9	**18**
Total	**397**	**3,689**	**776**	**3,730**	**4,964**	**179**	**3,526**	**6,402**	**23,663**

Figure 55. LabVISE reports, Australia, 2000



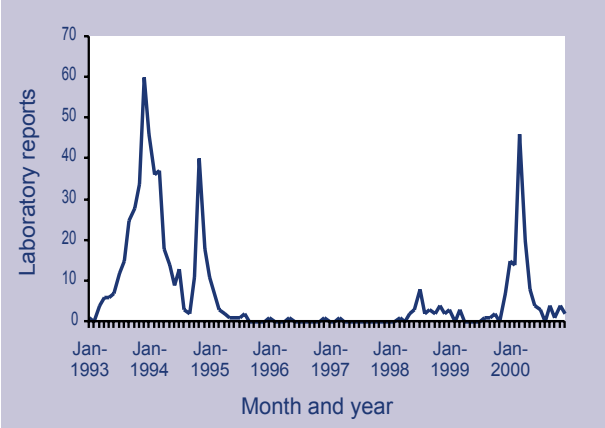
Parainfluenza viruses are an important cause of acute respiratory infection in infants and children. In March 2000, there was an outbreak of human parainfluenza type 1 (HPIV–1) in Australia. This is in keeping with biennial outbreaks of this parainfluenza strain in Autumn in Australia (Figure 56). Parainfluenza type 2 (HPIV–2) causes smaller outbreaks in alternate years to type 1, while there are annual outbreaks of parainfluenza type 3 (HPIV–3) annually in the Winter months. The majority of isolates (157/230, 68%) were from children aged 0–4 years.

Figure 56. Trends in laboratory reports of human parainfluenza virus strains 1, 2 and 3, Australia, 1991 to 2000, by month of report



Echovirus type 30 has caused large outbreaks of aseptic meningitis in many regions of the world in the past 40 years. LabVISE received 121 reports of echovirus 30 isolates in 2000. All but three of these were from New South Wales and Victoria. This is the first significant reporting of echovirus 30 to LabVISE since 1995 (Figure 57). Of the 121 cases 51 (42%) were in children under 10 years of age and 50/89 (56%) with diagnosis information were from individuals with a diagnosis of meningitis.

Figure 57. Trends in laboratory reports of Echovirus 30, Australia, 1991 to 2000 by month of report



An outbreak of pharyngoconjunctival fever occurred among school children in North Queensland in October 2000. Seven children who had attended a camp and presented with symptoms were investigated. Five of these children had positive viral cultures of adenovirus 3. An examination of the absenteeism rates at the school after the camp, however, suggested that 34 children had been infected. Fever, headache and sore throat were the most common symptoms.[108]

A review of LabVISE reports over the 10 years (1991 to 2000), examining data trends and quality will be published in the next issue of *Communicable Diseases Intelligence.*

Rotavirus Surveillance Programme 2000/2001[109]

A national rotavirus surveillance programme was commenced in June 1999 to undertake the surveillance and characterisation of rotavirus strains causing annual epidemics of severe diarrhoea in young children throughout Australia. Among 1,108 rotavirus isolates examined between June 2000 and May 2001, serotype G1 was the most common (49.5%) followed by serotypes G9 (18.1%), G2 (12.5%) and G4 (9.7%). Two outbreaks were detected, one of serotype G4 in Gove in the Northern Territory in September 2000 and another of serotype G9 in Alice Springs in May 2001.

Reports of the Australian National Polio Reference Laboratory[110,111]

Enterovirus testing of all cases of acute flaccid paralysis is an essential activity in the post-polio eradication era. The WPR, which includes Australia, was officially declared polio-free by the WHO in October 2000.

The Australian National Polio Reference Laboratory is responsible for processing and testing samples for poliovirus from all Australian patients with acute flaccid paralysis and for characterising polioviruses recovered from untyped enteroviruses submitted from Australian laboratories. Between 1 January and 31 December 2000, 35 specimens from 20 patients with acute flaccid paralysis were tested. Poliovirus type 3 Sabin-type was isolated from samples from 2 patients while the remaining samples from the other 18 patients were negative. In both AFP patients with positive culture for poliovirus, *Clostridium botulinum* toxin and/or other bacteria were detected in stool samples. The expert committee that reviews all cases did not consider polio to be the cause of the AFP.

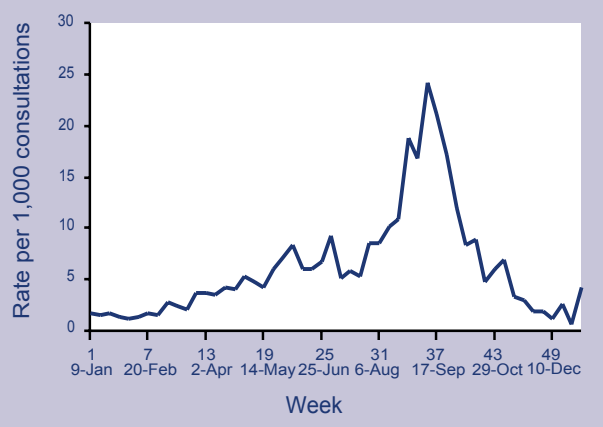
Australian Sentinel Practice Research Network

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

There were approximately 120 general practitioners participating in the scheme from all States and Territories in 2000. Approximately 75 per cent of these are located in metropolitan areas and the remainder are in rural areas. There were on average 5,000 consultations each week.

In 2000, 14 conditions were being monitored by the ASPREN management committee, and five of these conditions related to communicable diseases. These were influenza, chickenpox, gastroenteritis, gastroenteritis with stool culture and ADT immunisations. In total there were 392,896 consultations reported to ASPREN of which 9,005 (2.3%) were of communicable diseases related conditions. The majority of communicable diseases reported were gastroenteritis (4,000 presentations, 44% of the total), followed by influenza (2,481 presentations, 28%), ADT immunisations (2,005 presentations, 22%) and chickenpox (583 presentations, 6%) respectively. The weekly reporting of influenza as a rate per 1,000 consultations are shown in Figure 58. Presentations with symptoms of influenza-like illnesses peaked in the Winter months (week 36).

Figure 58. ASPREN Communicable disease surveillance presentations to GPs, 2000



National Influenza Surveillance Scheme

Based on Annual report of the National Influenza Surveillance Scheme 2000, (Communicable Diseases Intelligence 2000;25:107).

In 2000, influenza surveillance in Australia was based on three systems: laboratory diagnosis by virus isolation and serology from laboratories participating in LabVISE, consultation rates for clinically diagnosed influenza illness by sentinel general practitioners; and the absenteeism data of workers from a national employer. Sentinel general practice schemes were both State-based (in New South Wales, Victoria and the Northern Territory) and national through reporting to the Australian Sentinel Practice Research Network.

In 2000, participating laboratories of the LabVISE scheme reported a total of 1,916 laboratory isolations of influenza. These included 1,366 reports of influenza A and 550 reports of influenza B. The ratio of influenza A to B was 2.5:1. Total influenza reports showed a low level of activity until mid-June when there was an increase in reports to approximately 50 per week, followed by a major peak in mid-September, then a decline to baseline by late November. There were few temporal differences in the peaks of influenza A compared with influenza B activity through the year. The peak of influenza activity in 2000 was significantly later in the year than in 1999. The pattern in 2000 closely resembled that in 1997, when there was also a larger proportion of influenza B isolates (influenza A to B ratio of 1.5:1) and a later peak in disease reporting. The overall male to female ratio for influenza in 2000 was 1.2:1. The age and sex rates were highest among infants and children aged less than 5 years, with a second peak among men aged 70 years or more and women aged 75 years or more.

The Northern Territory Tropical Influenza Surveillance scheme data showed two peaks of influenza activity in March and October. The ASPREN data and that of New South Wales and Victorian sentinel schemes all showed a single peak in reporting in the week ending 17 September. Comparison of the ASPREN and LabVISE reports showed a similar pattern of activity, with the peak in laboratory reports one week later than that from general practitioner surveillance.

The WHO Collaborating Centre for Reference and Research on Influenza received a total of 1,116 influenza isolates of which 922 (83%) were suitable for analysis. Of these, 518 (56%) were influenza A (H3N2) subtype, 262 (28%) were influenza B and the remaining 142 (16%) were influenza A (H1N1) subtype.

The influenza A (H1N1) isolates were predominantly (73%) A/New Caledonia/20/99-like viruses with only 39 isolates characterised as A/Bayern/7/95-like. These two separate lineages of viruses have co-circulated in Australia for some time. Although 3 sporadic isolates of the A/New Caledonia lineage were isolated in 1999, this is the first year in which viruses of the lineage have been isolated in significant numbers in Australia. All but one of the 39 A/Bayern/7/95-like isolates came from an outbreak in South Australia.

The majority of the influenza A(H3N2) isolates (94%) were most closely related to the reference strain A/Moscow/10/99 and vaccine strain A/Panama/2007/99 and were distinguishable from the previous prototype and vaccine strain A/Sydney/5/97. Nevertheless, serological studies demonstrated that vaccines containing an A/Sydney/5/97-like strain used in the Australian 2000 Winter produced similar antibody responses to the Australian 2000 A (H3N2) isolates as vaccines containing an A/Moscow/10/99-like strain used in the 2000/2001 Northern Hemisphere Winter. Thus while some antigenic heterogeneity was observed in the influenza A (H3N2) isolates there was no evidence of significant antigenic drift beyond the A/Moscow/10/99 reference strain.

Influenza B strains isolated during the 2000 season showed a progressive drift away from the B/Beijing/184/93 strain. The majority (64%) were most closely related antigenitically to the new reference strain B/Sichuan/379/99.

In 1999/2000, there were a total of 2,591 admissions to Australian hospitals for influenza/pneumonia (Source: National Hospital Morbidity Database, 1990–2000: AIHW). Influenza virus was identified in 673 of these were cases. Altogether, influenza was responsible for 4,583 hospital patient days in 1999/2000.

Antibiotic resistance in Australia

The major event in the control of antibiotic resistance in Australia in 2000 was the publication of the *Commonwealth Government response to the report of the Joint Expert Technical Advisory Committee on Antibiotic Resistance*, August 2000 (the Government Response), which was endorsed by the then Minister for Health and Aged Care and the Minister for Agriculture, Fisheries and Forestries - Australia.

In October 1999, the JETACAR made 22 recommendations to the Commonwealth Government for an antibiotic resistance management program covering regulatory controls; monitoring and surveillance; infection prevention strategies; education; research; and communication. The Government Response acknowledges the threat from antibiotic resistant organisms to the health and economic prosperity of the Australian population, and supports the development of a national antibiotic resistance management program. The Government Response was to establish the Expert Advisory Group on Antimicrobial Resistance (EAGAR), to provide continuing advice on antibiotic resistance and related matters; and the Commonwealth Interdepartmental JETACAR Implementation Group (CIJIG) to oversee and coordinate the continuing government response to the JETACAR, to respond to the policy advice received from EAGAR; and to seek funding for implementation purposes.

CIJIG met for the first time in November 2000. CIJIG and EAGAR work collaboratively to further develop and implement the Government Response in consultation with stakeholders, including the States and Territories and industry, to ensure appropriate implementation. To assist formal consultation with the States and Territories and to monitor implementation, the Australian Health Minister's Conference (AHMC) appointed the AHMC JETACAR Taskforce in August 2000. To fill a similar role on the animal side and to aid consultation with industry, discussions are under way with the Department of Agriculture, Fisheries and Forestries — Australia to reactivate the Standing Committee on Agriculture and Resource Management JETACAR Taskforce.

As part of the implementation of the JETACAR recommendation DoHA commissioned a study of the current surveillance activities for healthcare associated infections. The National Surveillance of Healthcare Associated Infection in Australia study will provide vital information for future national planning for healthcare associated infection surveillance and inform public health action to alleviate the problem of antibiotic resistance. The report was made available for public consultation in early 2001 and was provided to the Australian Council for Safety and Quality in Healthcare for consideration and action.

Creutzfeldt-Jakob disease in Australia 2000

(Based on the report from the Australian CJD Registry, The University of Melbourne — update to January 2000)

The Australian Creutzfeldt-Jakob disease (CJD) Registry was established in October 1993 in response to the recognition of four probable human pituitary hormone related CJD deaths. The Registry is funded by the Commonwealth Department of Health and Ageing and is based in the Department of Pathology at the University of Melbourne. An inquiry into the use of human pituitary hormones under the Australian Human Pituitary Hormone Program suggested the expansion of some activities of the Registry.[112] These have been adopted, including retrospective case ascertainment from 1 January 1970. However, in parallel with monitoring possible iatrogenic cases of CJD, the Registry monitors all cases of transmissible spongiform encephalopathies, both sporadic and familial, within Australia. There is no systematic neuropathological or prion protein genetic assessment, but in line with non-domestic programs, evaluation of this type is now attempted in all prospective cases.

As of July 2001, Australia remains free of animal forms of prion disease, such as bovine spongiform encephalopathy and scrapie, and has no confirmed cases of variant CJD. As of the middle of 2001, there were 454 cases on the Registry, which included 237 definite cases, 168 probable cases and 49 incomplete cases (cases positive in an immunoassay but not finally classified).

In Australia, there has been a doubling of the average incidence of spongiform encephalopathies to one case per million since the mid-1980s. This mainly reflects case ascertainment bias due to improved recognition, confirmation and reporting. The composition of cases on the Registry is 90.4 per cent sporadic, 7.4 per cent familial and 2.2 per cent iatrogenic.

Appendices

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

Appendix 1a. Case definitions and mapping to ICD–10 code for notifiable diseases reported to NNDSS in 2000, bloodborne diseases

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

Appendix 1b. Case definitions and mapping to ICD–10 code for notifiable diseases reported to NNDSS in 2000, gastrointestinal diseases

Disease	Case definition (NHMRC 1994)	ICD–10 code(s)
Botulism	A clinically compatible illness (diplopia, blurred vision, muscle weakness, paralysis or bulbar palsy) with a history of exposure to a probable food source in the absence of a contaminated wound AND one of the following: isolation of *Clostridium botulinum* from faeces or other clinical specimens, *or* detection of *C. botulinum* toxin in serum, faeces or probable food source, *or* epidemiological linkage to other cases of confirmed foodborne botulism	A05.1
Campylobacteriosis	Isolation of *Campylobacter* species from a clinical specimen	A04.5
Haemolytic uraemic syndrome	Acute microangiopathic anaemia on peripheral blood smear AND acute renal impairment AND/OR thrombocytopaenia	D59.3
Hepatitis A	Anti-HAV IgM positive in the absence of recent vaccination OR demonstration of a clinical case of hepatitis (jaundice and/or elevated aminotransferase levels) without a non-infectious cause	B15
Hepatitis E*	A person who demonstrates anti-HEV IgM in sera collected less than 4 weeks after onset of acute hepatitis OR IgG seroconversion in paired sera OR HEV identified by nucleic acid test OR HEV identified by electron microscopy on stool OR a hepatitis-like illness in the absence of other causes of hepatitis and detection of antibodies to HEV	B17.2
Listeriosis	Isolation of *Listeria monocytogenes* from a site which is normally sterile, including foetal gastrointestinal contents	A32
Salmonellosis	Isolation of *Salmonella* species (excluding S. Typhi) from any clinical specimen	A02
Shigellosis	Isolation of *Shigella* species from any clinical specimen	A03
SLTEC, VTEC*	A person with bloody diarrhoea or HUS from whom in a clinical specimen: Shiga-toxin producing *E. coli* are isolated OR isolation of Shiga toxin from an *E. coli* isolate OR identification of the gene associated with the production of Shiga toxin in *E. coli*	A4.1, A4.4
Typhoid	Isolation of *Salmonella* Typhi or S. Paratyphi serotype A, B, or C from any clinical specimen	A01.1
Yersiniosis	Isolation of *Yersinia enterocolitica* or *Y. pseudotuberculosis* from blood or faeces OR detection of circulating antigen by ELISA or agglutination test OR positive *Yersinia* serology in the presence of clinical compatible illness	A04.6

Appendix 1c. Case definitions and mapping to ICD–10 code for notifiable diseases reported to NNDSS in 2000, quarantinable diseases

Disease	Case definition (NHMRC 1994)	ICD–10 code(s)
Cholera	An illness characterised by diarrhoea and/or vomiting AND isolation of toxigenic *Vibrio cholerae* serogroup 01 or 0139 from a clinical sample	A00
Plague	A fourfold or greater change in serum antibody titre for *Yersinia pestis* OR isolation of *Yersinia pestis* from a clinical specimen	A20
Rabies	Clinically compatible neurological illness AND *either* detection of viral antigens in tissue *or* isolation of rabies virus from saliva, skin snips, CSF or neural tissue	A82
Viral haemorrhagic fever	Sudden or insidious onset of fever, nausea, vomiting, diarrhoea, multifocal haemorrhages and shock. An appropriate travel history to an endemic country is supportive of diagnosis AND one of the following: demonstration of specific IgM antibody by ELISA, IFA or Western blot *or* isolation of the virus in cell culture *or* demonstration of viral antigen in a tissue specimen to Ebola virus, Lassa fever virus, Marburg virus or Crimean Congo virus.	A96, A98, A99
Yellow fever	A clinically compatible illness AND demonstration of yellow fever virus, antigen or genome in any clinical specimen OR a fourfold or greater change in serum antibody titre to yellow fever virus, OR a single elevated yellow fever specific IgM antibody titre, where cross-reaction with other flaviviruses has been ruled out and the patient has not received yellow fever vaccine during the previous 2 months	A95

Appendix 1d. Case definitions and mapping to ICD–10 code for notifiable diseases reported to NNDSS in 2000, sexually transmissible infections

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

Appendix 1e. Case definitions and mapping to ICD–10 code for notifiable diseases reported to NNDSS in 2000, vaccine preventable diseases

Disease	Case definition (NHMRC 1994)	ICD–10 code(s)
Diphtheria	Isolation of toxigenic *Corynebacterium diphtheriae* AND pharyngitis and/or laryngitis (with or without a membrane OR toxic (cardiac or neurological) symptoms	A36
***Haemophilus influenzae* type b**	An invasive clinically compatible illness (meningitis, epiglottitis, cellulitis, septic arthritis, osteomyelitis, pneumonia, pericarditis or septicaemia) AND *either* the isolation of *Haemophilus influenzae* type b (Hib) from blood *or* detection of Hib antigen (in a clinical case) or detection of Gram-negative bacteria where the organism fails to grow in a clinical case	A41.3, G00.0, J05.1
Measles	An illness characterised by all the following features: a generalised maculopapular rash lasting three or more days AND a fever (at least 38oC) AND cough or coryza or conjunctivitis or Koplik spots OR Demonstration of measles specific IgM antibody OR A fourfold or greater change in measles antibody titre between acute and convalescent-phase sera obtained at least 2 weeks apart, with tests preferably conducted at the same laboratory OR Isolation of the measles virus from a clinical specimen OR A clinically compatible case epidemiologically related to another case	B05
Mumps	Isolation of mumps virus from a clinical specimen OR significant rise in mumps antibody level by any standard serological assay, except following immunisation OR a clinically compatible illness (unilateral or bilateral swelling of the parotid or other salivary glands lasting 2 days or more without other apparent cause)	B26
Pertussis	Isolation of *Bordetella pertussis* from a clinical specimen OR elevated *Bordetella pertussis*-specific IgA in serum or *B. pertussis* antigen in a nasopharyngeal specimen using immunofluorescence with a history of clinically compatible illness	A37

Appendix 1e. Case definitions and mapping to ICD–10 code for notifiable diseases reported to NNDSS in 2000, vaccine preventable diseases continued

Disease	Case definition (NHMRC 1994)	ICD–10 code(s)
Poliomyelitis	Acute onset of a flaccid paralysis of one or more limbs with decreased or absent tendon reflexes in the affected limbs without other apparent cause, without sensory or cognitive loss	A80
Rubella	A generalised maculopapular rash and fever AND one or more of: arthralgia/arthritis *or* lymphadenopathy *or* conjunctivitis AND an epidemiological link to a confirmed case OR demonstration of rubella-specific IgM antibody, except following immunisation OR a fourfold or greater change in rubella antibody titre between acute and convalescent-phase sera obtained at least 2 weeks apart	B06
Tetanus	A clinically compatible illness without other apparent cause, with or without a history of injury and with or without laboratory evidence of the organism or its toxin	A33

Appendix 1f. Case definitions and mapping to ICD–10 code for notifiable diseases reported to NNDSS in 2000, vectorborne diseases

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

Appendix 1g. Case definitions and mapping to ICD–10 code for notifiable diseases reported to NNDSS in 2000, zoonoses

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

Appendix 1h. Case definitions and mapping to ICD–10 code for notifiable diseases reported to NNDSS in 2000, other bacterial infections

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

Appendix 2. Completeness of National Notifiable Diseases Surveillance System data received from States and Territories, 2000

Field	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
No. missing age	0	12	51	0	107	8	150	12	**340**
% complete for age	100.0	100.0	98.5	100.0	98.3	99.5	99.3	99.9	**99.6**
No. missing sex	3	64	13	3	1	6	307	23	**420**
% complete for sex	99.8	99.7	99.6	100.0	100.0	99.6	98.5	99.8	**99.5**
No. missing Indigenous status	567	16,727	331	18,829	966	1,251	18,669	3,896	**61,236**
% complete for Indigenous status	56.7	31.2	90.5	9.3	84.8	23.6	9.1	65.8	**31.8**

Appendix 3. Population totals for States and Territories, 2000*

ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
314,036	6,463,455	195,463	3,566,357	1,497,634	470,376	4,765,856	1,883,860	**19,157,037**

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

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Annual report of the National Influenza Surveillance Scheme, 2001

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Abstract

Surveillance of influenza in Australia in 2001 was based on data from national and state-based sentinel practice consultations for influenza-like illness, laboratory isolations of influenza virus and absenteeism rates from a national employer. In 2001, laboratory-confirmed influenza became a notifiable disease and was reported to the National Notifiable Diseases Surveillance System (NNDSS). Influenza A was the dominant type, 81 per cent of which were subtype H1N1 and 19 per cent were subtype H3N2. The influenza A (H1N1) analysed were all A/New Caledonia/20/99-like strains. The H3N2 isolates were antigenically similar to the reference strain A/Moscow/10/99 and the vaccine strain A/Panama/2007/99. The influenza B isolates, which made up only 10 per cent of all isolates, were mainly B/Sichuan/379/99-like strains but 10 per cent of isolates were more closely related to B/Harbin/7/94-like viruses, which circulated in previous years. The Australian 2001 influenza vaccine represented a good match for the circulating viruses and 77 per cent of persons over 65 years in Australia were vaccinated in 2001. *Commun Dis Intell* 2002;26:204–213.

Keywords: influenza, surveillance, vaccine, general practice, strain

Introduction

Influenza is an acute, self-limiting upper respiratory tract infection. Complications including lower respiratory tract infection (in particular secondary pneumonia and exacerbation of chronic obstructive pulmonary disease) and exacerbation of cardiopulmonary disease may occur.[1] Influenza-related morbidity (measured as excess hospitalisation) and mortality may result from these complications. Although influenza infection affects all age groups, the rates of serious morbidity and mortality tend to be highest among those aged 65 years and over, Aboriginal and Torres Strait Islanders and those with chronic medical problems. Young infants and pregnant women are also at increased risk of hospitalisation from influenza.

Influenza outbreaks usually occur during 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. Even though the complication rate may be low, the overall high attack rate during epidemics leads to a considerable increase in hospitalisations and mortality. In Australia in 1999, pneumonia and influenza accounted for 1,898 deaths (ICD–10 codes J10–J18; 1.5 per cent of all deaths, Australian Bureau of Statistics, 2001). Influenza pandemics occur every 10 to 30 years. During these pandemics, a quarter or more of the global population may be affected within a short period and the rates of illness and death from influenza can increase dramatically.

Influenza viruses are successful human pathogens because of their ability to vary their two external proteins, haemagglutinin (H) and neuraminidase (N). Mutations cause a gradual change in these proteins called 'antigenic drift', which results in annual epidemics of influenza. The greater the change in these proteins, the less likely it is that the virus will be recognised by immune cells primed by exposure to earlier infections or vaccines, and the greater the epidemic potential. At irregular intervals, there are more dramatic changes in the viral proteins, called 'antigenic shift', which are a result of either direct introduction of avian influenza viruses into the human population or a re-assortment between human and avian viruses which is believed to occur in intermediate hosts such as pigs. These 'shifts' result in the emergence of a new influenza virus. In the absence of immunity to these new viruses, there is rapid spread of influenza with dramatically increased rates of morbidity and mortality.

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After the pandemic of 1918 the H1N1 virus circulated widely in the human population until 1957. The Asian and Hong Kong pandemics in 1957 and 1968 introduced the H2N2 and H3N2 subtypes respectively, in each case replacing the previously circulating subtype of influenza A. There have been no major 'antigenic shifts' causing pandemics of influenza since 1968, however, the H1N1 subtype reappeared in the human population in 1977 and did not replace the H3N2 subtype. Since 1977, influenza A (H1N1), A (H3N2) and influenza B viruses have co-circulated and have been widespread globally, varying in frequency temporally and geographically.[2]

The formulation of influenza vaccines for use in Australia is determined annually by the Australian Influenza Vaccine Committee after review of the viruses circulating locally and internationally and after consideration of the World Health Organization (WHO) recommendations made in September. Influenza vaccination is provided free to non-Indigenous Australians aged 65 years and above and Indigenous Australians aged 50 years and above and is recommended for individuals with a range of underlying risk conditions, for pregnant women and for individuals who may transmit influenza to those with risk conditions.[3]

An effective national surveillance system is an essential component of a program for the control of influenza. Influenza surveillance is a mix of laboratory reporting of isolates and clinical diagnosis of influenza-like illness in sentinel practice schemes. Influenza surveillance aims 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. The major objectives of such surveillance include:

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

(ii) characterisation of the nature of the epidemic;

(iii) isolation and antigenic characterisation of circulating influenza viruses to assist in the formulation of the following season's vaccine; and

(iv) evaluation of the impact of the epidemic and associated public health measures.

This annual influenza report provides a summary of the surveillance methods and data for 2001.

Surveillance methods

Surveillance of influenza in Australia is based on six sets of data:

1. Notifications required by legislation to State and Territory health departments and nationally reported to the National Notifiable Diseases Surveillance System (NNDSS, from January 2001).
2. Laboratory diagnosis including virus isolation and serology by laboratories participating in the Laboratory Virology and Serology Reporting Scheme (LabVISE).
3. The WHO Collaborating Centre for Reference and Research on Influenza provides subtype data of influenza virus isolates forwarded by LabVISE laboratories.
4. Consultation rates for influenza-like illness diagnosed by sentinel general practitioners.
5. Absenteeism data of workers from a national employer.
6. Hospitalisation and mortality data.

National Notifiable Diseases Surveillance System

The Communicable Diseases Network Australia (CDNA) brings together communicable disease epidemiologists in all Australian States and Territories.[4] The CDNA has revised the list of diseases, to be notifiable across all jurisdictions. From January 2001, this included laboratory-confirmed influenza for the first time. Because some States needed to make legislative changes to make influenza a notifiable disease, complete reporting for influenza was not available from all States and Territories and no data were received from Tasmania.

Laboratory surveillance

The Laboratory Virology and Serology Reporting Scheme (LabVISE) is a national scheme of sentinel laboratories. In 2001, 16 laboratories contributed to this scheme, although not all provided reports each month. Laboratory reports of influenza are sent to LabVISE all year round. 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 the polymerase chain reaction (PCR) is now in use for diagnosis.[2]

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. This information is used to monitor the nature of influenza strains present in Australia and the rest of the world, to assess the suitability of the current vaccine (by measuring the degree of match between circulating strains and the current vaccine) and to determine the composition of vaccine for the following influenza season. Influenza viruses are named after the places where they were first identified. For example, A/Sydney/5/97 was first isolated in Sydney in 1997 and was influenza A isolate number 5 for that year.

The WHO Collaborating Centre for Reference and Research on Influenza conducts detailed antigenic analysis on all isolates received from Australian laboratories using conventional serological techniques. A geographically and temporally representative sample of isolates, together with any strains demonstrating uncharacteristic reactions during antigen characterisation were further analysed by genetic sequencing of the viral haemagglutinin antigen and, for a proportion of these, the neuraminidase antigen. Studies are also conducted with panels of pre-and-post vaccination human sera to determine the likely effectiveness of current vaccines against recently circulating viruses to provide data that assists in vaccine formulation decisions.

Sentinel general practitioner surveillance

Sentinel general practitioner surveillance schemes detect and record clinical diagnoses of influenza-like illness (ILI). The Australian Sentinel Practice Research Network (ASPREN) collects data at a national level. In addition, data are collected through the New South Wales Influenza Surveillance Scheme, the Victorian Influenza Surveillance Scheme, Western Australian sentinel general practices and the Northern Territory Tropical Influenza Surveillance Scheme.

Of sentinel general practices contributing to the ASPREN scheme, most are located in capital cities and larger regional centres, mostly on the east coast of Australia. In 2001, between 3,716 and 8,195 consultations were recorded each week. Participation is voluntary in all sentinel general practice surveillance systems, leading to variation in the number of contributors. In 2001, the number of contributing practices varied from 34 to 71 per reporting period.

The New South Wales Influenza Surveillance program collects clinical reports from New South Wales practitioners who are part of ASPREN and from three Area Health Services (AHS), two rural and one metropolitan (Southern AHS, North Sydney AHS and New England AHS). Reports were published weekly between 4 May and 28 September 2001. The total number of participating practices varied in 2001 from 6 to 37 and the number of consultations from 1,688 to 4,926 per week.

Table 1. Case definitions of influenza like illness used in different sentinel practice schemes

Program	Case definition
Victorian State program	Fever, cough, fatigue
Western Australia State program	Fever, cough, fatigue
New South Wales State program, Northern Territory and ASPREN	Six of the following criteria with sudden onset (<12 hours previously): cough, rigours or chills; fever; prostration and weakness; myalgia; redness of mucous membranes; influenza in close contacts.

(adapted from Watts and Kelly 2002 *Commun Dis Intell* 2002;26:8–12)

Absenteeism surveillance

Australia Post, a major nationwide employer, provided sick leave absenteeism data during 2001 between March and September. Absenteeism was defined as an absence due to illness for at least 3 consecutive days. Absenteeism was reported as the rate per 100 employees and rates were calculated on a weekly basis.

Hospitalisation data

To assess the impact of influenza on hospitalisation, the Australian Institute of Health and Welfare (AIHW) provide data on hospital separations and average length of stay in public and private hospitals. Information was accessed by ICD–10AM code that classifies influenza under two categories: cases of influenza where the virus is identified (J10) and cases where the virus is not identified (J11).

Results

The influenza surveillance data presented here are limited and should be interpreted with caution. Laboratory-confirmed influenza are a small proportion of all influenza cases in the year and consequently the estimation of the circulating strains is based on a small sample. Definitions of influenza-like-illness vary between sentinel practices (Table 1) which make comparisons of influenza incidence difficult. In addition, definitions of influenza-like illness have varied from year to year, so comparisons of data across years are complex.

National Notifiable Diseases Surveillance System

In 2001, 1,329 laboratory-confirmed cases of influenza were reported to the NNDSS. As noted above, not all jurisdictions submitted reports and the totals for some jurisdictions might represent less than 12 months data. The data collected are shown by jurisdiction in Table 2, along with the month from which influenza notifications commenced.

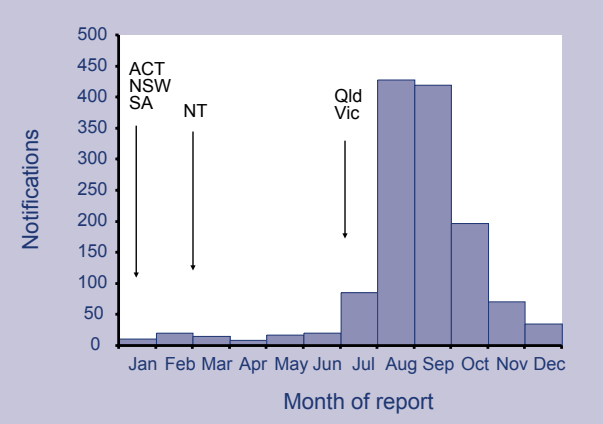
Table 2. Notifications of influenza to the National Notifiable Diseases Surveillance System, Australia, 1 January to 31 December 2001, by date of onset

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Notifications	15	282	95	392	135	NDR	174	236	1,329
Month when influenza notifications commenced	January	January	February	July	January	Did not report in 2001	July	January	

NDR: No data received. Updated as at 6 February 2002.

The notifications to NNDSS by month of report are shown in Figure 1. Notifications showed a peak in August (429 notifications) and September (420 notifications).

Figure 1. Notifications of laboratory-confirmed influenza, Australia, 2001, by month of report



The breakdown of laboratory-confirmed influenza cases to NNDSS by age and sex is shown in Figure 2. The overall male to female ratio for influenza in 2001 was 1.1:1. The age specific rates were highest among infants and children aged less than 5 years (32.1 cases per 100,000 population), 5 to 9 year olds (11.6 cases per 100,000 population) and among 80 to 84 year olds (7.9 cases per 100,000 population).

Laboratory surveillance

In 2001, a total of 1,076 laboratory diagnoses of influenza were made in participating laboratories of the LabVISE scheme. There were 869 reports of influenza A and 208 reports of influenza B, giving a ratio of influenza A to B of 4.2:1. This compared with 1,916 influenza reports to LabVISE in 2000, when the ratio of influenza A to B was 2.5:1. Total influenza reports showed a low level of activity until week 28 (15 July) when there was an increase in reports to approximately 35 per week, followed by a major peak of 64 reports per week in week 32 (12 August), then a decline to baseline (14 reports per week) by week 45 (11 November, Figure 3). There was a temporal difference in the peaks of influenza A (week 22) compared with influenza B (week 40) activity through the year. The peak of influenza activity in 2001 was earlier in the year than in 2000 (Figure 4).

The seasonal pattern of influenza A and B activity between 1996 and 2001 is shown in Figure 5. The pattern in 2001 closely resembled that in 1999 with a relatively small number of influenza B isolates and a large A:B ratio.

Figure 2. Notification rates of laboratory-confirmed influenza, Australia, 2001, by age and sex

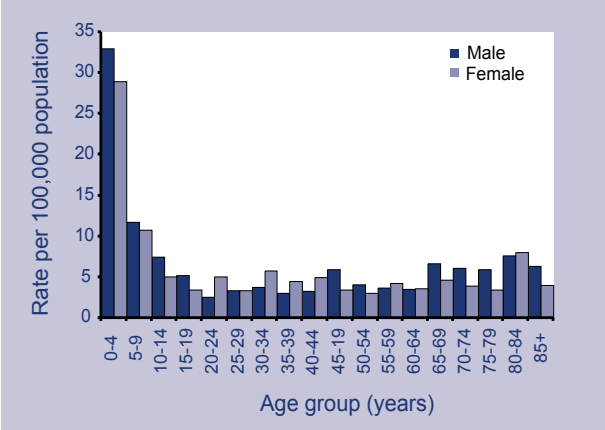


Figure 3. Laboratory reports of influenza, Australia, 2001, by type and week of specimen collection

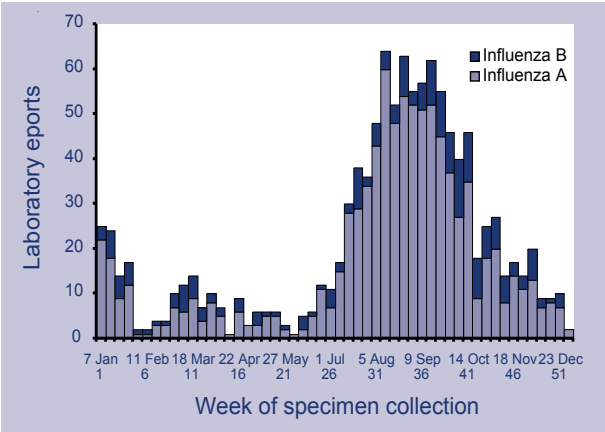


Figure 4. Laboratory reports of influenza, Australia, 2000 and 2001, by month of specimen collection

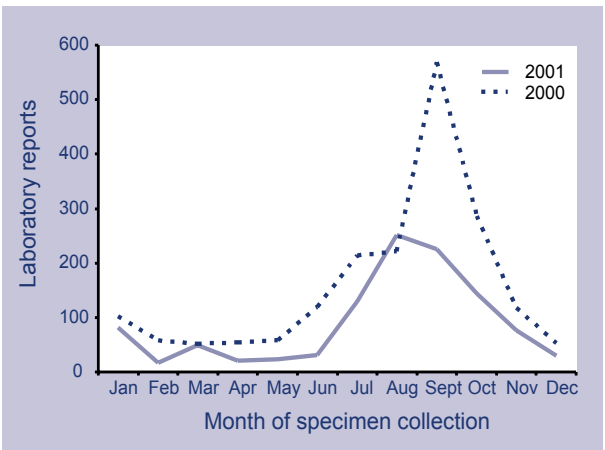
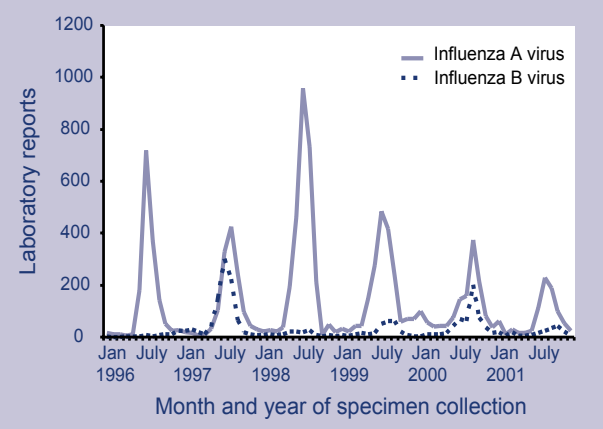


Figure 5. Laboratory reports of influenza, Australia, 1996 to 2001 by type and month of specimen collection



WHO Collaborating Centre for Reference and Research on Influenza

The Centre received 615 viable influenza isolates that could be analysed antigenically, approximately half the number received in 2000. Of these isolates 441 (71.7%) were influenza A(H1N1) subtype, 103 (16.7%) were A(H3N2), and 71 (11.5%) were influenza B. The variable region of the haemagglutinin antigen was sequenced in 83 (13%) isolates and the neuraminidase antigen was sequenced in 32 (5%) of the isolates. All of the A(H1N1) viruses were A/New Caledonia/20/99-like strains. Genetic analysis of these showed only minor changes from the reference strain. Viruses of the second A(H1N1) lineage (i.e. A/Bayern/7/97-like strains) were not seen among the Australian isolates or in isolates received from the Asia-Pacific region.The A(H3N2) isolates were antigenically similar to the reference strain A/Moscow/10/99 and the vaccine strain A/Panama/2007/99. However, as is usually observed for this subtype, there was some antigenic and genetic heterogeneity (Figure 6). There was no evidence of emergence of a representative new antigenic variant and antibodies induced in vaccine recipients by the current A/Panama vaccine strain were of similar titre and frequency to viruses isolated throughout the year as to those against the vaccine virus. The neuraminidase antigen showed some evidence of genetic but not antigenic drift.

The Australian influenza B isolates were mainly B/Sichuan/379/99-like strains however, approximately 10 per cent of isolates were more closely related to older influenza B strains such as B/Harbin/7/94 which circulated previously (Figure 7). Antisera prepared against B/Sichuan/379/99-like strains, and post-vaccination sera from people receiving B/Sichuan-like vaccine, reacted strongly with these B/Harbin-like viruses. None of the Australian influenza viruses belonged to the more distinct influenza B lineage (often referred to as the B/Victoria/2/87 lineage), which has persisted in Asia and has been seen in some other regions recently.

Based on antigenic and genetic analysis, and post-vaccination human serology of the Australian 2001 vaccine represented a good match for the circulating viruses.

Figure 6. Evolutionary relationships between influenza H3 haemagglutinins (HA1 region)

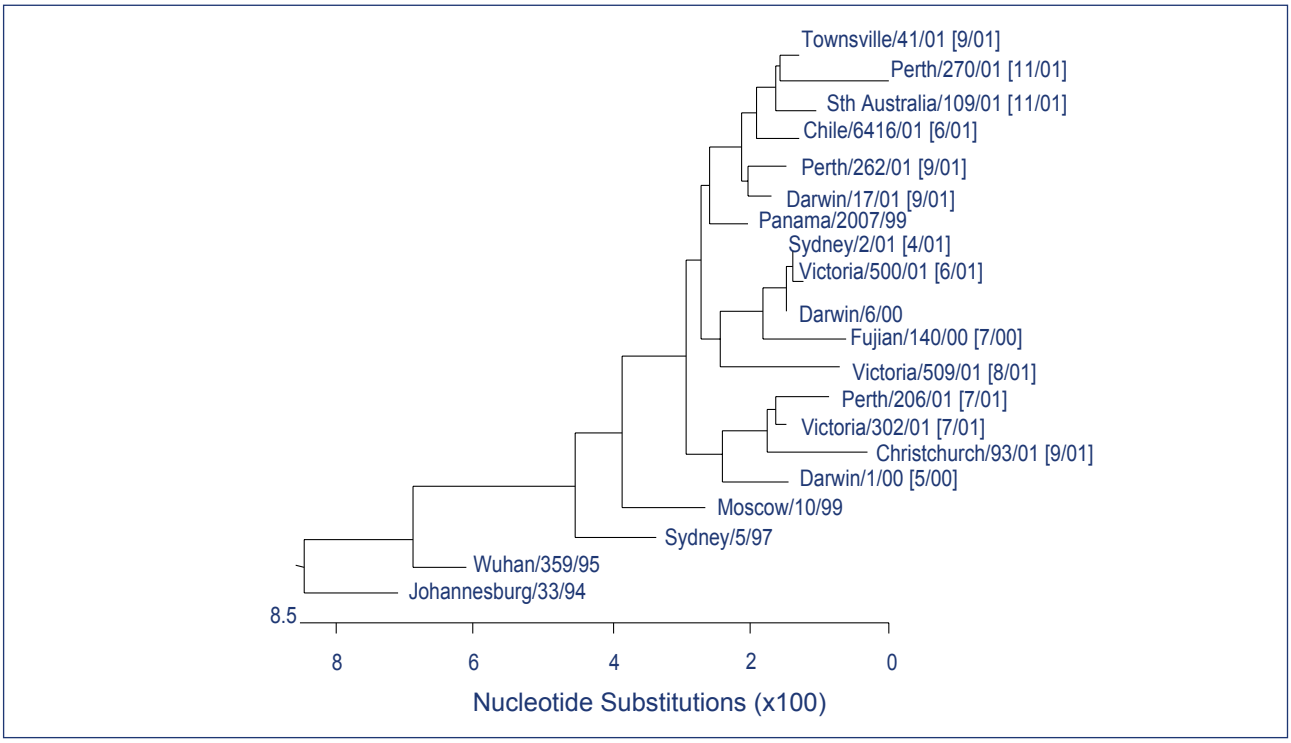
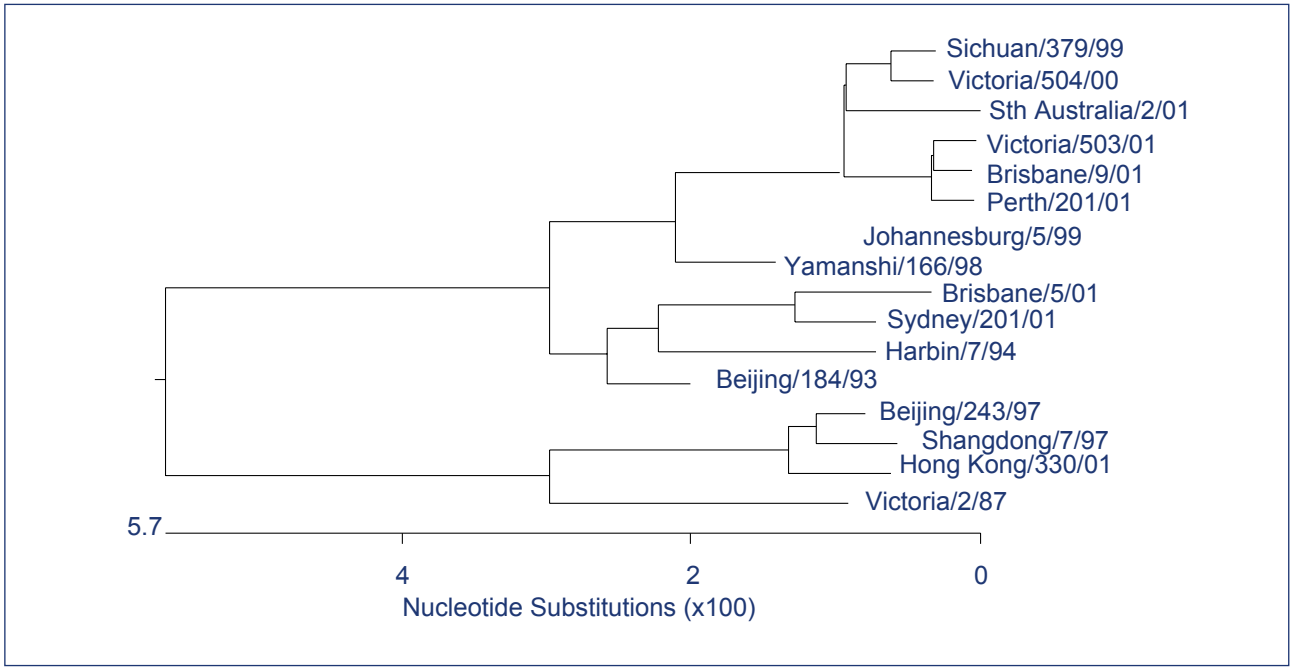


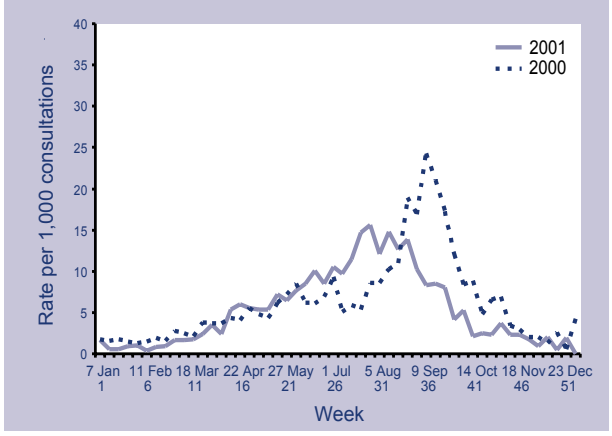
Figure 7. Evolutionary relationships between influenza B haemagglutinins (HA1 region)



Sentinel general practice surveillance

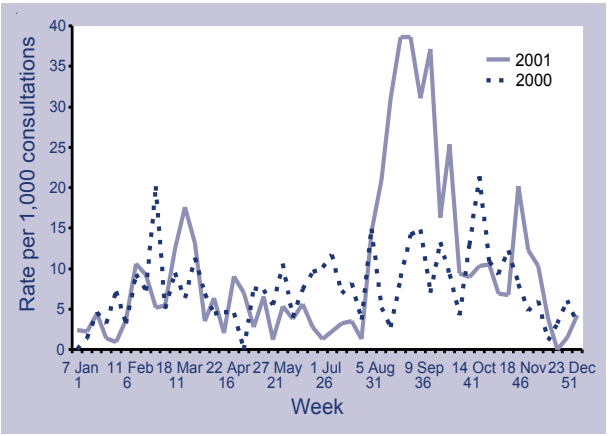
Reports of influenza-like illness to ASPREN practice sites showed a peak in week 30 (29 July, Figure 8) when reports reached a rate of 15.6 cases per 1,000 consultations. This peak in activity was lower and earlier in the year than the peak reporting in 2000 (24.3 cases per 1,000 consultations in week 36, 9 September).

Figure 8. ASPREN consultation rates for influenza-like illness, Australia, 2000 and 2001, by week of report



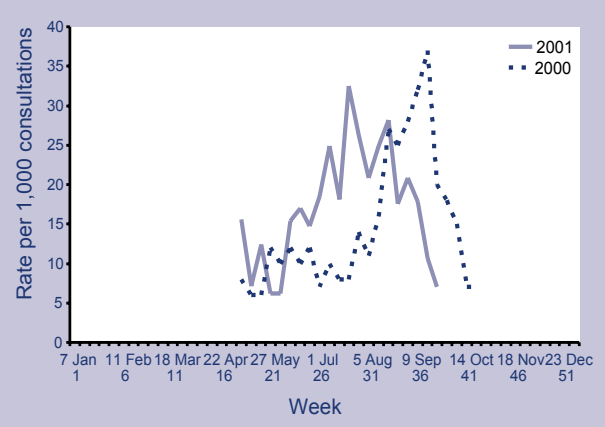
The Northern Territory Tropical Influenza Surveillance Scheme data showed two peaks of influenza activity in week 12 (25 March, 17.6 cases per 1,000 consultations) and weeks 34 and 35 (26 August to 2 September; 38.6 cases per 1,000 consultations). The influenza activity in the latter part of 2001 was greatly increased compared with 2000 (Figure 9).

Figure 9. Consultation rates for influenza-like illness, Northern Territory, 2000 and 2001, by week of report



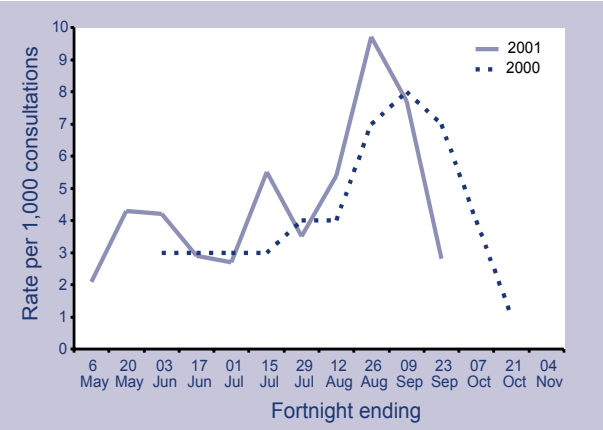
In New South Wales, influenza-like illness reports peaked in week 29 (22 July) at 32.5 cases per 1,000 consultations (Figure 10). In contrast with reports in 2000, the peak activity of influenza in New South Wales was earlier and lower – the peak in 2000 was in week 37 (16 September) at 37 cases per 1,000 consultations.

Figure 10. Consultation rates for influenza-like illness, New South Wales, 2000 and 2001, by week of report



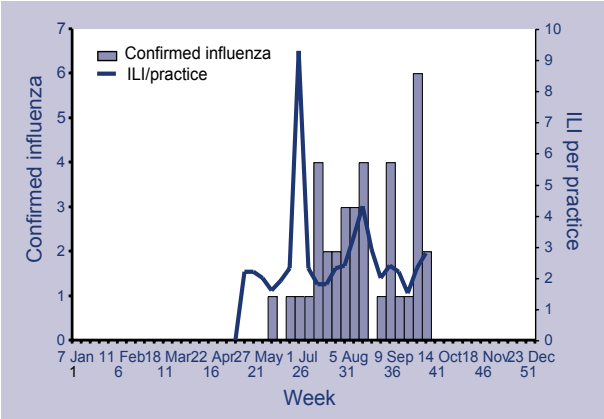
In Victoria, the reporting rate of influenza-like illness peaked at 9.7 cases per 1,000 consultations in the fortnight ending 26 August (Figure 11). The rate was higher in Victoria in 2001 and occurred earlier in the year than in 2000 (peak reporting in early September at 8 cases per 1,000 consultations).

Figure 11. Consultation rates for influenza-like illness, Victoria, 2000 and 2001, by fortnight of report



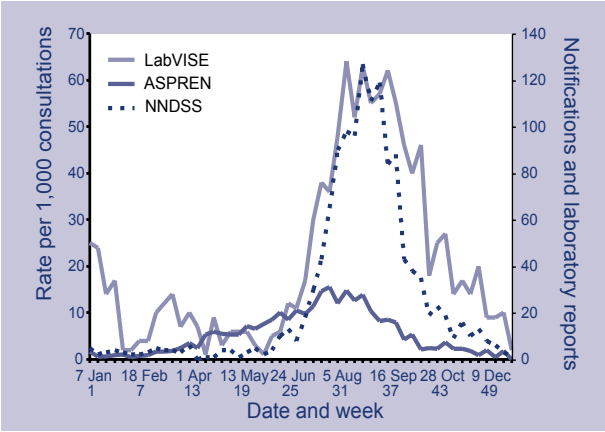
In Western Australia, the peak of reporting of influenza-like illness occurred in week 30 (29 July) at 9.3 cases per practice (Figure 12).

Figure 12. Consultation rate for influenza-like illness and laboratory reports of influenza, Western Australia, 2001, by week of report



A comparison of the NNDSS, ASPREN and LabVISE reports is shown in Figure 13. The peak in reports of influenza-like illness to ASPREN in week 30 preceded the peak of laboratory reports of influenza to LabVISE in week 32, which preceded the peak of notifications to NNDSS (week 34).

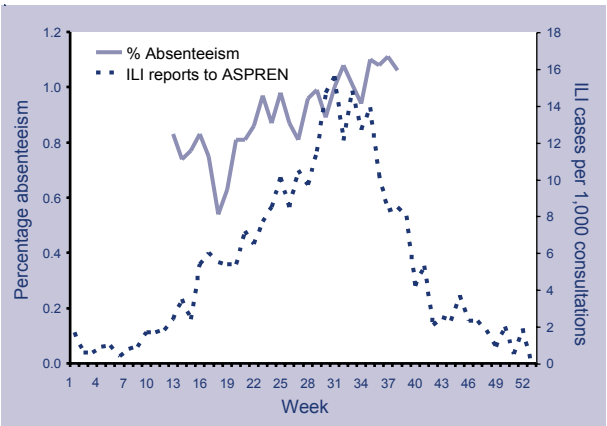
Figure 13. Laboratory reports to LabVISE, notifications to NNDSS and consultation rates in ASPREN of influenza, Australia, 2001, by week of report



Absenteeism surveillance

There was little evidence of any association between absenteeism and the peak in influenza activity in the data supplied by Australia Post. Data were only available up to week 37. Absenteeism was highest at 1.11 per cent in week 36 (Figure 14).

Figure 14. Rates of absenteeism and consultation rates for influenza-like illness, Australia, 2001, by week of report



Hospitalisation due to influenza

There were no data on hospitalisations or deaths due to influenza for 2001 available at the time of writing this report.

Discussion

In 2001, influenza activity in Australia was at low to moderate levels as assessed by all surveillance systems compared with activity in 2000. While influenza activity was moderate in temperate regions of Australia, a large outbreak was reported from the Northern Territory late in 2001. Two areas of the Northern Territory were affected — urban Alice Springs and an island off the north coast. While both areas reported a mixture of influenza types, influenza B was more common in Alice Springs and influenza A was more common on the island (Peter Markey, CDC Darwin, personal communication).

Overall, the influenza viral strains circulating in Australia in 2001 were similar to those seen in the previous season and were well matched to the strains in the 2001 vaccine. The proportion of influenza A H1N1 among circulating strains in Australia further increased in 2001, continuing a trend seen elsewhere in the world since 2000. The emergent A(H1N1) strains were A/New Caledonia/20/99-like. Influenza epidemics which are predominantly A(H1N1) have been shown to cause a lower rate of hospitalisation than epidemics which are predominantly A(H3N2).[5] The number of viral isolates analysed in 2001 were half that analysed in 2000. A larger, more representative sample of influenza isolates should be analysed so that surveillance of circulating strains includes infrequent variant strains.

The 2001 to 2002 Northern Hemisphere influenza season was late to commence and severity has been generally moderate. Reporting from the United States of America up to mid-January 2002 showed low influenza activity nationwide, although influenza activity normally peaks in February.[6] In contrast with the 2001 Australian influenza season, the majority of the circulating viruses in the Northern Hemisphere were H3N2 and were well matched by the vaccine strain.

Two variant influenza strains were noted in the Northern Hemisphere 2001 to 2002 influenza season.

Firstly, the emergence of some influenza B/Hong Kong/330/2001-like strains (B/Victoria/2/87 lineage) occurred in Europe. Viruses of this lineage are antigenically distinct from B/Sichuan/379/99 and during the past decade had circulated only in Asia. The current B/Sichuan/379/99-like vaccines induce antibodies that react poorly with the B/Hong Kong/330/2001-like strains. Secondly, a substantial number of genetic reassortant A(H1N2) viruses carrying an A/New Caledonia/20/99-like haemagglutinin and an A/Panama/2007/99-like neuraminidase were identified. Although A(H1N1) and A(H3N2) viruses have co-circulated in the human population since 1977, and genetic reassortment of influenza viruses occurs rather readily during mixed infections, viruses carrying mixed surface antigens from the two subtypes have been rather rare. Current vaccines and diagnostic reagents remain appropriate for the reassortant viruses.

Australian national influenza surveillance continues to rely on a number of different sources of data from independently operating schemes with distinct case definitions and surveillance practices. This complicates the analysis of national influenza activity and trends, as the data from different schemes are not always comparable. A survey of the detection of influenza-like illness in sentinel practice schemes in Australia has been recently published.[7] This study based on a telephone survey in August 2001, found major differences in the definitions of ILI used by different sentinel schemes (Table 1) and variable access to, or use of laboratory testing. These differences confound attempts to compare influenza activity as measured by ILI rates in different parts of Australia. There is a need to standardise clinical definitions of ILI and to measure the sensitivity and specificity of ILI definitions relative to the diagnosis of influenza by laboratory techniques.

Laboratory-confirmed influenza is now a nationally notifiable disease in Australia. This means there is a legal obligation to report laboratory-confirmed cases from all Australian medical practices, hospitals and laboratories to State and Territory health departments. These in turn have agreed to send data to the National Notifiable Diseases Surveillance System. Up to 2001, data on laboratory-confirmed influenza cases were reported through the Virology and Serology Reporting Scheme, a sentinel reporting scheme in which a group of laboratories voluntarily report on laboratory diagnoses of influenza. In 2001, the number of notifications to NNDSS (1,329) was greater than laboratory reports to LabVISE (1,076). NNDSS data are more representative than LabVISE as NNDSS reports come from a larger number of laboratories. The NNDSS dataset has been undergoing extensive revisions and from 2002, a larger set of data will be reported on each case. For cases of influenza, it will be possible to record the virus type and (if available) the virus strain, and whether the case was vaccinated. In addition, it may be possible to identify cases linked in an outbreak.

Preparations for an influenza pandemic in Australia continued in 2001 with the development of the Australian action plan for pandemic influenza. The pandemic plan, under consideration by CDNA in February 2002, focuses on the responsibilities of the Commonwealth and the States and Territories in the phases of an influenza pandemic.[8] The main priority in a pandemic will be to minimise morbidity and mortality. It will be essential to enhance influenza surveillance, ensure maintenance of health care and essential services, and implement a communication strategy for timely dissemination of information throughout the health care system and community in general. The action plan lists the main activities to be considered for these issues and the responsible agencies. It also recommends preparation of State and Territory action plans to increase coordination at all levels.

Influenza vaccination of vulnerable populations such as the elderly is an important public health activity to reduce the morbidity and mortality of annual influenza epidemics. The National Health and Medical Research Council recommends annual influenza vaccination for all Australians aged over 65 years and influenza vaccine is available free to Australians over 65 years. A national telephone survey conducted in October and November 2001 showed 77 per cent of Australians aged over 65 years received influenza vaccination in 2001. This is an increase of 3 per cent of the vaccination rate in this age group in 2000. The vaccination rates in Australians aged 65 years or more with a chronic disease reached 84 per cent in 2001.[9] The survey reported that under the National Influenza Vaccine Program, 89 per cent of the respondents in this age group had received the vaccine through the program.

The Australian 2002 influenza vaccine is composed of an A/New Caledonia/20/99(H1N1)-like strain, an A/Moscow/10/99(H3N2)-like strain and a B/Sichuan/379/99-like strain. Although the strain composition is the same as for vaccines issued in 2001, immunity to influenza vaccine is of limited duration and should be reinforced annually regardless of vaccine strain composition. Influenza vaccines have a limited shelf life and are intended for use during the year that they are distributed.

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Tuberculosis notifications in Australia, 2000

Ming Lin, Jenean Spencer, Paul Roche, Moira McKinnon and the National TB Advisory Committee (Ral Antic - Chair, Ivan Bastian , Amanda Christensen, Mark Hurwitz, Anastasios Konstantinos, Vicki Krause, Avner Misrachi, Graham Tallis, Justin Waring) for the Communicable Diseases Network Australia

Abstract

Australia has one of the lowest incidences of tuberculosis (TB) in the world. The annual incidence rate has remained stable at between 5 and 6 per 100,000 population, since 1991. In 2000, there were 1,060 TB notifications in Australia, of which 1,004 were newly diagnosed cases and 56 were relapse cases. The corresponding incidence rate for new and relapsed TB was 5.2 and 0.3 cases per 100,000 population, respectively. The highest incidence of TB disease in Australia continues to be among the overseas-born (18.0 per 100,000 population) and Indigenous Australians (15.3 per 100,000 population). By contrast, the incidence of disease in the non-Indigenous Australian-born population remains low (1.2 per 100,000 population). *Commun Dis Intell* 2002;26:214-225.

Keywords: tuberculosis, Mycobacterium tuberculosis

Introduction

Tuberculosis is a global public health threat. In 2000, there were an estimated 8.7 million new cases of TB worldwide, an increase from 8.4 million in 1999. In 2000, 3.7 million cases (42% of all estimated TB cases) were reported to the World Health Organization (WHO) Global Surveillance Programme, by 202 countries. Directly Observed Treatment – Short course (DOTS) is now used for 55 per cent of the world's TB patients in 148 countries.[1]

Although the global case notification rate for TB has remained stable since 1980, there are differences in the epidemiology of the disease in different regions. While TB incidence has been increasing in central and eastern Europe (8% per annum) and in eastern and southern African countries affected by HIV (10% per annum), TB is declining in developed countries.[1]

Poorly supervised and inadequately treated TB cases are the basis for the emergent problem of multi-drug resistant TB (MDR-TB). The status of drug resistant tuberculosis in Australia in 2000 is addressed in the TB laboratory annual report in this issue of *Communicable Diseases Intelligence*.[2]

A majority of the world's TB cases occur in South East Asia and the Western Pacific regions, geographically adjacent to Australia. Australia has maintained a low and stable rate of TB through effective pre-migration screening and the activities of specialised, multi-disciplinary TB services in the States and Territories.

The National Mycobacterial Surveillance System (NMSS), established in 1991, has monitored trends in the national rates of TB over the last 10 years. Future enhancements to the national TB surveillance system, such as use of a common database to facilitate timely collection of data from States and Territories will serve to better inform policy makers, public health practitioners and clinicians.

Methods

Data collection

In Australia, all jurisdictions have legislation requiring medical practitioners, laboratories and other health officials to report cases of TB to State and Territory health authorities. Notifications reported to State and Territory health authorities are collated on an annual basis and sent to NMSS in de-identified computerised format. Data fields include a unique identifier for each notification, disease code, postcode of residence, date of birth, sex, Indigenous status, dates of disease onset and report, country of birth, length of residence in Australia for overseas-born persons, species of the pathogen, principal site of disease, methods of diagnosis, antimicrobial therapy initiated at the time of notification, past Bacille Calmette Guerin (BCG) vaccination, HIV status and classification of TB as new or relapsed disease. The National Tuberculosis Advisory Committee (NTAC) of the Communicable Diseases Network Australia (CDNA) is responsible for determining what data are collected.

Data processing and quality control

TB notifications reported in 2000 were received by September 2001. The data were checked for completeness and accuracy. Missing data and apparent errors together with any queries arising from the data were returned to jurisdictions for review, correction of errors and ascertainment of completeness of case information for the year.

A number of data quality factors may affect the usefulness of the data, including the percentage of cases notified and the completeness and accuracy of data. In Australia, most cases of TB are thought to be reported to the notification system. The reason for this achievement includes an effective TB screening program, a high standard health care system (the Australian health care system provides free TB treatment to both Australians and non-Australian residents), and the activities of specialised, multi-disciplinary TB services in the States and Territories.

Case definition

The case definitions for tuberculosis used in Australia in 2000 are described below:

Tuberculosis (new case)

> A case which has been confirmed by the identification of *Mycobacterium tuberculosis* (or *M. africanum* or *M. bovis*) by smear, culture.
>
> or
>
> A case which has been diagnosed to be active clinically and which has been accepted as such by the State or Territory Director of Tuberculosis.

Tuberculosis (relapse)

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

Population estimates for 2000

The rates used in this report have been calculated using population figures provided by the Australian Bureau of Statistics (ABS). Denominator data for age and sex are based on mid-year population estimates for 2000. Population estimates of Australian-born Indigenous people are based on the Projected Indigenous Population 1997 to 2006, Australia.[3] In these projections, assumptions are made about future births, deaths and migrations in the Indigenous population and a 'low' and 'high' estimate of projected population numbers are produced. In this report, we have used the low estimate as the denominator for rates in Indigenous populations as in previous reports. Resident populations in Australia by birthplace of origin were based on estimates of the relevant populations as at 30 June 2000. Countries of birth were coded by the ABS Standard Classification of Countries for Social Statistics.

The population estimates of total Australian-born people were calculated by subtracting the overseas-born population subtotal (estimated in 2000) from the total population in 2000. The population estimates of non-Indigenous Australian-born people in 2000 were calculated by subtracting the total Indigenous population estimate from total Australian-born population estimate.

In this report, notifications may include persons who were visitors or non-permanent residents of Australia during the notification period. As the result, some incidence rates may be overestimated.

TB-related deaths

Mortality data for TB were obtained from the ABS. All the cases with the ICD-10 code A15 to A19 and B90 were classified as deaths caused by TB.

Results

Data quality

In 2000, most of the data fields were complete for cases reported to NMSS. Information on sex and age was reported for all TB notifications and country of birth was recorded for 1,050 (99%) of all TB notifications. In 2000, Indigenous status was reported for 230 (97%) of all notifications for people born in Australia. The principal site of tuberculosis disease was reported for 1,050 (99%) and the diagnosis method was reported for 936 (88%) of the notifications. The anti-TB drug regimen given at the time of diagnosis was reported for 962 (91%) cases of TB. Data fields that were incomplete in the 2000 data collection were HIV status (7% complete), BCG vaccination (32% complete) and length of residence in Australia for overseas-born persons (66% complete).

TB notification rates

In 2000, 1,060 cases of TB were notified nationally (5.5 cases per 100,000 population), representing a 9 per cent decrease from 1999 notification data. The national notification rate has remained at the current level since the mid-1980s (Figure 1). Of the 1,060 cases in 2000, 1,004 (95%) were new cases and 56 (5%) were relapsed cases. The corresponding incidence rate was 5.2 cases per 100,000 population for new cases and 0.3 cases per 100,000 population for relapsed cases (Table 1)

Crude incidence rates vary widely between jurisdictions (Table 2). In 2000, the notification rates of TB have remained below the national average in the Australian Capital Territory, Queensland, South Australia, Tasmania and Western Australia. There was a 41 per cent decrease in TB notifications in the Northern Territory, from 50.3 cases per 100,000 population in 1999 to 29.7 cases per 100,000 population in 2000. This was related to the large number of cases of TB among East Timorese refugees who were evacuated to Darwin in the latter part of 1999.

Figure 1. Incidence rate of TB Australia, 1950 to 2000

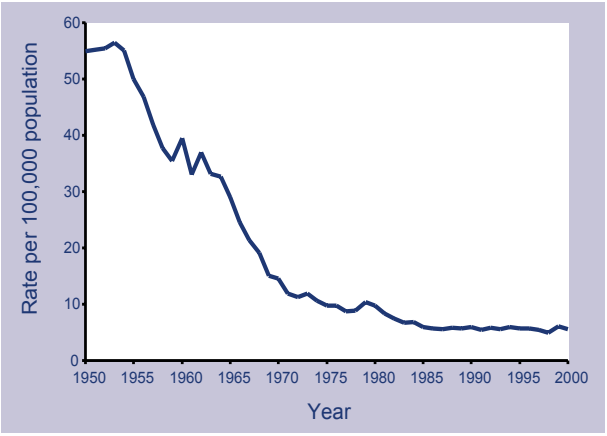


Table 1. Notifications of new and relapsed cases of TB, and rates per 100,000 population, Australia, 1990 to 2000, by year

Year	New cases		Relapsed cases		Total cases	
	Number	Rate	Number	Rate	Number	Rate
1990	979	5.7	37	0.2	1,016	5.9
1991	903	5.2	47	0.3	950	5.5
1992	983	5.6	28	0.2	1,011	5.8
1993	944	5.4	47	0.3	991	5.7
1994	996	5.6	61	0.3	1,057	5.9
1995	988	5.5	50	0.3	1,038	5.8
1996	983	5.4	54	0.3	1,037	5.7
1997	954	5.2	47	0.3	1,001	5.5
1998	884	4.7	39	0.2	923	4.9
1999	1,117	5.9	42	0.2	1,159	6.1
2000	1,004	5.2	56	0.3	1,060	5.5

Table 2. Notifications of new and relapsed cases of TB and rates per 100,000 population, Australia, 2000, by State or Territory

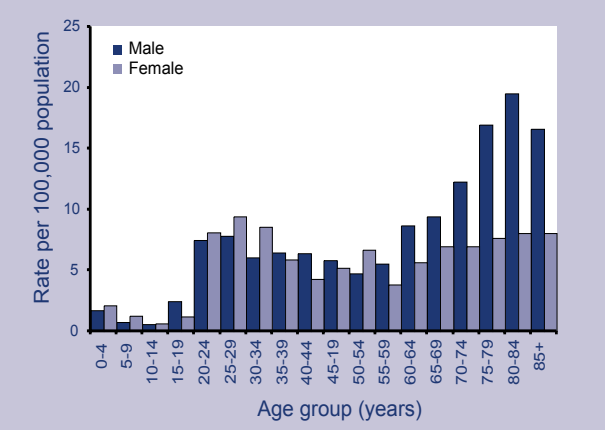
State/Territory	New cases		Relapsed cases		Total cases	
	Number	Rate	Number	Rate	Number	Rate
Australian Capital Territory	11	3.5	0	0.0	11	3.5
New South Wales	399	6.2	32	0.5	431	6.7
Northern Territory	57	29.2	1	0.5	58	29.7
Queensland	97	2.7	10	0.3	107	3.0
South Australia	55	3.7	1	0.1	56	3.8
Tasmania	10	2.1	0	0.0	10	2.1
Victoria	283	5.9	8	0.2	291	6.1
Western Australia	92	4.9	4	0.2	96	5.1
Australia	**1,004**	**5.2**	**56**	**0.3**	**1,060**	**5.5**

Note: Only 4 cases were not residents in the State of notification.

TB Incidence by age and sex

In 2000, sex and age was reported in all TB notifications. Notification rates by age and sex are shown in Figure 2. Of newly acquired TB cases, 526 (52%) were male and 478 (48%) were female giving a male to female ratio of 1.1:1. The corresponding incidence rates for newly acquired TB in males and females was 5.5 cases and 5.0 cases per 100,000 population, respectively. Of relapse cases, half were males and half females and all were aged more than 20 years.

Figure 2. Incidence rate of TB, Australia, 2000, by age and sex



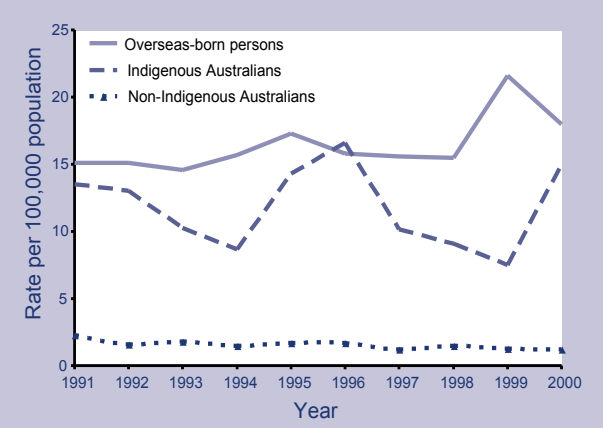
Tuberculosis occurred more commonly in men in both Indigenous and non-Indigenous Australian populations (38 Indigenous cases and 100 non-Indigenous cases) than in women (26 Indigenous cases and 72 non-Indigenous cases) with an overall male to female ratio in Australian born of 1.4:1. However, TB affected equal proportions of men and women in the overseas-born population (408 male cases and 406 female cases).

One of the aims of the recently released Australian TB Strategic Plan is to reduce TB among children. In 2000, there were 45 cases in individuals aged less than 15 years (incidence rate 1.1 cases per 100,000 population). All were newly diagnosed TB cases. Most cases in children occurred in the Indigenous Australians (12 cases; 7.4 cases per 100,000 population) and overseas-born population (14 cases; 6.5 cases per 100,000 population) compared with non-Indigenous Australians (19 cases; 0.5 cases per 100,000 population). Twenty-four cases of TB occurred in children under the age of 5 years. Of these cases, 14 (58%) were non-Indigenous Australians, 7 (29%) were Indigenous Australians and 3 (13%) were residents born overseas.

TB incidence by country of birth

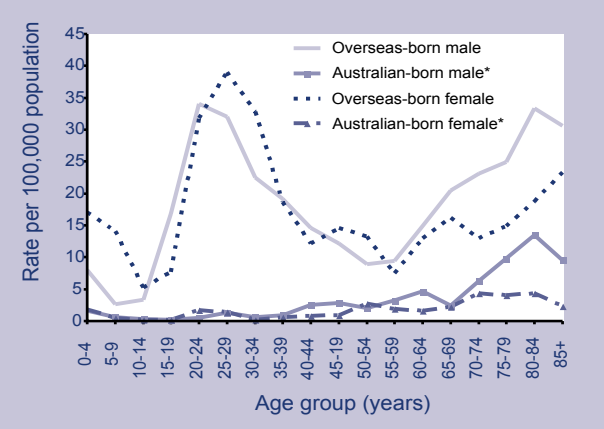
In 2000, information on country of birth was recorded for 1,050 (99%) of all TB notifications. The majority of TB notifications occurred among overseas-born Australian residents (814 cases, 78% of total). The incidence rate of TB in the Australian-born population (including both Indigenous and non-Indigenous Australians) and the overseas-born population was 1.6 cases and 18.0 cases per 100,000 population respectively. The incidence rate of TB in overseas-born persons was 15 per cent lower in 2000 compared with 1999 (21.3 cases per 100,000 population). This reduction was related to the effect that the large number of cases of tuberculosis among East Timorese refugees evacuated to Australia in 1999 had on the incidence rate in overseas-born persons in that year (Figure 3).

Figure 3. Trends of TB incidence rates, Australia, 1991 to 2000, by country of birth



The age- and sex-specific incidence rates in the Australian-born (including both Indigenous and non-Indigenous Australians) and overseas-born populations are illustrated in Figure 4. The overseas-born population shows high age-specific rates in both young adults and the elderly, whereas in the Australian-born population, there is a gradual increase in age-specific rates with advancing age. The highest rates among overseas-born persons were in the 20 - 24 year age group for men (34.1 cases per 100,000 population) and the 25 - 29 year age group for women (39.0 cases per 100,000 population). The highest rates among Australian-born persons were in the 80 - 84 year age group for men (13.4 cases per 100,000 population) and in the 70 - 74 year and 80 - 84 year age groups for women (4.3 cases per 100,000 population).

Figure 4. Incidence rates of TB in the Australian and overseas-born, by age and sex



* Including both Indigenous and non-Indigenous Australians

Table 3 shows the number of TB notifications and incidence rates per 100,000 in 2000 in the overseas-born resident population in Australia by country of birth, compared with the 2000 estimated TB incidence rates in these countries. In some countries, such as Indonesia and Burma, officially reported rates are considered underestimates. The incidence of TB was highest in Australian residents born in Somalia in 2000 (809 cases per 100,000 population). The rate, however, was calculated using the ABS 1996 Census population data due to lack of a current estimated resident population for people born in Somalia and resident in Australia. Australian residents born in India had the second highest TB incidence rate (84.4 cases per 100,000 population, Figure 5). In 1999, Indonesian-born residents had the highest rates (229.4 cases per 100,000 population) due to the large number of TB cases diagnosed in East Timorese-born refugees in that year. In 2000, the rate in Indonesian-born residents has fallen to 81.4 and the East Timorese-born residents are now counted separately (5 cases were reported in East Timorese-born persons in 2000).

Table 3. Notifications of tuberculosis, Australia, 2000. Number and estimated rate per 100,000 population for selected countries of birth

Country of birth	New cases	Relapsed cases	Total cases	Estimated Australian resident population* by country of birth, 2000	Rate per 100,000 population in Australia by country of birth, 2000	WHO incidence rate (per 100,000 population)† for country, 2000
Vietnam	130	8	138	174,449	79.1	112
India	91	2	93	110,190	84.4	111
Philippines	82	5	87	123,035	70.7	170
China	61	3	64	168,071	38.1	36
Indonesia‡	55	0	55	67,553	81.4	32
Korea§	25	6	31	41,357	75.0	Rep Korea (47) DPR Korea (153)
Hong Kong (SAR)	24	1	25	56,283	44.4	75
Papua New Guinea	18	2	20	27,380	73.0	252
UK/Ireland	17	3	20	1,160,039	1.7	10
Cambodia	16	2	18	23,711	75.7	144
Somalia	17	0	17	2,100	809.5	65
Malaysia	13	1	14	97,632	14.3	68
Sri Lanka	13	1	14	56,048	25.0	44
Thailand	14	0	14	22,327	62.7	54
Myanmar	10	3	13	41,357	31.4	65
Fiji	10	1	11	40,312	27.3	18
Italy	11	0	11	241,749	4.6	6
Overseas-born	769	45	814	4,517,267	18.0	
Australian-born	225	11	236	14,639,770	1.6	
Not stated	10	0	10			
Total	**1,004**	**56**	**1,060**	**19,157,037**		

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

* The Australian resident population for Somalia was the estimate from the ABS 1996 Census data.

† Rates from WHO 2002 Global Tuberculosis report.

‡ Population for Indonesia includes those born in East Timor.

§ The notifications for Korea included both Republic Korea and Democratic Peoples Republic Korea.

Fourteen (2%) of overseas-born TB cases were aged less than 15 years with an age specific rate of 6.5 cases per 100,000 population compared with 0.8 cases per 100,000 Australian-born population in the same age group. Of these TB cases in children, country of birth was reported as Papua New Guinea (4 cases), New Zealand (3 cases), China, Ethiopia, India, Indonesia, Somalia, the United States of America (USA) and Vietnam (1 case for each country).

TB incidence in Indigenous Australians

Indigenous status was reported for 230 (97%) of all notifications for people born in Australia. Indigenous Australians accounted for 64 TB cases in 2000, a 88 per cent increase on the 34 cases reported in 1999, but a similar number to that in 1996. The annual incidence rate for Indigenous Australians has increased from 8.3 cases per 100,000 Indigenous population in 1999 to 15.3 cases per 100,000 population in 2000. By contrast, the incidence rate remained low for non-Indigenous Australians in 2000 (1.2 cases per 100,000 population).

The largest increase in TB cases in Indigenous Australians occurred in the Northern Territory (17 cases in 1999 to 37 cases in 2000). Increases were also noted in Queensland (8 cases in 1999 to 14 cases in 2000), New South Wales (3 cases in 1999 to 5 cases in 2000) and South Australia (one case in 1999 to 2 cases in 2000). Western Australia reported 5 cases in both years and Tasmania reported one case in Indigenous Australians in this year. The Australian Capital Territory and Victoria reported no cases in Indigenous Australians in 2000. Table 4 shows the crude incidence rates of TB in the Indigenous population by jurisdiction.

Among the 64 TB cases in Indigenous Australians, 38 were male and 26 female with a male to female ratio of 1.5:1. Twelve (19%) notifications in Indigenous Australians were in children aged less than 15 years with an age specific rate of 7.4 cases per 100,000 Indigenous population. This is in contrast to the rate of 0.5 cases per 100,000 population in the same age group in the non-Indigenous Australian-born population.

The incidence rate in Indigenous males aged 75 years and over (159.1 cases per 100,000 population), was markedly higher than in the same age group in the non-Indigenous Australian-born population (10.2 cases per 100,000 population). The rates in Indigenous women in the 65 - 69 years age group (79.8 cases per 100,000 Indigenous population) was also markedly higher than the rate in the same age group of non-Indigenous Australian (1.3 cases per 100,000 population) (Table 5). Forty-two (66%) Indigenous Australians had pulmonary tuberculosis.

Figure 5. Incidence rate of TB per 100,000 resident population, Australia, 2000 by country of birth

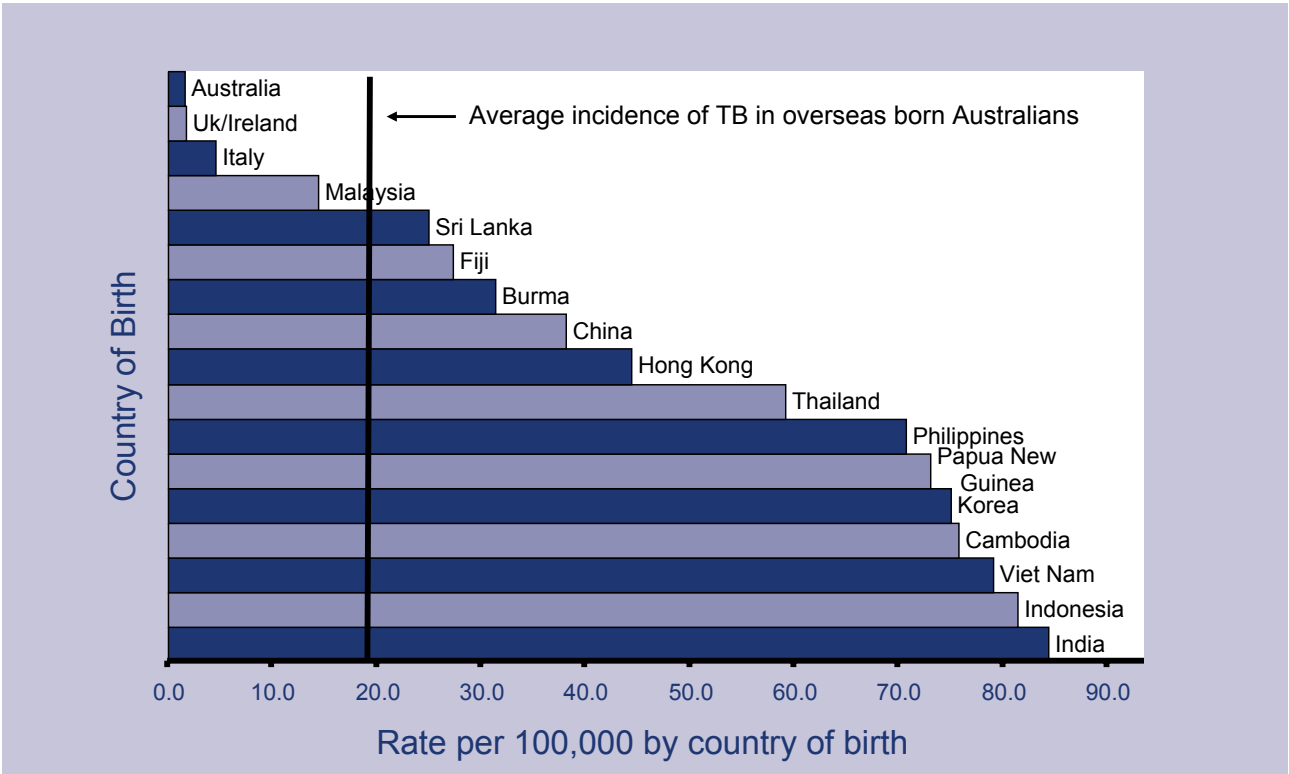


Table 4. Notifications of tuberculosis and incidence rates in Indigenous populations, Australia, 1999 and 2000, by jurisdiction

State/Territory	1999		2000	
	Number	Rate*	Number	Rate*
Australian Capital Territory	0	0.0	0	0.0
New South Wales	3	2.6	5	4.2
Northern Territory	17	31.1	37	66.7
Queensland	8	7.1	14	12.1
South Australia	1	8.8	2	17.3
Tasmania	0	0.0	1	6.1
Victoria	0	0.0	0	0.0
Western Australia	5	8.4	5	8.3
Australia	**34**	**8.3**	**64**	**15.3**

* Per 100,000 population

Table 5. Incidence rates of tuberculosis in Indigenous and non-Indigenous Australians, 2000, by age and sex

Age group (years)	Indigenous Australians				Non-Indigenous Australians			
	Male		Female		Male		Female	
	Number	Rate*	Number	Rate*	Number	Rate*	Number	Rate*
0-4	4	13.7	3	10.8	6	1.0	8	1.4
5-9	1	3.5	2	7.4	3	0.5	1	0.2
10-14	1	3.9	1	4.0	1	0.2	0	0.0
15-19	1	4.4	0	0.0	0	0.0	1	0.2
20-24	3	16.4	2	10.9	0	0.0	7	1.4
25-29	3	17.1	2	10.9	5	0.9	6	1.1
30-34	1	6.7	1	6.0	2	0.4	0	0.0
35-39	3	23.2	1	6.9	2	0.4	2	0.4
40-44	7	64.8	3	25.2	6	1.2	1	0.2
45-49	4	48.2	3	32.4	9	2.0	1	0.2
50-54	1	15.8	2	29.1	7	1.8	9	2.3
55-59	2	46.7	1	21.8	8	2.7	5	1.6
60-64	3	101.2	1	29.1	8	3.4	3	1.2
65-69	1	49.2	2	79.8	4	2.0	3	1.3
70-74	1	83.0	1	59.1	11	5.8	9	3.9
75+	2	159.1	1	51.7	28	10.2	16	3.5
Total	**38**	**18.4**	**26**	**12.3**	**100**	**1.4**	**72**	**1.0**

* Per 100,000 population

HIV status of TB cases

HIV test status was not provided for the majority (982, 93%) of notified cases of TB. Of the 78 cases in which HIV status was reported, 17 were positive and 61 were negative. Of the HIV positive cases, seven were Australian-born (all were male), and 10 were born overseas (6 male and 4 females). All HIV positive cases had newly acquired TB and 12 of the 17 had pulmonary disease. The age range of the HIV positive TB patients was 25 - 64 years for men and 20 - 44 years for women.

Principal sites of TB disease

The principal site of tuberculosis disease was reported for all but 9 cases of newly acquired TB and all but one case of relapsed TB (Table 6). The most common site for infection was pulmonary (683 cases; 64%), followed by lymphatic (179 cases; 17%).

Pulmonary disease accounted for 64 per cent (640 cases) of the newly acquired cases, and 78 per cent (43 cases) of the relapse cases. Rates of pulmonary TB in non-Indigenous Australians was 0.8 cases per 100,000 population, compared with 10.0 cases per 100,000 population in Indigenous Australians and 11.4 cases per 100,000 population in those born overseas.

The rate of extra-pulmonary TB in non-Indigenous Australians was 0.4 cases per 100,000 population, compared to 4.3 cases per 100,000 population in Indigenous Australians and 6.5 cases per 100,000 population in those born overseas.

Methods of diagnosis of TB

The diagnosis method was reported for 936 (88%) notifications. Of these cases, 47 (5%) were diagnosed by clinical examination only. The diagnostic modalities employed were reported for 648 cases of the 683 pulmonary cases. A positive result by one or more investigation was reported for 535 of these: 441 were tested positive by culture, 300 by microscopy, 105 by radiography, 48 by histology and 29 by nucleic acid testing. The diagnostic modalities were reported for 254 of 367 extra-pulmonary cases. Of these, 235 had a positive result by at least one method: 131 cases confirmed by culture, 100 by histology, 83 by microscopy, 23 by radiography and 12 by nucleic acid testing.

BCG status of TB cases

BCG vaccination status was reported for 340 (32%) of the 1,060 TB notifications. Of these, 245 (72%) reported receiving BCG vaccination and 95 had no history of BCG vaccination. It was noted that of the 7 Indigenous children with TB, 5 had received BCG vaccination and only 2 of the cases were pulmonary disease.

Table 6. Notifications of new and relapsed cases of TB in Australia, 2000, by principle site of disease

Site	New cases	Relapsed cases	Total cases	Total%
Pulmonary	640	43	683	64.4
Lymphatic	172	7	179	16.9
Pleural	70	2	72	6.8
Bone/joint	41	2	43	4.1
Peritoneal	23	0	23	2.2
Genitourinary	17	1	18	1.7
Miliary	4	0	4	0.4
Meningeal	12	0	12	1.1
Other	16	0	16	1.5
Unspecified	9	1	10	0.9

Anti-TB drug regimens

The anti-TB drug regimen given at the time of diagnosis was reported for 962 (91%) cases of TB (Table 7). There was no information available for 98 notifications. The reasons for incomplete follow-up data may include death, transfer overseas or interstate. Patients who are managed in private practice may also not report back to TB services.

A four-drug regimen of isoniazid (INH), rifampicin (RIF), pyrazinamide (PZA) and ethambutol (EMB) was the most commonly prescribed anti-TB therapy for new cases (737/913; 81%) and relapsed cases (37/49; 76%). The four-drug regimen was the most frequently prescribed treatment for overseas-born persons (642/749; 86%), Indigenous Australians (44/60; 73%) and non-Indigenous Australians (109/148; 73%). The three-drug combination of INH, RIF and PZA was commonly prescribed for children under the age of 5 years (18/23; 78%).

Deaths due to TB

In 2000, the ABS registered 55 deaths for which TB was recorded as the primary cause of death. The crude mortality rate was 0.3 deaths per 100,000 population, which was similar to the 0.2 deaths per 100,000 population in 1999 (Figure 6). Of the total deaths, 35 (64%) were in the Australian-born population and 20 (36%) in the overseas-born resident population. The corresponding mortality rates were 0.2 deaths per 100,000 Australian-born population and 0.4 deaths per 100,000 overseas-born Australians.

Age and sex specific mortality rates showed the largest number of deaths due to TB among the 80 - 84 year age group (7 deaths in men and 6 deaths in women), giving a rate of 4.3 deaths per 100,000 population.

Figure 6. Tuberculosis incidence and mortality rates, Australia, 1970 to 2000

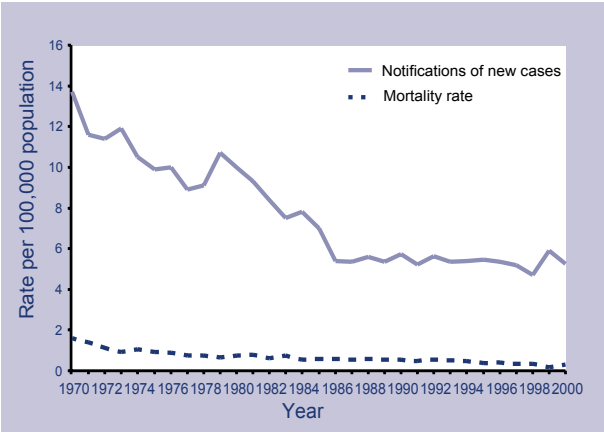


Table 7. Initial drug regimen given for treatment of tuberculosis, Australia, 2000

Drug regimen	New cases	Relapsed cases	Total cases
Eight drug regimen	1	1	2
Seven drug regimen	2	0	2
Six drug regimen	3	0	3
Five drug regimen	5	3	8
Four drug regimen	761	39	800
Three drug regimen	123	6	129
Two drug regimen	18	0	18
Total	**913**	**49**	**96225**

Discussion

In 2000, Australia continued to report one of the lowest TB incidence rates in the world. The incidence rate of TB in Australia has stabilised at less than 6 cases per 100,000 population since 1986, except in 1999, when the rate reached 6.1 per 100,000 population. The reason for the increase in cases of tuberculosis in Australia in 1999 was the significant numbers of cases of tuberculosis found among refugees from Kosovo and East Timor given temporary resident visas under the 'Safe Havens' programs.

Despite the decrease in notifications in the overseas-born population in the year, 78 per cent of TB cases in Australia in 2000 occurred in this population, and the incidence rate was 15 times the rate in the non-Indigenous Australian-born population. The proportion of overseas-born cases is highly dependent on global international circumstances. The high incidence of TB among the overseas-born in Australia ought to alert physicians to consider TB in patients from these communities presenting with compatible symptoms. The proportion of TB cases occurring in the overseas-born in Australia is similar to that in other developed countries. For example in the USA in 2000, 46 per cent of TB cases were reported in the foreign-born and the incidence rate was more than seven times higher than in the USA-born population.[4] The fact that Australia's incidence rate has remained stable since the mid-1980s despite increasing migration, reflects effective screening policies and programs currently in place. However, it also reinforces the need to remain vigilant within our national and state surveillance, management and control programs. For example, health screening of 7,000 illegal entrants to Australia between 1 January 2000 and 30 June 2001 resulted in the diagnosis of 11 cases of tuberculosis.[5]

The burden of TB disease remains high in the Indigenous Australian population. Over the last 10 years, rates of TB have remained 10 to 15-fold higher in Indigenous Australians compared to the non-Indigenous Australian-born population. In 2000, the TB incidence rate in Indigenous Australians increased from 8.3 cases per 100,000 population in 1999 to 15.3 cases per 100,000 population in 2000. In part, this is due to an extended outbreak of TB in the Northern Territory. Among the risk factors for TB in Indigenous Australians are poor socio-economic status, diabetes, renal disease, smoking, alcohol abuse and poor nutrition.[6] These factors need to be addressed in concert with accessible TB control programs. As programs improve Indigenous status reporting in notifiable diseases there may be an apparent increase in rates of TB among Indigenous Australians.

HIV associated TB is widely recognised as the important issue for TB control. The global burden of TB has been further exacerbated by HIV co-infection in many regions of the world, especially Asia and Africa.[1] Australia has the lowest rate of TB/HIV co-infection in the world. A previous study estimated that 2 per cent of Australian TB patients were HIV positive.[7] HIV status, however, was only recorded for 7 per cent of the notifications in 2000. Further collaborative efforts are necessary to improve surveillance practice to gain a better understanding of the extent of TB/HIV co-infection in Australia.

For Australia to maintain a low incidence of TB and to reach the goal of TB elimination, there is a continued need to enhance the capacity and expertise to respond to persons with TB nationally and internationally. CDNA recently endorsed the National Strategic Plan for TB control in Australia beyond 2000, to ensure TB management and control activities are undertaken with optimal coordination among national and international public health partners. Future annual reports will focus on progress towards the performance indicators developed in the strategic plan.[8]

There are few indications that the global TB threat is abating, which reinforces the need for all nations to remain vigilant. Having a surveillance system in place that can accurately report on trends, and important changes in the epidemiology of TB, alerts public health authorities and policy makers to emerging problems and appropriate action.

Acknowledgments

The members of the Communicable Diseases Network Australia are thanked for their co-operation with this surveillance initiative, together with the State and Territory Directors of Tuberculosis, and other health department personnel in the States and Territories involved in compiling the individual datasets. Special thanks is offered to Louise Carter, Hillary McClure and Joyce Della in the Australian Capital Territory, Rob Menzies and Mohammed Habib in New South Wales, David Peacock in the Northern Territory, Patrick Derhy in Queensland, Sara Noonan in South Australia, David Coleman in Tasmania, Trevor Lauer and Lynn Browne in Victoria, and Jag Atrie in Western Australia. In addition, a note of appreciation is extended to the many physicians and medical practitioners and nurses who contribute to the collection of these data.[7]

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A message from the Branch Head

On behalf of the *Communicable Diseases Intelligence* (*CDI*) staff and the Commonwealth Department of Health and Ageing I'd like to acknowledge and thank Dr Angela Merianos for her dedicated contribution to *CDI* and her work as Medical Director of the Surveillance Section of the Communicable Diseases Branch for the period 2000 — 2002.

As a senior member of staff and the Commonwealth representative to the Communicable Disease Network Australia (CDNA), Angela advanced many aspects of a national agenda for improved communicable disease surveillance and response and brought to the Commonwealth a breadth of skills and experience in Public Health Medicine, particularly in the areas of vaccine preventable and arboviral disease control, and international health. She also strengthened the epidemiology and research skills within the department, in part reflected in the positive production developments of *CDI*. Most recently, Angela headed the Commonwealth Task Force related to Biosecurity and played a pivotal role in assisting the national response to anthrax and other bio-terrorist threats.

I wish her well in her new role as a Medical Advisor at the Australian Agency for International Development (AusAID) and am pleased that she will be able to continue to contribute to the cause of communicable disease control as the AusAID representative to CDNA.

Greg Sam
Head
Communicable Diseases and Health Protection Branch

Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2000:

Report of the Australian Mycobacterium Laboratory Reference Network

Richard Lumb,[1] Ivan Bastian,[1] David Dawson,[2] Chris Gilpin,[2] Frank Havekort,[2] Peter Howard,[2] Aina Sievers[2]

Abstract

The Australian Mycobacterium Reference Laboratory Network collected and analysed laboratory data on new diagnoses of disease caused by *Mycobacterium tuberculosis* complex in the year 2000. A total of 765 cases were identified, representing an annual reporting rate of 4.0 cases of laboratory-confirmed tuberculosis (TB) per 100,000 population. Pulmonary disease was diagnosed in 64.9 per cent of cases with a male:female ratio of 1.5:1. Smears were positive for 209/365 (57.3%) of sputum isolates and 39/117 (33.3%) bronchoscopy isolates. Sputum from males was more likely to be smear-positive (63.3%) than from females (47.5%). Isolates from lymph node accounted for 136 (17.7%) of all cases; only 28.7 per cent were smear-positive. Eighty-four (11.0%) isolates, comprising 82 *M. tuberculosis* and 2 *M. bovis* strains, demonstrated *in vitro* resistance to at least one of the standard anti-TB medications. Resistance to at least isoniazid and rifampicin (defined as multidrug-resistant TB) was observed for only 8 (1.0%) strains, a rate similar to previous years. Almost all (96.3%) of patients with drug resistant strains were classified as having initial resistance. The country of birth was known for 76 (92.7%) of 82 patients with a drug resistant strain of *M. tuberculosis*; 6 were Australian-born and 70 (92.1%) had migrated from a total of 17 countries. Of these 70 migrants with drug-resistant disease, 68.6 per cent had migrated from one of the following countries: Vietnam (n=15), China (n=11), Philippines (n=11), India (n=6), and Indonesia (n=5). *Commun Dis Intell* 2002;26:226–233.

Keywords: Mycobacterium tuberculosis, Mycobacterium bovis, *laboratory diagnosis, tuberculosis, TB, drug resistance, initial resistance*

Introduction

Australia's notification rate for tuberculosis (TB) has remained stable at between 5-6 cases per 100,000 population since 1991. A rise to 6.1 per 100,000 population in 1999 was largely due to the influx of refugees from Kosovo under the 'Safe Havens' program and evacuees from East Timor to Darwin in September 1999.[1] Data from the World Health Organization (WHO) reveals that Australia has one of the lowest notification rates in the world.[2] Continued collection of accurate, comprehensive and timely statistics for tuberculosis will help ensure strategic directions are identified, that outcomes are achieved, and that Australia's enviable record of TB control is maintained.[3,4]

Since 1991, the National Mycobacterial Surveillance System (NMSS) of the Communicable Diseases Network Australia has provided statistics on cases of tuberculosis reported to public health authorities in Australia's States and Territories.[5] The Australian Tuberculosis Reporting Scheme has been conducted by the Australian Mycobacterium Reference Laboratory Network (AMRLN) since 1986.[6] Statistics compiled by the AMRLN relate to cases of bacteriologically confirmed tuberculosis whereas NMSS data will have a proportion of cases that are identified on the basis of clinical and epidemiological information, or on non-bacteriological laboratory investigations.[7] This report describes the bacteriologically confirmed diagnoses for the year 2000.

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Methods

The data are based on clinical specimens that were culture-positive for *Mycobacterium tuberculosis* complex (MTBC). Although the Bacille Calmette Guerin (BCG) strain of *M. bovis* is a member of the MTBC, no information on this organism is included in the present report. Almost 80 laboratories performed culture for mycobacteria in 2000 (Royal College of Pathologists of Australasia Quality Assurance Program) with nearly all isolates of MTBC being referred to one of the five laboratories comprising the AMRLN for specific identification and drug susceptibility testing. Comparable laboratory methodologies are used in the reference laboratories. Relapse cases, as defined in the TB notifications in Australia, 1999 report,[1] were included in the laboratory data as laboratories are frequently unable to differentiate relapses from new cases. Temporary visitors to Australia were included as were illegal immigrants within correctional services facilities and asylum seekers located in detention centres or on temporary visas within Australia.

For each new bacteriologically confirmed case, the following information was collected:

- demography: patient identifier, age, sex, HIV status and State/Territory of residence;
- specimen: type, site of collection, date of collection and microscopy result;
- isolate: species of mycobacterium and results of drug susceptibility testing; and
- drug resistant strain: patient country of origin or risk factors, and history of previous TB treatment to determine whether resistance was initial or acquired.

Data from contributing laboratories were submitted in standard format to the scheme coordinator for analysis. Duplicate entries (indicated by identical patient identifier and date of birth) were deleted prior to analysis. Rates were calculated using the respective mid-year estimates of the population supplied by the Australian Bureau of Statistics.[8] For each patient, the nature of the first clinical specimen that yielded an isolate of MTBC was used to record the nominal site of disease. Culture-positive specimens collected at bronchoscopy or by gastric lavage were considered as pulmonary disease. In cases of multi-site disease, provided a sputum specimen was culture-positive, these cases were listed as pulmonary disease, the most important category for public health purposes. Multiple-site isolations were not categorised as having miliary or disseminated disease as differentiation is based on clinical findings that were frequently not available to the reporting laboratories. Initial drug resistance was defined as the presence of drug resistant strains of *M. tuberculosis* in new cases of tuberculosis. Patients who had begun anti-TB treatment and had developed resistance to one or more of the drugs used during treatment were said to have developed acquired drug resistance.[9]

Results

Total reports and distribution by State or Territory

There were 765 bacteriologically confirmed cases of TB in 2000, representing an annual rate of 4.0 cases per 100,000 population (Figure 1). State-specific reporting rates varied from less than one (Tasmania) to 23.0 cases per 100,000 population (Northern Territory) (Table 1). Of the 45 culture positive cases in the Northern Territory, 13 were associated with one remote Aboriginal community and 3 were foreign nationals working in East Timor transferred to Darwin for diagnosis and initial treatment (Dr Vicki Krause, personal communication). There were 9 patients from Papua New Guinea who were diagnosed in Australia and who are included in the Queensland laboratory data, and 5 persons identified as illegal immigrants (Western Australia n=4, Northern Territory n=1).

Figure 1. Comparison between TB notification rates and laboratory data, Australia, 1990 to 2000

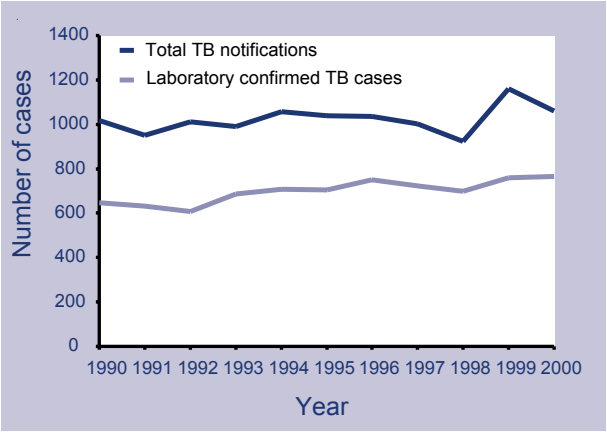


Table 1. Bacteriologically confirmed cases of tuberculosis, Australia, 1990 and 1997 to 2000, cases and rate per 100,000 population, by State or Territory

State/ Territory	1990[11]		1997[7]		1998[10]		1999[10]		2000	
	n	rate	n	rate	n	rate	n	rate	n	rate
NSW*	275	4.5	329	5.0	289	4.4	291	4.3	307	4.5
NT	32	19.4	28	15.0	22	11.6	21	10.9	45	23.0
Qld	78	2.7	74	2.2	85	2.5	75	2.1	76	2.1
SA	31	2.2	39	2.6	40	2.7	46	3.1	41	2.7
Tas	14	3.0	8	1.8	6	1.3	2	0.4	2	0.4
Vic	177	4.1	193	4.2	192	4.1	261	5.5	231	4.8
WA	41	2.5	51	2.8	66	3.6	64	3.4	63	3.3
Total	**648**	**3.8**	**722**	**3.9**	**700**	**3.7**	**760**	**4.0**	**765**	**4.0**

* Data from the Australian Capital Territory are included with those from New South Wales.

Causative organism

Almost all isolates were identified as *M. tuberculosis* (763) with only 2 isolates of *M. bovis*, and no cases of disease caused by *M. africanum*.

Distribution by gender, age and site of disease

Complete information for gender and age were submitted for 756 of the 765 cases. Eleven children under 10 years of age had bacteriologically confirmed tuberculosis (lymph node n=6, CSF n=2, respiratory n=2, pleural n=1). The overall male:female ratio was 1.15:1. The overall age/sex rates are shown in Figure 2. Age and gender rates varied depending on the site of infection (Figures 3 and 4). The male:female ratio for pulmonary disease was 1.5:1.

Figure 2. Laboratory confirmation of MTBC disease, Australia, 2000, by age and sex

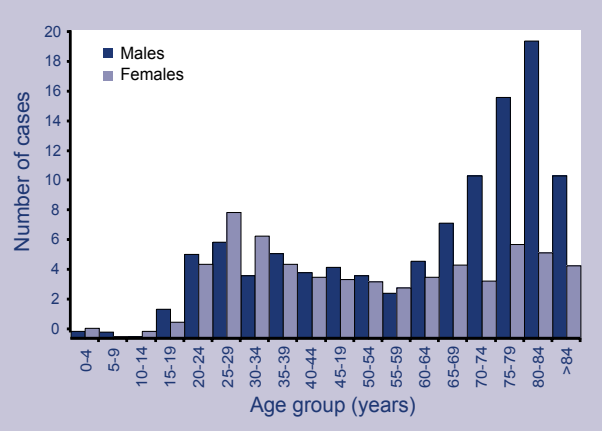


Figure 3. Isolation of MTBC from the respiratory tract, Australia, 2000, by age and sex

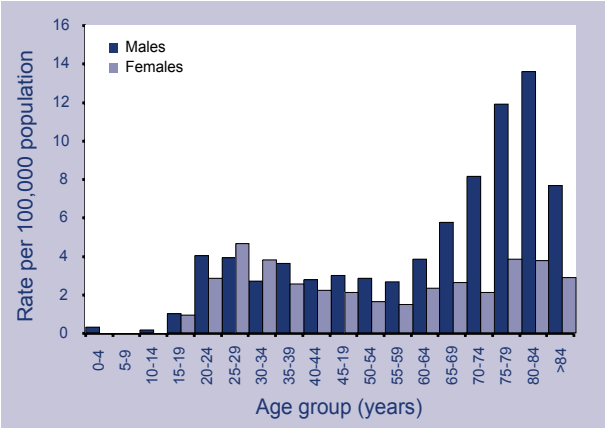
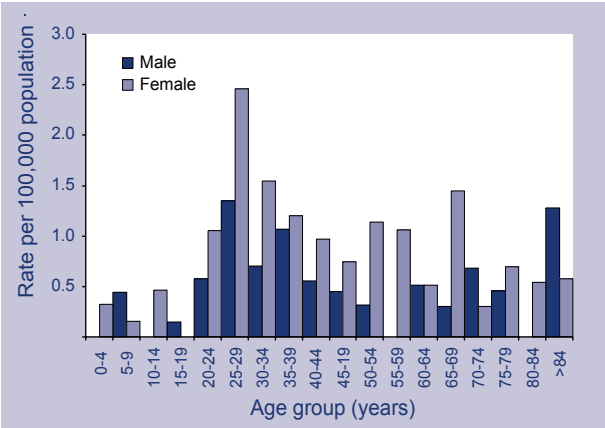


Figure 4. Isolation of MTBC from lymph node, Australia, 2000, by age and sex



Sputum (n=365, 73.7%) was the predominant specimen type; a further 117 (23.6%) were from bronchoscopy specimens and 13 were lung tissue/biopsy samples. Forty-four isolates were of pleural origin: 31 being pleural fluid, 12 from pleural biopsy or tissue, and the source of one pleural specimen was not further identified. Isolates from lymph node accounted for 136 (17.7%) of the total number of isolates with a male:female ratio of 1:1.9. There were 16 isolates from other sites, including usually sterile fluids (pericardial, blood), abscesses (psoas, groin, ischiorectal), faeces, and tissue (colon, caecal, adrenal, pericardium, aorta, subparietal). For 12 isolates, there was insufficient information to determine the site of disease.

Association with HIV

The AMRLN databases had access to the HIV status of only 76 (9.9%) patients. Six patients were identified as HIV seropositive, all were infected with *M. tuberculosis*. One HIV-positive patient was sputum smear-positive with a multidrug resistant strain of *M. tuberculosis*.

Microscopy

Results of microscopy were available for 755 of 765 isolates (Table 2). Microscopy was not performed for 6 specimens and results for a further 4 samples were unknown. Smears were positive for 209 of 365 (57.3%) sputum isolates and 39 of 117 (33.3%) bronchoscopy isolates. Sputum from males was more likely to be smear-positive (63.3%) than from females (47.5%). A total of 44 pleural specimens were culture positive for *M. tuberculosis* with only four (9.1%) smear-positive for acid fast bacilli (AFB). Of the 136 isolates from lymph node, 39/136 (28.7%) were smear-positive for AFB.

Table 2. Site of specimens

	Number*	Smear positive (%)	
All specimens	765	324	(42.4)†
Sputum	365	209	(57.3)
Bronchoscopy	117	39	(33.3)
Lymph node	136	39	(28.7)
Pleural	44	4	(9.1)
Genito-urinary	30	7	(23.3)
Peritoneal	10	3	(30.0)
Skin	8	4	(50.0)
CSF	7	0	(0.0)
Bone/joint	7	3	(42.8)

* Specimens not tabulated: 13 pulmonary tissue samples, 16 specimens from miscellaneous sites, and 12 of unknown site

† Excludes microscopy not performed (6) or result unknown (4).

Drug susceptibility testing

In 2000, results of *in vitro* drug susceptibility testing were available for all 765 isolates for isoniazid (H), rifampicin (R), ethambutol (E), and pyrazinamide (Z). Results of testing for streptomycin (S) were available for 230/765 (30.0%) of isolates. A total of 84 isolates (11.0%), comprising 82 *M. tuberculosis* and two *M. bovis* strains, were resistant to at least one of the above anti-tuberculosis agents. Resistance to H and/or R was noted for 79 isolates (10.3%), with resistance to both H and R (i.e. defined as multidrug-resistant (MDR) disease) observed in eight (1.0%) strains. All of the MDR isolates were *M. tuberculosis* (MDR-TB). Of the eight MDR-TB isolates, seven were from the respiratory tract (sputum 5, bronchoscopy 2); the remaining isolate was from lymph node (Table 3).

The two *M. bovis* isolates were fully susceptible apart from their inherent resistance to Z. Of the 82 *M. tuberculosis* isolates, 56 (7.3%), 4 (0.5%), and 2 (0.3%) demonstrated mono-resistance to H, Z and R, respectively. There was no mono-resistance to ethambutol. Seventy-seven strains demonstrated resistance to H at a concentration of 0.1 mg/L in the radiometric BACTEC system. Twenty strains were not tested at the higher concentration of 0.4 mg/L. Of the remaining 57 strains resistant at 0.1 mg/L, 33 (57.9%) demonstrated resistance at the higher level.

Thirty-six of 82 (43.9%) specimens culture-positive for drug resistant *M. tuberculosis* were also smear-positive for AFB. Importantly, five of the 7 patients with pulmonary MDR-TB were smear-positive.

Initial or acquired resistance and country of origin

There were 82 *M. tuberculosis* isolates resistant to at least one of H, R, E or Z. Of these, 79/82 (96.3%) were classified as having initial resistance, one case had probable acquired resistance, and no data were available on the presence or absence of previous treatment for 2 patients. The country of birth was known for 76/82 (92.7%) of patients with a drug resistant strain of *M. tuberculosis*; six were Australian-born and 70 (92.1%) had migrated from a total of 17 countries.

Of these 70 migrants with drug-resistant disease, 68.6 per cent had migrated from one of the following countries: Vietnam (n=15), China (n=11), Philippines (n=11), India (n=6), and Indonesia (n=5). For the 6 Australian-born persons identified as having a drug-resistant strain, risk factors were not identified for 3 patients, one person was a Torres Strait Islander, one had travelled in South East Asia, and another person was a work contact of a known TB case.

Table 3. Drug resistance patterns in multidrug-resistant strains, Australia, 1996-2000

Resistance pattern (standard drugs)[1]	1996[14]	1997[7]	1998[10]	1999[10]	2000
H+R only	10	6	2	2	3
H+R+E	1	1	1	1	1
H+R+Z	4	5	2	1	3
H+R+E+Z	0	2	1	0	1
Total (%)	**15 (2.0)**	**14 (1.9)**	**6 (0.9)**	**4 (0.5)**	**8 (1.0)**

H = Isoniazid

R = rifampicin

E = ethambutol

Z = pyrazinamide

Discussion

The rate of 4.0 cases of laboratory-confirmed tuberculosis per 100,000 population for the year 2000 falls within the range of 3.7–4.1 cases per 100,000 population reported in the past decade.[10] The annual incidence rates for the States and Territories varied from a low of 0.4 cases per 100,000 population in Tasmania to 4.8 cases per 100,000 population in Victoria, which is also consistent with previous years.[7,10,11–14] However, the Northern Territory incidence rate of 23.0 cases per 100,000 population was higher than in recent years. Almost 30 per cent of the Northern Territory population are Aboriginal or Torres Strait Islanders, a group identified with a far higher incidence rate for TB than the Australian-born, non-Indigenous population.[1]

The respiratory tract was the primary site of disease for 495 (64.7%) patients with only 12 (2.5%) reports noting the isolation of MTBC from other sites concomitantly. Overall, 57.3 per cent of sputum specimens were smear-positive for AFB; a finding consistent with previous reports.[10] Interestingly, a gender difference was noted, with males more likely to be sputum smear-positive than females (63.3% vs 47.5%).

Seven patients (3 male, 4 female) had culture-confirmed TB meningitis, all smear-negative, and caused by *M. tuberculosis*. Three cases were children aged 15 or less. In 2000, there were 12 cases of TB meningitis reported by the NMSS.[15] Laboratory diagnosis of meningeal TB is problematic with smear positivity rates typically reported between 10–40 per cent although higher rates are reported when multiple, large (10–20 mL) volumes of CSF are examined.[16,17] Pleural TB was confirmed bacteriologically in only 44 cases. This diagnosis is seldom confirmed by culture of pleural fluid; the diagnostic yield being increased by pleural biopsy.[18,19] Lymphatic TB was confirmed almost twice as frequently in females than in males, especially in the 25–39 year age group.

Tuberculosis notification data provided by the NMSS have consistently reported incidence data higher than that provided by the AMRLN.[15] A comparison of the two sources of data for the past decade reveals that, on average, there are 34 per cent (range 24–40%) more notifications by NMSS where the bacteriological status was either negative or unknown (Figure 1). Possible reasons for the gap between the two data sources include:

- diagnoses made on clinical and radiological findings only;
- difficulties obtaining specimens from young children and the elderly;
- failure to submit appropriate specimen(s);
- sample(s) being placed into histological fixative; and
- faulty or insensitive laboratory culture techniques.

Eighty-four isolates (11.0%) demonstrated *in vitro* resistance to at least one of the standard anti-TB medications; isoniazid, rifampicin, ethambutol, or pyrazinamide. This figure is marginally higher than that of previous years which, with the exception of 1996 data (11%), has been at less than 10 per cent. For the year 2000, *in vitro* resistance to at least H+R (i.e. multidrug-resistant TB) was observed for only 8 (1.0%) strains, a finding consistent with previous annual reports.[6,7,10–14]

The National Committee for Clinical Laboratory Standards (NCCLS) in the United States of America has recommended that *M. tuberculosis* isolates be tested at two concentrations of isoniazid (e.g. 0.1 and 0.4 mg/L in the BACTEC radiometric system),[20] and this practice has been adopted by the AMRLN. Of 57 isoniazid-resistant isolates tested at both concentrations, 24 (42.1%) demonstrated low-level resistance (i.e. 0.1<MIC<0.4 mg/L). Serum isoniazid levels within this range are obtainable, and continuation of isoniazid treatment in patients with low-level resistance may therefore be beneficial.[21,22] Hence, NCCLS suggests that laboratories reporting *M. tuberculosis* isolates with low-level resistance should append a comment such as, 'These test results indicate low-level resistance to isoniazid. Some evidence indicates that patients who are infected with strains exhibiting this level of resistance to isoniazid may benefit from continuing therapy with isoniazid. A specialist in the treatment of tuberculosis should be consulted regarding the appropriate therapeutic regimen and dosages'.[20] The AMRLN has not reached consensus on explanatory comments for low-level isoniazid resistance and any laboratory proposing to introduce such a comment is advised to consult with their relevant State or Territory TB Control Unit. Regardless, any patient with an isolate which shows resistance to isoniazid or rifampicin should be treated in close co-operation with the State or Territory TB Control Unit.

Previous AMRLN reports have not been able to identify cases of primary or acquired drug resistance because the NMSS and the AMRLN databases remained unlinked. WHO and the International Union Against Tuberculosis and Lung Disease (IUATLD) studies use the term *primary resistance* to define transmission of a drug resistant strain from one person to another, who then develops disease caused by the drug resistant strain. The rates of primary and acquired drug resistance may provide a measure of the past and current quality, respectively, of a tuberculosis control program. International TB authorities therefore prefer that drug resistance data be sub-divided based on the patients' previous exposure to anti-tuberculosis therapy.[9] With the assistance of members of the National TB Advisory Committee, this report has attempted this categorisation for the first time. However, the difficulties in categorising 'primary' and 'acquired' drug resistance from a laboratory database with limited clinical information must be appreciated.

Some patients may not recall previous anti-TB therapy or may not divulge such information. Determining whether drug resistance resulted from previous treatment or from infection by a drug resistant strain then becomes problematic. The WHO/IUATLD now recommend the use of the term *Resistance Among New Cases* when patients deny any prior anti-TB treatment and, in countries where adequate documentation is available, no documented evidence of prior treatment exists. Acquired resistance is defined as the emergence of a drug resistant strain from a person whose initial strain was drug susceptible, which can be determined only in countries with the resources to perform serial susceptibility testing.[9] An alternative approach to estimate acquired drug resistance is obviously necessary. A proxy for acquired drug resistance is to measure *Resistance among previously treated patients*. The WHO/IUATLD recommend that this group be subdivided into 4 subgroups and reported as such whenever feasible:

- patients failing anti-TB treatment (treatment failure);
- patients who become smear positive after completion of treatment and declared cured (treatment relapse);
- patients who interrupt their treatment for more than 2 months after having received a total of at least one month of treatment, then returning with bacteriologically confirmed TB (return after default);
- patients who continue to be smear-positive after completion of a treatment regimen (chronic case).

With the minimal clinical information available in the AMRLN database, the terms 'initial' and 'acquired' drug resistance were used in this report recognising the inherent limitations of these terms. More detailed and worthwhile analyses will only be possible when the AMRLN and NMSS databases are linked.

Of 82 patients with drug-resistant disease, 79 (96.3%) were classified as having initial resistance However, 70 (85.4%) of these 82 patients with drug-resistant TB were overseas born. Determining whether migrants have previously received anti-tuberculosis treatment is problematic. Previous medical records are usually not available and drug susceptibility testing facilities are generally not present in the country of origin. The large predominance of supposed initial resistance in this report is unusual and counter-intuitive, and suggests that a significant proportion of patients categorised as having initial resistance actually have acquired resistance. Since the large proportion of initial drug resistance occurs among the overseas born, a better performance indicator for the Australian National TB Control Program would be to monitor patients who relapse or fail to respond after treatment in Australia where the drug susceptibility profiles of the original and subsequent isolates will also be available.

Acknowledgments

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

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

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

Victorian Infectious Diseases Reference Laboratory, North Melbourne.

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

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

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Report on the Second Technical Advisory Group Meeting to Stop TB in the Western Pacific Region, Beijing, China 4–6 June 2001

Angela Merianos, formerly Director, Surveillance and Epidemiology Section, Department of Health and Ageing

"A world free of poverty will remain a mere dream, unless we join hands to overcome major global threats to the poor and marginalized people around the world. Without question, tuberculosis is one such threat, and its control must be on the global development agenda. TB and poverty are closely linked. Poor living and working conditions stimulate transmission and disease, and disease exacerbates economic and social distress... The public health strategy known as DOTS (Directly-observed Treatment, Short course) provides the cornerstone for action in fighting TB. But for such a strategy to stop TB, it must be an integral part of poverty reduction strategies of developing countries, strategies that are of these nations' own conviction and political will, and strategies that are owned by these countries and supported by the international community."

Source: World Bank Vice President for the South Asia region, Mieko Nishimizu at the *Tuberculosis and Sustainable Development Ministerial Conference*, 22–24 March 2000, Amsterdam. World Bank Group News Release No: 2000/254/HD.

Australia is fortunate to have one of the lowest rates of tuberculosis (TB) in the world. Rates have remained consistently around 6 cases per 100,000 population or lower since the 1986, with a rate in the Australian born population of less than 2 cases per 100,000 population.[1] Australia's *National Strategic Plan for TB Control in Australia Beyond 2000*[2] was launched this year on World TB Day (24 March) as a statement of directions and priorities for TB control. The Plan was developed by the National Tuberculosis Advisory Committee (NTAC) of the Communicable Diseases Network Australia and includes a number of verifiable performance indicators against which to monitor the next phase of the TB control program.

The most important issues that currently affect the control of TB in Australia are increased migration from countries with very high rates of TB and socioeconomic inequalities within Australia reflected in higher rates of TB in indigenous Australians. In 1999, the rate of pulmonary tuberculosis in non-Indigenous Australian-born persons was 0.9 cases per 100,000 population compared to 13.6 cases per 100,000 population in the overseas-born and 6.6 cases per 100,000 population in Indigenous Australians.[1] While the proportion of overseas born cases represented in annual TB notifications has increased over the last decade, the rates of TB have not, a testament to the success of our TB control program which provides for free diagnosis and treatment, contact tracing and preventive treatment for eligible Mantoux positive contacts among other strategies. In 1986, 60 per cent of TB notifications were overseas-born, compared to 70 per cent in 1990, 75 per cent in 1996, 77 per cent in 1998 and 83 per cent in 1999.[1] Australia is also fortunate that despite the impact on global TB control of the HIV/AIDS epidemic and the emergence of multi-drug resistant TB (MDR-TB), these factors have had a limited effect on TB control in Australia. Appropriately, one of the recommendations of the National Strategic Plan is to "liaise with regional partners to assist TB control programs in neighbouring countries."[2]

Annually, over 2 million deaths worldwide were attributable to TB, with 95 per cent of these occurring in developing countries. It is estimated that there were over 8 million new cases of TB in 1998 worldwide with over 3.6 million reported to the World Health Organization (WHO) Global Surveillance Program by 189 countries.[3] Of these TB notifications, 39 per cent were managed under the WHO Directly Observed Treatment-Short course (DOTS) strategy for TB control and 1.4 million of these notifications (40%) were new sputum-positive pulmonary cases.[3] There are an estimated 2 million cases of TB in the World Health Organization's Western Pacific Region (WPR), of which some 850,000 are infectious sputum smear-positive cases.[4]

The Region's countries can be grouped according to the burden of tuberculosis and DOTS coverage. There are four groups in the Western Pacific Region (Box),[5] with countries representing some of the

highest and lowest burden countries globally. Nearly one third of the global burden of TB is found in the WPR which has a population of approximately 1.7 billion people and includes seven high burden countries – Cambodia, China, Lao People's Democratic Republic, Mongolia, Papua New Guinea, the Philippines and Viet Nam. China accounts for over half of the regional TB cases followed by the Philippines and Viet Nam. The 1999 estimated average rate of all types of TB in the high burden countries was 50.7 cases per 100,000 population and 25 cases per 100,000 population for new smear positive TB compared to 49.2 and 23.5 respectively for the WPR as a whole. The WHO reported that the case detection rate in the Region in 1999 was 44 per cent and 47 per cent for all types of TB and new smear positive TB respectively, with an estimated 792,914 new smear positive cases in that year. Although overall TB/HIV co-infection rates are still low in the WPR, some countries, notably Cambodia and PNG, have reported an increase in co-infected cases with an expected rise over the next few years.

In 1999 a 'tuberculosis crisis' was declared in the WPR which led to the establishment of a WHO sponsored Special Project to Stop TB.[6] The STOP TB Initiative was presented as a global partnership being mobilised to serve the needs of countries, particularly high burden countries. Participants expressed their interest and expectations that such an initiative would provide them needed support to advocate the case for TB control. As a result, participants developed outline action plans to lobby their governments about pushing TB control higher in the domestic political agenda and participating in building the foundation of the STOP TB Initiative.

A Technical Advisory Group (TAG)[4] comprising of tuberculosis experts from around the world met for the first time in February 2000. The meeting endorsed the Regional Strategic Plan, including the regional objectives to reduce tuberculosis prevalence and mortality by half by 2010. The TAG and Member Countries committed to reach regional targets for the Stop TB Special Project. The Second TAG Meeting, reported here, was held in Beijing in June 2001. In recognition of the increasing incidence of TB/HIV co-infection in the Region, the development of a regional strategy for TB/HIV co-infection control was initiated and the WHO Western Pacific Regional Office (WPRO) participated in the first meeting of the Global TB/HIV Working Group meeting in April 2001.

There is an increasing recognition of the relationship between chronic diseases, such as TB, and a spiralling descent into abject poverty among the infected poor unable to work because of their disease. This has serious implications not only for the affected individual, but also their family, community, and at a population level, to national economic growth and development. Approximately 23 per cent of the world's poor live in the WPR. Globally, over 60 per cent of TB deaths occur among the poorest 20 per cent of the population. At the Second TB TAG Meeting,[4] a presentation by Ms Christy Hanson from the World Bank presented on behalf of the World Bank's East Asia group on 'Poverty and TB Control' raised concerns that although the WHO's DOTS program was a 'pro-poor' initiative in principle, there was a need for further investigation to determine how well DOTS programs were actually reaching the poor and whether health systems and financing policies in some countries were further restricting access to health care by the poor.

The Stop TB Special Project aims to treat all diagnosed patients with TB within the DOTS program. Currently DOTS uptake in the Region is 60 per cent with a wide range between countries. Dr Mario Raviglione, Coordinator of Tuberculosis Strategy and Operations in WHO, Geneva, reported that the WPR notified 25 per cent of sputum smear positive TB cases globally while having the highest treatment success rates of all WHO regions.[4] The cure rate and treatment success rate of new smear-positive cases under DOTS were 93.3 per cent and 94.8 per cent, respectively, in the Region. For China, data strongly influence the regional figures, particularly the high regional treatment success rate (96.6% success rate in DOTS areas in China).[7] However, in order to reach the Region's 2005 target of 70 per cent case detection rate an acceleration of DOTS uptake was required; the target would otherwise be delayed until around 2007.[4] All participants acknowledged the need for even greater political commitment to reaching these targets.

In addition to the ongoing challenge of sustained political and financial support for the Stop TB Program, the current challenges to TB control in the Region were summarised at the Second TAG Meeting as the need to:

- secure additional funding to reduce financial shortfall;
- strengthen DOTS management in-country and secure adequate human resources to run programs effectively;

- strengthen laboratory networks in high burden countries and the Pacific Islands and develop a Regional Laboratory Quality Control Strategy;
- secure a regular uninterrupted supply of TB drugs to treat all diagnosed patients;
- improve TB burden estimates by conducting national TB prevalence surveys;
- improve cure rates to block the emergence of MDR-TB; and
- liaise more effectively with HIV/AIDS control programs at a national and regional level to minimise the risk of TB/HIV co-infection.

Australia's international aid program in health for 2001 to 2002 is estimated at around $205 million of direct health assistance to developing countries, approximately 12 per cent of overall aid expenditure, and is disbursed through the Australian Agency for International Development (AusAID).[8]

Approximately 12 per cent of the health budget will be directed to programs aimed at reducing the burden of communicable and vectorborne diseases, including tuberculosis and malaria. A further 19 per cent is earmarked for the control of sexually transmitted infections, including HIV. In addition, Australia's aid program assists developing countries in reducing their burden of communicable diseases by providing training to professionals in the government and non-government sectors and community-based organisations in health services management, strategic planning, monitoring and evaluation, and in technical aspects of their work, supporting health sector reform and promoting gender and poverty awareness. Australia works bilaterally with partner governments and also funds multilateral organisations such as WHO to achieve health gain in developing countries.

The Third TAG Meeting was held in February 2002 in Osaka, Japan. We await the proceedings of the meeting with interest.

Box. Grouping of countries in the Western Pacific Region according to TB burden

Group 1: *Countries with high tuberculosis burden:*

Cambodia, China, the Lao People's Democratic Republic, Mongolia, Papua New Guinea, the Philippines, Viet Nam.

Group 2: *Countries with intermediate tuberculosis burden and good health infrastructure:*

Brunei Darussalam, Hong Kong, China, Japan, the Republic of Korea, Macao, Malaysia, Singapore.

Group 3: *Pacific Island Countries (PICs) with populations of less than 1 million and initial stage of DOTS implementation:*

All small PICs: American Samoa, Cook Islands, Fiji, French Polynesia, Guam, Kiribati, the Commonwealth of the Northern Mariana Islands, the Marshall Islands, the Federated States of Micronesia, Nauru, New Caledonia, Niue, Palau, the Pitcairn Islands, Samoa, Solomon Islands, Tokelau, Tonga, Tuvalu, Vanuatu, Wallis and Futuna.

Group 4: *Industrialized countries with low tuberculosis burden and low incidence:*

Australia, New Zealand.

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Towards global polio eradication: Australia's commitment to the global eradication of poliovirus by 2005

- Do you have or have you ever stored samples of poliovirus?
- Do you have or have you ever stored samples that might potentially harbour such virus (eg. faecal or throat swab samples)?

If the answer is yes to either of these questions, the stores of poliovirus and potentially infectious materials should be included on a national inventory currently being compiled by the Commonwealth Department of Health and Ageing (the Department) as part of Australia's commitment to the global polio eradication process.

The laboratory containment of poliovirus is a vital step towards Australia receiving World Health Organisation certification as a polio-free nation. The WHO is aiming to globally certify global eradication of poliovirus by 2005. To ensure this happens, the Department needs to make contact with all organisations in Australia that store all existing sources of poliovirus.

Who should contact us?

As Australia's last reported case of locally acquired poliomyelitis was in 1972, the only known sources of poliovirus now remaining are within laboratories. These sources need to be appropriately contained and identified for the purposes of the national inventory.

The Department needs to contact laboratories that may hold:

- stocks of poliovirus;
- reference strains of poliovirus;
- potentially infectious materials such as clinical samples (eg. faecal samples, throat swabs);
- environmental samples (eg. water or sewage samples);
- research materials (eg. wild poliovirus capsid); and
- untyped enterovirus.

If you are an organisation that has laboratories that may store some of the materials listed above and you have not already been contacted by the Department, or you are unsure as to whether or not the samples you hold are potentially infectious, please contact: The Infection Management Section on (02) 6289 8951 or email at jo.pendergast@ health.gov.au for more information and to complete the appropriate questionnaire.

National Strategic Plan For TB Control in Australia Beyond 2000

March 2002

Introduction

Tuberculosis (TB) is a global emergency. The World Health Organization (WHO) estimates 8 million new cases and 2 million deaths occur each year from this treatable disease. This emergency exists for various reasons including: the reduction in health services dealing with the control and treatment of TB; poverty and conflict; migration (both in-country and between-country); the emergence of HIV/AIDS; and the rise of multi-drug resistant (MDR) TB. If control is not further strengthened WHO predicts that between the years 2000 and 2020, nearly one billion people will be newly infected with TB, 200 million people will get sick, and 35 million will die from TB.[1]

Australia has one of the lowest rates of TB in the world. However, specific subgroups, such as Indigenous people and persons born overseas, have rates many times those of non-Indigenous Australian-born persons. The present rates in Australia can be attributed to the improved socio-economic circumstances that occurred over the last century and the success of the post-World War II National TB Campaign. The low rate has been maintained in the presence of large-scale migration from countries with higher TB rates than Australia, largely because of effective pre-migration screening and the activities of specialised, multi-disciplinary TB services in the States and Territories.

Recent difficulties in TB control in other industrialised nations, the lack of a defined national TB structure and a perceived decline in TB expertise within Australia, have highlighted the need for vigilance and continued action. Therefore, in 1999 the Communicable Disease Network Australia (CDNA) endorsed the formation of the National TB Advisory Committee (NTAC) which has representation from each jurisdiction and the Commonwealth. NTAC has two terms of reference:

1. To provide strategic, expert advice to CDNA on a co-ordinated national and international approach to TB control.
2. To develop and review nationally agreed strategic and implementation plans for the control of TB in Australia.

Recommendations

The key recommendations of the National Strategic Plan for TB control are to:

1. Maintain and enhance national surveillance of important epidemiological TB control indices including; disease incidence, drug resistance and treatment outcomes.
2. Ensure all States and Territories:
    - Have programs that are consistent with national TB guidelines.
    - Maintain a close working relationship among public health practitioners, laboratory services and clinical activities in the delivery of TB services.
3. Ensure there continues to be no financial barrier to achieving diagnosis, management and treatment of TB.
4. Continue the commitment of the Department of Health and Ageing to:
    - National TB surveillance.
    - Support NTAC.
    - Liaise with national and international bodies involved in TB control.
    - Ensure that NTAC is consulted by any national body considering issues that may impact on TB control.
5. Co-ordinate screening programs for people from high-risk countries and high-risk groups.
6. Improve awareness-raising of TB among health care workers in order to promote early detection.
7. Maintain a national advisory committee for TB that includes representatives with expertise in TB control from each of the States and Territories and the Commonwealth Mycobacteriology Reference Laboratory Network, and which reports to the Communicable Disease Network Australia.
8. Liaise with regional partners to assist TB control programs in neighbouring countries.

TB control in Australia

The most important issues that currently affect the control of TB in Australia are increased migration from countries with very high rates of TB and socio-economic inequalities within Australia. Despite the impact on global TB control of the HIV/AIDS epidemic and the emergence of MDR-TB, to date these factors have had a limited effect on TB control in Australia.

WHO recommends the 5-point strategy known as Directly Observed Treatments - Short Course (DOTS) to control TB world-wide. This strategy is implemented for TB control in Australia with suitable modifications for a low incidence industrialised country. The continued success of the National TB Control Program requires States, Territories and the Commonwealth to work together to ensure that their complementary roles are successfully undertaken and that Australia remains a country with one of the lowest rates of TB in the world.

Currently, States and Territories have responsibility for the provision and management of TB services in Australia and for ensuring a close working relationship between public health laboratories, clinicians and TB services. TB reference laboratories have a range of responsibilities including the undertaking of antibiotic susceptibility testing. The Commonwealth monitors the incidence and prevalence of TB on a national basis using information provided by State and Territory health authorities and laboratory services.

The Department of Health and Ageing needs to continue to maintain national TB surveillance and to liaise with national bodies considering issues which will impact on TB control, by referring them to NTAC for review.

Given that the natural history and transmission of TB predicates against the elimination of TB in a single geographical area, it is important that Australia has a vision for TB control globally and a commitment to be involved and assist in reducing the regional burden of TB.

Aim

The aim of the TB control program in Australia is to minimise the burden and human impact of TB and prevent the transmission of TB through early detection and treatment.

Goals

The goals of the National TB control program are to:

- Eliminate the transmission of TB in Australia.
- Reduce the incidence of TB in the Indigenous Australian population to that of the Australian-born non-Indigenous population.
- Reduce the burden of disease from TB in overseas-born persons and other high-risk groups.
- Maintain the current low level of drug resistance in Australia.
- Foster research and development in TB control.
- Strengthen partnerships that contribute to the global control of TB with particular emphasis on regional countries.

Strategies

The three key strategies to advance the goals of the National TB control program are:

- Active and passive case finding that allows the early and accurate diagnosis of persons with TB through effective clinical and laboratory services.
- Prompt and effective free treatment of persons with active TB in supervised programs.
- Timely surveillance and reporting of disease incidence, drug resistance and treatment outcomes nationally to inform program evaluation.

Other important strategies that assist in achieving the goals are:

- Effective contact tracing.
- Pre-migration screening and treatment.
- Targeted screening of high-risk groups.
- Appropriate mycobacteriological investigations, which includes susceptibility testing from all cases of TB.
- Appropriate use of diagnostic molecular methods.
- Appropriate treatment of latent TB infection.
- Use of BCG vaccination in specific subgroups.
- Education and training of health care workers and other key stakeholders.
- Increasing the awareness of TB through education and empowerment of high-risk groups and their families/carers.

- Advocacy for social and economic issues.
- Advocacy for and an active role in assisting TB control programs in countries in our region including programs to monitor MDR-TB.
- Undertaking research and development consistent with the goals of the TB control program.

Performance Indicators

National TB performance indicators have been developed and need to be regularly reviewed at both the State and National level. States and Territories may need to develop other indicators by which to assess additional aspects of their TB control programs, such as delays in diagnosis and quality of public health response.

All indicators can be measured as part of the expanded data collection for TB.

Annual incidence of TB in:

- General population.
- Overseas-born persons.
- Australian-born persons.
- Indigenous Australians.
- Relapse cases initially treated in Australia.
- Children less than 15 years of age analysed by risk group.
- HIV sero-positive persons.

Laboratory surveillance measures (percentage of):

- Pulmonary cases which are sputum-smear positive.
- Total notifications where laboratory culture of sputum was attempted.
- Total notifications confirmed by culture.
- Culture-confirmed cases for which susceptibility testing has been performed.
- Culture-confirmed cases with drug resistance strains including MDR organisms.
- Sputum-smear positive cases that are sputum negative by the third month of treatment.

Treatment outcome measures (percentage of):

- Cases evaluated for outcomes.
- Cases that have had treatment completed and are cured.
- Cases recorded as treatment failures.

Completed incident, laboratory and outcome data provided by all States and Territories by September of the following year.

Publication of the TB annual report including disease incidence, outcome measures and drug-resistance testing by December of the following year.

TB data provided to WHO annually or as required.

- Annual reporting on regional/global TB activities.

Epidemiology

Prior to 1950 the incidence of active TB in Australia was over 45 notified cases per 100,000 population. During the period of the National TB Campaign from 1950 to 1976 there was a rapid and sustained decline in the notification rate of new cases[2] despite continued large-scale migration from countries with higher TB rates than Australia.[3,4]

Since 1986, there has been a slowing in the rate of decline to plateau at 4.9 to 5.7 new notifications per 100,000 population. These low rates compare favourably with other developed countries.[5] Over the same period the mortality from TB has declined substantially from 10 per 100,000 population in 1954 to 0.2 per 100,000 population in 1999.[2,5]

Of the 1,159 new cases notified in 1999, 18 percent were in Australian-born persons (1.2 cases per 100,000 population) and 82 per cent were in overseas-born persons (20.6 cases per 100,000 overseas-born population) predominately from south-east Asian countries.[6] TB notifications in the overseas-born population occurred equally in males and females and across all age groups peaking in early adulthood. The rates were much higher than those of the Australian-born population across all age groups.

The rate of TB in Indigenous Australians is higher than in non-Indigenous Australians. In 1999 the rate was estimated to be approximately 8.3 per 100,000 population,[6] ie eight times greater than in non-Indigenous Australians. Indigenous Australians have higher rates of infection, disease, hospitalisation and mortality from TB than non-Indigenous Australians.[7] These data should be interpreted recognising that the numbers are small and Indigenous status reporting is incomplete. The overall rate in Indigenous Australians also masks significant geographic variability in the incidence of

TB amongst Indigenous Australians, e.g. in Far North Queensland the rate in Indigenous Australians is 16 times that of non-Indigenous Australians in that region.[8]

In 1999, 767 (66%) of the notifications were pulmonary disease and 37 per cent of these were sputum-smear positive. Notification rates of new extra-pulmonary disease have declined since the 1980s, although there has been a slowing in the rate of decline similar to that seen in pulmonary disease.[2,6]

Although high rates of drug resistant TB have been reported overseas, drug resistance in Australia has remained low. Between 1989 and 1992 average annual resistance rates of 14 per cent were recorded for all isolates tested including 8 per cent to isoniazid alone or in combination, 1.9 per cent to rifampicin alone or in combination and 0.8 per cent to both isoniazid and rifampicin in combination.[9] Published data for 1993 to 1998 show no notable changes in the prevalence of drug-resistant strains in Australia, and the average proportion of MDR isolates (defined as resistance to both isoniazid and rifampicin) has remained below one per cent.[10]

HIV/AIDS is becoming increasingly recognised as an important co-factor for TB. Internationally, TB is one of the major causes of death amongst people infected with HIV. However, several studies concerning HIV/AIDS and TB in Australia show that while the risk of TB is high for people infected with HIV, the absolute numbers remain small.[11,12,13] As such, while it is expected the numbers of TB/HIV co-infection would remain low in view of the low rates of both HIV and TB, this needs to be closely monitored as the increased potential exists in certain TB risk groups with adverse social, economic and environmental conditions.

In summary, Australia currently has an enviable position with low rates of disease, low rates of MDR-TB and relatively little overlap between the TB infected communities and the HIV community. However, continued vigilance and effective control programs are required to reduce the human impact of TB in the sub-groups of the population who are particularly at risk of this disease, and to quell the possible slow emergence of drug resistance.

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Further copies of this document are available from the Internet at: http:/www.health.gov.au/pubhlth/cdi/pubs/tb-plan.htm

Annual report of the Australian Gonococcal Surveillance Programme, 2001

The Australian Gonococcal Surveillance Programme

Abstract

The Australian Gonococcal Surveillance Programme monitors the antibiotic susceptibility of *Neisseria gonorrhoeae* isolated in all States and Territories. In 2001 the *in vitro* susceptibility of 3,641 isolates of gonococci from public and private sector sources was determined by standardised methods. Antibiotic susceptibility patterns again varied considerably between regions. Resistance to the penicillins remained high in larger urban centres and warrants close attention in those rural centres where treatment with the penicillins continues. Quinolone resistance in gonococci (QRNG) became more widespread in Australia in 2001 and MICs increased. Nationally, 17.5 per cent of all isolates were QRNG. Endemic cycles of transmission of QRNG in homosexually active men declined further, but heterosexual transmission increased substantially. All isolates remained sensitive to spectinomycin. A small number of isolates in a number of jurisdictions again showed some decreased susceptibility to ceftriaxone. A high proportion of gonococci examined in larger urban centres were from male patients, and rectal and pharyngeal isolates were common. In other centres and in rural Australia the male to female ratio of cases was lower, and most isolates were from the genital tract. *Commun Dis Intell* 2002:26:242-247.

Keywords: Surveillance, Neisseria gonorrhoeae, *antimicrobial resistance, gonorrhoea, antibiotics, quinolones, penicillins, spectinomycin, cephalosporins*

Introduction

The Australian Gonococcal Surveillance Programme (AGSP) was established in 1979 to monitor the susceptibility to antibiotics of gonococci isolated throughout the country. The need for such a program is arguably now more important than ever as both the rates of gonococcal disease and levels of antibiotic resistance in *Neisseria gonorrhoea* continue to increase.

The AGSP is a long-term collaborative program conducted by reference laboratories in each State and Territory. Data from this program were published in *Communicable Diseases Intelligence* (*CDI*) from 1981 and annual reports have been produced since 1996. Prior to 1996, consolidated data were published elsewhere. This report is based on data obtained during the 2001 calendar year.

Methods

The AGSP is a component of the National Neisseria Network of Australia and comprises participating laboratories in each State and Territory (see acknowledgments). This collaborative network of laboratories obtains isolates for examination from as wide a section of the community as possible and both public and private sector laboratories refer isolates to regional testing centres. Although the sources of isolates remained relatively unchanged in 2001, the increasing use of non-culture based methods of diagnosis reduces the number of cultures available for susceptibility testing, and the details of the numbers of organisms examined are provided in order to indicate sample size and not disease incidence.

Gonococci isolated in and referred to the participating laboratories were examined for antibiotic susceptibility to the penicillins, quinolones, spectinomycin and third generation cephalosporins and for high level resistance to the tetracyclines by a standardised methodology.[1] The AGSP also conducted a program-specific quality assurance (QA) program.[2] Antibiotic sensitivity data were submitted quarterly to a coordinating laboratory which collated the results and also conducted the QA program. Additionally, the AGSP received data on the sex of the patient and site of isolation of gonococcal strains. The geographic source of acquisition of resistant strains was ascertained whenever possible.

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Results

Numbers of isolates

There were 3,725 gonococcal isolates referred to or else isolated in AGSP laboratories in 2001. The number and percentage of isolates from each State and Territory (except for the Australian Capital Territory and Tasmania, n=19) for 1999 to 2001 are shown in Table 1.

The source and site of infection of these isolates are shown in Table 2. Of these 3,641 remained viable for susceptibility testing.

Compared with data from the same sources in recent years, there were decreases in the number of isolates tested from Victoria (from 744 in 1999 and 802 in 2000 to 701) and in Western Australia. Increases were recorded in South Australia (124) and New South Wales (1,505). The number of isolates available from the Northern Territory and Queensland was stable. Numbers in other centres were low.

Table 1. Trends in sample size of gonococcal isolates analysed by the AGSP, Australia (except the Australian Capital Territory and Tasmania) 1999 to 2001, by State or Territory

Jurisdiction	Number (% of annual total) gonococcal isolates					
	1999		2000		2001	
	n	%	n	%	n	%
New South Wales	1,528	41.0	1,255	35	1,505	40
Northern Territory	453	12.1	445	13	460	12
Queensland	589	16.0	620	17	619	17
South Australia	93	2.5	93	3	124	4
Victoria	744	20.0	802	23	701	19
Western Australia	313	8.4	317	9	297	8
Total	**3,720**		**3,532**		**3,706**	

Note: data on the number and source of isolates tested are reported to provide information on the sample available for susceptibility testing, not disease incidence.

Table 2. Source and sample size of gonococcal isolates tested for antibiotic susceptibility, Australia, 2001, by sex, site and State or Territory*

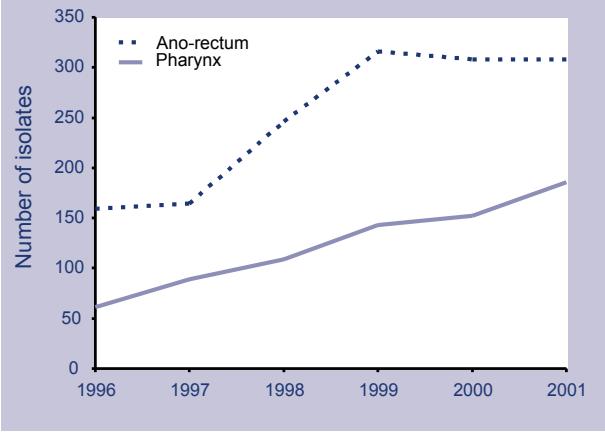
	Site	NSW	NT	Qld	SA	Vic	WA	Aust
Male	Urethra	1,040	270	408	61	539	222	2,550
	Rectal	206	1	26	13	50	9	308
	Pharynx	126	3	9	11	35	0	186
	Other/NS	34	47	16	7	22	2	130
	Sub total	1,406	321	459	92	646	233	3,174
Female	Cervix	87	129	153	30	44	62	507
	Other/NS	12	10	7	2	11	2	44
	Subtotal	99	139	160	32	55	64	551
Total		**1,505**	**460**	**619**	**124**	**701**	**297**	**3,725**

* The site of isolation of some infected patients was not known.

Source of isolates

There were 3,174 strains isolated from men and 551 strains isolated from women, giving a male to female (M:F) ratio of 5.7:1. This ratio changed little from the previous year. The M:F ratio remained highest in New South Wales (14.2:1) and Victoria (11.7:1) where a higher proportion of strains were obtained from urban populations. The lower ratios in Western Australia (3.6:1), Queensland (2.8:1) and the Northern Territory (2.3:1) reflected the large non-urban component of gonococcal disease in those regions. Male rectal and pharyngeal isolates were most frequently found in New South Wales (23% of isolates from men) and Victoria (13%). This pattern is similar to that noted in recent years, but may also reflect clinical sampling practices in those States (Figure 1). About 5 per cent of isolates are shown as being isolated from 'other' sites. These included 13 cases of disseminated gonococcal infection, most of these in men in New South Wales. Not all infected sites were identified. Isolates from urine samples were regarded as genital tract isolates. Most of the other unidentified isolates were probably from this source, although they were not so specified. There were a small number of isolates from the eyes of both newborn and older infants and also adults.

Figure 1. Male rectal and pharyngeal isolates by year 1996 - 2001, in New South Wales and Victoria



Antibiotic susceptibility patterns

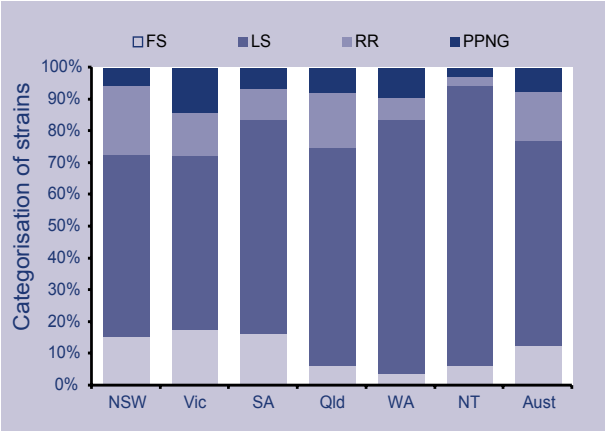
In 2001, the AGSP reference laboratories examined 3,641 gonococcal isolates for sensitivity to penicillin (representing this group of antibiotics), ceftriaxone (representing later generation cephalosporins), ciprofloxacin (representing quinolone antibiotics) and spectinomycin and for high level resistance to tetracycline (TRNG). As in past years the patterns of gonococcal antibiotic susceptibility differed between the various states and territories. For this reason data are presented by jurisdiction as well as aggregated for Australia as a whole.

Penicillins

Resistance to the penicillin group (penicillin, ampicillin, amoxycillin) may be mediated by the production of beta-lactamase (penicillinase-producing *N. gonorrhoeae* - PPNG) or by chromosomally-controlled mechanisms (CMRNG).

Chromosomal resistance is expressed as the minimal inhibitory concentration in mg/L (MIC) which is the least amount of antibiotic which inhibits *in vitro* growth under defined conditions. The categorisation of strains in Australia in 2001 by penicillin MIC is shown in Figure 2. The MIC reflects the expression of multiple and different chromosomal changes present in an organism.[3] These multiple changes result in incremental increases in the MIC and strains are classified as fully sensitive (FS, MIC ≤ 0.03 mg/L), less sensitive (LS, MIC 0.06 - 0.5 mg/L) or relatively resistant i.e. CMRNG (RR, MIC ≥ 1 mg/L). PPNG are a separate (resistant) category. Infections with strains in the less sensitive or fully sensitive categories usually respond to therapy with standard treatment regimens with the penicillins. Infections caused by strains which are PPNG or in the relatively resistant category (CMRNG) usually fail to respond to treatment with the penicillins.

Figure 2. Penicillin resistance of gonococcal isolates, Australia, 2001 by jurisdiction



FS	Fully sensitive to penicillin, MIC ≤ 0.03 mg/L
LS	Less sensitive to penicillin, MIC 0.06 – 0.5 mg/L
RR	Relatively resistant to penicillin, MIC ≥ 1 mg/L
PPNG	Penicillinase producing *N. gonorrhoeae*

The number (558) and proportion (15.3%) of isolates resistant to penicillin by CMRNG, in 2001 was higher than the 377 (10.6%) recorded in 2000 but approximated the 525 (14.3%) recorded in 1999. Strains of this type were concentrated in New South Wales (321 CMRNG, 21.5% of all isolates) Victoria (91 CMRNG, 13.4%), and Queensland (101 CMRNG, 17.3%). An increase in CMRNG was noted in Western Australia to 20 (6.9%) from 6 (2%) CMRNG in 2000. In the Northern Territory, 13 strains represented 2.9 per cent of all isolates, twice the number and proportion seen in 2000. In South Australia, nearly 10 per cent of isolates were CMRNG.

PPNG decreased slightly in 2001 both in number and as a percentage of all isolates (to 274 and 7.5% from 302 and 8.7% in 2000) but was similar to the number and proportion of PPNG in 1999. The distribution of PPNG differed significantly by jurisdiction. Victoria had the highest number (96) and proportion (14.2%) of PPNG. New South Wales had 86 PPNG (5.7%), Queensland 45, (7.7%), Western Australia 27, (9.3%) and South Australia 8, (6.5%). Twelve PPNG were found in the Northern Territory (2.7%). PPNG were acquired locally in a minority of cases and contact in countries close to Australia accounted for the source of most PPNG infections. Indonesia, the Philippines, Thailand, Vietnam and China were the most frequently nominated countries of PPNG acquisition. PPNG acquisition was also reported from contact in Dubai, Ethiopia, Korea, Malaysia, Mexico, Norway, Russia, Singapore, Hong Kong, Cambodia and Taiwan.

Ceftriaxone

Ceftriaxone resistance leading to treatment failure has yet to be encountered. A small but increasing number of strains in a number of jurisdictions showed a small increase in ceftriaxone MICs. The mechanisms by which these alterations occurred have not been determined but are presumed to be the result of changes in penicillin binding sites.

Spectinomycin

All isolates were susceptible. Resistance most often occurs as a result of a single step ribosomal change.

Quinolone antibiotics

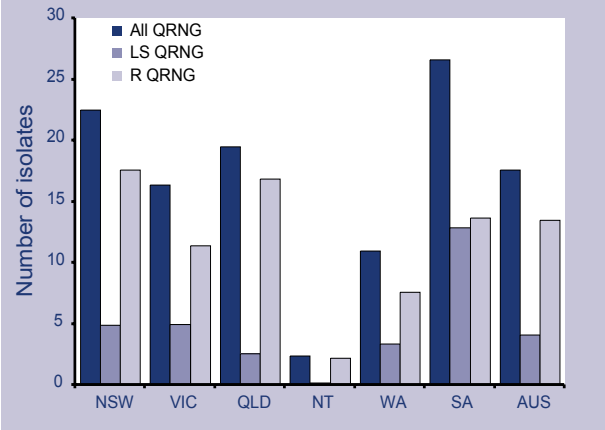
Resistance to the quinolone antibiotics is mediated only by chromosomal mechanisms so that incremental increases in MICs are observed. The AGSP uses ciprofloxacin as the representative quinolone and defines altered resistance as an MIC of 0.06 mg/L or more. Treatment with currently recommended doses of 500 mg of ciprofloxacin is effective for strains with this level of developed resistance in about 90 per cent of cases, but lower doses of the antibiotic will more often result in treatment failure. The proportion of treatment failures increases exponentially as MICs rise. Treatment failure occurs in about 60 per cent of infections with strains with MICs of 1 mg/L or more, even when higher doses are used. Currently, gonococci with MICs up to 16 and 32 mg/L are being seen in Australia.

In 2001, a total of 638 gonococci (17.5%) displayed altered sensitivity to the quinolones (QRNG) (Figure 3). This is about the same number and proportion of QRNG seen in 2000 (619, 17.8%) and 1999 (628, 17.2%) but more than the three times the number of QRNG seen in 1998 (186, 5.2%). Up to 1999, QRNG were particularly concentrated in homosexually active men (HAM) in New South Wales and Victoria. In 2001, the distribution of QRNG became more widespread, with larger numbers found in more jurisdictions while a slight decline in QRNG numbers occurred in the two larger States. The QRNG seen previously in HAM were predominantly in the lower MIC range, namely, 0.06 – 0.5 mg/L. In 2001 in all centres, the predominant QRNG were those with MICs of 1 mg/L or more.

In South Australia the 33 QRNG represented 26 per cent of all gonococci and 114 QRNG represented 20 per cent of all isolates in Queensland. The 337 QRNG in New South Wales comprised 22.5 per cent of all isolates. In Victoria 111 QRNG accounted for 16.4 per cent of all isolates and 32 QRNG represented 11 per cent of gonococci in Western Australia. The Northern Territory recorded 11 QRNG (2.4%).

In most centres endemic transmission of QRNG was prominent. Importation of QRNG continued from many countries, and as for PPNG was most often from acquisition in nearby countries.

Figure 3. Percentage of gonococcal isolates which were less sensitive to ciprofloxacin* or with higher level ciprofloxacin resistance* and all strains with altered quinolone susceptibility, by jurisdiction, Australia, 2001



* LS QRNG, MIC 0.06 – 0.5 mg/L
R QRNG, MIC 1 mg/L or more

High level tetracycline resistance

The spread of TRNG is examined as an epidemiological marker of drug resistance and tetracyclines are not a recommended treatment for gonorrhoea. Three hundred and forty-three high level tetracycline resistant *N. gonorrhoeae* (TRNG, 9.4 % of isolates) were detected throughout Australia in 2001, similar to that observed in 2000. Most TRNG were found in New South Wales where local spread was more evident. Victoria had the highest proportion of TRNG (98 TRNG, 14.4%) with Queensland 78 TRNG (13.3 %). Lower numbers and proportions were found in Western and South Australia, the Australian Capital Territory and the Northern Territory. Overall the number of TRNG detected altered little in 2001.

Discussion

Patterns of gonococcal disease in Australia show considerable regional differences. A considerable proportion of the gonorrhoea contracted in larger urban centres is in HAM and antibiotic resistant gonococci are often encountered. In contrast, gonorrhoea in rural settings is more often heterosexually transmitted and antibiotic resistance is a lesser concern.[4] The considerable regional variation in susceptibility of gonococci to antibiotics noted in AGSP reports over many years was again present in 2001. Standard treatment regimens are thus best derived from a consideration of local patterns of susceptibility rather than aggregated national data.

The World Health Organization guidelines suggest that a rate of gonococcal resistance to an antibiotic of 5 per cent or more is an indication to change treatment schedules, although this 'acceptable' resistance rate is much lower in groups with a high rate of disease transmission. A high proportion of the gonococci isolated in urban centres has been resistant to the penicillins for many years. This situation was unaltered in 2001, and these agents should not be used in these settings. Rates of penicillin resistance in New South Wales, Victoria, South Australia, Queensland and Western Australia ranged between 16 and 27 per cent. Most of this resistance was chromosomally mediated, but in Western Australia and Victoria PPNG were prominent. The proportion of CMRNG in the Northern Territory remains low, but there has been a continuing shift upwards in MICs so that close surveillance needs to be maintained if penicillins are to remain the preferred treatment option.

Quinolone resistance remained a major problem in 2001. More QRNG were found in more centres and the MICs were higher. High levels of endemic transmission of QRNG continue together with continuing importation of QRNG from other countries. This suggests that the spread of QRNG in the regions close to Australia, noted in WHO based surveillance,[5] continues to be relevant to treatment of individuals who acquire gonorrhoea overseas but present locally. The increasing numbers of locally and overseas acquired QRNG severely diminishes the effectiveness of quinolone based treatments for gonorrhoea in Australia. Newer quinolone agents, while marginally more effective for some types of QRNG, are unlikely to be sufficiently efficacious for satisfactory treatment of gonorrhoea in Australia.[6]

Most gonococcal isolates were fully susceptible to the third generation cephalosporin, ceftriaxone, although an increasing number of strains had slightly increased MICs. This too is a trend noted in other reports,[5,7] and warrants close monitoring. It is emphasised that there have been no treatment failures with later generation cephalosporins attributable to antibiotic resistance. However, because of decreasing efficacy of quinolones, this group of antibiotics is now the first line treatment for gonorrhoea in a number of Australian centres.

The sample of available isolates in 2001 was maintained, and was sufficient for the purpose of susceptibility surveillance. However, the increasing use of non-culture based methods for the diagnosis of gonorrhoea may well pose significant problems in the future unless there is a continuing

commitment to maintaining culture-based systems. Use of non-culture based diagnostic methods has meant that commentary by the AGSP on comparative rates and trends in gonorrhoea is now difficult. It is now established however, that rates of gonorrhoea are again increasing in developed countries, and this coupled with emergence and spread of antibiotic resistance in *Neisseria gonorrhoeae* suggests that efforts for control of this disease must be vigorously maintained. An important element in control is the use of optimal antibiotic treatment and this is best determined by use of data from programs of antibiotic susceptibility surveillance.

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OzFoodNet: enhancing foodborne disease surveillance across Australia: Quarterly report, October to December 2001

The OzFoodNet Working Group[1]

Introduction

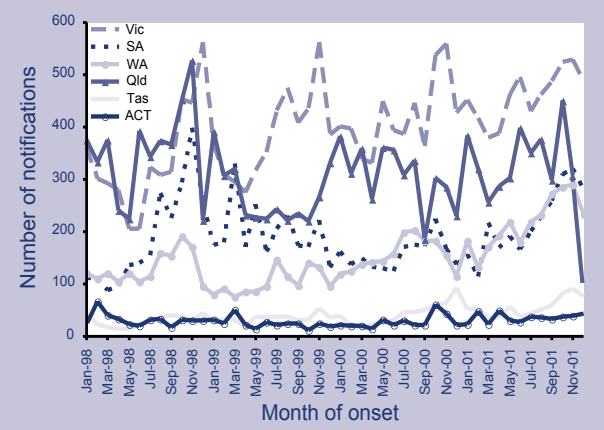
OzFoodNet is a collaborative network of epidemiologists and microbiologists conducting applied epidemiological research into foodborne disease and improving existing surveillance mechanisms for foodborne disease. The Commonwealth Department of Health and Ageing established OzFoodNet in 2000 and the network has had representation on the Communicable Diseases Network Australia (CDNA) since 2001. All Australian jurisdictions collaborate in OzFoodNet. The New South Wales Health Department has enhanced surveillance in the Hunter Region. The Northern Territory participates as an observer and data are only included where specified. Historical comparisons use notifications by date of onset. All other data are reported using the date the report was received by the health agency.

This fourth quarterly report of OzFoodNet summarises the incidence of foodborne disease in the 6 States of Australia and the Australian Capital Territory between October and December 2001. During the fourth quarter of 2001, OzFoodNet continued to collect data on the incidence of diarrhoea and it's causes around Australia.

Notifications in the fourth quarter

In the last 3 months of 2001, notifications of *Campylobacter* infection were elevated across Australia, with the major peak in October and November (Figure). In recent years *Campylobacter* incidence has continued to increase in Australia, which follows similar patterns in other countries. Infections are most common in Spring and Summer. During the fourth quarter 2001, OzFoodNet sites reported 4,492 notifications of campylobacteriosis, which represented a 20.2 per cent increase over the mean for the same quarter for the years 1998 to 2000. For Western Australia and Tasmania, the increase over the mean for 1998 to 2000 was 89 per cent and 67 per cent respectively. The median age of cases ranged between 29 to 33 years. All States reported that the male to female ratio of cases ranged from 1.2:1 to 1.4:1. There were two separate outbreaks of *Campylobacter* infection in Victoria which affected 76 people in total.

Figure. Notifications of campylobacteriosis in OzFoodNet Sites, by month of onset, 1998 to December 2001



OzFoodNet sites reported a total of 1,537 cases of salmonellosis during the fourth quarter, which represented a 6.6 per cent increase over the mean for the same quarter for the years 1998 to 2000. The median age of cases ranged from 15 to 36 years in OzFoodNet sites. The median age of cases in Queensland was higher (15 years) compared to

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the previous two quarters (9.0 years). The male to female ratio was approximately 1:1 in all sites, except Tasmania (1:1.6) and South Australia (1:1.3) where females predominated. Sites reported five *Salmonella* outbreaks during the quarter.

During the quarter, there were 5 serovars that were among the most common in three or more States: *Salmonella* Typhimurium (phage types 9, 126, 135 and 170), and S. Infantis (Table 1). Health departments across Australia continued to receive *Salmonella* Typhimurium phage type 126 notifications. In October, the Victorian Department of Human Services reported an increase in *S*. Typhimurium 170. Increases in this serovar were also reported for New South Wales and Queensland. Queensland reported that there were 29 cases of *S*. Birkenhead this quarter compared to 25 cases for the same time period in 2000. There is an endemic focus of this serovar in the southern region of Queensland and northern parts of New South Wales. During the first quarter of 2002, the Hunter OzFoodNet site also reported notifications of *S*. Birkenhead. The National Enteric Pathogen Surveillance Scheme reported that during the fourth quarter of 2001 the five most common *Salmonella* infections nationally were *S*. Typhimurium phage types 135, 126, 170 and 4, and *S*. Virchow 8. (Personal communication. Joan Powling, University of Melbourne, 17 January 2002).

State health departments received 14 notifications of listeriosis during the fourth quarter of 2001, which was similar to the mean number of notifications for the last 3 years (15 cases). Queensland reported five of these cases, four of which were in older people and one materno-foetal case. Western Australia reported 3 cases, two of which were pregnancy-associated infections in foetuses at 15 and 23 weeks gestation, respectively. Nationally, the range of ages for non-pregnancy associated cases was 36–89 years old.

OzFoodNet sites reported 11 cases of shiga toxin producing *E. coli* infections during the quarter; with cases notified from South Australia (5), Western Australia (2), Queensland (3), and New South Wales (1). There were no common links identified between cases, except for one case in Western Australia. The Western Australian Health Department reported isolation of *E. coli* O157:H7 from a patient stool and a locally produced salami product containing a mix of meats. The age range of all cases was 1 to 76 years, with females predominating (1:1.4). New South Wales reported one case of haemolytic uraemic syndrome in an 11-month-old male.

OzFoodNet sites reported that during the quarter there were 74 cases of shigellosis, 14 cases of typhoid, and 10 cases of yersiniosis.

Foodborne disease outbreaks

During the fourth quarter of 2001, OzFoodNet sites reported 28 outbreaks of gastrointestinal infections with a probable food source, affecting an estimated 990 people, of whom 11 were hospitalised (Table 2). There were no reported deaths from these outbreaks.

There were several outbreaks associated with pre-Christmas functions, often where the meals were catered. This may be due to an overall increase in the number of functions, or unsafe practices used during this time. At least 10 of the 28 outbreaks were associated with catered events. Six of the outbreaks were associated with meals served at restaurants or hotels.

The Australian Capital Territory reported 4 outbreaks of gastroenteritis that were associated with meats cooked on a spit roast. All 4 meals were prepared by the same company in Sydney and transported to the Australian Capital Territory the next day. Investigators did not identify a responsible agent, but suspected bacterial toxins as the cause.

The South Australian Department of Human Services reported an outbreak of *S*. Typhimurium 135a associated with a dessert containing raw eggs. This was the fourth outbreak associated with eggs during 2001, two of which were due to *Salmonella* Typhimurium 135.

Table 1. Top five *Salmonella* infections reported to OzFoodNet sites, October to December 2001, by date of receipt of notification at each health department

OzFoodNet site	Top 5 Salmonella infections	Number of notifications				
		4rd Qtr 2001	4rd Qtr 2000	Total 2001	Total 2000	Ratio*
ACT	S. Typhimurium 9	3	3	10	31	1.0
	S. Bovismorbificans 14	2	0	4	0	–
	S. Enteritidis RDNC 11	2	0	2	0	–
	S. Typhimurium 64	1	0	1	1	–
	S. Para B bv Java Dundee	1	0	2	1	–
Hunter	S. Typhimurium 135	8	4	15	10	2.0
	S. Birkenhead	4	9	5	9	0.4
	S. Typhimurium U290	3	0	3	0	–
	S. Typhimurium 126	2	2	9	3	1.0
	S. Typhimurium 64	2	3	9	14	0.7
New South Wales	S. Typhimurium 135	48	34	202	115	1.4
	S. Typhimurium 126	34	17	98	56	2.0
	S. Birkenhead	27	30	87	73	0.9
	S. Typhimurium 9	23	38	133	138	0.6
	S. Typhimurium 170	14	3	35	8	4.7
Queensland	S. Saintpaul	43	27	164	184	1.6
	S. Typhimurium 135	38	35	137	118	1.1
	S. Virchow 8	32	36	177	189	0. 9
	S. Birkenhead	29	25	130	102	1.2
	S. Typhimurium 126	21	0	73	2	–
South Australia	S. Typhimurium 126	23	2	110	5	11.5
	S. Typhimurium 64var	21	0	21	0	–
	S. Typhimurium 108	20	3	31	11	6.7
	S. Typhimurium 9	11	6	49	26	1.8
	S. Infantis	8	0	19	8	–
Tasmania	S. Mississippi	12	22	98	73	0.5
	S. Typhimurium 9	2	11	11	22	0.2
	S. Typhimurium 135	2	2	5	5	1.0
	S. Infantis	2	1	3	4	2.0
	S. Saintpaul	2	1	2	2	2.0
Western Australia	S. Typhimurium 135	15	2	80	68	7.5
	S. Chester	9	4	31	12	2.3
	S. Typhimurium 9	8	0	15	14	–
	S. Stanley	8	3	21	5	2.7
	S. Kiambu	5	0	20	9	–
Victoria	S. Typhimurium 170	46	11	73	36	4.2
	S. Typhimurium 9	22	41	127	186	0.5
	S. Typhimurium 135	19	9	96	70	2.1
	S. Typhimurium 4	14	23	79	37	0.6
	S. Infantis	8	3	28	14	2.7

* Ratio of cases for the fourth quarter 2001 to the fourth quarter 2000

Table 2. Outbreaks reported by OzFoodNet sites, October to December 2001

State	Month of outbreak	Setting	Agent responsible	Number exposed	Number affected	Evidence*	Epidemiological study	Responsible vehicles or mode of transmission
ACT	Dec	Catered event	Suspected toxin	24	22	D	Case series	Spit roast
	Dec	Catered event	Suspected toxin	141	63	D	Case series	Spit roast
	Dec	Catered event	Suspected toxin	130	50	D	Case series	Spit roast
	Dec	Catered event (school)	Suspected toxin	56	31	D	Not done	Spit roast (at school)
	Dec	Restaurant	Suspected toxin	43	15	D	Case series	Unknown
	Oct	Conference centre	Campylobacter	129	48	S	Cohort	Tomato and cucumber salad
Hunter	Oct	Conference	Escolar wax-ester oils	47	20	D	Cohort	Escolar
QLD	Nov	Home	Ciguatoxin	4	4	D	Case series	Coral trout
	Nov	Home	Ciguatoxin	9	9	D	Case series	Spanish mackerel
	Dec	Hotel	Unknown	35	6	D	Case series	Unknown
SA	Nov	Function centre	Human calicivirus	331	90	D	Case series	Unknown
	Nov	Private function	Human calicivirus	15	13	D	Case series	Unknown
	Nov	Restaurant	Suspected viral	47	44	D	Case series	Unknown
	Dec	Private function	S.Typhimurium 135a	19	11	S, M	Cohort	Tiramisu dessert
	Dec	Restaurant	S. Typhimurium 64 var	Unknown	28	S, M	Cohort	Mango pudding
Vic†	Nov	Aged care	Campylobacter	243	49	D	Case series	Unknown
	Dec	Restaurant	Unknown	115	33	D	Cohort	Unknown
	Dec	Hotel	C. perfringens	9	9	D	Case Series	Suspected potato and bacon soup
	Dec	Private residence	S. Virchow 34	17	11	M	Cohort	Barbequed chicken or beef
	Dec	Convention centre	Suspected toxin	533	269	D	Cohort	Suspected soup or roast beef
	Dec	Community	S. Mississippi	Unknown	6	D	Case series	Suspected seafood
	Dec	Community	S. Hvittingfoss	Unknown	5	D	Case series	Unknown
WA	Oct	Wedding	Unknown	93	50	D	Cohort	Unknown
	Nov	Fast food restaurant	Unknown	21	10	D	Cohort	Unknown
	Dec	Catered event	Norwalk-like virus	36	23	D	Cohort	Unknown
	Dec	Catered event	Norwalk-like virus	90	56	D	Cohort	Unknown
	Dec	Christmas breakfast	Unknown	14	4	D	Cohort	Unknown
	Oct	Respite farm for handicapped	Human calicivirus	31	11	D	Cohort	Unknown

* D = Descriptive evidence implicating the suspected vehicle or suggesting foodborne transmission;
S = Statistical association between illness and one or more foods;
M = Microbiological confirmation of agent in the suspect vehicle and cases.

† In Victoria, where investigators conducted a cohort study and did not interview the whole cohort, the number affected is calculated from the proportion of people interviewed that were ill multiplied by the number exposed.

Applied research

In the fourth quarter of 2001, three OzFoodNet sites interviewed patients infected with *Campylobacter* and controls for the national *Campylobacter* case control study. The national *Salmonella* Enteritidis case control study, which will identify travel history from human cases, and examine risk factors for infection also commenced this quarter. OzFoodNet sites also commenced case control studies of local endemic serovars of *Salmonella* to identify risk factors for infection, including *S.* Typhimurium 135 (Hunter Public Health Unit and surrounding Public Health Units (PHU), Victoria and Western Australia), *S.* Mississippi (Tasmania), and *S.* Birkenhead (Queensland and Northern Rivers PHU).

During the quarter, the OzFoodNet-Hunter site coordinated a microbiological typing project to supplement the *Campylobacter* case control study. Several laboratories, using different microbiological techniques, analysed *Campylobacter* isolates collected in an earlier case control study conducted by the Hunter PHU. Risk factor data from this study will by analysed by *Campylobacter* subtype, and the most relevant microbiological test selected for use in the OzFoodNet national *Campylobacter* case control study.

The national OzFoodNet gastroenteritis survey showed that 11.6 per cent of people experienced gastroenteritis in the month prior to interview (Table 3). The overall response rate for the 3 months was 67 per cent. During the quarter, Northern Territory residents reported the highest crude proportion of people experiencing gastroenteritis in the previous month (19.5%), and Queensland residents reported the lowest (9.7%). Nationally, the prevalence of gastroenteritis was highest for respondents interviewed in the month of December (12.9%). This is self-reported gastroenteritis and does not distinguish foodborne illness from other causes of gastroenteritis.

This gastroenteritis survey covers all States and Territories and will run for a year. It will provide important information about the burden of gastrointestinal disease. These preliminary results suggest that gastroenteritis is very common and affects millions of people each year in Australia. The data collected in this survey will contribute to OzFoodNet's calculation of an estimate of the proportion of gastroenteritis due to food.

Table 3. Unweighted results of the national OzFoodNet gastroenteritis survey between October and December 2001 showing the proportion of respondents reporting an episode of gastroenteritis in the previous month (n = 1,829).

State or Territory	Proportion with gastroenteritis (%)			
	October	November	December	Mean prevalence
New South Wales*	11.9	8.2	12.1	10.8
Northern Territory	22.2	13.1	23.1	19.5
Queensland	15.5	8.1	5.5	9.7
South Australia	11.4	11.1	11.6	11.4
Tasmania	10.0	8.6	13.9	10.8
Victoria	13.0	7.1	12.6	10.9
Western Australia	4.7	14.1	13.3	10.7
Total	**12.1**	**9.7**	**12.9**	**11.6**

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

Australia declared polio free

Rennie M. D'Souza,[1] Margery Kennett,[2] Charles Watson[3]

Abstract

For Australia to be declared polio free, evidence of the absence of circulation of wild poliovirus was required by the Regional Commission for the Certification of Eradication of Poliomyelitis in the Western Pacific in August 2000. Data on surveillance of poliomyelitis, acute flaccid paralysis (AFP), vaccine associated paralytic polio and enteroviruses were provided to document the absence of circulation of wild poliovirus. The last wild poliomyelitis virus case in Australia was in 1972. AFP surveillance has improved since it was initiated in 1995 and achieved a rate of 0.94 per 100,000 population in 1999. No wild polioviruses have been isolated from stool samples of AFP cases. Australia has in place a comprehensive network of laboratories for enterovirus surveillance and this provides further evidence for the absence of wild poliovirus infection. The immunisation coverage in the country has been over 80 per cent over the last 3 years. If there were an importation of a case of poliomyelitis into Australia, a national outbreak response would be coordinated through the Communicable Diseases Network Australia. Plans for containment of laboratory stocks of wild poliovirus are being implemented. The evidence provided was sufficient to satisfy the Regional Commission that there was no wild poliovirus circulating in the region and enabled Australia to be declared polio free on October 29, 2000 along with the other 36 countries in the Western Pacific Region. Australia must remain vigilant against importations of wild poliovirus from endemic countries and maintain high immunisation coverage and sensitive surveillance systems. *Commun Dis Intell* 2002;26:253-260.

Key Words: poliomyelitis eradication, Australia, acute flaccid paralysis surveillance, laboratory containment, enterovirus surveillance

Introduction

The Regional Commission for the Certification of Eradication of Poliomyelitis in the Western Pacific[1] had determined that the Region would only be certified as poliomyelitis-free after all countries of the Region had met the following criteria:

- no evidence of indigenous wild poliovirus transmission had been detected for a period of at least 3 years during which surveillance had been maintained at the level of performance needed for certification;
- a National Certification Committee in each country had validated and submitted the certification documentation required by the Regional Commission; and
- appropriate measures were in place to detect and respond to importations of wild poliovirus.

In 1997, the Australian National Polio Certification Committee was set up, comprising of a public health physician, a virologist and a neurologist. The National Certification Committee reviews progress made on activities related to certification of poliomyelitis eradication and has submitted documentation annually since 1998 to the Regional Commission for the Certification of Eradication of Poliomyelitis in the Western Pacific.

In 1998, the Polio Expert Committee was set up, comprising of a paediatrician, an epidemiologist and a virologist. This committee was to review all cases of acute flaccid paralysis (AFP) that were investigated through the AFP surveillance system and to undertake a retrospective review of hospital cases and classify them according to the World Health Organization's (WHO) virological classification.[2]

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Surveillance of poliomyelitis

The surveillance strategy for confirming poliomyelitis eradication in Australia consists of 5 components:

1. the National Notifiable Diseases Surveillance System;
2. surveillance of acute flaccid paralysis cases;
3. surveillance of vaccine associated paralytic polio (VAPP) cases;
4. surveillance of enteroviruses; and
5. intratypic differentiation of all polioviruses isolated in Australia.

1. National Notifiable Diseases Surveillance System

Poliomyelitis has been a notifiable condition in Australia since 1922. The National Notifiable Disease Surveillance System (NNDSS) was established in 1990 under the auspices of the Communicable Diseases Network Australia New Zealand (CDNANZ).[3] The last 2 epidemics of polio were in 1956 and 1961 to 1962. The number of polio cases dropped substantially since the introduction of the inactivated vaccine in 1956 and subsequently the oral polio vaccine.[4]

The last 3 cases of poliomyelitis diagnosed as such on clinical grounds occurred in Victoria in 1972 but were not confirmed virologically. Apart from the imported case in 1977, other AFP cases initially notified have been reclassified as VAPP. Virological investigations of stored viruses from Victoria indicate that the last wild poliovirus was isolated from a patient with clinical poliomyelitis in 1967. If these Victorian stored polioviruses are representative of all Australian isolates it is possible that wild poliovirus may have disappeared from Australia in the 1960s and that cases notified later were all VAPP or imported cases, as were all the cases notified after 1972. However, in the absence of virological testing and without further investigation of the 1972 cases, 1972 must be considered the year in which Australia had its last cases of indigenously acquired wild poliovirus infections.

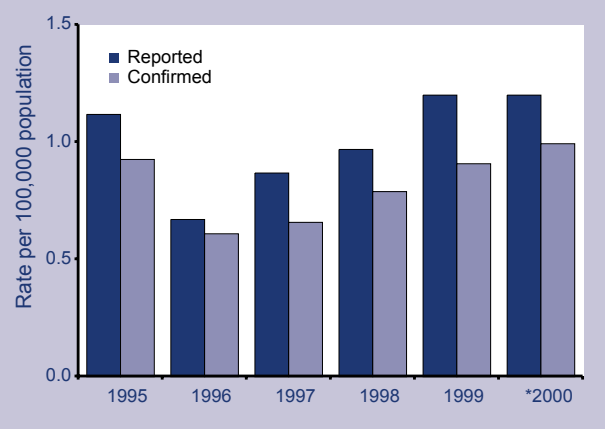
2. Surveillance of acute flaccid paralysis

While it is unlikely that Australia has indigenous wild poliovirus, adequate investigation of cases of AFP is required for the certification process. The surveillance of AFP was initiated in Australia in March 1995 through the Australian Paediatric Surveillance Unit (APSU).[5] The expected number of AFP cases in Australia in children less than 15 years is 1 case per 100,000 population per year i.e. 39-40 cases in a year. This is an internationally accepted indicator of a highly sensitive surveillance system for acutely paralysed children and is based on the experience of other non-endemic countries.[1] The methods adopted for AFP surveillance and findings from 1995 to 1997 have been reported.[4,6]

There have been a total of 161 AFP cases investigated over the five-year period (March 1995 to April 2000) after excluding duplicates and errors. The main causes of AFP in Australia in the last 5 years have been Guillain Barré Syndrome (GBS) and transverse myelitis (TM), which represent 63 per cent of the cases, followed by trauma and encephalitis.

Since there is no wild poliovirus in Australia, the non-polio AFP rate is the same as the AFP rate. The average reporting rate of AFP cases for 1995 to 1999 based on the notifications received by the APSU and the Surveillance and Epidemiology Section, Department of Health and Ageing (DoHA) is 1.2 cases per 100,000 population under the age of 15 years. The average AFP rate based on investigated cases i.e. those reports with completed questionnaires for 1995 to 1999 is 0.79 cases per 100,000 population under 15 years of age. The rate of AFP in Australia has increased over the last 5 years and reached 0.94 cases per 100,000 population under 15 years of age in 1999[7] and the adjusted rate for 2000 (adjusted for the number of months of surveillance) is 0.99 cases per 100,000 population under 15 years of age (Figure 1). This rate, which is lower than the benchmark of 1 case per 100,000 population under 15 years of age, is due to paediatricians reporting and investigating only those AFP cases that they consider to be poliomyelitis compatible. The Australian Poliomyelitis Expert Committee has adhered strictly to the case definition of AFP and some mild Guillain Barré cases have been excluded as non-AFP cases. Therefore the AFP rate in Australia is based on a very narrow AFP case definition. If the committee was not so strict, the non-poliomyelitis AFP rate would be higher.

Figure 1. AFP rate per 100,000 children under the age of 15, Australia, 1995 to 2000



* Data as at April 2000.

The average annual AFP rate for the years 1997 to 1999 is 0.79 cases per 100,000 population under the age of 15 years of age. Only two jurisdictions, New South Wales and the Australian Capital Territory reached the expected rate of 1 case per 100,000 population under 15 years of age. Victoria has performed poorly over the last 5 years. Western Australia achieved a rate of 1 case per 100,000 population under 15 years of age in 1998 but did not report all acute flaccid paralysis cases in 1999. When the 4 cases picked up in a review of the hospital records are included, the rate for Western Australia is 1 case per 100,000 under 15 years of age in 1999. The Northern Territory has not reported a case of AFP in the last 5 years and therefore will need further investigation. Tasmania and the Northern Territory are very sparsely populated and therefore the small number of AFP cases may make the estimation of rates unreliable.

At least 80 per cent of AFP cases should have 2 stool specimens collected at least 24 hours apart and within 14 days of onset of paralysis.[2] The proportion of AFP cases from whom 2 stool samples were collected increased from 11.5 per cent in 1997 to 40.5 per cent in 1999 and 54 per cent in 2000. There has been considerable reluctance by the paediatricians to order stool specimens when they have a confirmed clinical diagnosis based on other investigations. This has resulted in a failure to reach the expected target of 80 per cent stool collection rate of AFP cases. There have been numerous efforts to improve the notification of AFP cases and stool collection over the last 5 years.

Based on the clinical and laboratory information provided by the paediatricians, the Poliomyelitis Expert Committee was able to exclude 94.4 per cent of cases as being non-poliomyelitis even in the absence of stool specimens (Figure 2). There are 9 residual cases for whom additional information has not been forthcoming and which cannot be formally classified as non-poliomyelitis.

Evaluation of AFP surveillance system (retrospective hospital review of AFP cases)

As the AFP surveillance in Australia had not reached the expected rate of 1 case per 100,000 population under the age of 15 years during the period 1995 to 1997, a search of medical records was made in 2 main hospitals in Victoria[8] and one hospital each in the Australian Capital Territory and the Northern Territory. In addition, a state wide search of hospital separation data was carried out in New South Wales and Western Australia.[9] The rationale for doing state-wide searches in these 2 States which had reached the expected rate of AFP, was to identify additional cases missed by the active AFP surveillance and therefore establish the true AFP rate.

There were 61 additional cases of AFP found in the hospital record review for the period 1995 to 1998 that were not investigated through the AFP surveillance (Table). More than 50 per cent of the cases were identified in 2 hospitals from Victoria, which had a poor AFP reporting rate. The hospital searches in New South Wales and Western Australia identified GBS and TM cases that were not reported to the active AFP surveillance.

The code for GBS was the most reliable in identifying true AFP cases and almost 99 per cent of cases of GBS were true AFP cases as compared to TM which is coded as unspecified myelitis and had a much higher false positive rate.

There were no poliomyelitis cases found in the hospital reviews. As the AFP surveillance and hospital reviews both missed cases, the combined total gives a better approximation of the true AFP rate, which is in the range of 1.14 cases per 100,000 population under the age of 15 years (Figure 3).

Table. AFP cases identified from hospital review (1995 to 1998)

Additional AFP cases from hospitals	1995	1996	1997	1998	Total
ACT (1995-98)	2	1	2	0	5
NSW (1995-98)	4	6	3	3	16
WA (1995-98)	4	1	2	2	9
Vic (1995-97)	12	8	10		31*
Total	**22**	**16**	**17**	**5**	**61**

* Includes 1 case with unknown year

Figure 2. Virological classification of AFP cases and case outcome following Poliomyelitis Expert Committee

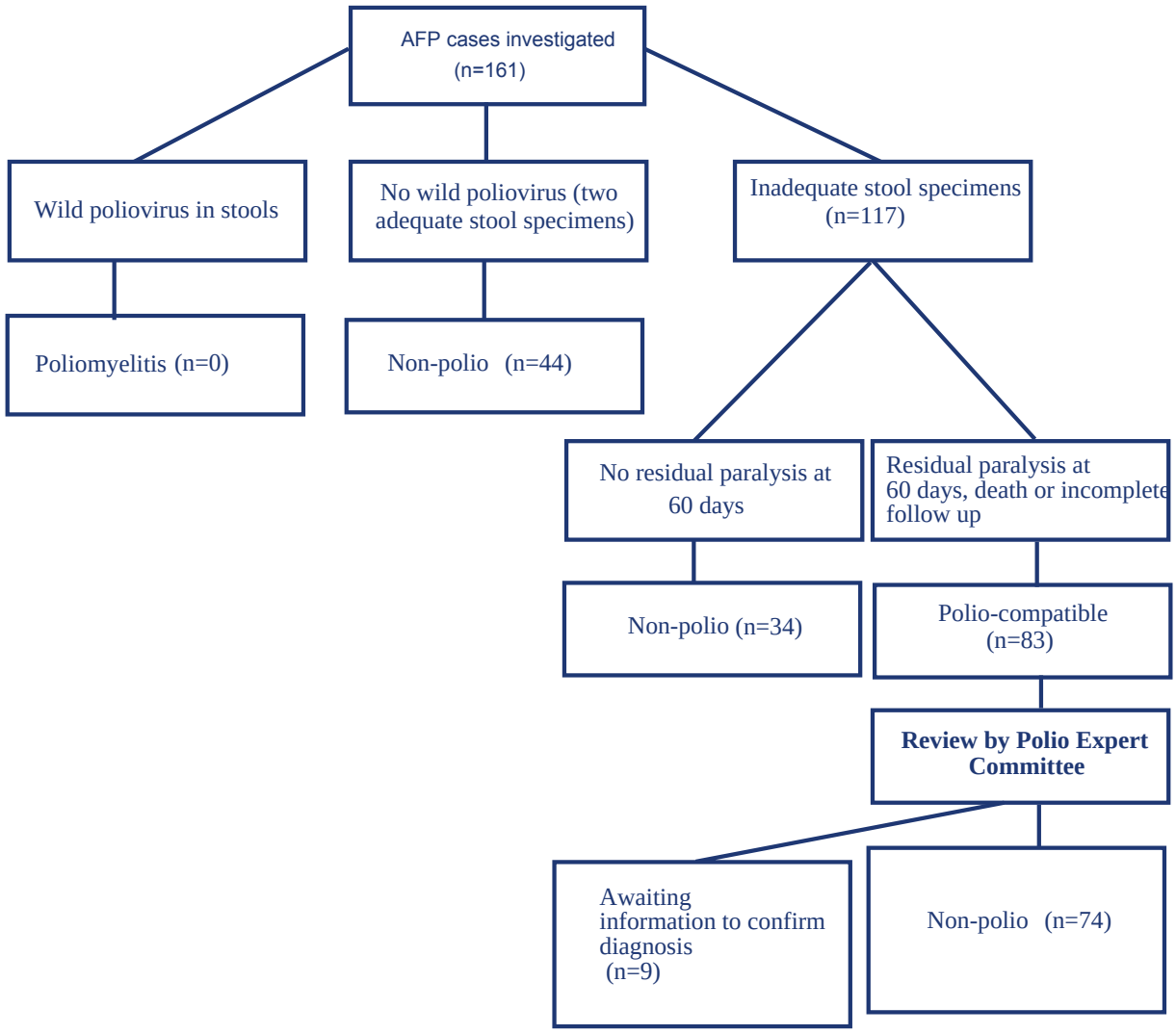
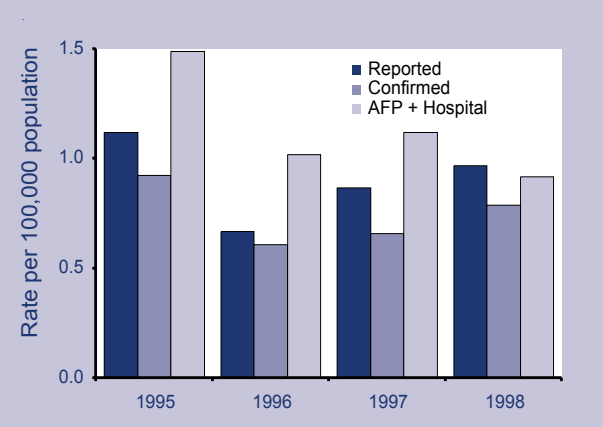


Figure 3. AFP rate per 100,000 children under the age of 15, Australia, (AFP surveillance hospital review)



The information obtained from the medical records was adequate for the Poliomyelitis Expert Committee to review and exclude 88.5 per cent of the AFP cases as being non-poliomyelitis.

3. Surveillance of vaccine associated paralytic poliomyelitis

In rare instances, oral poliomyelitis vaccine (OPV) may cause a paralytic illness in healthy recipients and their contacts. Estimates of the frequency of this risk suggest that it occurs at the rate of one case of paralytic disease in immunologically healthy vaccine recipients for every 6.8 million doses of OPV distributed, and one case of paralytic disease among household and community contacts for every 6.4 million doses distributed. The reported risk for VAPP in the United States of America is 1/700,000 per first dose; 1/6.9 million per subsequent doses for all children and is greater in adult contacts than child recipients.[10]

The ability to detect VAPP, if reported separately, is a useful indicator of sensitivity of a surveillance system. Australia can expect to find about one case of VAPP every 3 years. One case of VAPP in Australia was reported in 1994 in a mother of a child who was immunised with OPV.[11]

There have been 2 parallel national surveillance systems for reporting serious adverse events following vaccination including VAPP. One was the Surveillance of Serious Adverse Events Following Vaccination (SAEFVSS) and the other is the Adverse Drug Reaction Advisory Committee (ADRAC).

The SAEFVSS[12] was a national surveillance system that is managed by the National Childhood Immunisation Program. Reports from providers of adverse events related to childhood vaccinations were forwarded by States and Territories and classified according to a list of defined serious adverse events. This system was modified on 1 January 2000 to allow greater integration of information collected through the SAEFVSS and ADRAC systems and is now known as the Adverse Events Following Immunisation (AEFI) Surveillance System. Since the modification, AEFI receives data on all adverse events via ADRAC. This enables the AEFI system to utilise the causality classification allocated to each report by the ADRAC system.

The ADRAC[13] system is operated through the Therapeutic Goods Administration and monitors adverse events after the administration of all drugs including vaccinations. This system accepts all reports, including those from parents, which are later reviewed by a committee and causality is assigned to the reports.

To date neither of these systems has reported a VAPP in the last 5 years. A patient with TM was detected during AFP surveillance in 1996. Poliovirus type 3 Sabin vaccine-like isolated from a stool sample may have been an incidental finding. It is possible that some AFP cases could have been VAPP cases but could not be proved as stool specimens were not taken.

4. Surveillance of enteroviruses

The laboratory activities for certification can be divided into 2 components; AFP surveillance and enterovirus surveillance. For AFP surveillance, at least 80 per cent of AFP cases must have an examination of 2 adequate stool specimens by an accredited laboratory. Since there should only be approximately 80 samples from 40 cases each year in Australia, it was agreed in 1996 that all samples would be transported to the National Polio Reference Laboratory (NPRL) at the Victorian Infectious Diseases Reference Laboratory (VIDRL) for enterovirus culture and poliovirus identification and characterisation. The laboratory which also serves as one of the Western Pacific Regional Reference Laboratories has been funded to carry out this work by the Commonwealth Department of Health and Ageing, since 1994.[14] In 1998 and 1999 the laboratory was accredited by the World Health Organization (WHO) as both a regional and national polio laboratory.

From January 1997 to mid-June 2000, 92 stool samples were processed and inoculated into WHO-supplied cell lines for enterovirus culture. No polioviruses were isolated. Samples were not collected from contacts of AFP patients and stool

surveys or environmental testing were not performed. Serological investigations were performed on patients with paralysis when stool samples were not collected within 14 days of onset of paralysis. There were no cases with evidence of poliovirus infection.

Two sero-surveys to determine immunity to polioviruses were carried out in the national laboratory. One was to determine the immunity to polio and other viruses in fully vaccinated Aboriginal and Torres Strait Island children.[15] The other compared immunity to childhood preventable diseases in local and recently arrived immigrant children in a Melbourne suburb (J Buttery, M Kennett, personal communication). Although over 90 per cent of all children had protective antibody levels to poliovirus types 1 and 2, only 60 per cent of the Aboriginal and Torres Strait Island and local Melbourne children had antibodies to type 3.

Documentation is also required to demonstrate that laboratory services are in place to identify wild polioviruses. Polio and also coxsackie A and B, ECHO and enteroviruses type 68 to 71 belong to the enterovirus family in the genus picornaviridae. Enterovirus culture is performed on a wide range of clinical specimens in state reference laboratories and some hospital, diagnostic and environmental laboratories in Australia. In earlier years, enterovirus isolates would have been referred to state reference laboratories for identification in neutralisation tests utilising type specific antisera but more recently many remain unidentified. In recent years, some laboratories have replaced culture by direct nucleic acid detection, a broad-based method in which complete identification is not performed.

5. *Intratypic differentiation of all polioviruses isolated in Australia*

The Sabin live attenuated OPV is the most commonly administered poliomyelitis vaccine in Australia. The three types, 1, 2 and 3, may be present in respiratory or stool samples from children who have recently received OPV for up to several weeks after immunisation. If samples are collected from such children for any reason, these Sabin vaccine strains may be isolated.

Wild and Sabin vaccine polioviruses are differentiated using 2 WHO recommended tests, nucleic acid hybridisation and enzyme immunoassay, at the NPRL. The NPRL has established an informal poliomyelitis laboratory network to facilitate in the collection and submission of stool samples from AFP patients and to ensure that all polio or untyped enteroviruses are transported to it for identification and characterisation. Fifteen laboratories in all States and the Australian Capital Territory report their virus isolations and serology findings to the Virology and Serology Laboratory Reporting Scheme (LabVISE) each month. As only positive results are reported, it is difficult to determine the number of specimens tested for enterovirus diagnosis in Australia.[16] From January 1997 to May 2000, 5,128 specimens were positive for enteroviruses of which 229 were polioviruses. Attempts have been made by the polio laboratory network and DoHA staff to contact reporting laboratories to ensure that all polio and untyped enterovirus isolates are referred to, identified and characterised at the NPRL.

From January 1994 to May 2000, 1,173 such isolates have been tested at VIDRL. Six hundred and fifty-seven were identified as Sabin polioviruses, 491 were non-polio enteroviruses and 24 were not enteroviruses or were not recovered. One referred poliovirus type 2 was shown to be non Sabin-like and on further investigation was found to be identical with an attenuated poliovirus (Koprowski) used in the original laboratory so was considered as a laboratory contaminant.[17]

Immunisation coverage

The immunisation coverage of OPV has generally been high in Australia over the last 10 years, as there has been commitment by the Federal government to promote immunisation. This rate has improved since 1997, in part due to financial incentives provided to general practitioners, better ascertainment of coverage and the use of financial disincentives (parents are to have family payments withheld if their child is not immunised).

The overall coverage of OPV in infants has been steadily increasing over the last 3 years and reached 88 per cent in 1999. Every State or Territory had achieved at least a minimum coverage of OPV of 86.6 per cent in 1999.[18] This reduces the risk of spread of poliomyelitis in the case of an importation.

Outbreak response

The risk of importation exists in Australia, as many tourists and immigrants enter from, or transit through, 'high-risk' (endemic) countries. Adult immigrants from high-risk countries are unlikely to be a source of infection because most would have acquired immunity in childhood. If there were an

importation of a case of poliomyelitis into Australia, it would be considered a public health emergency. Australia has the capacity, infrastructure and expertise to investigate and mount a disease control response to a suspected case of wild poliovirus. A rapid response would be co-ordinated by the Communicable Diseases Network Australia (CDNA). In addition, to the organised surveillance systems, the Public Health Laboratory Network, which provides laboratory based surveillance and technical expertise to CDNA, would alert virological laboratories about the need for stool collection to be forwarded to VIDRL for confirmation and intratypic differentiation.

Isolation of wild poliovirus constitutes a public health emergency and appropriate control efforts must be initiated in consultation with local health-care providers, State and Territory Health Authorities, DoHA and CDNA.

Australia has a history of tourism and other visits by people from polio endemic countries (India, Pakistan, Bangladesh etc). Despite these risks there have been no poliomyelitis outbreaks since the 1960s and no known transmission of poliomyelitis, even though it is probable that cases of acute flaccid paralysis have been missed through the existing surveillance system.

Laboratory containment of wild poliovirus in Australia

In countries like Australia where wild poliovirus has not circulated in the community for more than 20 years, laboratories are likely to be amongst the only places where wild poliovirus still exists. The laboratory containment of wild poliovirus and materials which may be potentially infectious for wild poliovirus is therefore a crucial part of the process of certification of a country as being free of wild poliovirus. In addition to the standard certification criteria, the regional certification committee requested all countries to provide documentation on progress towards implementation of the pre-eradication phase of wild poliovirus containment.

The Commonwealth has supported the appointment of a national containment co-ordinator based in the epidemiology division at VIDRL. The coordinator is responsible for preparing a national plan, coordinating its implementation and preparing the national inventory of poliovirus infectious materials.

A national workshop attended by representatives of States and Territories and all types of laboratories was held in March 2000 to discuss strategies for identifying laboratories and institutions in which wild poliovirus infectious materials might be stored and the practicalities of containment. A national advisory committee and State representatives were appointed to assist VIDRL staff in implementation of the plan. Two pilot surveys were conducted to evaluate the effectiveness of the documentation and survey materials. Subsequent surveys were based on the findings.[19]

The database of laboratories and institutions which may manage laboratories in which poliovirus materials may be stored included diagnostic, reference, research, regulatory, environmental and manufacturing laboratories, universities and independent research institutions.[17] Two thousand one hundred and eighty-four organisations were surveyed to determine which of these had at least one laboratory which may store biological materials for more than 4 weeks. Those organisations which did so were surveyed to determine if their stored materials were likely to contain wild poliovirus. The third stage was to prepare an inventory of all stored wild poliovirus materials. The project is still proceeding as intensive follow-up is required to complete all 3 stages.

Conclusion

The evidence to date confirms that there is no transmission of wild poliovirus in Australia. The evidence provided to the Regional Commission was sufficient to determine that there was no wild poliovirus circulating in the region and enabled Australia to be declared polio free on 29 October 2000 along with the other 36 countries in the Western Pacific Region. There is a strong commitment on behalf of the Commonwealth to continue to maintain high levels of OPV immunisation and AFP surveillance at an acceptable level until global certification is achieved.

Australia must remain vigilant against importations of wild polioviruses from endemic countries. There are surveillance systems in place to identify an importation of a case of poliomyelitis and laboratory surveillance is extremely good. The importance of the sensitivity of a surveillance system cannot be fully appreciated until the time when a disease is targeted for eradication. It was the sensitivity of the AFP surveillance system that was one of the main criteria for certification of eradication of polio from the Western Pacific Region. Secondly, in Australia, it was the national coordination of various sectors like surveillance

and immunisation sections in DoHA, APSU, VIDRL, laboratories, paediatricians and public health professionals in the country that provided the evidence of polio-free status in this country. This did require a major effort in increasing awareness of overall but especially in clinicians as some of the certification initiatives like AFP surveillance for polio were being instituted almost 20 years after the last case of polio was seen.

In addition, plans for containment of laboratory stocks of wild poliovirus are being implemented. This again would require the cooperation, honesty and good will of all laboratories within the country. Although polio is only the second vaccine preventable disease to be eradicated from the Western Pacific Region, it has provided an infrastructure in the region to be working towards elimination of measles which is the next disease targeted for eradication. Hopefully the lessons learnt from the polio eradication initiatives can be applied to measles elimination and eradication.

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The authors would like to acknowledge the contributions of the APSU for facilitating the AFP surveillance, all paediatricians who reported and investigated cases of AFP, staff of hospitals, the National Polio Reference Laboratory and other laboratories for collection, transportation and processing of stool and serum samples. Lastly, we would like to acknowledge the contribution by Heath Kelly and Nittita Prasopa-Plaizier for the containment data, staff of the National Polio Reference Laboratory and the Department of Health and Ageing in preparing the certification documentation.

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A large, prolonged outbreak of human calicivirus infection linked to an aged–care facility

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Abstract

This report investigates an outbreak of acute gastrointestinal illness, microbiologically and epidemiologically linked to an aged–care facility and seeks to determine if there was a point source of infection. A register of cases that included onset date and time of illness and symptoms was maintained by nursing staff. Faecal specimens were tested for conventional gastrointestinal pathogens and for human calicivirus (HuCV). There were 81 cases reported. Specimens were received for testing from 25 cases. Twenty–three of the 25 (92%) specimens were positive for HuCV RNA by reverse transcriptase polymerase chain reaction (RT–PCR). The 2 negative samples contained RT–PCR inhibitors. Descriptive epidemiology suggested that staffing practices were important in prolonging the outbreak. No point source of infection was identified. Instead environmental contamination, aerosol transmission and work practices that fail to take account of the natural history of HuCV infection probably contributed to the size (81 cases) and duration (3 weeks) of this outbreak among the residents, staff and visitors of an aged–care facility and their contacts. Institutional outbreaks caused by HuCV, formerly called Norwalk–like or small round structured viruses, are extremely difficult to control. Infected staff may contribute significantly to the amplification of outbreaks. Rapid confirmation of HuCV infection is now routinely possible using polymerase chain reaction diagnostics but progress in laboratory technology has not yet translated into faster or more effective interventions. *Commun Dis Intell* 2002;26:261–264.

Keywords: outbreak, Norwalk-like virus; calicivirus, small round structured virus

Introduction

Human caliciviruses (HuCVs), formerly called Norwalk–like viruses (NLVs) or small round structured viruses (SRSVs), have long been suspected to cause outbreaks of acute gastroenteritis.[1] Until recently determining the specific aetiology of these outbreaks has been hampered by the insensitivity of microbiological diagnostics. In deciding whether cases of acute gastroenteritis were caused by HuCVs, epidemiologists have had to depend on the combination of laboratory tests being negative for all other pathogens and the occurrence of characteristic symptoms of HuCV infection: an illness duration of 12 to 60 hours, an incubation period 15 to 48 hours and vomiting being more prominent than diarrhoea.[2] The development of antigen detection methods and, more significantly, polymerase chain reaction (PCR) diagnostics now make it possible to definitively determine the cause of many outbreaks previously only suspected as being the result of HuCV infection. We present the investigation of a 3 week long outbreak of acute HuCV gastroenteritis which affected 81 people associated with an aged–care facility in Adelaide.

The outbreak

On 24 August 2000, the Communicable Disease Control Branch (CDCB) was notified of an acute outbreak of gastrointestinal illness in an aged–care facility in metropolitan Adelaide. The initial report was of gastrointestinal illness among several hostel residents and staff but not among residents of the associated nursing home. The symptoms were consistent with the classic presentation of HuCV.[2]

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Methods

Case definition

A case was defined as a person living, working, visiting or epidemiologically linked to the aged–care facility with acute onset of diarrhoea or vomiting between 14 August and 3 September 2000.

Epidemiological investigation

Staff of the aged-care facility maintained an illness register. Staff from CDCB visited the aged–care facility and gathered data on resident seating arrangements at meal–time and residents' room numbers. Residents were not interviewed because of cognitive impairment.

Environmental investigation

A local environmental health officer reviewed food preparation, food storage and hygiene practices among food handlers at the facility and arranged the collection of faecal specimens.

Microbiological investigation

Faeces from 25 symptomatic residents, staff and their contacts were tested for conventional parasitic, bacterial and viral (rotavirus and adenovirus) pathogens by the Institute of Medical and Veterinary Science (IMVS), using routine methods. In addition, tests were conducted for HuCVs by RT–PCR assays using primers specific for each of the three main groups of HuCV: Norwalk–like virus group 1 (NLV–1), NLV–2 and Sapporo–like viruses (SLV) and for astroviruses.[3]

Infection control measures

Standard infection control practices were routine at this institution. Additional infection control measures were initiated on 24 August 2000 when the outbreak was first reported to the CDCB in accordance with published recommendations.[2] As well, ill hostel residents were isolated and signs were erected informing visitors about the outbreak. Staff were advised not to return to work for 48 hours after symptoms resolved.

Results

Epidemiological investigation

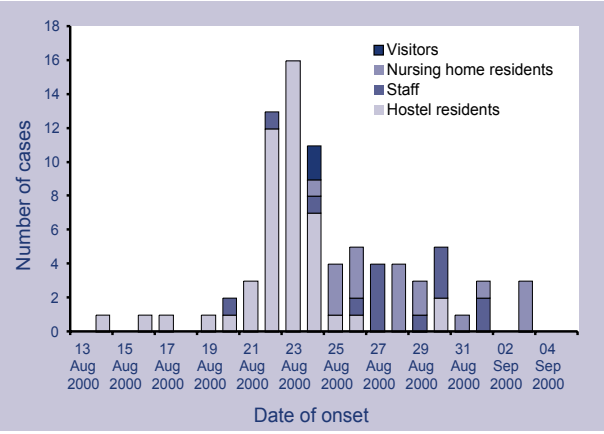
Setting

The aged-care facility had 107 (17 males, 90 females) residents, of whom 64 (60%) were resident in the hostel. The other residents lived in the nursing home section. Hostel residents had their own rooms, however, meals were eaten in a common dining room and residents had specific seating arrangements. Nursing home residents slept in single rooms with the exception of two, who shared a room. Only 16 (37%) of the 43 nursing home residents ate meals in a dining room (separate to the dining room used by hostel residents). Food was prepared in a central kitchen on site for both the nursing home and hostel residents. Seventy–five staff members were employed at the aged–care facility. Nursing staff worked between the 2 areas, particularly during periods of staff shortages.

Outbreak description

Of the 107 residents, 65 (61%) reported gastrointestinal illness. The epidemic curve (Figure) shows a slow start and protracted course (over a 3 week period). The first person to become ill was a hostel resident who had not left the facility prior to illness. The first wave, observed from 14 to 23 August 2000, occurred mainly among hostel residents and included 2 nursing staff members. The second wave from 24 August to 3 September 2000 affected mainly nursing home residents and staff. One visitor and an indirect contact (daughter of a case from the hostel and the daughter's grandson who did not visit the hostel) reported illness at the beginning of the second wave.

Figure. Cases of human calicivirus in an aged-care facility, 14 August to 3 September 2000, by date of onset.



Hostel residents

The attack rate among hostel residents was 73 per cent (47/64). Of these, 31 (66%) experienced diarrhoea and vomiting, 8 (17%) vomiting and 8 (17%) had symptoms of diarrhoea. The mean age was 85 years.

Nursing home residents

Of the 43 nursing home residents, 18 (42%) were ill. Of these, 8 (44%) experienced vomiting, 8 (44%) diarrhoea and 2 (11%) had diarrhoea and vomiting. The mean age was 87 years.

Staff

Of the 75 staff, 14 (19%) were ill. Of these, 7 (50%) experienced diarrhoea, 5 (36%) vomiting and diarrhoea and 2 (14%) had vomiting. The first 2 staff members to become ill were a nurse who would visit all areas of the facility and a nurse who worked in the hostel. The onset of illness in kitchen staff did not occur until the beginning of the second wave. Staff who worked in the nursing home were the last to become ill. The only staff member with NLV–2 detected in faeces was a nurse who worked in the hostel section prior to onset of illness.

Visitor

A visitor to the facility reported contact with vomit on her mother's nightdress and on the bedroom floor. The soiled nightdress was removed from the facility and washed at home in the presence of a grandson. The grandson had not accompanied his grandmother to the complex earlier in the day. Within 18 hours, the daughter and grandson experienced vomiting and diarrhoea. In both, the duration of illness was 24 hours.

Environmental investigation

The environmental health inspection revealed problems with food quality and food handling practices. Serving utensils had been left in foods on the stove, in the cool-room and on the preparation bench. Cracked wooden spoons were in use accumulating food particles and potentially harbouring microbes. Inadequate cleaning was observed in the kitchen, for example, the accumulation of grime and dirt on surfaces and equipment. A further follow–up inspection was conducted by the environmental health officer.

Microbiological investigation

No parasites or bacterial pathogens were detected in any of the 25 individual case specimens. All adenovirus antigen assays were negative as were RT–PCR assays for NLV–1, SLV and astroviruses. The RT–PCR assay for NLV–2 RNA was positive in 23 of 25 (92%) specimens, with specimens from the remaining 2 cases (1 from a hostel resident and 1 from a nurse) containing RT–PCR inhibitors which could not be removed. Two hostel residents who were positive for NLV–2 RNA were also positive for rotavirus antigen.

Discussion

We can expect in Australia that institutional outbreaks of acute gastroenteritis proven definitively to be caused by HuCVs will now be reported regularly.[4,5] The chief aim of investigating such outbreaks is to determine if transmission has occurred person to person or through contaminated food or drink. HuCVs in oysters and orange juice have caused large outbreaks in Australia.[6] Kitchen workers infected with HuCV may cause more circumscribed outbreaks through food contamination.[7] Although the investigation reported here revealed problems with food quality and handling, the descriptive epidemiology of this outbreak strongly suggests that transmission occurred by person-to-person contact. It was particularly significant that, although the hostel and the nursing home residents were fed the same food from the same kitchen, the outbreak followed a biphasic pattern: only 4 new cases occurred in hostel residents after the first case in a nursing home resident. Most of the affected staff only became ill after the outbreak spread to the nursing home, perhaps because of the necessarily closer contact between staff and these dependent, ill residents. The sudden increase of cases among residents of the hostel only after the first staff member became ill suggests that staff had a crucial role in amplifying the outbreak. By contrast, contact between residents at meal times was unrelated to the time of onset of disease. Single room accommodation, which would reduce resident-to-resident contact, did not prevent spread of the infection suggesting the importance of ill staff in prolonging the outbreak.

Controlling person-to-person transmission of HuCV gastroenteritis in an institution is very difficult. It is almost certain that these viruses can be spread by airborne transmission, so in contrast to other

gastrointestinal pathogens which spread strictly by the faecal–oral route, handwashing alone is not efficacious. Also because these viruses are relatively hardy,[8] they can survive for some time on contaminated bedding and clothes and presumably could be re–aerosolised.[9] The grandson of the visitor who became ill after contact with the nightdress of one of the residents illustrates the extreme infectivity of the agent.

Because virus excretion commences some hours prior to the onset of symptoms it is recommended that staff from a ward affected by an HuCV outbreak not be transferred to new work areas for 48 hours after their last shift in the affected ward.[2] Also HuCVs continue to be excreted some days after symptoms have ceased and ill health care workers and food–handlers should not recommence duty until at least 48 hours after their last symptoms.[10] The public health message is that conditions of employment that encourage early return to work after HuCV illness may contribute to prolonging outbreaks. In practice, especially when symptomatic illness reduces staff numbers, it is probably difficult for administrators to adopt these restrictions with the result, as in the situation reported here, that the outbreak may spread further and be prolonged.

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Evaluation of the Australian CJD Surveillance System

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Abstract

An evaluation of the surveillance capacity of the Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR) was undertaken. It focused on the ability of the Registry to detect CJD in Australia and, in particular, to identify cases that require public health responses. The Registry relies on a complex reporting system and staff with expertise to accurately identify and classify cases of CJD. The Registry satisfies the criteria of flexibility and acceptability and has a high positive predictive value and representativeness. The sensitivity of the system could not be evaluated, as the rarity of the condition precludes an independent assessment of the incidence of CJD, but the incidence of CJD is comparable to that found in other countries. The time required to establish a definite diagnosis of CJD is approximately 2 months, impacting negatively on the timeliness of the system. In order to maximise the likelihood of detecting all cases of CJD in Australia in a timely fashion, suggestions are made for improving the system's sensitivity and timeliness of reporting as well as for using methods that allow meaningful comparisons of incidence between populations with different age structures. *Commun Dis Intell* 2002;26:265–272.

Keywords: CJD, evaluation, surveillance

Introduction

Classical Creutzfeldt-Jakob disease (CJD) is a rare degenerative disease of the central nervous system, with an annual incidence of about one in a million people worldwide.[1] It is invariably fatal, with a median duration of illness of 4 months.[2]

CJD is the most common human form of a group of diseases, transmissible spongiform encephalopathies (TSEs), pathologically characterised by a loss of neurons, proliferation of astrocytes and the development of microscopic vacuoles in the brain.[1,2] Animal TSEs include scrapie in sheep and bovine spongiform encephalopathy (BSE) in cattle, popularly known as 'mad cow disease'.[2]

The transmissibility of the TSEs has, in general terms, been demonstrated through inoculation experiments in animals, but for over 85 per cent of human cases the specific cause is unknown.[1,2] Inherited syndromes account for a small proportion of cases and an iatrogenic aetiology has been proven for a further group of cases.[3,4,5,6] In one part of Papua New Guinea, a human TSE known as kuru was transmitted through ritual cannibalism.[7,3]

Concerns about iatrogenic CJD transmission led to the establishment of Australia's National CJD Registry in 1993. More recently, the discovery in the United Kingdom (UK) of a new form of CJD linked to consumption of BSE-contaminated beef has stimulated heightened interest in surveillance for human TSEs.[8]

Public health surveillance for CJD in Australia has therefore been largely motivated by the need to have a mechanism for early detection of cases that may reflect transmission either by iatrogenic means or by consumption of contaminated food products. The occurrence of any such case may have major public health consequences and should be notified to health authorities in a timely fashion.

In order to assess the surveillance capacity of the ANCJDR, an evaluation was undertaken by the author, as part of the course requirements for the Master of Applied Epidemiology degree at the National Centre for Epidemiology and Population Health (NCEPH). The evaluation focused on the ability of the Registry to detect all cases of CJD in Australia, and in particular, to identify cases that may have public health importance.

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Evaluation methods

The evaluation of the CJD surveillance system was carried out using guidelines published by the Centers for Disease Control and Prevention (CDC), Atlanta and the World Health Organization (WHO).[9,10,11] (Data from the ANCJDR has recently been published in this journal.[12])

CJD case definition

The Registry uses published criteria to define CJD cases, which are classified as definite, probable or incomplete.[13,14,15]

A definite CJD case has a clinical picture of progressive dementia, with spongiform encephalopathy confirmed by histopathologic examination.

A probable CJD case has similar clinical features, but no pathological confirmation.[13] Probable cases include people alive and some who are deceased and did not have a post-mortem pathological examination. Therefore, the figures for definite and probable cases are subject to retrospective adjustment.[15] If a post mortem examination does not take place (for example if the relatives of the patient refuse consent), the case remains permanently in the probable category.

Incomplete CJD cases are cases for which a clinical suspicion of CJD exists, but further information is needed to enable final classification. For example, cases of progressive dementia where CJD is suspected due to the finding of characteristic proteins in the CSF, may be later classified as definite or probable cases depending on results of further investigations becoming available (for example, post-mortem examination results).

Variant CJD was first reported by the UK National CJD Surveillance Unit in 1996.[8] The case definition for vCJD was developed by the UK Registry and includes clinical and investigational criteria. This case definition was adopted by the World Health Organization and by the ANCJDR.

In addition to clinical history and ancillary investigations such as EEG and MRI of the brain, several laboratory tests are being used to assist in the diagnosis and classification of CJD subtypes.[4,16] Since 1997, the Registry has made available a Western Blot assay for the detection of 14-3-4 proteins in the CSF, as they act as markers of neuronal injury in some forms of CJD.[16,17] The utility of the test is limited in cases with a slower progression of the disease, such as most vCJD cases and some iatrogenic and sporadic cases.[18,19] Other tests used to distinguish subtypes of CJD are glycoform typing of prion proteins and tests for genetic susceptibility.[19,20,21,22]

Description of the CJD surveillance system

Surveillance for CJD in Australia is conducted through the ANCJDR. The Registry is located in the Department of Pathology at the University of Melbourne.

CJD has never been notifiable in any State or Territory in Australia. The Registry collects information directly from clinicians and pathologists and conducts searches of death certificates and hospital separation records (Figure).

Figure. The Australian national CJD surveillance information flow chart

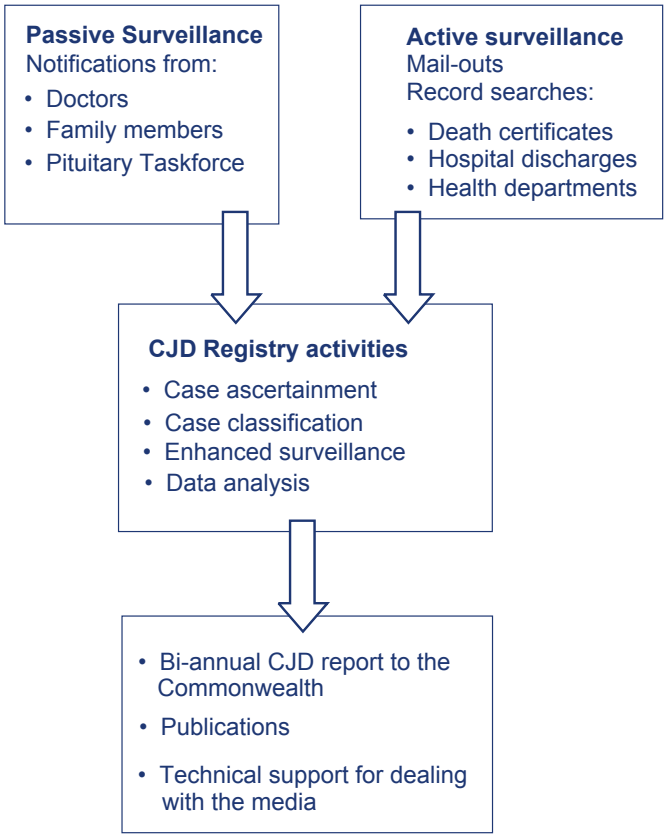

Table 1. The objectives of the CJD Registry

1. Collation and analysis of all cases of CJD identified in Australia since 1970.
2. Monitoring trends in incidence of CJD in Australia and overseas.
3. Identification of clinical features, possible risk factors and geographic distribution of CJD in Australia.
4. Establishment of diagnostic expertise in the pathological diagnosis of CJD in suspected cases.
5. Collaboration with CJD registries overseas.
6. Providing specialist advice to expert committees relating to infection control procedures.
7. Providing advice to the Commonwealth Government on scientific and medical developments relating to CJD.
8. Monitoring vCJD case diagnoses worldwide.

Objectives of the system

When first established in 1993, the Registry's brief was to record all suspected cases of CJD identified in Australia so that iatrogenic cases could be identified. In 1994, the Allars report on the use of pituitary-derived hormones in Australia recommended the expansion of the Registry's activities to include case ascertainment retrospectively from 1970 and prospectively to 2010.[23] Soon after its inception, the Registry's brief was extended to encompass monitoring of all forms of transmissible spongiform encephalopathies. The aim of the Registry was to identify all incident cases of TSEs, obtain detailed clinical information and study possible risk factors for disease development. The stated objectives of the CJD Registry are listed in Table 1.

Population under surveillance

The Registry collects data from all Australian States and Territories; it therefore covers the entire population of Australia. Prospective surveillance has been undertaken since 1993 and cases were sought retrospectively for the period between 1970 and 1992.

Data sources

As CJD is so rare, multiple and overlapping sources are used for the reporting of cases, in order to maximise the likelihood of case detection.[12] They include: personal communications with neurologists and pathologists; regular mail-outs of reminder cards to all these medical specialists; hospital medical records searches; death certificates searches; and referrals from the Pituitary Taskforce and the CJD Counseling Service. The system combines features of a passive surveillance system (e.g. personal communications by neurologists) and an active system (case ascertainment through searches of hospital records and death certificates).

For just over half of the cases reported to the Registry since its inception, the first report has come directly from neurologists and neuropathologists. Of these cases, a relatively small proportion (an additional 7%) was reported as a result of the reminder cards sent to medical specialists twice a year. The mailing list includes all practising neurologists and neuropathologists registered with the respective professional bodies in Australia. On average, 70 per cent of these specialists return the cards whether they see a case or not.

An annual search for CJD-related codes in the National Death Index maintained by the Australian Institute for Health and Welfare has been used for retrospective case finding. This was carried out by the Registry for the period from 1980 to 1994 and retrieved 21 per cent of all cases included in the Register. In the last 5 years, as more cases were notified by clinicians, the contribution of other sources to case ascertainment has proportionally decreased.[12]

Information collected

Upon notification of a possible case, a Registry member contacts the referring source and obtains detailed clinical information. The patient's family is then contacted and informed consent is sought for the completion of a questionnaire, which collates all relevant information obtained from interviewing

families and from patients' medical records. Since the Registry's inception, 936 questionnaires have been completed, with a response rate of 89 per cent. Information collected pertains to possible risk factors for CJD, socio-demographic information, as well as symptoms, clinical signs and results of relevant ancillary and laboratory investigations.

Data storage

A customised database is used to enter this information and for many fields, the Registry's comments are included as free text, which is essential for the documentation of unusual or as yet unclassified cases. Data entry errors are corrected on an informal basis each time new information becomes available and/or changes in classification are made.

Data analysis

Data are analysed twice a year by a full-time Registry member and the following calculations are carried out: the number of TSE-related deaths in Australia; the crude incidence rates of TSE; age-adjusted incidence rates for the Australian population over 45; and tabulations of cases by age, sex and aetiology (Table 2).

Dissemination of results

The information is disseminated through semi-annual reports, publications in peer-reviewed journals and presentations at scientific meetings in Australia and overseas. The semi-annual reports are sent to the Department of Health and Ageing, the National Health and Medical Research Council Special Expert Committee on TSEs, to 15 overseas collaborative TSE research units and to other organisations, on a need to know basis.

Performance of the surveillance system

Qualitative attributes

Simplicity

The Registry is recognised as the sole national repository of surveillance data on CJD. Through personal contact and regular mail-outs, the potential providers of data for the Registry can be reminded of the reporting protocols to the ANCJDR.

The ANCJDR relies on a complex network of reporting sources and conducts regular searches of hospital and death records to ensure complete case ascertainment. The Registry staff are required to have a thorough understanding of CJD pathology and of the diagnostic algorithms used for case classification. Additionally, experience with laboratory techniques involved in making the diagnosis of CJD and special skills in family counselling and interviewing are also needed. This means that although the system is simple in design, it has inherent operational complexities.

Flexibility

A surveillance system should have the ability to adapt to changing needs and/or objectives. The recent discovery of vCJD has provided a potential test of this ability. The Registry incorporated the vCJD case definition and introduced corresponding new data collection elements relating to the diagnosis and possible aetiology of vCJD, proving that the system was able to rapidly adapt to changes. The occurrence of one or more vCJD cases in Australia will be the only practical way to formally test the flexibility of the system in this new role.

Table 2. Data tabulation

Annual incidence in the general Australian population (per million inhabitants).
Annual incidence adjusted for age over 45 years (the population at highest risk).
Annual incidence by State.
Number of cases by age at onset and at death (tabulated by sex and aetiology).
Average age at death (tabulated by sex and aetiology).
Average duration of illness (tabulated by aetiology).
Tabulation of cases by occupation.
Tabulation of cases by country of birth.

Acceptability

A surveillance system depends crucially on the willingness of health professionals and those affected by the disease of interest to contribute relevant and accurate information.

Evidence for the support received by the Registry from targeted specialist doctors comes from the high proportion of cases (over half) that are received as unprompted communications. The response rate to the annual mail-outs to neurologists and neuropathologists has stabilised in the 60–70 per cent range for the last 3 or 4 years.

The acceptability of the surveillance system to patients and their families can be measured to some extent by the completion rates of questionnaires seeking information on personal, professional and medical histories. For the cases reported since surveillance was implemented prospectively in 1993, questionnaires have been completed for some 90 per cent of referred cases. According to staff, no complaints have been brought to the attention of the Registry specifically about its procedures, or the nature of the information sought.

Quantitative attributes

Sensitivity

The proportion of cases detected by a surveillance system is affected by several factors, including: the likelihood that the disease requires medical attention and is correctly diagnosed; the availability of a diagnostic test; and the chance that the case is then reported to the surveillance system.

Due to the severity of the condition, a case of CJD would come to the attention of the health system and would be referred to a specialist medical practitioner. Nevertheless, there is no information available on whether all such cases would actually be referred. It is conceivable that dementia occurring in an older person will not be fully investigated, particularly if the person dies soon after the onset of dementia.

Similarly, there has been no quantitative assessment of the proportion of potential CJD cases seen by different categories of medical specialists. It may be possible that some cases are seen by psychiatrists and general physicians who are not contacted by the Registry card system and may be unaware of the Registry's procedures or existence.

Ideally, assessment of the sensitivity of the surveillance system would require information on the true occurrence of CJD in Australia to compare with the rates being reported by the Registry. Given the rarity of CJD, it is not possible to measure its incidence independent of the Registry. However, Davanipour found that rates of sporadic CJD vary little across populations, so the sensitivity of the Australian reporting system can to some extent be assessed by comparisons with the incidence reported from other countries.[1] An increase in the notification of cases to the Registry has been observed since 1997, when diagnostic testing for CSF 14-3-3 proteins became available.[12] The annual recorded incidence of CJD has approximately doubled since the mid-1980s, from 0.564 cases per million prior to 1988, to 1.092 cases per million in 1999. Similarly, in France the annual incidence rate was 0.68 per million for 1992 to 1994 and 1.19 per million during 1995 to 1997.[24] The European Union Collaborative study found that the overall incidence rate for 1993 to 1995 in the 6 European participating countries was 0.69 per million, with rates by year and country ranging from 0.37 in 1995 in Slovakia to 1.18 cases per million in 1994 in the Netherlands.[25] The United States (US) figures for the annual age-adjusted mortality rates for CJD for 1979 to 1990 was 0.9 deaths per million; the rate remained stable through to 1994.[26,27] With the exception of the US, all other surveillance systems reported an increase in CJD incidence in the last 10 years, and attributed it to improved recognition and reporting of cases rather than an increase in the number of affected individuals.[24,25]

Positive Predictive Value

As the criteria required for diagnosis are very specific, the number of reported cases that are incorrectly labelled as CJD is very small. Through good communication between the Registry and its reporting sources, information relevant to the diagnosis of CJD is updated and the Positive Predictable Value of the Registry is likely to be high.

Representativeness

Similar to the assessment of sensitivity, the representativeness of the surveillance system can only be measured with reference to the real occurrence of CJD in different Australian subpopulations. Assessment of representativeness can be made indirectly by comparing reported rates across Australian jurisdictions, under the assumption that there is little variation in the true rate of sporadic CJD.[1] Table 3 shows little variation in reported rates across Australian States and Territories.

Table 3. Reported rates of sporadic CJD, 1990 to 1999, by jurisdictions

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA
Number of cases 1990-1999	1	64	1	38	19	2	50	23
Incidence/million	1.00	1.03	0.52	1.18	1.29	0.42	1.09	1.30
95% CI	0.025, 5.57	0.793, 1.339	0.013, 2.896	0.837, 1.14	0.776, 2.01	0.0508, 1.516	0.80, 1.438	0.824, 1.95

Confidence intervals are calculated using Poisson regression.[28]

Timeliness

Delays in CJD reporting may occur between the time it takes to report a case to the Registry (which is dependent on the time elapsed between the onset of symptoms and the time a provisional diagnosis of CJD is made) and the time it takes for the Registry to confirm the diagnosis. From a public health perspective, timeliness is important if specific actions are required to stop routes of transmission indicated by new cases.

The timeliness of reporting a case to the Registry has been improved by the introduction of the 14-3-3 assay, although this investigation is most useful in supporting the diagnosis of sporadic CJD, which does not carry public health implications. The time required for neuropathologic confirmation of a case is on average 2 months. However, if the reporting source communicates a clinical suspicion of vCJD or raises a public health concern, the Registry's prioritising of the investigations can reduce this duration to a few weeks. At the time of referral, detailed information on possible exposures that could be implicated in the transmissibility of TSEs (including surgery, hormone administration, farm work, reception/donation of transplanted organs, country of birth and occupational history) is sought from the referring doctors and families to determine whether a case has public health implications.

The usefulness of the system

The usefulness of the system can be judged by the extent to which it is supporting public health policies and interventions related to the prevention of TSEs. Since the inception of the surveillance system, the monitoring of CJD has resulted in the identification of 10 iatrogenic cases (the last one in 1999), half of them related to the use of dura mater grafts. The use of these grafts was discontinued in 1987, prior to the establishment of the Registry.[6] Half the cases were associated with the administration of human-derived growth hormone or human pituitary gonadotrophins, which ceased in 1985.[6,23] The biannual Registry reports are used to inform decision-making in the area of TSEs, primarily in confirming the absence for the moment of iatrogenic CJD or vCJD in Australia.

Discussion

The ANCJDR is detecting sporadic CJD cases at rates that are comparable to those detected in other countries with comprehensive surveillance systems. It is using sophisticated diagnostic techniques and generally benefits from a high degree of cooperation from reporting physicians and the families of people diagnosed with CJD.

By using qualitative criteria of evaluation, the CJD surveillance system was found to have high degrees of flexibility and acceptability. Although simple in design, the system relies on a complex reporting system and experienced staff, to accurately identify and classify cases of CJD and recognise those of public health importance. Evaluating the system against quantitative criteria, the PPV and representativeness were also found to be high. An independent evaluation of the system's sensitivity could not be made due to the rarity of the condition, but as the incidence of CJD is comparable to that in other countries, the sensitivity is likely to be high and some suggestions are made to validate this further. The timeliness of reporting is not high for sporadic cases, but for suspected cases of vCJD the time to diagnosis has been reduced by 75 per cent, as a result of improved lines of communication with the reporting sources and prioritisation of cases. It is unlikely that the time to diagnosis can be reduced for all referred cases with the current level of staffing, due to the high workload involved in case ascertainment and classification.

To ensure the sensitivity of the system, repeat mail-outs, or personal contact with non-responding practitioners may prove useful. An initiative of this kind would serve to assess the system's sensitivity even if it does not contribute additional cases. Another approach could be the broadening of search terms for dementia when checking hospital discharge records or death certificates to increase the yield of potential cases.

Including psycho-geriatricians and psychiatrists in the biannual mail-out may help identify possible CJD cases occurring in the elderly and improve the timeliness of reporting of possible vCJD cases, as they will most likely first present with psychiatric symptoms.

Using age standardised rates for reporting, rather than crude rates, will allow comparisons between different populations and may help differentiate whether the incidence of CJD does indeed fall with advancing age, or whether this is just under-ascertainment in some areas. To assess age-specific trends, consideration could be given to the use of age-specific rates for the entire Australian population, as applying them to the individual States and Territories would result in numbers too small to reach statistical significance.

The Australian National CJD surveillance system is designed as a stand-alone system, which contrasts with the UK system, based on a more formal collaboration between the National CJD Surveillance Unit, the Public Health Medicine Environmental Group, the Public Health Laboratory Service and the UK Health Departments.[15] The higher level of integration in the UK system was prompted by the specific requirements imposed by the vCJD epidemic, which called for a local response for the prevention of potential secondary transmission and the protection of the entire community in the context of an evolving public health threat.[15] The implementation of a formal cooperation program with health authorities, similar to the UK model, may be beneficial in the light of possible vCJD cases being detected by the Registry, to provide technical support for a national response to a suspected or confirmed case and to provide specialised information to the health sector and the wider community.

When the ANCJDR was established, the issue of whether CJD should be notifiable was debated. This issue may need to be revisited in the light of the public health implications of vCJD, which became apparent only after the Registry's inception. It can be argued that making CJD notifiable under State and Territory legislation would not improve the completeness or the timeliness of reporting, as diagnosis would still require the input of a specialist unit, due to the complex diagnostic process entailed. It can be argued that it would be best for the notifying sources to continue to report directly to the ANCJDR, rather than to the State and Territory health departments. Another option would be for reporting to continue in the present form, but that the confirmed cases be notified to a coordinating national health agency, leading to a higher degree of integration of the system and an increased awareness of TSEs for all interested parties.

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A measles outbreak among young adults in Victoria, February 2001

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On behalf of the outbreak investigation team.

Abstract

In January 2001 a 19-year-old Sydney resident, who had recently returned from India, visited Melbourne for 4 days while infectious with measles. A further 50 measles cases were subsequently identified, mainly among young adults. Thirty-eight cases (75%) were in the same birth cohort (born between 1968 and 1981). This cohort was identified as being at high risk of measles infection after a previous outbreak in Victoria involving 75 cases. These individuals are now aged between 20 and 33 years. A high proportion of cases, 22 (43%) were hospitalised after multiple visits to various healthcare providers. None of the cases had documentation of receiving the recommended number of doses of measles-containing vaccine for their age. Repeated outbreaks clearly demonstrate that young adults remain the group at highest risk of measles infection in Victoria. More targeted strategies for young adults and healthcare workers are required to better protect these groups against measles. *Commun Dis Intell* 2002;26:273–278.

Keywords: measles, outbreak, young adults

Introduction

In 1999, Victoria experienced a large outbreak of measles when a returned traveller was identified as the primary case with further transmission to 74 mainly young adult cases.[1] Together with serologic evidence which indicated a relatively low level of immunity among this cohort,[2,3] this outbreak as well as outbreaks in other States[4,5] were the impetus for a national campaign recommending measles immunisation for 18–30 year olds. The results of an investigation of another measles outbreak in Victoria that occurred less than 2 years later[6] are reported. Once again the measles virus was introduced by a returned traveller and the majority of cases occurred among young adults. The epidemiological characteristics of this outbreak which highlight the risks associated with multiple presentations to healthcare providers prior to diagnosis and the continued failure to vaccinate young adults, are described.

In Victoria, medical practitioners and diagnostic laboratories who suspect that a person may have measles are required to notify the Department of Human Services. In collaboration with the Victorian Infectious Diseases Reference Laboratory (VIDRL), the Victorian Department of Human Services has been conducting enhanced surveillance for measles since 1997 which includes home visits to collect specimens for laboratory confirmation of the diagnosis and, where possible, identification of the virus genotype.[7,8] The specimen collection service is offered for all notified cases not just those that meet the definition of 'suspected infection' (see below). Specimens are forwarded to VIDRL and tested for IgM and IgG antibodies to measles, rubella and parvovirus B19. Testing for viruses commonly causing rash illnesses improves the turn-around time of an alternative diagnosis to measles and adds a specificity check for a positive IgM result. All notifications are followed-up with the medical practitioner, patient (or the parent/guardian if the patient is a child) and, if applicable, the primary diagnostic laboratory. Demographic data, clinical details, vaccination history, exposures during the incubation period and contacts during the infectious period are recorded using structured telephone interviews.

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Methods

Measles cases were defined in accordance with the national guidelines:[9]

Confirmed infection

- A laboratory-confirmed case defined as the presence of measles specific IgM antibody in an appropriate specimen[9] (excluding those serologically diagnosed cases who received a measles-containing vaccine 8 days to 8 weeks before testing who were not linked to another laboratory-confirmed case).
- A person with signs and symptoms consistent with measles (see 'suspected infection') epidemiologically linked to a laboratory-confirmed case.

Suspected infection

- A person with an illness including all of the following features: morbilliform rash, cough and fever present at the time of rash onset.

On identification of this outbreak, active surveillance was instituted to identify additional cases and protect susceptible contacts. Health alerts were distributed to medical practitioners, hospitals, local councils and child-care centers. Regular press releases were issued.

Where appropriate, advice was given about the need for personal isolation during the infectious period, such as exclusion from school and child-care centres. Measles-mumps-rubella vaccination or immunoglobulin was offered to contacts in accordance with the guidelines.[9]

If initial serology was performed elsewhere the original sample was requested to be forwarded to VIDRL for confirmatory testing. During the outbreak the turnaround time for measles IgM and IgG results was approximately 4 hours from receipt of specimen. Polymerase chain reaction (PCR) for the detection of measles virus RNA was performed for virus genotyping at VIDRL.[10]

Results

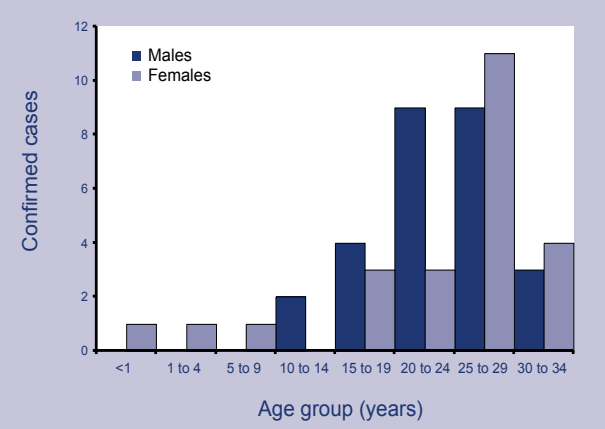
The primary case

On 29 January 2001 the New South Wales Health Department reported that a 19-year-old Sydney resident with no documented history of previous measles vaccination had been serologically confirmed with measles after returning from India on 4 January 2001. Rash onset for this patient was 20 January 2001. During the infectious period the patient visited Melbourne for 4 days (from 17 to 20 January 2001) and attended numerous locations including restaurants, a nightclub and shopping centres, and had travelled on public transport.

The outbreak

The first Victorian case was notified on 1 February 2001 and the following day 2 more cases were reported. All three were young adults who had been admitted to hospital. By 31 March 2001, 151 notified cases of measles had been investigated. The diagnosis of measles was confirmed in 52 patients, although one case was not considered part of the outbreak as the person had been overseas for the entire incubation period (United States of America) and the virus identified was a distinctly different genotype (see below). Of the 51 confirmed cases considered part of the Victorian outbreak, 50 were laboratory-confirmed (including the primary case) and one was epidemiologically linked (a housemate of a laboratory-confirmed case). The median age of these cases was 25 years (range 10 months – 34 years) with most (75%) aged 20–34 years (Figure 1). Twenty-seven were males.

Figure 1. Confirmed cases of measles, Victoria, February to March 2001 (n=51), by age group



Of the 151 notifications, 70 cases were rejected on the basis of serological evidence.[7,9] Two of these cases were confirmed as parvovirus B19, one was confirmed as rubella virus, and two were vaccine reactions (a 14-month-old child vaccinated 7 days prior to rash onset and confirmed by PCR to be shedding vaccine strain measles virus, and a 20-year-old person with no epidemiological link to a laboratory-confirmed case who was vaccinated 14 days prior to rash onset). There was no alternative diagnosis for the remaining 65 cases.

A further 29 cases did not have an epidemiological link to a laboratory-confirmed case and could not be confirmed or rejected on laboratory evidence. Only four of these cases met the clinical definition of 'suspected infection': three were unvaccinated infants who were measles IgM and IgG negative but the specimen was collected less than 4 days after rash onset and the offer to collect a convalescent specimen was declined; the parent of the fourth case (an unvaccinated child aged 9 years) did not consent to specimens being collected for laboratory confirmation of the diagnosis. Twenty-five cases did not meet the clinical definition of 'suspected infection', 20 of these were measles IgM and IgG negative but the specimen was collected less than 4 days after rash onset so they and were excluded on clinical grounds.[9]

Utility of clinical definition for suspected measles

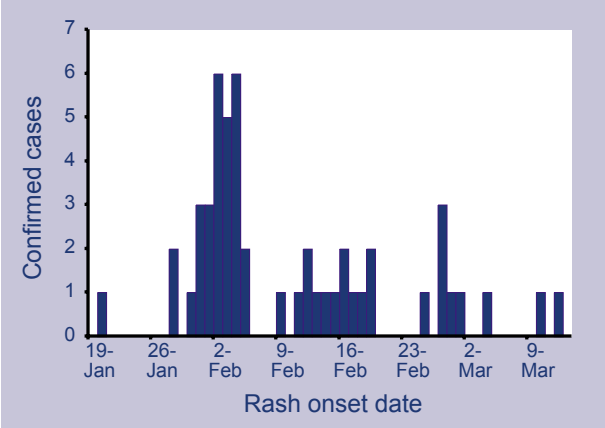
In order to assess the utility of the clinical definition of suspected measles infection,[9] the clinical symptoms for the 50 laboratory-confirmed cases were compared to the clinical symptoms of the 70 cases that were serologically-confirmed as not being measles. Of those cases who met the suspected case definition, 71 per cent (47/66) were confirmed as measles whereas 94 per cent of cases who did not meet the clinical criteria (51/54) were serologically-confirmed as not being measles (sensitivity 94%, specificity 73%).

Transmission

The first Victorian case developed a rash on 28 January 2001, 8 days after rash onset in the primary case. The last case developed a rash on 13 March 2001 (Figure 2). Direct links between the primary case and a further 8 cases were identified in the first wave of transmission. These secondary cases were known to have attended the same restaurant, nightclub or airport terminal at the same time as the primary case during his infectious period. Probable links with another 9 patients were established. These patients had either been in the same area, visited a common shopping centre or travelled on the same train line as the primary case. Further chains of transmission were established for 24 additional cases. Two of the identified chains of transmission included:

- An unvaccinated 21-year-old female who may have traveled on the same train line as the primary case, visited a nightclub during her infectious period (prior to rash onset). A singer at the nightclub subsequently developed measles with symptoms of rash and fever. He attended a hospital emergency department during his infectious period at the same time as an unvaccinated 11-year-old who was presenting with a broken arm. The 11-year-old subsequently developed measles and infected his unvaccinated 9-year-old sibling. Every case in this chain of transmission was laboratory-confirmed and none had been vaccinated against measles.
- A 29-year-old woman, in whom measles had not been suspected, had 2 visits to a hospital emergency department and was subsequently admitted to a ward but was not isolated. A healthcare worker at the hospital, who had remained unvaccinated despite being identified as susceptible in the previous outbreak, subsequently became infected and was also hospitalised.[11]

Figure 2. Epidemic curve for measles outbreak, Victoria, February to March 2001 (n=51)



All but two of the 51 confirmed cases lived in the metropolitan area of Melbourne. One of the non-metropolitan cases was at the same Melbourne Airport terminal as the primary case when the latter was infectious. No direct epidemiological link was identified for the other non-metropolitan case (aged 13 months), however, the infant had been at a wedding at which a number of guests were young adults from metropolitan Melbourne.

Genotyping

Serum from the primary case was negative for measles virus RNA by PCR. However, measles virus RNA was detected by PCR from clinical specimens from 24 patients all of whom were infected with the same novel genotype, proposed as new genotype 'D8'.[12] During the course of the outbreak, we identified one laboratory-confirmed case that was a different genotype (D3). This case, a 23-year-old female, developed a rash and fever while flying back from the United States of America and was admitted directly to hospital.

Hospitalisations

Of the 51 confirmed cases, 22 (43%) were hospitalised for a total of 91 in-patient days (range one to 10 days). All but two of the hospitalised cases were in the 17–34 year age range. Clinical presentations requiring admission included a combination of dehydration, lethargy, diarrhoea, nausea, vomiting, headache, rash and fever of unknown origin. One case also presented with suspected appendicitis and had an appendectomy the day prior to rash onset. Two cases developed pneumonia. There were no deaths.

The 22 hospitalised cases accessed health services (either hospitals, GPs or health centres) a total of 62 times. All 22 had sought treatment at GP clinics before being admitted to hospital. Three of the cases had been admitted and discharged without diagnoses before being readmitted and subsequently diagnosed. Only 4 of the 22 hospitalised cases (13%) were suspected as having measles on the initial presentation to a healthcare provider. An early case sought treatment at four different GPs before being admitted to hospital 11 days after the initial presentation. Another case visited 3 hospitals with no clear diagnoses before being admitted 4 days after rash onset. Information regarding access to healthcare from non-hospitalised patients was not collected.

During the 1999 measles outbreak 6 healthcare workers were infected,[1] however, despite the large number of cases hospitalised during this outbreak only one healthcare worker was infected. Of concern is the fact that this healthcare worker was identified during the 1999 outbreak as being susceptible to measles infection but did not receive any measles containing vaccine. During infection control investigations following possible exposure of staff relating to a hospital admission of a confirmed case, exposure and infection of this healthcare worker were confirmed. This case highlights the need for continued vigilance on the part of healthcare workers and healthcare employers to ensure staff protection from vaccine preventable diseases.

Vaccination status

One case was too young to have received a first dose of measles vaccination (10 months of age). Four cases (age range 17–29 years) had received one documented dose of a measles-containing vaccine when they were aged between 13–24 months but none of these cases had received a second dose as currently recommended.[13] None of the other cases had a documented history of prior measles vaccination, 2 cases were siblings whose parents were conscientious objectors to vaccination.

Discussion

In 1999, a 21-year-old unvaccinated person who returned from holidays in Bali was the index case in an outbreak in which 75 cases of measles were identified.[1] In January 2001, another returned traveller, this time a 19-year-old who had returned from India, triggered an outbreak of 51 cases among a similar age group. The primary case in this outbreak developed a rash 16 days after returning from India and was only in Melbourne for 4 days during the infectious period. We were able to identify exposure to the primary case for eight of the cases in the first wave of transmission and probable exposure for another 9 cases. Genotyping established that 24 cases had the same novel genotype virus recently described as an endemic strain in Nepal.[12] This continues the pattern of rapidly changing measles virus genotypes in Victoria over the last 15 years without evidence of sustained transmission,[10] which is highly suggestive of the interruption of indigenous measles transmission.

Lambert, et al highlighted the changing epidemiology of measles in Victoria and suggested that young adults[1] born between 1968 and 1981, were the group most at risk of measles in Victoria. Of the 51 cases identified in the 2001 outbreak, 38 (75%) were in this birth cohort and none of these cases had documentation of receiving 2 doses of a measles-containing vaccine. Persons born prior to 1970 are generally presumed to be immune to measles,[13] however in this outbreak 6 confirmed cases (12%) were born between 1966 and 1970.

In both the 1999 and 2001 outbreaks, a large proportion of the young adults with measles were hospitalised. The increased severity of the disease in adults has been documented elsewhere.[14,15] A third outbreak has now been identified among a similar age group in Victoria and was being investigated at the time this manuscript was prepared. Repeated outbreaks clearly demonstrate that young adults remain the group at highest risk of measles infection in Victoria.

Highly sociable, mobile living patterns within this group together with the fact that they were born during a period of introduction of vaccination into routine use makes them more susceptible to infection. This age group were either not vaccinated or because of the introduction of vaccines reducing the circulation of the virus, they were less likely to have come into contact with the virus and therefore not vaccinated or infected. The variety of settings and passing of incidental contact of most cases highlights the infectiousness of this disease and the need for prompt implementation of public health measures.

In this age of relatively inexpensive and rapid travel between countries and within regions, importation of measles, particularly in young adults, will continue to be a public health problem. Vaccination of international travellers to endemic areas should be emphasised until global elimination of measles has been achieved.

Young adults are a particularly difficult group to target effectively for widespread immunisation programs because of their mobility and diversity of employment. GPs, travel clinics and university and defence forces health services should be advised to take every opportunity to vaccinate this age group. Because of the continued threat of reintroduction of the virus, vaccination of travellers in this age group should be a high priority. Strategies should also target young adults who travel to measles endemic countries, as they are at increased risk of being exposed to measles.

Difficulties in clinical diagnosis of measles are well recognised now that measles has become a much rarer disease.[16,17] Even though 71 per cent of confirmed cases in this outbreak could have been diagnosed as suspected measles on the clinical grounds of rash, cough and fever at rash onset, many cases experienced delay in diagnoses. All but one of the 22 hospitalised cases experienced delayed diagnoses and there were multiple presentations among this group, presenting opportunities for further transmission within the healthcare setting. Laboratory confirmation is an integral component of enhanced surveillance for measles in Victoria,[7] but the disease must first be suspected before it can be reported.

Outbreaks will continue to occur in Victoria as imported cases introduce the virus into a high-risk young adult population. Not only are young adults a susceptible population, they are also a group which is highly mobile and may travel to areas where they are at higher risk of acquiring measles. Awareness amongst practitioners and the community that measles is now a disease of young adults as opposed to children, needs to be reinforced. The high number of hospitalisations also suggests that young adults are more likely to have severe disease. The single most effective strategy is to ensure that all young adults, healthcare workers included, have at least 2 documented doses of a measles-containing vaccine. It is not enough to rely on self-reported history of previous vaccination or exposure to wild virus.[18]

Outbreak investigation team

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Melioidosis in the Torres Strait Islands of Far North Queensland

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Abstract

During the six-year period from 1995 to 2000, 23 cases of melioidosis were diagnosed from the Torres Strait islands that lie between northern Queensland and Papua New Guinea. This represents an average annual incidence of 42.7 per 100,000 population, the highest documented to date in this region. This probably reflects the extremely high prevalence of diabetes, the high seasonal rainfall in the area, and the lifestyle of Torres Strait Islanders. The majority of patients (20 out of 23) acquired their disease in one of the more remote outer island indigenous communities. Most patients presented with a community-acquired pneumonia or with deep seated abscesses. One patient presented with the first case of suppurative parotitis due to melioidosis recorded in Australia. Diabetes was the overwhelming risk factor, being present in over three-quarters of all cases. Five patients (22%) died. Strategies to try to minimise illness and death due to melioidosis in the Torres Strait are discussed. *Commun Dis Intell* 2002;26:279-283.

Keywords: melioidosis, Burkholderia pseudomallei, *Torres Strait, Papua New Guinea, incidence, diabetes, prevention*

Introduction

Melioidosis is a well-recognised infection caused by the bacteria *Burkholderia pseudomallei*, which is a free-living organism present in the soil, especially mud, and surface water. The disease is endemic in northern Australia and South-East Asia,[1] where it is a significant cause of morbidity and mortality. Most infection is asymptomatic and disease usually occurs in people with risk factors. Latent infection and relapse are not uncommon. This study reports on 23 cases of melioidosis that presented in the Torres Strait between 1995 and 2000.

The Torres Strait, which separates Cape York peninsula from mainland Papua New Guinea (PNG), has a population of about 9,000 mainly Indigenous inhabitants. About half of these people live on or close to Thursday Island which is the main commercial and government centre. The island has a 38 bed general hospital. The rest of the population live in communities on 15 inhabited outer islands that stretch up to the northernmost Australian border. This runs very close to the PNG mainland. A health centre is located on each of these islands and is staffed by remote area nurses and/or indigenous health workers. These clinics are also serviced by regular visits by a doctor, but sick patients requiring in-patient care must be evacuated by air to Thursday Island. The closest referral hospital is Cairns Base Hospital more than 800km away. This area has two distinct seasons with a monsoonal wet season from December to May each year. The main health problem in the Torres Strait is type 2 diabetes mellitus and its associated complications.

Methods

We retrospectively (using systematic hospital record review) and prospectively (from 1999) analysed patients from the Torres Strait who presented with culture-proven melioidosis in the 6 years from January 1995 to December 2000. In all patients *B. pseudomallei* was isolated from clinical microbiological specimens. All patients were admitted to the Thursday Island hospital for initial management, however, most were transferred to Cairns Base Hospital or Townsville General Hospital for further investigation or treatment. One case, a child from PNG was excluded from the analysis which only looked at patients who resided in the Torres Strait. Cases with presumed cure of previous melioidosis that subsequently recurred (i.e. were reinfected) were considered new episodes. However, patients with a relapse of melioidosis (i.e. recrudescence or treatment failures) were only included once in the series. This study was approved by the Torres Strait and Northern Peninsula Area Health Services District.

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Results

Melioidosis was confirmed in 23 cases during the study period. This represented an average annual incidence of 42.7 cases per 100,000 population in Torres Strait Islanders. The median age was 43 years and sex distribution was equal. All cases were in Indigenous Torres Strait Islanders.

Eighteen of the 23 cases occurred during the wet season months from December to May, confirming the seasonal nature of the disease (Table). There was some clustering of cases particularly during the 1998-1999 wet season (8 cases), however, new cases were diagnosed during six of the seven wet seasons included in the survey.

Table. Melioidosis cases, Torres Strait, 1995 to 2000

Case	Date	Age	Sex	Community	Risk factor	Presentation	Outcome
1	Feb 95	46	F	St Pauls	Diabetes	Pneumonia	Recovery
2	May 95	66	M	St Pauls	Diabetes	Cerebral abscesses	Disability
3	Aug 95	63	F	Murray	Diabetes + PHx	Osteomyelitis tibia	Chronic infection
4	Oct 95	28	M	Yam	Diabetes + CHD	Liver abscess	Recovery
5	Feb 96	8	M	Horn	None	Pneumonia†	Death
6	Mar 96	35	F	St Pauls	Diabetes + PHx	Splenic abscess	Relapse
7	Jul 96	45	M	Murray	Diabetes	Septic arthritis elbow†	Death
8	Oct 96	71	M	Saibai	Diabetes	Pneumonia†	Relapse
9	Feb 98	17	M	Kubin	Diabetes	Parotid abscess	Relapse
10	Mar 98	22	M	Badu	None	Puo	Recovery
11	May 98	39	F	Mabuiag	Diabetes	Septic arthritis ankle	Recovery
12	Jun 98	59	M	St Pauls	Diabetes + PHx	Retro-orbital abscess†	Death
13	Dec 98	61	F	St Pauls	Diabetes	Pneumonia	Recovery
14	Jan 99	24	F	Hammond	Pregnancy	Intraspinal abscess	Recovery
15	Jan 99	61	F	Thursday Is.	Diabetes + PHx	Septic arthritis knee	Disability
16	Feb 99	43	F	St Pauls	Diabetes + IS	Epidural abscess	Disability
17	Feb 99	47	F	Saibai	Diabetes + CRF	Pneumonia†	Death
18	Feb 99	39	M	Mabuiag	Diabetes	Liver abscesses	Recovery
19	Feb 99	45	F	St Pauls	Diabetes	Groin abscess	Recovery
20*	Feb 99	8	F	PNG	IS	Pneumonia	Death
21	Mar 99	40	F	Badu	Diabetes	Liver abscesses	Recovery
22	Jan 00	30	M	Badu	None	Pneumonia	Recovery
23	Feb 00	39	F	Badu	None	Septic arthritis knee	Disability
24	Dec 00	76	M	Yam	Diabetes + CRF	Pneumonia	Death

* This patient from PNG was not included in the analysis of cases
† Also presented clinically septicaemic
PUO Pyrexia of unknown origin
CHD Congenital heart disease
PHx Previous history of melioidois
IS Immunosuppression = immunosupressant medication or malnutrition
CRF Chronic renal failure

Diabetes was by far the most common risk factor, being present in 18 cases (78%) (Table). Four of these patients had previously been diagnosed with melioidosis and 4 patients (17%) had no recognisable risk factors for melioidosis. Pneumonia was the most common form of presentation (7 cases) followed by internal organ abscesses (5 cases, including 3 liver abscesses). Five patients died, representing a 22 per cent mortality. Four of these patients were clinically septicaemic at presentation. One death was in an 8-year-old boy with no risk factors for melioidosis.

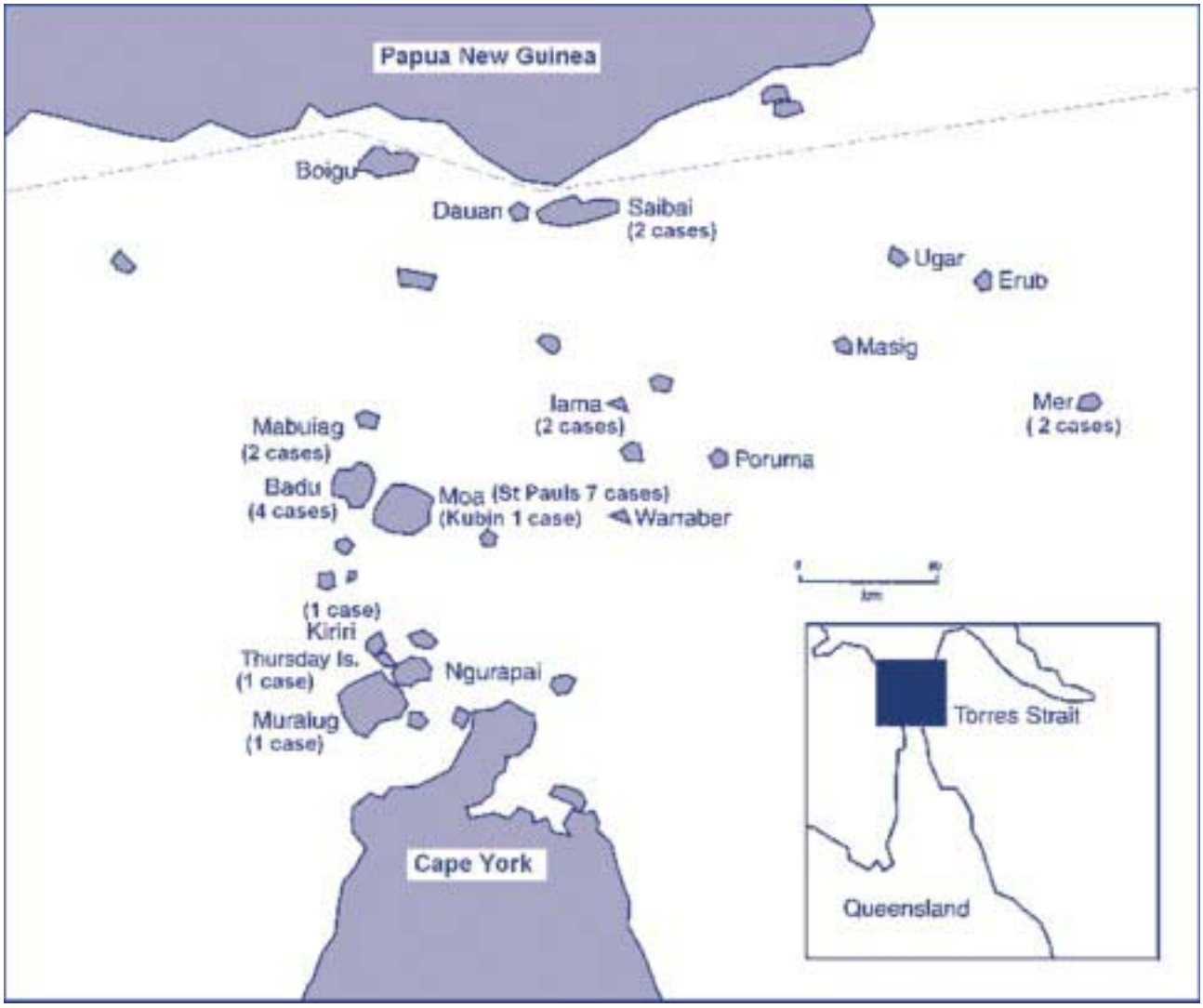
The geographical distribution of cases is shown in Figure 1. Of note is that only 3 cases occurred on or close to Thursday Island where approximately half the population lives. Most patients presented from the outer island Indigenous communities with 7 cases recorded from St Pauls community on Moa Island.

Discussion

Melioidosis is endemic in the northern part of Australia.[2] It was first described in humans in Australia in north Queensland in 1962.[3] The first cases of melioidosis in the Torres Strait were reported in 1967[4] and 4 cases from Thursday Island were included in a clinical and epidemiological review of 20 patients investigated at the Cairns Base Hospital in 1984.[5] The seroprevalence for melioidosis in the Torres Strait islands has been documented at 7.8 per cent.[2]

It has been suggested that the incidence of melioidosis is highest in the north of the Northern Territory, with two hyperendemic areas for melioidosis being the 'Top End' of the Northern Territory and Ubon Ratchatani province in north-east Thailand.[6] The average annual incidence of melioidosis in the Torres Strait islands during the

Figure 1. Map of Torres Strait showing distribution of melioidosis cases, 1995 to 2000



period of our study was 42.7 per 100,000 population. This compares with an annual incidence of 16.5 per 100,000 population in the Top End[6] and 4.4 per 100,000 population in Ubon Ratchatani.[7] Several factors may explain the remarkably high incidence of melioidosis in the Torres Strait. Diabetes is well recognised as the most important risk factor for melioidosis[5,7,8,9,10] and the prevalence of type 2 diabetes in adults is about 24 per cent in the Torres Strait island population, the highest rate in Australia and also one of the highest rates internationally.[11,12] Over three-quarters (78%) of cases of melioidosis in our series were in diabetic patients, which is much higher than in most other series (37% in Currie, 2000;[10] 21% in Suputtamongkol, 1994;[7] 32% in Chaowagul, 1989;[8] 30% in Guard, 1984;[5] 24% in Rode and Webling, 1981[13]) except for Suputtamongkol, 1999,[9] where 61 per cent of melioidosis cases were in diabetics. The Torres Strait also has a very high rainfall concentrated during the summer wet season. This area has an average annual rainfall of 2,041mm compared with 1,713mm in Darwin (Bureau of Meteorology data) and it has been shown that there is a strong association between the incidence of melioidosis and average rainfall in some endemic areas.[7,14] Finally, Torres Strait islanders have a basic and outdoor lifestyle, especially those living on the outer islands where often footwear is not worn. This is conducive to the acquisition of melioidosis which is most likely transmitted from percutaneous inoculation or exposure to wet soil and surface water.[6] Indeed, it has been demonstrated that there is a synergistic interaction between diabetes and the extent of exposure to wet soil as risk factors for melioidosis.[9]

It has been recognised that discrete foci of melioidosis infection has occurred.[2] A cluster of cases was recorded in St Pauls village, one of the 2 communities on Moa Island. Seven cases (30%) occurred over a 4 year period in a population of only 237 people, which represents 3 per cent of the total population in the Torres Strait. These included 3 patients presenting over a 7 week period during the 1998-1999 wet season. Previous outbreaks of melioidosis that may have been related to contamination of the community water supply have been recognised.[15] The clustering of cases on St Pauls was not recognised at the time and no further investigation was undertaken. However, since February 1999 there have been no more cases reported from this community.

As with most other recent series in Australia,[5,10,13] pneumonia was the most common form of presentation. Our series included a 17-year-old boy who presented with a parotid abscess requiring surgical drainage. Although suppurative parotitis has been noted as a characteristic form of melioidosis in north-east Thailand[16] where it accounts for about 6 per cent of cases in adults and up to 38 per cent of melioidosis in children, it has previously never been reported in Australia.[6,10]

An 8-year-old-girl who crossed the border from PNG was admitted to the Thursday Island Hospital with fulminant septicaemic melioidosis and died shortly after admission. The Torres Strait shares an international border with the Western Province of Papua New Guinea and over recent years 12 culture-confirmed cases of melioidosis have been isolated from a small community in the Fly region of the Western Province. The average age of these cases was 7 years (Dr J Warner, James Cook University, Townsville, personal communication, August 2000). It is interesting that the only case of melioidosis diagnosed in a Papua New Guinea national in the Torres Strait was also in a child.

This study has identified melioidosis as an important cause of illness and death in the Torres Strait. Strategies to try and decrease morbidity and mortality must stress the importance of early diagnosis and treatment.[17] A high index of suspicion for melioidosis should be maintained in any undiagnosed infective illness especially in a diabetic during the wet season, and appropriate antibiotic and supportive treatment must be started early. Prevention of melioidosis is based on reducing exposure to the organism in the soil. This basically involves public education, especially of diabetics. After the 1999 wet season when an increased number of melioidosis cases occurred in Far North Queensland, a public education program and associated health promotion pamphlet and poster aimed at informing indigenous communities about melioidosis and its prevention (Figure 2) was initiated by the Tropical Public Health Unit in Cairns. Topics emphasised included avoiding contact with soils or muddy water especially in the wet season; wearing protective footwear and gloves especially whilst gardening; covering open wounds; and general diabetes and foot care.

Figure 2. Public education poster for melioidosis prevention (courtesy of the Tropical Public Health Unit, Cairns, Queensland)

Acknowledgements

We acknowledge all the medical, nursing and health worker staff who have helped in the management of patients, including Blair Koppen. Thank you to John McBride and Clive Hadfield for managing patients in Cairns Base Hospital and providing clinical advice. We thank Robyn McDermott, Ann Richards, Di Brooks, Dianne James and Jeff Hanna at the Tropical Public Health Unit in Cairns for their help and for permission to reproduce the melioidosis poster, and to Ashim Sinha and Sid Selvanayagam for comments on the manuscript. We also acknowledge the Torres Strait and Northern Peninsula Area Health Council and the people of the Torres Strait.

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The National Public Health Partnership

The National Public Health Partnership was established in 1996 through a Memorandum of Understanding between the State, Territory and Commonwealth Health Ministers. It is the peak intergovernmental forum on public health issues reporting directly to the Australian Health Ministers Advisory Council (AHMAC).

The Partnership group members include senior public health officials from the States, Territories and the Commonwealth health departments and representatives of the Australian Institute of Health and Welfare and the National Health and Medical Research Council. A representative of the Ministry of Health of New Zealand attends Partnership meetings as an observer. The Partnership has an advisory group of representatives of peak non-government and professional organisations with an interest in Public Health, and the chair of the Advisory Group also attends Partnership meetings.

In the early years of its work the Partnership focused its attention on public health infrastructure issues such as information, public health practice and planning issues, workforce issues, research and legislative reform and established the National Public Health Information Working Group and the Legislative Reform Working Group.

In 1999 a decision was made by AHMAC that all public health committees reporting to AHMAC would become committees of the Partnership, hence the then Communicable Diseases Network Australia New Zealand became a sub committee of the Partnership and a member of the Partnership was appointed as the Chair of the Network.

This decision coincided with a general shift in the Partnership's agenda to include specific priority public health issues along with issues of public health practice and infrastructure. The Partnership now has a number of subcommittees addressing the range of public health priority issues in addition to Communicable Diseases Network Australia (CDNA), including:

- enHealth Council, addressing environmental health matters;
- SIGNAL, the Strategic Intergovernmental Nutrition Alliance;
- SIGPAH, the Strategic Intergovernmental Forum on Physical Activity;
- SIPP, the Strategic Injury Prevention Partnership;
- CHIP, the Child and Youth Health Intergovernmental Partnership; and
- PHGWG, the Public Health Genetics Working Group.

The Partnership has always sought to work in partnership with other key groups and has joint working groups in general practice, mental health and healthy ageing with other key national bodies.

The Partnership has a number of major ongoing projects including:

- Development of the Public Health Evidence Schema. This project is developing a schema for considering evidence in relation to public health interventions which is broader than assessment based on a hierarchy of evidence more suitable for clinical practice and more readily addresses issues such as context.
- Development of public health performance indicators. A consultation process is currently underway in the States and Territories to assess appropriate indicators for public health for inclusion in national health performance reporting.
- The Public Health Expenditure Project which reports on expenditure in a number of categories of public health practice across the States, Territories and the Commonwealth.
- Development of a framework for public health workforce studies.
- A project to develop guidelines for National Public Health Strategies to improve their capacity to address Aboriginal and Torres Strait Islander health issues.

In addition, each sub-committee has an extensive work program.

A review was conducted of the Partnership's activities over the first 5 years of its operations. This review reported to AHMAC in February 2002. At that meeting AHMAC endorsed the Partnership's continuing operation for the next 5 years and endorsed a framework for its agenda and priorities. The current Chair of AHMAC, Dr Rob Stable from Queensland, was appointed as Chair of the Partnership from June this year.

Communicable diseases remain a key priority for the Partnership under its new work program. The CDNA is strongly supported by the Partnership with project funds for some key initiatives of CDNA being funded through a cost sharing arrangement between Partnership members.

A number of key themes have been endorsed to guide the work of all sub-groups of the partnership over the next few years. These themes include:

- addressing health inequalities through public health programs and interventions;
- improving the quality of public health practice;
- engaging effectively with non-government organisations, key experts, major national structures and key consumer groups;
- strengthening the evidence for public health interventions;
- integrating key risk groups, settings and priority areas into all work programs;
- collaborating and priority setting in public health research; and
- regulatory reform.

Details of the Partnership's activities and its publications can be accessed on the Partnership's Website at: www.nphp.gov.au.

Dr Cathy Mead
Executive Officer
National Public Health Partnership

Outbreak of gastroenteritis due to *Salmonella* Typhimurium phage type 135a following consumption of raw egg

Robert Hall[1]

On 19 and 20 December 2001, 3 notifications of infection with *Salmonella* sp. were received by the Communicable Disease Control Branch for persons who had attended the same Christmas function on 14 December. On 21 December 2001 a voluntary service organisation reported that 4 of about 14 people attending a Christmas function had reported illness with gastrointestinal disease. The function was for staff of the organisation and was self-catered in a private home, with each of the guests contributing food and drink items. Interview of the hostess on 21 December 2001 established that 20 people (17 adults and 3 children), including the 2 notified cases, had attended the function. A list of persons attending the function was obtained from the voluntary organisation and 6 were interviewed to compile a list of menu items. On 24 December 2001 an additional case was notified. This case had also attended the function. Organisms from all cases were subsequently typed as *Salmonella* Typhimurium phage type 135a.

A questionnaire was developed inquiring after symptoms before and after the function and foods and drinks consumed at the function. A case was defined as a person attending the function who had reported diarrhoea after 14 December 2001. Nineteen persons were available for interview. Responses to the questionnaire were entered into a database constructed in EpiData 2.0b and analyzed using Stata 7 and Excel 2000.

Eleven of the 19 persons attending were ill, 4 were admitted to hospital. The epidemic curve is presented in the Figure.

Figure. Cases of *Salmonella* Typhimurium phage type 135a, 14 to 19 December 2001, by date of onset.

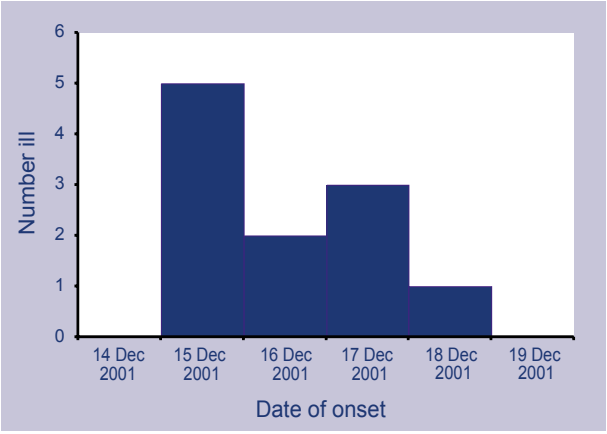

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Table 1. Foods consumed and risk ratios for developing gastroenteritis

Food	Risk ratio	95% Confidence interval
Chicken wings	0.89	0.21, 3.80
Lasagna	1.88	0.36, 9.71
Salmon roulade	0.75	0.35, 1.62
Party pies	1.65	0.77, 3.53
Sausage rolls	undefined	-, -
Cocktail frankfurts	2.00	0.60, 6.64
Quiche	0.97	0.41, 2.32
Cherries	1.18	0.28, 4.97
Tiramisu	undefined	-, -
Hazelnut torte	1.48	0.57, 3.90
Rum balls	0.00	-, -
Chocolate slice	1.80	1.19, 2.72
Apricot slice	0.00	-, -
Almond bread	1.24	0.58, 2.64
Toffeed almonds	1.25	0.55, 2.84
Nibbles	1.81	0.91, 3.59
Nuts	0.98	0.44, 2.18

Foods consumed at the function and risk ratios for diarrhoea are presented in Table 1.

These results indicate a risk due to sausage rolls, tiramisu and chocolate slice. The sausage rolls were of 2 types: home-made and store-bought. Eighteen persons reported eating sausage rolls, and 11 became ill. Fourteen persons reported eating the tiramisu and 10 became ill. Only 1 person reported eating the chocolate slice.

Some leftover foods were available for sampling and these were cultured at the Food Laboratory of the Institute of Medical and Veterinary Sciences. Culture results are presented in Table 2.

The microbiology results support the hypothesis that the primary vehicle for infection was the tiramisu, with cross-contamination of some of the other foods. It is possible that the other foods may have been contaminated after the function, since all the foods available for culture were stored in the same fridge after the function.

The tiramisu was made by one of the guests, who denied illness before or after the party. It was made from Italian sponge biscuits, percolated coffee, port, free-range eggs, cream, sugar and cocoa. The port came from a flagon won in a raffle at a pub, the eggs were obtained from either a family friend or a relative (who kept layer chickens) and the other ingredients were commercial products obtained from supermarkets. The tiramisu was made by soaking the biscuits in the coffee and port, and whipping the cream and eggs together. Cocoa was added as a topping. It was set in the fridge and there was no cooking step. No eggs were available for culture.

These results indicate that it is likely that the eggs used in preparing the tiramisu were the original source of this outbreak. Outbreaks of *Salmonella* gastroenteritis where raw eggs have been implicated as the original source are not uncommon. In the United Kingdom and United States of America *Salmonella* Enteritidis phage type 4

Table 2. Microbiology of foods available for culture

Food	Result	Colony count
Fruit and nut slice	*Salmonella* Typhimurium PT 135a	<3cfu/g
Hazelnut torte	*Salmonella* Typhimurium PT 135a	<3cfu/g
Mixed nuts	*Salmonella* Typhimurium PT 135a	<3cfu/g
Tiramisu	*Salmonella* Typhimurium PT 135a	>1,000 cfu/g
Chocolate slice	*Salmonella* Typhimurium PT 135a	<3cfu/g
Toffeed almonds	Negative	
Almond bread	Negative	
Apricot/marshmallow roll	Negative	

has been associated with consumption of raw egg.[1,2] In South Australia, an unrelated outbreak of gastroenteritis due to *Salmonella* Typhimurium phage type 135a due to the consumption of a pie glazed with raw egg and rice pudding made with raw egg was reported in 2001.[3] An outbreak associated with raw egg due to contamination of mock ice cream containing raw egg with *Salmonella* Typhimurium phage type 135 was reported in Western Australia in 2001.[4] In 2001,[4] outbreaks of *Salmonella* (of different serovars) infection related to eggs were reported to OzFoodNet, including the South Australia and Western Australia outbreaks noted above (Martyn Kirk, OzFoodNet, personal communication). Between 1995 and 2000, 9 outbreaks of *Salmonella* infection have been identified where there has been exposure to eggs. Four of these have involved *Salmonella* Typhimurium phage type 135 (Craig Dalton, Hunter Public Health Unit, personal communication). These reports, and the present report, indicate a potential risk of *Salmonella* infection from raw egg and that foods containing raw egg should not be consumed by vulnerable groups such as the elderly and the immunocompromised.

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Erratum

The following corrections to *Communicable Diseases Intelligence* Vol 26 No 1 should be noted.

Page 11, Table 1. 'Fragmentation of influenza surveillance in Australia' by Watts and Kelly.
The headings for columns 5, 6 and 7 were incorrect.

Column 5 should read 'record influenza-**like illness**'

Column 6 should read 'Virological **surveillance with laboratory support**'

Column 7 should read '**Collation of data**'

Page 75: Table 2. Notifications of diseases received by State or Territory health authorities in the period 1 October to 31 December 2001, by date of notification

Diphtheria results were incorrectly inserted in the 'Sexually transmissible diseases' and 'Other bacterial infections' sections of the Table.

The data for 'Ornithosis' was entered into the row for 'Other Lyssavirus' and visa versa. There were no reports of other lyssavirus and 37 cases of ornithosis notified in the quarter.

Page 79: Table 3. Notification rates of diseases by State or Territory, 1 October to 31 December 2001 (rate per 100,000 population)

The data for 'Ornithosis' was entered into the row for 'Other Lyssavirus' and visa versa. There were no reports of other lyssavirus and the rate for ornithosis was 0.8 per 100,000 for Australia in the quarter.

Page 81: Table 4. Virology and serology laboratory reports by State or Territory for the reporting period, 1 October to 31 December 2001, and total reports for the year

The data section for 'Ortho/paramyxoviruses' was duplicated in the Table.

Page 87: Childhood Immunisation coverage

The text was truncated above Figure 1. The missing text was:

Figure 1 shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months and 24 months. However, the rate of increase in coverage is slowing with the curve beginning to flatten out for estimates at 12 months of age.

Page 88

Tables 9 and 10 contained incorrect data. The correct data are reproduced on the next page.

Table 9. Percentage of children immunised at 1 year of age, preliminary results by disease and State for the birth cohort 1 October to 31 December 2000; assessment date 31 March 2002

Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	1,049	21,750	792	12,373	4,362	1,477	15,190	6,256	**63,249**
Diphtheria, Tetanus, Pertussis (%)	92.4	91.8	88.8	92.7	92.5	92.6	93.1	90.7	**92.2**
Poliomyelitis (%)	92.2	91.7	89.1	92.6	92.4	92.4	93.1	90.6	**92.1**
Haemophilus influenzae type b (%)	93.7	93.8	93.1	94.7	94.5	95.5	94.8	93.9	**94.3**
Hepatitis B (%)	94.3	94.4	93.1	94.9	95.4	94.8	93.9	93.2	**94.3**
Fully immunised (%)	90.9	89.9	87.3	91.5	90.5	91.3	91.0	89.1	**90.4**
Change in fully immunised since last quarter (%)	+1.8	-0.8	-2.1	-0.3	-1.1	+0.3	-1.0	-0.4	**-0.8**

Table 10. Proportion of children immunised at 2 years of age, preliminary results by disease and State for the birth cohort 1 October to 31 December 1999; assessment date 31 March 2002[1]

Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	1,065	22,173	786	12,660	4,578	1,534	15,838	6,245	**64,879**
Diphtheria, Tetanus, Pertussis (%)	92.0	89.1	85.8	91.9	91.7	92.6	91.0	88.3	**90.3**
Poliomyelitis (%)	95.3	93.7	93.9	94.4	95.4	96.0	95.3	93.1	**94.3**
Haemophilus influenzae type b (%)	96.4	95.0	91.9	95.0	96.2	96.6	96.2	94.0	**95.3**
Measles, Mumps, Rubella (%)	94.0	92.4	93.1	94.0	94.4	94.1	94.0	91.6	**93.2**
Hepatitis B(%)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Fully immunised (%)1	90.1	86.4	83.5	90.2	89.9	90.1	88.8	85.5	**88.0**
Change in fully immunised since last quarter (%)	+3.5	+0.7	+3.7	+1.6	+0.8	+1.4	+1.3	-0.5	**+1.0**

1. The 12 months age data for this cohort was published in *Commun Dis Intell* 2001;25:94.
2. These data relating to 2 year-old children should be considered as preliminary. The proportions shown as 'fully immunised' appear low when compared with the proportions for individual vaccines. This is at least partly due to poor identification of children on immunisation encounter forms.

Communicable Diseases Surveillance

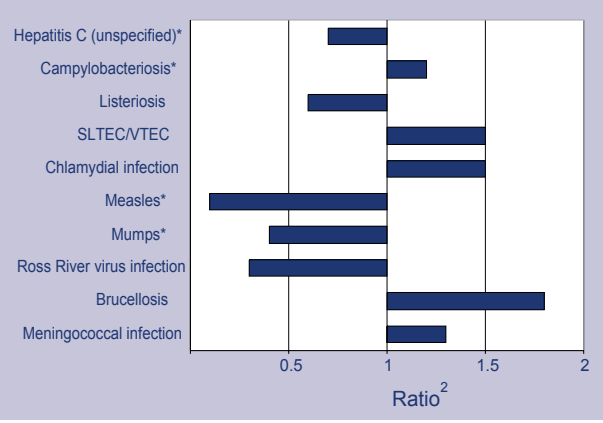
Highlights for 1st quarter, 2002

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

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia. NNDSS collates data on notifiable communicable diseases from State or Territory health departments. The Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme which collates information on laboratory diagnosis of communicable diseases. In this report, data from the NNDSS are referred to as 'notifications' or 'cases', and those from ASPREN are referred to as 'consultations' or 'encounters' while data from the LabVISE scheme are referred to as 'laboratory reports'.

Figure 1 shows the changes in disease notifications with an onset in the first quarter of 2002, compared with the 5-year first quarter mean. Disease notifications above or below the 5-year mean, plus- or minus- two standard deviations are marked with an asterisk. Diseases where the number of cases reported was two standard deviations above the mean of the same reporting period in the last 5 years in this quarter were campylobacteriosis and brucellosis. Diseases where the number of reports were two standard deviations below the 5 year mean in this quarter were unspecified hepatitis C, measles and mumps. These and other disease trends are discussed below with additional commentary provided by State and Territory health authorities.

Figure 1. Selected[1] diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 January to 31 March 2002 with historical data[2]



1. Selected diseases are chosen each quarter according to current activity.
2. Ratio of current quarter total to mean of corresponding quarter for the previous five years.

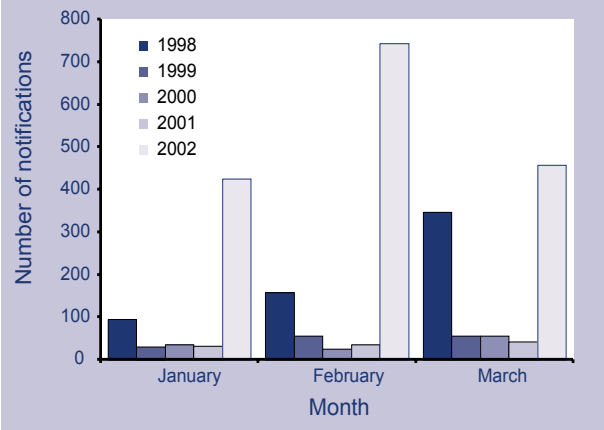
* Notifications above or below the 5-year mean for the same period plus- or minus- two standard deviations

Gastrointestinal disease

Cryptosporidiosis

Reports of cryptosporidiosis to NNDSS commenced in January 2001, although reporting in some jurisdictions was for only part of 2001. Cryptosporidiosis reports from Queensland, where the disease has been notifiable at a State level for some years is at a historic high (Figure 2). Year to date cases of cryptosporidiosis reported from the Australian Capital Territory, New South Wales, the Northern Territory and South Australia suggest that disease activity in these jurisdictions have also been increased above 2001 levels in this quarter.

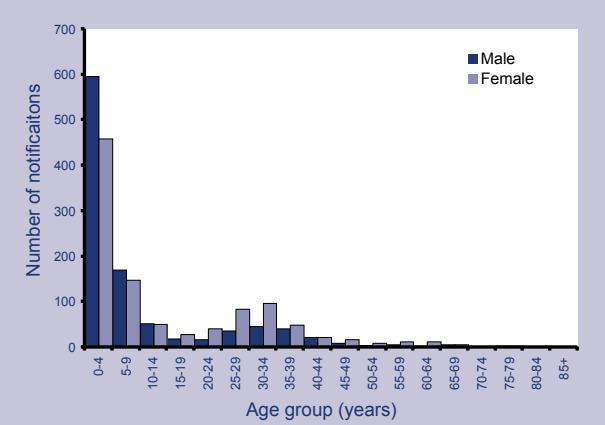
Figure 2. Notifications of cryptosporidiosis, Queensland, 1998 to 2002, by month of report



Of the 2,094 cases of cryptosporidiosis reported in this quarter, 1,377 (66%) were in children less than 10 years of age and 1,058 (50%) were aged under 5 years (Figure 3). The number of male and female cases was relatively equal. The notification rates varied widely between jurisdictions with the highest rates in the Northern Territory (218 cases per 100,000 population) and Queensland (179 cases

per 100,000 population) and lower rates in Victoria (7 cases per 100,000 population) and Tasmania (6 cases per 100,000 population) (Table 3). This reporting period covers the summer months, which is the most popular vacation period in Australia and when cryptosporidiosis infections are most commonly reported.

Figure 3. Notifications of cryptosporidiosis, Australia, 1 January to 31 March 2002, by age and sex



In Queensland, cases were reported from across the State and the number of reports show a broad peak from late January to mid-March. The majority of cases seem to be linked to recreational water exposure or person to person contact.

An outbreak of cryptosporidiosis in a caravan park in a rural area Victoria was reported on 5 February 2002. Probable cases were defined as a person who attended the caravan park between 26 and 29 January 2002 and had onset of a gastrointestinal illness consisting of two or more symptoms of diarrhoea, abdominal pain and nausea. Cases were confirmed if *C. parvum* was isolated from a faecal specimen. Eleven confirmed and eight probable cases were identified amongst a group of 21 persons attending the park over the weekend. The suspected source was the park's swimming pool where all cases had been swimming. The two people who were not ill had not been swimming. Environmental investigations suggested there were ongoing problems with ducks swimming in the pool. Water and duck faecal samples were negative for *C. parvum*. The pool was closed until results of water samples were obtained and superchlorination and other pool hygiene procedures were undertaken by the pool owners.

Since point source outbreaks of cryptosporidiosis account for a minority of cases, researchers have attempted to identify risk factors for sporadic cases. A recent study of cryptosporidiosis in Australia[1] identified person to person contact, specifically, contact with young children with diarrhoea as the strongest risk factor for acquiring cryptosporidiosis infection. Other risk factors identified included the use of public swimming pools and drinking untreated water from a rural river, lake or dam. While this suggests drinking water may be a hazard, another recent study[2] has provided evidence to discount the contribution of drinking water to gastroenteritis in Melbourne. Melbourne drinking water is drawn from a protected catchment area and undergoes minimal treatment (chlorination only). A randomised-controlled trial over 68 weeks using real or sham water treatment units in 600 Melbourne households showed no difference in the rates of gastroenteritis between the two groups. Of the 795 faecal samples from 2,669 cases of gastroenteritis which occurred during the study period, *Cryptosporidium* was only isolated in 13 (1.6%) samples. There was no significant difference in the rates of cryptosporidiosis between those drinking treated and untreated tap water.

Salmonellosis

In February, an increase in *Salmonella* Typhimurium phage type 9 was identified in New South Wales. As of late March, 82 cases were reported with onset in 2002, compared with 126 for all of 2001. Fifty five cases with onset in February 2002 were reported, compared with only 16 reported in February 2001. Compared with other types of salmonellosis in 2001 and 2002, a higher proportion of the 82 cases identified in 2002 were female and a lower proportion were less than 5 years old. In both years, cases of *S.* Typhimurium 9 occurred more frequently in the Sydney area.

Two clusters of *S.* Typhimurium 9 cases were reported in February 2002. The first cluster of seven cases were students of a boarding school identified in an outbreak of gastrointestinal disease that involved 105 students. A case-control study suggested an association between illness and eating chilli con carne and baked beans, although the mechanism of contamination remains unclear. The second cluster of cases was among people who ate at a restaurant in late February. In interviews with 19 people who were at the restaurant on 20 and 21 February, 8 reported illness within 48 hours of eating there and in 2 of these *S.* Typhimurium 9 infection was confirmed on stool testing. In a retrospective cohort study, an association between illness and eating deep fried ice cream was found. The ice cream had been battered using a tray that had earlier been used to prepare raw pork and chicken. This practice has since ceased. NSW Health staff interviewed

another 37 people infected with S. Typhimurium 9 cases and seven of these reported eating deep fried ice-cream at the restaurant implicated in the outbreak. The investigation continues.

Nine cases of *Salmonella* Aberdeen were notified in Victoria in the first quarter with onset of illness between 2 December 2001 and 5 January 2002. A case series investigation failed to identify a source.

Other *Salmonella* reported in the quarter include reports from around the country of infections with *Salmonella* Typhimurium phage type 170. Since late 2001 there have been an increase in the numbers of reports of this serovar, which was previously rarely isolated in Australia. Despite investigations, no disease clusters have been identified or food vehicles implicated. Some clustering of kanamycin resistant STM 170 in Victoria has been noted but without any associations with food.

Hepatitis A

On 9 January 2002, the Communicable Diseases Section was notified of a case of hepatitis A in a teacher at a child care centre in southern Victoria. Between 9 January and 11 February, 11 confirmed cases were identified amongst teachers, siblings and parents of children who attended the centre, with onsets of illness between 28 December 2001 and 9 February 2002. Control measures including providing information to families, primary schools and teachers in the area about the outbreak and prevention measures. Environmental investigations and clean-up procedures were undertaken at the centre. Recommendations for testing of potentially exposed persons for hepatitis A IgM and the receipt of immunoglobulin were based on the last possible day of exposure, incubation period of hepatitis A, and the onset dates of the confirmed cases. While immunoglobulin was recommended for the families of six cases, it was not given as parents refused or because treating doctors had given them hepatitis A vaccine instead. The NHMRC Immunisation Handbook[3] recommends the administration of hepatitis A immunoglobulin in an outbreak in such a setting to all at risk. Hepatitis A vaccine may be considered as an alternative in settings where recurrent outbreaks of hepatitis A are anticipated.

Other foodborne disease

A large outbreak of food poisoning was reported in Melbourne in late March. More than 272 people sought medical care and although 15 were admitted to hospital overnight, symptoms were short-lived. The onset of gastroenteritis was within one and four hours after consumption of a meal of rice, lamb and potatoes served at a New Year Islamic festival. *Bacillus cereus* and *Staphylococcus aureus* were confirmed as the cause of the outbreak. Inadequate storage and handling of leftover food was thought to be cause of the outbreak.[4]

A single case of a non-01, non-139 *Vibrio cholerae* peritonitis was reported in a 48-year-old man from South Australia.

Clostridium perfringins is the suspected cause of a series of foodborne disease outbreaks in several jurisdictions linked to spit-roast meals served at functions. Investigations are continuing.

Vaccine preventable diseases

Measles

No cases of measles were reported from the Australian Capital Territory, the Northern Territory, Queensland, South Australia Western Australia or Tasmania. Prior to a single case of imported measles reported in this quarter, New South Wales had not had a case of measles for 5 months.

Pertussis

Reports of pertussis were increased overall in the first quarter compared to historical data (Table 2). Totals for January and February were the highest since 1998. Pertussis notifications in 2001 were the highest on record for New South Wales, the highest since 1995 for the Northern Territory, since 1997 for Queensland and South Australia and since 1998 for Western Australia. Pertussis activity was lower in 2001 than 2000 in the Australian Capital Territory and Tasmania and only moderately increased in Victoria.

Influenza

Two outbreaks of influenza A in aged care facilities in Victoria were reported in this quarter.[5] The first outbreak in January occurred in a hostel with 25 cases identified (23 lab confirmed and 6 suspected). Of 42 residents, 38 (90%) had been vaccinated but only 2 of 29 (7%) of staff had received influenza vaccination. The second outbreak in March occurred in a nursing home, where 28 cases were identified (16 laboratory-confirmed and 12 suspected). Of 20 patients on whom vaccination history had been collected 18 (90%) had been vaccinated against influenza, while only 3 of 31 (10%) of staff had been vaccinated. Both outbreaks were due to influenza A (H3N2) strains related to A/Moscow/10/99, which have been components of the 2001 and 2002 Australian influenza vaccine.

In the elderly residents, influenza vaccine efficacy was calculated to be 61 per cent in preventing disease and 84 per cent in preventing hospitalisation. These figures are consistent with estimates of vaccine efficacy in the elderly. According to the NHMRC Immunisation Handbook,[3] when the match between the vaccine and the circulating viral strains is close, influenza vaccination has a 70–90 per cent efficacy against illness in healthy adults aged less than 65 years. Among the over 65-year-olds, efficacy of the vaccine is 30–70 per cent in the non-institutionalised in preventing hospitalisation. In elderly people residing in nursing homes, influenza vaccine efficacy can be 50–60 per cent effective in preventing hospitalisation and up to 80 per cent effective in preventing death. Vaccine efficacy in preventing disease in institutionalised elderly, however, may be much lower.

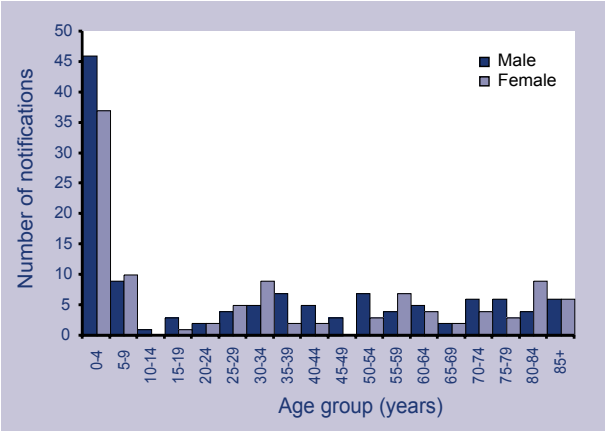
These outbreaks highlight the importance not only of maintaining high levels of vaccination in the elderly but also in their carers and contacts. A recent telephone survey of vaccination rates in health-care workers in Victoria showed that only 48 per cent overall were vaccinated with the current influenza vaccine.[6]

Invasive pneumococcal disease

Since 2001, invasive pneumococcal disease (IPD) has been a nationally notifiable disease in all jurisdictions in Australia. In total 1,663 cases were reported in 2001, although since data collection started later in some jurisdictions, figures from some States and Territories do not represent a full years total. Two hundred and forty two cases were reported in this quarter with reports from all jurisdictions except the Australian Capital Territory (Table 2). The notification rate was highest in the Northern Territory (22.2 cases per 100,000 population). The age and sex distribution is shown in Figure 4. The male to female ratio was 1.2:1. Eighty-five (35%) of cases were in children aged less than 5 years and 52 (21%) were in people aged more than 65 years.

Invasive pneumococcal disease (defined as the isolation of *Streptococcus pneumoniae* from a sterile site), most commonly presenting as meningitis or bacteraemia is a significant disease of children and the elderly in Australia.[7] In non-Indigenous urban settings the rates of IPD are estimated at 50–100 cases per 100,000 population in the under 2 year olds and 8 to 15 cases per 100,000 population in the over 65 year olds. Indigenous communities, have higher rates of IPD disease than other settings for all age groups. Indigenous children in Central Australia have some of the highest rates of IPD disease in the world (>1,500 cases per 100,000 population).

Figure 4. Notifications of invasive pneumococcal diseases, Australia, 1 January to 31 March 2002, by age and sex



The possibility of control of pneumococcal disease by vaccination has been greatly improved by the licensing in December 2000 of a seven-valent pneumococcal conjugate vaccine (7vPCV) for use in Australia. The 7vPCV vaccine has shown an efficacy of approximately 95 per cent in preventing IPD disease due to the vaccine serotypes in children in the USA.[8] Earlier polysaccharide pneumococcal vaccines were poorly protective in young children. A vaccination schedule has been implemented from July 2001 providing free vaccine to children identified to be at high-risk of IPD. Australian States and Territories have agreed to implement enhanced surveillance of pneumococcal disease to assess the impact of the conjugate vaccine on the rates and clinical types of invasive pneumococcal disease and the prevalence of circulating pneumococcal serotypes and levels of antibiotic resistance.

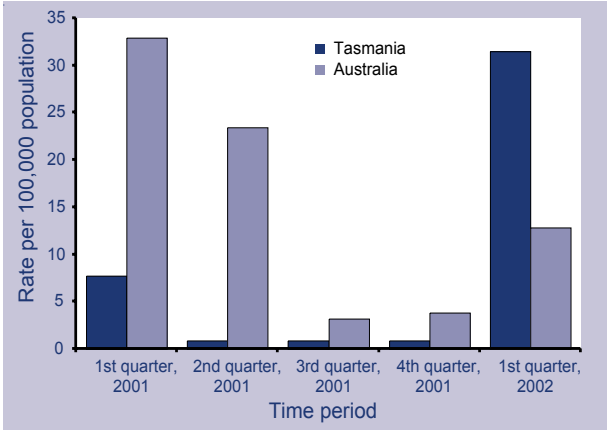
Vectorborne disease

There was an outbreak of Barmah Forest virus (BFV) disease in the Gippsland region of Victoria. Victoria received 36 notifications of BFV with onset dates between 1 January and 31 March, of which the majority came from the Gippsland region. This compares to 10 notifications with onset dates in the same period in 2001.

Reports of Ross River virus (RRV) infection were markedly reduced compared with the five-year average (Figure 1). Despite this national trend, 37 cases including 25 reports in a single week of RRV infection were recorded in Tasmania.[9] In 2002, the year to date total for RRV in Tasmania exceeds the full year totals for each of the past two years. The notification rates for RRV in Tasmania compared with rates for Australia are shown in Figure 5. The increase is believed to be partly due to heavy spring rains last year which increased mosquito breeding.

Municipalities in southeast coastal areas of Tasmania have reported the largest proportion of cases.[10]

Figure 5. Rate of notification for Ross River virus, Australia and Tasmania, 2001 to 2002, by quarter of report



Three suspected cases of Murray Valley encephalitis virus (MVE) infection reported from Western Australia await confirmation. Sentinel chicken seroconversions to MVE were reported in Western Australia and the Northern Territory in February and health authorities warned residents in tropical areas to take precautions to avoid being bitten by mosquitoes.[11]

A cluster of 18 confirmed cases of dengue occurred in March in North Queensland. All the cases were dengue serotype 2 and transmission was localised to an area with high numbers of *Aedes aegypti*, the vector for the disease. The index case was infected overseas as dengue is not endemic in Australia. Because the mosquito vector is present in North Queensland, imported cases have previously caused local outbreaks as occurred in Townsville and Charters Towers in 1992/1993 (dengue type 2) and in Cairns in 1997/998 (dengue type 2 and 3).[12] Mosquito control activities in response to the present outbreak were instituted and there were no more cases after the end of March. During the first quarter of 2002, dengue has been reported at record numbers in Brazil, Indonesia and the South Pacific.[13]

Zoonoses

In this quarter, the Northern Territory reported the first ever case of Q fever since NNDSS began in 1991. The case appears to be locally acquired and although no risk factors were identified, the patient was a delivery driver who handled frozen and packaged meat and who travels regularly near stockyards.

Other bacterial infections

Meningococcal disease

On Friday 25 January 2002, South Western Sydney Public Health Unit (SWSPHU) notified the Communicable Diseases Unit of the death of a 21-year-old South Western Sydney man from suspected invasive meningococcal disease. The man was taken by ambulance to the hospital on 24 January after collapsing at his home. He had a 3-day history of sore throat but had been otherwise well. A rash was noted and a diagnosis of meningococcal disease was made. Despite aggressive intervention, the man died. In the 7 days prior to the onset of his illness, the man had been on a cruise to the South Pacific. The cruise ship carried over a thousand passengers from all over Australia.

SWSPHU identified over 50 close contacts of the man who may have been at increased risk of disease, and provided them with information about the disease and with antibiotics to help prevent its further spread. The Communicable Diseases Unit informed other local Public Health Units (PHUs) and other States and Territories about the case. Shortly after, the South Australian Health Department reported that a South Australian man on the same cruise had been diagnosed with meningococcal disease on 22 January 2002. The man's close contacts had been contacted and given antibiotics.

No direct personal link between the cases was established. The cruise operator agreed to contact all passengers and crew from the ship to tell them about these events and about meningococcal disease. NSW Health set up a hotline providing general information to the public, issued media releases, and conducted regular media interviews to update the public on events. Passengers were alerted to seek medical attention if they develop symptoms of the disease. As a result of the public warnings, several other passengers were investigated for possible meningococcal infection, but in none of these was the diagnosis confirmed.

LabVISE

Chlamydia trachomatis

In this quarter, there were reports of an infectious conjunctivitis in remote communities in the Northern Territory that was subsequently confirmed as *Chlamydia trachomatis*. Thirty-three cases were recorded in LabVISE. Although trachoma has been eradicated from many communities in Australia, the disease persists in areas where living standards are inadequate and where personal and community hygiene is poor. The implementation of the 'SAFE' strategy (Surgery, Antibiotics, Facial cleanliness and Environmental improvement) in communities where trachoma persists is an urgent priority.[14]

Other communicable diseases

Melioidosis

Melioidosis, a disease caused by infection with *Burkholderia pseudomallei*, is endemic in tropical northern Australia. The disease is notifiable in Queensland, Western Australia and the Northern Territory but is not notifiable to NNDSS. The prevalence of the disease is increased in areas of high rainfall and underlying conditions such as diabetes are important risk factors. The article by Faa *et al* (*CDI* this issue) shows that the incidence of melioidosis in the Torres Strait is among the highest in the world. In the first quarter 2002, there were 12 cases reported in Queensland, including 2 deaths. This compares with 8 cases in all of 2001 and 38 cases in 2000.[15] By contrast, 9 cases were reported from the Northern Territory, a lower rate than usual. These differences would seem to be reflective of high rainfall in Queensland and late and reduced rainfall in the Northern Territory during this period. Three cases were reported in WA including a death in a 22-year-old male who had been backpacking in the Northern Territory.

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Tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 24,806 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 January and 31 March 2002 (Table 2). The notification rate of diseases per 100,000 population for each State or Territory is presented in Table 3.

There were 4,744 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 January to 31 March 2002 (Tables 4 and 5).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 1-4 to 9-13, ending 31 March 2002, are included in this issue of *Communicable Diseases Intelligence* (Table 6).

Table 1. Reporting of notifiable diseases by jurisdiction (1st quarter 2002)

Disease	Data received from:*
Bloodborne diseases	
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)	All jurisdiction, except NT
Hepatitis C (incident)	All jurisdictions except Qld and NT
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except NSW
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
SLTEC,VTEC	All jurisdictions
Typhoid	All jurisdictions
Quarantinable	
Cholera	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions except SA
Gonococcal infection	All jurisdictions
Syphilis	All jurisdictions
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumoccocal disease	All jurisdictions
Poliomyeltis	All jurisdictions
Rubella	All jurisdictions
Tetanus	All jurisdictions
Vectorborne diseases	
Arbovirus infection NEC	All jurisdictions
Barmah Forest virus infection	All jurisdictions
Dengue	All jurisdictions
Japanese encephalitis	All jurisdictions
Kunjin	All jurisdictions except ACT†
Malaria	All jurisdictions
Murray Valley encephalitis	All jurisdictions†
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions except SA
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Ornithosis	All jurisdictions
Other lyssaviruses (NEC)	All jurisdictions
Q fever	All jurisdictions
Other diseases	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

* Jurisdictions not yet reporting on diseases either because legislation has not yet made some diseases notifiable in that jurisdiction or data are not yet being reported to the Commonwealth.

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

Table 2. Notifications received by State and Territory health authorities in the period 1 January to 31 March 2002, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 1st quarter 2002[1]	Total 4th quarter 2001	Total 1st quarter 2001	Last five years mean 1st quarter	Ratio†
Bloodborne diseases													
Hepatitis B (incident)	0	15	2	12	3	5	35	11	83	72	96	84	1.0
Hepatitis B (unspecified)	18	656	NN	182	39	10	443	96	1,444	1,835	1,487	1,759	0.8
Hepatitis C (incident)	2	21	0	NN	9	1	14	32	79	91	82	88	0.9
Hepatitis C (unspecified)	48	930	42	773	131	108	1,167	201	3,400	3,727	4,309	5,006	0.7
Hepatitis D	0	1	0	0	0	0	1	0	2	4	5	4	0.5
Gastrointestinal diseases													
Botulism	0	0	0	0	0	0	0	0	0	0	1	0	0.0
Campylobacteriosis[2]	95	_	51	949	626	188	1,234	553	3,696	4,693	3,394	3,151	1.2
Cryptosporidiosis	21	136	108	1,634	33	7	85	70	2,094	445	255	N/A	N/A
Haemolytic uraemic	0	1	0	0	0	0	1	0	2	1	2	4	0.5
Hepatitis A	3	50	9	29	3	2	34	7	137	155	96	581	0.2
Hepatitis E	0	0	0	0	0	1	0	0	1	1	1	1	0.8
Listeriosis	0	2	0	5	0	0	2	5	14	14	21	22	0.6
Salmonellosis	35	683	128	1,076	131	62	404	246	2,765	1,825	2,180	2,536	1.1
Shigellosis	0	13	33	22	12	0	16	40	136	117	116	171	0.8
SLTEC,VTEC[3]	0	0	0	1	12	0	0	3	16	11	16	11	1.5
Typhoid	0	9	0	5	1	0	10	4	29	15	33	27	1.1
Quarantinable diseases													
Cholera	0	0	0	0	1	0	0	0	1	0	0	1	1.3
Plague	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0	0.0

Table 2. Notifications received by State and Territory health authorities in the period 1 January to 31 March 2002, by date of notification* continued

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 1st quarter 2002[1]	Total 4th quarter 2001	Total 1st quarter 2001	Last five years mean 1st quarter	Ratio[†]
Sexually transmissible diseases													
Chlamydial infection	125	1,067	286	1,494	383	110	1,200	745	5,410	4,910	4,696	3,495	1.5
Donovanosis	0	0	5	3	NN	0	0	0	8	6	2	6	1.3
Gonococcal infection[4]	4	310	373	238	21	5	194	334	1,479	1,539	1,457	1,432	1.0
Syphilis[5]	4	96	90	20	3	3	1	40	257	324	278	390	0.7
Vaccine preventable diseases													
Diphtheria	0	0	0	0	0	0	0	0	0	0	1	0	0.0
Haemophilus influenzae type b	0	3	1	3	1	0	2	0	10	2	5	7	1.4
Influenza	1	11	4	24	4	0	43	20	107	200	12	N/A	N/A
Measles	0	1	0	0	0	0	5	0	6	37	70	82	0.1
Mumps	0	5	0	2	1	0	6	2	16	13	31	43	0.4
Pertussis	15	505	29	555	183	22	231	85	1,625	3,210	1,217	1,464	1.1
Pneumococcal disease	0	77	11	33	15	6	61	39	242	388	87	N/A	N/A
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Rubella[6]	0	7	1	34	2	0	7	2	53	75	55	153	0.3
Tetanus	0	0	0	1	0	0	0	1	2	2	1	2	1.1
Vectorborne diseases													
Arbovirus infection NEC	0	3	0	3	0	0	1	0	7	0	9	24	0.3
Barmah Forest virus infection	0	63	12	124	3	0	36	13	251	159	324	239	1.0
Dengue	1	15	18	24	2	2	7	4	73	26	33	107	0.7
Japanese encephalitis	0	0	0	0	0	0	0	0	0	0	0	N/A	N/A
Kunjin virus infection	0	0	0	0	0	0	0	0	0	0	0	N/A	N/A
Malaria	3	40	8	57	1	2	19	12	142	130	230	251	0.6
Murray Valley encephalitis	0	0	0	0	0	0	0	3	3	0	2	N/A	N/A
Ross River virus infection	0	40	32	427	23	37	9	55	623	184	1,577	2,131	0.3

Table 2. Notifications received by State and Territory health authorities in the period 1 January to 31 March 2002, by date of notification* continued

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 1st quarter 2002[1]	Total 4th quarter 2001	Total 1st quarter 2001	Last five years mean 1st quarter	Ratio†
Zoonoses													
Anthrax	0	0	0	0	NN	0	0	0	0	0	0	N/A	N/A
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	N/A	N/A
Brucellosis	0	0	0	13	0	0	0	0	13	4	6	7	1.8
Leptospirosis	0	11	1	45	0	0	4	1	62	42	70	63	1.0
Other lyssavirus	0	0	0	0	0	0	0	0	0	37	0	N/A	N/A
Ornithosis	0	3	0	2	2	0	5	1	13	0	29	17	0.8
Q fever	0	51	1	87	2	0	11	6	158	155	169	139	1.1
Other bacterial infections													
Legionellosis	0	14	0	5	7	0	22	6	54	76	65	67	0.8
Leprosy	0	0	0	0	0	0	1	1	2	1	1	1	1.4
Meningococcal infection	4	32	2	20	8	4	35	9	114	148	128	85	1.3
Tuberculosis	1	76	2	9	11	0	63	13	175	179	163	252	0.7
Total	**380**	**4,947**	**1,249**	**7,911**	**1,673**	**575**	**5,409**	**2,660**	**24,804**	**24,853**	**22,812**	**24,031**	**1.0**

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

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

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

N/A Not calculated as only notifiable for under 5 years.

NN Not Notifiable

NEC Not elsewhere classified.

\- Elsewhere classified.

Table 3. Notification rates of diseases by State or Territory, 1 January to 31 March 2002 (Rate per 100,000 population)

	State or Territory								
Disease[1]	**ACT**	**NSW**	**NT**	**Qld**	**SA**	**Tas**	**Vic**	**WA**	**Australia**
Bloodborne diseases									
Hepatitis B (incident)	0.0	0.9	4.0	1.3	0.8	4.3	2.9	2.3	**1.7**
Hepatitis B (unspecified)	22.9	40.1	NN	20.0	10.4	8.5	36.6	20.0	**30.0**
Hepatitis C (incident)	2.5	1.3	0.0	NN	2.4	0.9	1.2	6.7	**2.0**
Hepatitis C (unspecified)	61.1	56.8	84.8	84.9	34.8	91.9	96.4	41.9	**70.0**
Hepatitis D	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	**0.0**
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Campylobacteriosis[2]	120.9	_	103.0	104.2	166.5	160.0	101.9	115.4	**114.8**
Cryptosporidiosis	26.7	8.3	218.2	179.4	8.8	6.0	7.0	14.6	**43.1**
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	**0.0**
Hepatitis A	3.8	3.1	18.2	3.2	0.8	1.7	2.8	1.5	**2.8**
Hepatitis E	0.0	0.0	0.0	0.0	0.0	0.9	0.0	0.0	**0.0**
Listeriosis	0.0	0.1	0.0	0.5	0.0	0.0	0.2	1.0	**0.3**
Salmonellosis	44.6	41.7	258.6	118.2	34.8	52.8	33.4	51.3	**56.9**
Shigellosis	0.0	0.8	66.7	2.4	3.2	0.0	1.3	8.3	**2.8**
SLTEC,VTEC[3]	0.0	0.0	0.0	0.1	3.2	0.0	0.0	0.6	**0.3**
Typhoid	0.0	0.5	0.0	0.5	0.3	0.0	0.8	0.8	**0.6**
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.3	0.0	0.0	0.0	**0.0**
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Sexually transmissible diseases									
Chlamydial infection	159.1	65.2	577.8	164.1	101.9	93.6	99.1	155.5	**111.4**
Donovanosis	0.0	0.0	10.1	0.3	NN	0.0	0.0	0.0	**0.2**
Gonococcal infection[4]	5.1	18.9	753.5	26.1	5.6	4.3	16.0	69.7	**30.5**
Syphilis[5]	5.1	5.9	181.8	2.2	0.8	2.6	0.1	8.3	**5.3**

Table 3. Notification rates of diseases by State or Territory, 1 January to 31 March 2002. (Rate per 100,000 population) continued

	State or Territory								
Disease[1]	**ACT**	**NSW**	**NT**	**Qld**	**SA**	**Tas**	**Vic**	**WA**	**Australia**
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Haemophilus influenzae type b	0.0	0.2	2.0	0.3	0.3	0.0	0.2	0.0	**0.2**
Influenza	1.3	0.7	8.1	2.6	1.1	0.0	3.6	4.2	**2.2**
Measles	0.0	0.1	0.0	0.0	0.0	0.0	0.4	0.0	**0.2**
Mumps	0.0	0.3	0.0	0.2	0.3	0.0	0.5	0.4	**0.3**
Pertussis	19.1	30.8	58.6	60.9	48.7	18.7	19.1	17.7	**33.5**
Pneumococcal disease	0.0	4.7	22.2	3.6	4.0	5.1	5.0	8.1	**5.0**
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Rubella[6]	0.0	0.4	2.0	3.7	0.5	0.0	0.6	0.4	**1.1**
Tetanus	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.2	**0.0**
Vectorborne diseases									
Arbovirus infection NEC	0.0	0.2	0.0	0.3	0.0	0.0	0.1	0.0	**0.1**
Barmah Forest virus infection	0.0	3.8	24.2	13.6	0.8	0.0	3.0	2.7	**5.2**
Dengue	1.3	0.9	36.4	2.6	0.5	1.7	0.6	0.8	**1.5**
Japanese encephalitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Kunjin virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Malaria	3.8	2.4	16.2	6.3	0.3	1.7	1.6	2.5	**2.9**
Murray Valley encephalitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.6	**0.1**
Ross River virus infection	0.0	2.4	64.6	46.9	6.1	31.5	0.7	11.5	**12.8**
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	NN	0.0	0.0	0.0	**0.0**
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Brucellosis	0.0	0.0	0.0	1.4	0.0	0.0	0.0	0.0	**0.3**
Leptospirosis	0.0	0.7	2.0	4.9	0.0	0.0	0.3	0.2	**1.3**
Other lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Ornithosis	0.0	0.2	0.0	0.2	0.5	0.0	0.4	0.2	**0.3**
Q fever	0.0	3.1	2.0	9.6	0.5	0.0	0.9	1.3	**3.3**
Other bacterial infections									
Legionellosis	0.0	0.9	0.0	0.5	1.9	0.0	1.8	1.3	**1.1**
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.2	**0.0**
Meningococcal infection	5.1	2.0	4.0	2.2	2.1	3.4	2.9	1.9	**2.3**
Tuberculosis	1.3	4.6	4.0	1.0	2.9	0.0	5.2	2.7	**3.6**

1. Rates are subject to retrospective revision.
2. Not reported for New South Wales because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
3. Infections with Shiga-like toxin (verotoxin) producing *E. coli* (SLTEC/VTEC).
4. Northern Territory, Queensland, South Australia , Victoria and Western Australia: includes gonococcal neonatal ophthalmia.
5. Includes congenital syphilis.
6. Includes congenital rubella.

NN Not Notifiable
NEC Not Elsewhere Classified.
- Elsewhere Classified.

Table 4. Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 January to 31 March 2002, and total reports for the year[2]

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2002	This period 2001	Year period 2002	Year to date 2001
Measles, mumps, rubella												
Measles virus	-	-	-	-	-	-	4	1	5	70	5	70
Mumps virus	-	1	-	1	-	-	1	-	3	4	3	4
Rubella virus	-	-	-	10	2	-	6	-	18	10	18	10
Hepatitis viruses												
Hepatitis A virus	-	1	4	9	4	-	2	1	21	5	21	5
Hepatitis D virus	-	-	-	1	-	-	-	-	1	1	1	1
Arboviruses												
Ross River virus	-	1	18	116	16	8	3	40	202	228	202	228
Barmah Forest virus	-	5	3	49	2	-	1	8	68	59	68	59
Dengue type 2	-	-	-	-	-	-	-	1	1	-	1	
Dengue not typed	1	1	90	1	1	-	1	13	108	-	108	-
Murray Valley encephalitis virus	-	-	-	-	-	-	1	2	3	-	3	-
Kunjin virus	-	-	-	-	-	-	-	2	2	-	2	-
Flavivirus (unspecified)	-	-	1	4	-	-	4	-	9	3	9	3
Adenoviruses												
Adenovirus type 3	-	-	-	-	-	-	1	-	1	2	1	2
Adenovirus type 4	-	-	-	-	-	-	2	-	2	-	2	-
Adenovirus type 7	-	-	-	-	-	-	5	-	5	2	5	2
Adenovirus type 8	-	-	-	-	-	-	2	-	2	1	2	1
Adenovirus type 19	-	-	-	-	-	-	2	-	2	-	2	-
Adenovirus type 37	-	-	-	-	-	-	1	-	1	1	1	1
Adenovirus type 40	-	-	-	-	-	-	-	9	9	-	9	-
Adenovirus not typed/pending	-	30	-	9	41	-	35	33	148	98	148	98
Herpes viruses												
Cytomegalovirus	2	47	1	31	148	3	39	5	276	176	276	176
Varicella-zoster virus	4	48	17	144	32	2	80	149	476	248	476	248
Epstein-Barr virus	-	21	15	170	126	1	36	118	487	207	487	207
Other DNA viruses												
Molluscum contagiosum	-	-	-	-	-	-	-	5	5	-	5	-
Parvovirus	-	3	1	8	47	-	12	23	94	30	94	30
Picornavirus family												
Coxsackievirus B1	-	2	-	-	-	-	-	-	2	-	2	-
Echovirus type 6	-	9	-	1	-	-	1	-	11	-	11	-
Echovirus type 9	-	5	-	1	1	-	-	-	7	2	7	2
Echovirus type 13	-	4	-	-	-	-	-	-	4	-	4	-
Echovirus type 30	1	1	-	-	-	1	-	-	3	-	3	-
Poliovirus type 1 (uncharacterised)	-	2	-	-	-	-	-	-	2	3	2	3
Poliovirus type 2 (uncharacterised)	-	1	-	-	-	-	-	-	1	3	1	3
Poliovirus type 3	-	2	-	-	-	-	-	-	2	-	2	-
Rhinovirus (all types)	-	46	3	-	2	-	-	29	80	28	80	28
Enterovirus not typed/pending	1	-	13	2	-	1	17	76	110	29	110	29
Picorna virus not typed	-	-	-	-	-	-	12	-	12	-	12	-

Table 4. Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 January to 31 March 2002, and total reports for the year[2] continued

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2001	This period 2000	Year period 2001[3]	Year to date 2000
Ortho/paramyxoviruses												
Influenza A virus	-	1	-	4	35	-	39	16	95	58	95	58
Influenza B virus	-	2	-	8	5	-	1	8	24	16	24	16
Parainfluenza virus type 1	1	32	-	5	1	-	2	2	43	4	43	4
Parainfluenza virus type 2	-	1	-	2	4	-	-	2	9	3	9	3
Parainfluenza virus type 3	-	8	-	-	35	-	1	19	63	47	63	47
Respiratory syncytial virus	-	27	2	19	21	-	10	36	115	43	115	43
Other RNA viruses												
Rotavirus	-	16	-	2	29	3	22	14	86	82	86	82
Calici virus	-	-	-	-	-	-	-	8	8	-	8	-
Norwalk agent	-	3	-	-	-	-	27	-	30	46	30	46
Other												
Chlamydia trachomatis not typed	10	116	33	303	173	4	4	271	914	442	914	442
Chlamydia psittaci	-	-	1	-	-	-	6	5	12	12	12	12
Chlamydia spp typing pending	-	-	-	-	-	-	1	-	1	1	1	1
Mycoplasma pneumoniae	-	19	4	46	81	1	87	40	278	103	278	103
Coxiella burnetii (Q fever)	1	3	1	22	9	-	7	15	58	18	58	18
Rickettsia spp - other	-	-	-	-	-	-	-	4	4	-	4	-
Streptococcus group A	-	9	8	63	-	-	16	-	96	79	96	79
Yersinia enterocolitica	-	1	-	-	-	-	-	-	1	2	1	2
Brucella species	-	-	-	2	-	-	-	-	2	-	2	-
Bordetella pertussis	-	32	11	98	121	-	78	25	365	140	365	140
Legionella pneumophila	-	2	-	-	-	-	14	-	16	3	16	3
Legionella longbeachae	-	-	-	-	2	-	3	2	7	-	7	-
Legionella species	-	-	-	-	-	-	2	-	2	-	2	-
Cryptococcus species	-	-	-	3	4	-	-	-	7	3	7	3
Leptospira species	-	2	1	7	-	-	-	1	11	3	11	3
Treponema pallidum	-	34	75	91	61	-	-	27	288	163	288	163
Entamoeba histolytica	-	-	-	1	-	-	1	4	6	1	6	1
Toxoplasma gondii	-	3	-	-	4	-	2	1	10	4	10	4
Echinococcus granulosus	-	-	-	-	4	-	2	4	10	-	10	-
Total	**21**	**541**	**302**	**1,233**	**1,011**	**24**	**593**	**1,019**	**4,744**	**2,483**	**4,744**	**2,483**

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

– No data received this period.

Table 5. Virology and serology laboratory reports by laboratories for the reporting period 1 January to 31 March 2002[1]

	Laboratory	January 2002	February 2002	March 2002	Total this period
Australian Capital Territory	The Canberra Hospital	-	-	-	-
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	96	59	36	191
	New Children's Hospital, Westmead	24	12	55	91
	Royal Prince Alfred Hospital, Camperdown	30	15	9	54
	South West Area Pathology Service, Liverpool	21	59	68	148
Queensland	Queensland Medical Laboratory, West End	512	533	425	1,470
	Townsville General Hospital	-	-	-	-
South Australia	Institute of Medical and Veterinary Science, Adelaide	562	445	-	1,007
Tasmania	Northern Tasmanian Pathology Service, Launceston	-	-	14	14
Victoria	Monash Medical Centre, Melbourne	20	7	11	38
	Royal Children's Hospital, Melbourne	105	58	22	185
	Victorian Infectious Diseases Reference Laboratory, Fairfield	126	112	138	376
Western Australia	PathCentre Virology, Perth	391	300	320	1,011
	Princess Margaret Hospital, Perth	21	6	35	62
	Western Diagnostic Pathology	35	47	15	97
Total		**1,943**	**1,653**	**1,148**	**4,744**

1. The complete list of laboratories reporting for the 12 months, January to December 2002, will appear in every report regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.

\- Nil reports

Table 6. Australian Sentinel Practice Research Network reports, weeks 1-4 to 9-13, 2002

Week number	**1-4**		**5-8**		**9-13**	
Ending on	**27 January 2002**		**24 February 2002**		**31 March 2002**	
Doctors reporting	250		239		229	
Total encounters	26,272		26,113		25,932	
Condition	**Reports**	**Rate per 1,000 encounters**	**Reports**	**Rate per 1,000 encounters**	**Reports**	**Rate per 1,000 encounters**
Influenza	46	1.8	33	1.3	49	1.9
Gastroenteritits	239	9.1	245	9.4	239	9.2
Acute cough with chest and systemic signs	46	1.8	52	2.0	51	2.0
Acute cough with chest signs	137	5.2	162	6.2	205	7.9
Acute cough with systemic signs	45	1.7	61	2.3	75	2.9
Acute cough without signs	259	9.9	226	8.7	255	9.8

Additional reports

Rotavirus surveillance

The National Rotavirus Reference Centre (NRRC) undertakes surveillance and characterisation of rotavirus strains causing annual epidemics of severe diarrhoea in young children throughout Australia.

Reduction in funding after June 2001 has limited that national scope of surveillance. Priority has been given to comprehensive surveillance of strains infecting children admitted to hospital in Western Australia, the Northern Territory and Victoria. Previous experience has shown Western Australia and Norhtern Territory to show differing epidemiological patterns from those of the eastern states and to be sites where 'new' strains have appeared. Melbourne's epidemiological patterns in the past have been similar to those in Brisbane, Adelaide and Hobart, and is currently regarded as representative of those locations.

The NRRC retains an interest in providing a service available to all sites if unusual epidemic patterns are observed and can be contacted at the Murdoch Childrens Research Institute, Department of Gastroenterology and Clinical Nutrition, Royal Children's Hospital, Flemington Road, Parkville, Victoria, 3052. Contact: Ruth Clark, Telephone: +61 3 9345 5069. Facsimile: +61 3 9345 6240. E-mail: clarkr@cryptic.rch.unimelb.edu.au. For more information see Commun Dis Intell 2000;24:10.

The National Rotavirus Reference Centre (NRRC) conducted rotavirus surveillance Australia-wide in 2001. One thousand and eighteen samples were collected from children admitted to hospital with acute gastroenteritis, of which 865 were confirmed as rotavirus positive. Serotype analysis of these samples was conducted using a combination of enzyme immunoassays, PCR and Northern hybridization. This analysis revealed that serotype G1 was the major serotype, representing 42.4 per cent of all strains, followed by serotype G9 (36.5% of all strains). All other serotypes represented less than 2.5 per cent of strains (Table 7). However, there was variation in the prevalence rates in several of the participating centres, with serotype G1 being the dominant strain in Melbourne and Perth, whereas serotype G9 was the dominant strain in Alice Springs, Darwin and Mt Isa.

There was an increase in the prevalence of serotype G4 in Melbourne during 2001. Whether the Melbourne serotype G4 strains identified in 2001 are related to the earlier serotype G4 strains prevalent in Darwin and Sydney during 2000, requires further analysis.

A major outbreak in the Northern Territory started in May 2001, and persisted through the year.[1] Serotype G9 was the dominant strain. This 'new' serotype has been reported world-wide since 1998 and its incorporation in candidate rotavirus vaccines is under discussion. It is important to keep track of changing strains, so that Australia is well placed to implement an appropriate vaccine when one reaches licensure.

Rotavirus collection continues and the National Rotavirus Reference Centre welcomes any notifications of rotavirus outbreaks.

Reference

1. Armstrong P. NT Disease Control Bulletin 2001;8:1-5.

Table 7. Rotavirus G types, January to December, 2001

Centre	G serotype (% of rotavirus positive)													Number of rotavirus positive samples
	G1		G2		G3		G4		G9		NR*		Mix	
Melbourne	85	(48.3)	8	(4.6)	0		12	(6.8)	18	(10.2)	50	(28.4)	3 (1.7)	176
Perth	201	(65.7)	1	(0.33)	1	(0.33)	0		57	(18.6)	42	(13.7)	4 (1.3)	306
WA Pathcentre	35	(34.3)	1	(1)	0		1	(1)	46	(45.1)	14	(13.7)	5 (4.9)	102
Darwin	1	(3.3)	0		0		0		28	(93.3)	1	(3.3)	0	30
Darwin W. Path	3	(6.8)	0		0		1	(2.3)	32	(72.7)	8	(18.2)	0	44
Alice Springs	40	(24.9)	0		0		0		111	(68.9)	10	(6.2)	0	161
Mt Isa	0		0		0		0		23	(92)	2	(8)	0	25
Adelaide	1	(50)	0		0		0		0		1	(50)	0	2
Brisbane	1	(25)	2	(50)	0		0		0		1	(25)	0	4
Hobart	0		6	(46.2)	0		0		0		7	(53.8)	0	13
West Sydney	0		1	(50)	0		0		1	(50)	0		0	2
Total	**367**	**(42.4)**	**19**	**(2.2)**	**1**	**(0.1)**	**14**	**(1.6)**	**316**	**(36.5)**	**136**	**(15.7)**	**12 (1.4)**	**865**

* NR – unable to be serotyped with monoclonal antibodies.

1018 specimens were forwarded to the NRRC, 865 were confirmed as positive

HIV and AIDS surveillance

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

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in HIV/AIDS, Viral Hepatitis and Sexually Transmissible Infections in Australia Annual Surveillance Report. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Telephone: +61 2 9332 4648. Facsimile: +61 2 9332 1837. Internet: http://www.med.unsw.edu.au/nchecr. For more information see Commun Dis Intell 2002;26:59.

HIV and AIDS diagnosis and deaths following AIDS reported for 1 October to 31 December 2001, as reported to 31 March 2002, are included in this issue of Communicable Diseases Intelligence *(Tables 8 and 9).*

Table 8. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 October to 31 December 2001, by sex and State or Territory of diagnosis

	Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Totals for Australia			
										This period 2001	This period 2000	Year to date 2001	Year to date 2000
HIV diagnoses	Female	1	7	0	5	3	0	7	2	25	14	94	78
	Male	1	76	1	25	12	0	54	5	174	140	680	664
	Not reported	0	0	0	0	0	0	0	0	0	1	2	1
	Total[1]	2	83	1	30	15	0	61	7	199	156	777	746
AIDS diagnoses	Female	0	0	0	0	2	0	2	0	4	2	16	22
	Male	0	14	0	5	2	0	6	1	28	56	127	214
	Total[1]	0	14	0	5	4	0	8	1	32	58	144	236
AIDS deaths	Female	0	0	0	0	0	0	3	0	3	1	11	8
	Male	0	11	0	1	1	0	3	0	16	29	70	123
	Total[1]	0	11	0	1	1	0	6	0	19	30	81	131

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

Table 9. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 31 March 2002, by sex and State or Territory

	Sex	State or Territory								
		ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Australia
HIV diagnoses	Female	28	672	10	180	72	5	250	132	**1,349**
	Male	231	11,562	112	2,153	727	80	4,203	981	**20,049**
	Not reported	0	244	0	0	0	0	24	0	**268**
	Total[1]	259	12,500	122	2,340	799	85	4,493	1,119	**21,717**
AIDS diagnoses	Female	9	208	0	51	28	3	79	27	**405**
	Male	88	4,823	37	883	363	45	1,725	364	**8,328**
	Total[1]	97	5,043	37	936	391	48	1,813	393	**8,758**
AIDS deaths	Female	4	118	0	35	16	2	57	18	**250**
	Male	70	3,281	25	588	242	29	1,313	260	**5,808**
	Total[1]	74	3,407	25	625	258	31	1,377	279	**6,076**

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

Childhood immunisation coverage

Tables 10 and 11 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR).

The data show the percentage of children fully immunised at age 12 months for the cohort born between 1 October to 31 December 2000 and at 24 months of age for the cohort born between 1 October to 31 December 1999 according to the Australian Standard Vaccination Schedule.

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

Commentary on the trends in ACIR data is provided by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS). For further information please contact NCIRS at: telephone +61 2 9845 1256, E-mail: brynleyh@chw.edu.au.

The percentage of Australian children 'fully immunised' by 12 months increased marginally from the last quarter by 0.1 percentage points to 90.5 per cent (Table 10). The change in the percentage 'fully immunised' varied by State and Territory. New South Wales (+0.7%), the Australian Capital Territory (+0.5%), the Northern Territory (+2.4%), and South Australia (+0.1%) showed an increase in coverage. Queensland, Western Australia, Tasmania and Victoria experienced no change or a marginal decrease in coverage in the quarter. Coverage is now below 90 per cent in only two jurisdictions, the Northern Territory (89.7%) and Western Australia (88%). Immunisation coverage for DTP and OPV by 12 months in Australia decreased marginally from the previous quarter whilst coverage for Hib and hepatitis B increased marginally. The biggest improvement in coverage by 12 months was seen in the Northern Territory, where coverage for DTP increased by 1.9 per cent, OPV by 1.4 per cent, Hib by 3 per cent and hepatitis B by 3.2 per cent.

Coverage measured by the percentage of Australian children 'fully immunised' at 24 months decreased marginally from the last quarter by 0.2 percentage points to 87.8 per cent (Table 11). Coverage increased compared with the previous quarter in three states and territories, the Northern Territory (2.4%), New South Wales (0.5%) and Western Australia (0.8%). Queensland, South Australia, Tasmania and Victoria experienced no change or a small decrease in coverage with South Australia experiencing the largest decrease (2.4%).

Coverage for individual vaccines by 24 months for Australia however, is much greater than for 'fully immunised', with coverage for Hib greater than 95 per cent and coverage for OPV and MMR approaching 95 per cent.

Figure 6 shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months and 24 months. However, the rate of increase in coverage is slowing with the curve beginning to flatten out for estimates at 12 months of age.

Figure 6. Trends in vaccination coverage, Australia, 1997 to 2001, by age cohorts

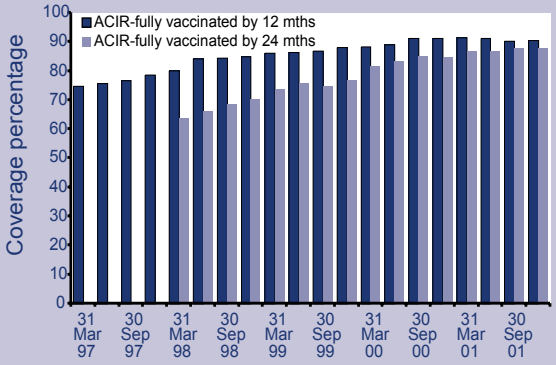

Table 10. Percentage of children immunised at 1 year of age, preliminary results by disease and State for the birth cohort 1 October to 31 December 2000; assessment date 31 March 2002

Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	1,084	21,340	845	12,019	4,231	1,535	15,258	5,848	**62,160**
Diphtheria, Tetanus and Pertussis (%)	92.9	91.9	90.7	92.0	92.2	92.1	92.8	90.1	**92.0**
Poliomyelitis (%)	92.8	91.8	90.5	91.9	92.0	92.1	92.8	90.0	**91.9**
Haemophilus influenzae type b (%)	94.7	94.5	96.1	94.3	94.5	95.7	95.0	93.1	**94.5**
Hepatitis B (%)	95.0	94.7	96.3	94.8	94.9	94.9	94.1	92.2	**94.4**
Fully immunised (%)	91.4	90.6	89.7	90.8	90.6	91.0	91.0	88.0	**90.5**
Change in fully immunised since last quarter (%)	-0.5	+0.7	+2.5	-0.7	+0.1	-0.3	+0.0	-1.1	**+0.1**

Table 11. Proportion of children immunised at 2 years of age, preliminary results by disease and State for the birth cohort 1 October to 31 December 1999; assessment date 31 March 2002[1]

Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	999	20,711	759	11,714	4,417	1,483	15,149	6,132	**61,364**
Diphtheria, Tetanus, Pertussis (%)	89.9	89.7	86.8	91.1	90.0	90.4	90.9	89.1	**90.2**
Poliomyelitis (%)	95.0	94.1	94.6	94.0	94.6	96.1	95.2	93.8	**94.4**
Haemophilus influenzae type b (%)	95.8	95.4	94.1	95.0	95.4	96.6	96.1	94.7	**95.4**
Measles, Mumps, Rubella (%)	94.4	92.8	94.2	93.2	93.2	95.1	94.1	92.9	**93.4**
Fully immunised (%)[2]	88.5	86.9	85.9	88.8	87.5	89.6	88.8	86.3	**87.8**
Change in fully immunised since last quarter (%)	-1.6	+0.5	+2.4	-1.4	-2.4	-0.5	-0.0	+0.8	**-0.2**

1. The 12 months age data for this cohort were published in *Commun Dis Intell* 2001;25:94.
2. These data relating to 2 year-old children should be considered as preliminary. The proportions shown as 'fully immunised' appear low when compared with the proportions for individual vaccines. This is at least partly due to poor identification of children on immunisation encounter forms.

Acknowledgment: These figures were provided by the Health Insurance Commission (HIC), to specifications provided by the Commonwealth Department of Health and Ageing. For further information on these figures or data on the Australian Childhood Immunisation Register please contact the Immunisation Section of the HIC: Telephone: +61 2 6124 6607.

National Enteric Pathogens Surveillance System

The National Enteric Pathogens Surveillance System (NEPSS) collects, analyses and disseminates data on human enteric bacterial infections diagnosed in Australia. These pathogens include Salmonella, E. coli, Vibrio, Yersinia, Plesiomonas, Aeromonas *and* Campylobacter. Communicable Diseases Intelligence *reports only on* Salmonella.

Data are based on reports to NEPSS from Australian laboratories of laboratory-confirmed human infection with Salmonella. Salmonella *are identified to the level of serovar and, if applicable, phage-type. Infections apparently acquired overseas are included. Multiple isolations of a single* Salmonella *serovar/phage-type from one or more body sites during the same episode of illness are counted once only. The date of the case is the date the primary diagnostic laboratory isolated a Salmonella from the clinical sample.*

Note that the historical quarterly mean count should be interpreted cautiously, and is affected by surveillance artefacts such as newly designated and incompletely typed Salmonella.

We thank contributing laboratories and scientists. Joan Powling (NEPSS Co-ordinator) and Mark Veitch (Public Health Physician), Microbiological Diagnostic Unit — Public Health Laboratory, Department of Microbiology and Immunology, University of Melbourne. For further information please contact NEPSS at the above address or on Telephone: +61 3 8344 5701, Facsimile: +61 3 8344 7833.

Reports to the National Enteric Pathogens Surveillance System of Salmonella *infection for 1 January to 31 March 2002 are shown in Tables 12 and 13. Data includes cases reported and entered by 15 April 2002. Counts are preliminary, and subject to adjustment after completion of typing and reporting of further cases to NEPSS.*

Table 12. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 January to 31 March 2002, as reported to 15 April 2002

	Australia	ACT	NSW	NT	Qld	SA	Tas	Vic	WA
Total all *Salmonella* for quarter	2,585	39	708	101	965	117	55	423	177
Total contributing *Salmonella* types	225	20	106	43	119	43	15	100	62

Table 13. Top 25 *Salmonella* types identified in Australian States and Territories, 1 January to 31 March 2002

National rank	*Salmonella* type	Total 1st quarter 2002	Last 10 years mean 1st quarter	Year to date 2002	Year to date 2001	Total 2001	ACT	NSW	NT	Qld	SA	Tas	Vic	WA
1	S. Typhimurium 9	279	148	279	160	398	14	165	0	31	8	3	42	16
2	S. Typhimurium 135	262	176	262	276	638	1	83	2	49	6	8	71	42
3	S. Saintpaul	145	117	145	98	288	0	10	1	108	2	1	14	9
4	S. Virchow 8	137	48	137	82	245	0	10	0	119	0	0	8	0
5	S. Typhimurium 170	132	31	132	19	148	0	59	0	24	0	0	48	1
6	S. Birkenhead	109	84	109	99	248	0	45	2	58	0	0	4	0
7	S. Aberdeen	67	32	67	33	87	0	4	0	54	0	0	9	0
8	S. Hvittingfoss	59	17	59	25	89	1	5	3	46	2	0	1	1
9	S. Typhimurium 126	58	26	58	58	314	0	13	1	11	11	1	21	0
10	S. Chester	48	58	48	67	166	1	9	6	25	2	0	2	3
11	S. Waycross	48	37	48	19	53	0	16	1	31	0	0	0	0
12	S. Muenchen	43	57	43	52	125	0	2	5	28	2	0	1	5
13	S. Virchow 34	41	28	41	32	87	1	17	0	16	0	1	6	0
14	S. Infantis	37	47	37	44	123	3	12	0	9	3	0	6	4
15	S. Mississippi	35	31	35	67	124	0	1	0	0	0	32	2	0
16	S. Anatum	32	32	32	20	58	0	3	5	19	0	0	1	4
17	S. Typhimurium 4	31	16	31	62	141	2	6	0	6	5	0	12	0
18	S. Montevideo	30	5	30	6	27	1	22	2	4	0	0	1	0
19	S. Mgulani	29	12	29	12	66	0	1	0	27	0	0	1	0
20	S. Potsdam	28	20	28	22	60	0	14	0	13	0	0	0	1
21	S. Typhimurium RDNC	22	43	22	30	102	0	11	0	2	1	0	8	0
22	S. Typhimurium U290	22	1	22	4	27	1	14	0	0	0	1	4	2
23	S. Agona	21	17	21	13	56	1	7	1	1	7	0	2	2
24	S. Typhimurium 12	21	6	21	18	62	0	6	1	6	7	0	1	0
25	S. Singapore	19	19	19	14	64	0	10	0	4	3	0	1	1
	Total of 25 most common types	1,755					26	545	30	691	59	47	266	91

Overseas briefs

ProMED–mail

This material has been summarised from information provided by ProMED–mail (http://ww.promedmail.org). A link to this site can be found under 'Other Australian and international communicable diseases sites' on the Communicable Diseases Australia homepage.

Dengue update

Hawaii

Source: Centers for Disease Control and Prevention, Travellers' Health; *Released 2 October 2001; updated 4 March 2002 (edited)*

As of 15 March 2002, Hawaii state health officials reported one additional recent case of dengue fever and 6 cases that occurred last year but were not confirmed by laboratory testing until 2002. The single recent case occurred in February 2002 in Haiku, Maui, and 5 of the cases from last year occurred among residents of Hana, Maui. The other case from last year occurred in early December 2001 in a visitor from New Mexico who stayed in a private residence in a forested area of Hana. A total of 118 cases of dengue fever have been reported in Hawaii since 10 June 2001. Of these, 77 occurred in the Hana area. Overall, the severity of illness has been relatively mild and the outbreak appears to be waning. Available data suggest that the risk of dengue infection to most visitors to Hawaii is very low.

Ecuador

Source: El Comercio (Ecuador), *26 March 2002 (edited)*

According to a spokesperson from the State Tropical Medicine Secretariat in Guayaquil, the intense rainstorms that have hit the coastal provinces of Ecuador beginning in February 2002, have unleashed epidemics of dengue and malaria. The spokesperson reported that, to date, there were 816 cases of suspected dengue fever, 23 of which have been confirmed, including 2 cases of dengue haemorrhagic fever (DHF). The Ministry of Health indicated that an additional 21 cases of suspected DHF were being investigated. The Tropical Medicine Secretariat revealed that cases of Asiatic (serotype 2) and serotype 3 dengue virus infection have been identified, and that they probably reached Ecuador through Venezuela and Central America. The severity of the winter season has forced the government to declare a state of public health and economic emergency in the coastal states as well as in 2 Andean states also affected by torrential rains.

Cuba

Source: Associated Press report, 28 March 2002 (edited)

"The dengue virus has been eradicated in our homeland," said President Castro on 27 March 2002. The Cuban leader, on 12 January 2002, launched a highly organised and widespread education and fumigation campaign aimed at wiping out the mosquito that transmits the virus. During the campaign in Cuba, homes and other buildings, especially in the hard-hit capital of Havana, were repeatedly sprayed over a period of more than 3 months, sometimes several times a week. Cuba's government-controlled newspapers printed educational articles about clean-up efforts. Because the virus was seen here as a matter of national security, cooperation in the campaign was obligatory. Residents who refused to let their homes be fumigated were sometimes fined. Cuba suffered a serious dengue epidemic in 1977 that made more than 400,000 people sick. During another dengue epidemic 10 years later, Cuba reported 350,000 cases.

Brazil

Source: Agencia EFE 31 March 2002 (edited)

Dengue cases reported in Brazil in the first quarter of the 2002 were more than twice the number of those in the same period last year. Between January and March 2002, 317,787 cases of dengue were reported in Brazil, compared with 153,148 in the same period last year, based on health department data from each of the States in the country. Haemorrhagic dengue has killed 59 people around the country in the first quarter of 2002, more than doubling the 28 deaths last year, and could exceed the total of 75 deaths reported between 1990 and 2001, according to Health Ministry data. The campaign against dengue has mobilised various sectors of Brazilian society, including the army, whose members in some States go house-to-house to eliminate breeding sites of *Aedes aegypti.*

Poliomyelitis in Haiti and Dominican Republic

Source: Reuters Health eLine, 14 March 2002 (edited)

A recent outbreak of poliomyelitis in Haiti and the Dominican Republic has been traced to a strain of oral polio vaccine (OPV) that mutated back to virulence. Based on genetic analysis of viral samples, the outbreak, which struck nearly 2 dozen children in both countries between 2000 and 2001, arose from OPV given to one child in 1998/1999. The cases in Haiti and the Dominican Republic illustrate a potential risk with OPV when it is given in a population where many people are unvaccinated. After a person receives OPV, virus from the vaccine is shed in the stools for a short period of time. In this outbreak, shed virus from a single OPV dose spread and mutated back to a virulent state, causing paralytic disease in a group of children who had either not been vaccinated or had not received a complete course of OPV. The findings were published in Science (http://www.sciencemag.org/sciencexpress/recent.shtml).

Variant CJD is confirmed as a new disease

Source: Reuters report, 25 March 2002 (edited)

A systematic review of Welsh death records and autopsy samples confirms the description of variant Creutzfeldt-Jakob disease (vCJD) as a new disease entity, rather than a result of better case ascertainment. Researchers at the University Hospital of Wales in Cardiff obtained death certificate data from the Office of National Statistics for deaths of individuals aged 15 to 45 between 1985 and 1995, to search for cases of vCJD that might not have been recognised before the first cases were reported in 1996. They compiled a list of International Classification of Diseases version 9 (ICD–9) codes that might be mistaken for vCJD, which they called 'non-specific fatal disorders compatible with vCJD.' For cases that fit within this classification, the group obtained postmortem brain material whenever possible, for examination by immunocytochemical staining for prion protein. Of 12,091 deaths, excluding external injury and poisoning, 3,322 fit within the ICD–9 classification scheme. Deaths were excluded where the maximum duration of illness from onset of symptoms exceeded 36 months, given that the first published vCJD cases had a maximum duration of 35 months. They also excluded illnesses for which clinical, laboratory, or pathological evidence for the disease existed. None of the more than 250 brain tissue specimens examined exhibited the pattern of prion protein immunoreactivity associated with vCJD, even though nearly half exhibited some immunoreactivity. In the few cases in which vacuolation was observed, it appeared to result from brain edema, recent lack of oxygen, or procedural artefacts. No spongiform changes or pathological plaques were observed.

Based on these data, the upper 95 per cent confidence interval for the incidence of death from vCJD would be 0.12 per million/year, which is markedly different from the actual mortality rate for 1995 to 2000 of 0.29 per million/year. This indicates "an increase in incidence in the past 5 years which is unlikely to be due to chance alone", the investigators write. "It suggests that vCJD is indeed a new disease."

Reference

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Brucellosis in New Zealand

Source: Official news release, New Zealand MAF (edited)

In early March 2002 public health authorities notified the Ministry of Agriculture and Forestry (MAF) of a case of human brucellosis. Blood cultures confirmed the infection, and biochemical profiling suggests the isolate was *Brucella suis*. The isolate was sent to Veterinary Laboratory Agency (VLA), Weybridge UK, for typing. The VLA report it is likely to be *Brucella suis* biovar 3 (confirmation pending). Officials believe on clinical and epidemiological grounds, that the case most likely acquired the infection in December 2001 in New Zealand. At that time the case purchased 2 pigs that were killed and butchered in a home-kill situation for consumption at 2 family feasts. The case had not travelled outside New Zealand for 10 years. The case did visit a country 10 years ago where *Brucella suis* is endemic. *Brucella suis* has not previously been recorded in New Zealand livestock. A previous human case of *Brucella suis* in New Zealand has been attributed to infection acquired in the Pacific Islands,[1] where the disease is known to be present in some countries.[2] Imported human brucellosis cases caused by other species are also occasionally diagnosed.

Animal surveillance performed by MAF since notification of the human case, includes trace back of the 2 pigs to the point of purchase, from there to a sale-yard, and to likely farms of origin. Testing using the competitive ELISA (sensitivity 90.8%, specificity of 96.6%) has produced all negative results to date. Public health authorities in conjunction with clinicians are following up people associated with the confirmed case, with the putative exposure event, or possible farms of origin. The index case remains the only confirmed case.

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Ebola haemorrhagic fever in Gabon and the Republic of the Congo

Source: World Health Organisation (WHO) Disease outbreaks report, 22 March 2002 (edited)

On 20 March 2002, the Gabonese Ministry of Health reported 60 confirmed cases of Ebola haemorrhagic fever, including 50 deaths. Suspected cases continue to be investigated. As of 22 March 2002, 32 confirmed cases, including 19 deaths, have been reported in villages in Cuvette region, Republic of the Congo. Eighteen contacts are being followed up in the Republic of the Congo. Ebola haemorrhagic fever has now been laboratory confirmed in the Kelle area. WHO, Medecins sans Frontieres, International Federation of Red Cross and Red Crescent Societies, and other partners in the Global Outbreak Alert and Response Network are responding.

A candidate vaccine for West Nile virus

Source: Newsday.com, 12 March 2002 (edited)

The most recent research, published last week in the Proceedings of the National Academy of Sciences, details the efforts of scientists from the National Institute of Allergy and Infectious Diseases in Bethesda, Maryland, and Walter Reed Army Institute of Research in Silver Spring, Maryland. The collaborators spliced 2 genes for West Nile coat proteins into a dengue virus backbone stripped of its own corresponding genes. West Nile virus and dengue virus belong to the flavivirus family of tick- and mosquito-borne viruses, a family that also includes yellow fever virus and Japanese encephalitis virus. The vaccine is still in the early stages of development, with only a few animal experiments conducted and human trials still a long way off.

Eosinophilic meningitis in Jamaica

Source: Emerging Infectious Diseases, *March 2002, vol 18 no3 (edited)*

A recent study reported that an outbreak in 2000 of eosinophilic meningitis in tourists visiting Jamaica was due to *Angiostrongylus cantonensis* and that the parasite was recently found in rats and snails on the island. Overall, 22 per cent (24/109) of rats harboured adult worms, and 8 per cent (4/48) of snails harboured *A. cantonensis* larvae. This report is the first of enzootic *A. cantonensis* infection in Jamaica. The paper describes how 12 persons in a group of 23 United States of America tourists who visited Jamaica for a week developed eosinophilic meningitis within 6–30 days (median 11) of returning home. Though 9 persons required hospitalisation, there were no deaths. There was serologic evidence of exposure to *A. cantonensis* in 8 persons who had eaten salad at the same restaurant, a common exposure that might account for all cases.

Unexplained rash in the USA

Source: MMWR *2002;51;161. 1 March (edited)*

The first reported rash occurred in Indiana on 4 October 2001, followed by cases in Virginia that began on 20 November 2001. Subsequent cases of rashes began in late January and occurred as recently as 21 February 2002. Rashes have been reported primarily from elementary schools but also among students in a few middle and high schools. The number of affected students in each state ranges from <10 to about 600. Characteristics of the rashes vary, but onset has generally been acute, typically with maculopapular erythematous lesions (possibly in a reticulated pattern) on the face, neck, hands, or arms. Duration of the rash varied but in most reports it was highly pruritic. The rashes were not attributed to a defined environmental exposure or infectious agent. Children with rashes were afebrile and usually had no other associated signs or symptoms. The rashes lasted from a few hours to 2 weeks and appeared to be self-limiting.

Secondary transmission has not been reported, but in-school 'sympathy' cases have reportedly occurred. Diagnoses by clinicians who have examined children have included viral exanthem, contact or atopic dermatitis, eczema, chemical exposure, impetigo, and poison ivy. About 40 serum samples collected in 4 states have been PCR or IgM negative for parvovirus B19 and 22 nasal swab samples have been negative for enterovirus. Environmental assessments have not identified any environmental causes.

The CDC is working with state and local health and education agencies in these investigations to determine if affected children within and between schools have developed rash as a result of a common etiology.

Hospital-acquired malaria in England

Source: Eurosurveillance *21 February 2002 (edited)*

An elderly man admitted for an orthopaedic procedure in late December 2001 to a hospital in England developed *Plasmodium falciparum* malaria at the end of January. As the patient had no known risk factors for malaria, this was deemed to be a hospital acquired infection. Investigations focused on transmission from healthcare workers or inpatients with malaria. There were no patients with malaria whose admission overlapped that of the index case. Blood samples were obtained from all healthcare workers (HCWs) who were potentially chronic carriers and had participated in invasive procedures on the index patient, with the exception of one HCW who has not yet been contacted. All samples obtained tested negative for malaria antibodies and on film.

On the assumption that infection was probably transmitted in the operating theatre or ward where the patient was treated, all other patients exposed to these 2 areas are being traced. Test results from this group of patients have all been negative for malaria thus far. These patients have also been given advice about seeking medical attention should they develop a fever in the following 3 months.

The HCW who has yet to be contacted was subsequently employed at a second hospital in the NHS South West Region and currently cannot be excluded as the source of infection. Patients who may have been exposed to this person in the second hospital have also been contacted and advised to seek medical attention in case they develop a fever.

This is the second case of hospital-acquired malaria in England in the past 3 years. In the previous incident, transmission is believed to have occurred through repeated use of a bottle of intravenous saline contaminated with blood from a malarious patient. This resulted in 3 cases of infection and one death. These 2 cases mean that exotic bloodborne infections should be considered when inpatients with no personal history of travel develop pyrexia of unknown origin. In the case described here the source of infection has not been determined; possibly transmission occurred from an infected HCW during an exposure-prone procedure.

Plague in India

Source: WHO 20 February 2002 Disease outbreaks reported

As of 19 February 2002, the Ministry of Health, India has reported a total of 16 cases of pneumonic plague including 4 deaths in Hat Koti village, Shimla district, Himachal Pradesh State, since the onset of the outbreak on 4 February 2002. The series of tests carried out by the National Institute of Communicable Diseases (NICD) confirm the presence of *Yersinia pestis* in clinical samples. A team from the NICD visited the village, and found that all the cases could be linked to residents of one hamlet. Under the guidance of the team, the local health administration has taken the following measures:

- administration of chemoprophylaxis to contacts of the patients, to residents of the affected and neighbouring village and to doctors/paramedics and health workers;
- fumigation in the affected villages and transport vehicles to kill infected fleas;
- public education campaign.

The last reported case had a date of onset of 8 February 2002. Careful surveillance in the area and in the state is continuing. WHO recommends no special restrictions on travel or trade to or from India.

Identification of a new subtype of influenza virus A(H1N2)

Source: Eurosurveillance Weekly, *Issue 6, 7 February 2002 (edited)*

A meeting of influenza experts at the World Health Organization (WHO) in Geneva this week has considered the recent isolation of a new subtype of the influenza A virus, A(H1N2).[1] The meeting was held to review the global influenza situation and decide the composition for the influenza vaccine for the northern hemisphere for winter 2002/03. This was based on information from the WHO global influenza surveillance programme and the Public Health Laboratory Service (PHLS) surveillance of influenza in England and Wales.

In recent years, 2 subtypes of influenza A have been circulating and causing illness in humans: the H1N1 subtype and the H3N2 subtype. A new subtype, H1N2, has emerged which contains a haemagglutinin (H) component which is very similar to that contained in the recent H1N1 strains and a neuraminidase (N) component which is very similar to that contained in the currently circulating H3N2 strains. The new subtype appears to have arisen by reassortment of the 2 human viruses. The new subtype of the influenza A virus has so far been isolated from humans in England, Israel, and Egypt in the last few weeks. A similar event occurred in China during the 1988/89 influenza season when a number of influenza A(H1N2) isolates were detected which were determined to have arisen as a result of reassortment. Further spread of these reassortant viruses in humans did not occur at that time.[2,3]

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Nasopharyngeal Corynebacterium ulcerans diphtheria in the Netherlands

Source: Dam A. van (M.M.)

A 59-year-old woman was admitted to the hospital with a 3-day history of a sore throat and increasing dysphagia during treatment with oral penicillin for 1 day . On admission, the patient was afebrile, the soft palate and uvula were swollen and a membraneous exudate was seen on the soft palate and nasopharynx. There was no palpable cervical lymphadenopathy or soft tissue swelling. The patient had not recently travelled abroad and had no contact with people who had recently travelled. The patient was not vaccinated against diphtheria. Diphtheria was included in the differential diagnosis.

The patient was barrier nursed and treated with intravenous penicillin. She recovered fully within 4 days. A throat smear grew no *Corynebacterium diphtheriae*, but *Corynebacterium ulcerans* was cultured from this specimen. The strain contained the gene encoding diphtheria toxin, as shown by PCR performed at the Dutch National Institute for Public Health. Although person-to-person transmission has not been documented, a contact investigation was initiated, but no *C. ulcerans* was grown from the 2 household contacts of the patient. The patient had not recently been in contact with horses, cattle or other animals except her domestic cat. Therefore, the source of the infection is unclear.

The most recent case of diphtheria caused by *C. diphtheriae* was reported in the Netherlands in 1991. We conclude that in countries where *C. diphtheriae* is no longer endemic, one should also be aware of the possibility of diphtheria caused by *C. ulcerans*.

UK and US on verge of eliminating rubella

Source: The Times, *23 January 2002 (edited)*

Rubella has been almost eliminated from Britain and the United States of America (USA), thanks to vaccination. The study, published in *JAMA*, shows that in the USA, the number of cases of the disease has declined from 57,600 in 1969 when vaccination began, to a few hundred cases a year, mostly in immigrants from countries where vaccination is newly established (http://jama.amaassn.org/issues/v287n4/abs/joc11125.html). Almost all cases of the disease in the United States now are among Hispanic adults born in other countries, primarily Mexico, meaning that the virus may no longer be circulating in the general USA population.

In Britain in the last quarter of 2000 (the most recent period for which data have been published by the Public Health Laboratory Service) not a single case of rubella was confirmed in England or Wales. General practitioners (GPs) diagnosed 1600 or so cases in 2000, of which only 9 cases were confirmed. It has become so rare that GPs are no longer skilled at diagnosing it. There has also been a sharp decline in the number of children born with birth defects as a result of their mother catching rubella in pregnancy.

Progress towards a malaria vaccine

Source: Bojang, KA et al. Lancet *2001; 358: 1927–1934 (edited)*

A study in Gambia of a new malaria vaccine construct including the major surface protein of the *P. falciparum* circumsporozoite protein, CSP, fused to the hepatitis B surface antigen HBsAg and adsorbed to the adjuvant AS0, showed partial short-time protection. Adult Gambians received 3 injections with the vaccine and were followed for 15 weeks. During the first 9 weeks, the protective efficacy against parasitemia was 71 per cent, but there was no protective efficacy during the last 6 weeks of the observation period. A fourth dose was given a year later and the subjects were followed for 9 weeks. The protective efficacy in this period was 47 per cent. The study identified the vaccine as immunogenic, safe, and the first to show any protection against the pre-erythrocytic stages of *P. falciparum*.