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Editorial: Introduction of the National Meningococcal C Vaccination Program

Natalie J Cohen
Commonwealth Department of Health and Ageing

In January 2003, the Commonwealth Government commenced the National Meningococcal C Vaccination Program. This program provides free meningococcal C conjugate vaccine over the next four years to all children and adolescents who turn one to 19 years of age in 2003, a target group of almost 6 million. The meningococcal C vaccine has also been added to the Australian Standard Vaccination Schedule at 12 months of age. A rise in the incidence of meningococcal disease in Australia, an increasing mortality rate due to the C strain of the disease and success in England and Wales with meningococcal C vaccination, have contributed to the decision by the Australian Government to implement this program.

The meningococcal C vaccine is a new conjugate vaccine, more effective and immunogenic than the polysaccharide vaccine previously available. The polysaccharide vaccine has been available for a number of years. However, it provides protection only for a limited time against serogroups A, C, W135 and Y, and is not effective in infants under two years of age.

The new conjugate vaccine (conjugation involves attaching a carrier protein to the oligosaccharide antigen formed from the coat of the bacteria[1]) was approved for use in Australia in 2001. It induces a T-cell dependent antibody response and immunological memory, and is immunogenic in children under two years of age. It is expected that immunity induced by the conjugate vaccine will be long term.

Notifications of meningococcal disease in Australia to the National Notifiable Diseases Surveillance System (NNDSS) have been gradually increasing since 1991 (337 cases) to 677 cases in 2002.[2] Meningococcal disease is caused by invasive infection with *Neisseria meningitidis*, resulting in meningitis and/or septicaemia. While meningococcal disease may affect all age groups, there is a bimodal age distribution with the highest incidence rates in children under five years of age and adolescents and young adults.[3] Approximately 32 per cent of cases in Australia are serogroup C (the proportion of serogroup C isolates has been rising since 1995) and the majority of the remaining cases are serogroup B.[4] Unfortunately, no effective vaccine is currently available for serogroup B disease.

A similar vaccination program was launched in England and Wales in November 1999, targeting 18 million children and adolescents.[5] Results have been very promising. High vaccine coverage (around 90%) has been achieved and there has been a 90 per cent decrease in meningococcal C disease notifications in the targeted age groups, and a 90 per cent decrease in deaths.[5] Estimates from England and Wales indicate vaccine efficacy ranges of 88–92 per cent in toddlers; and 96–97 per cent in 15–17 year olds.[6] The data also indicate a good safety record for meningococcal C conjugate vaccine. Post-licensure surveillance has indicated that adverse vaccine events include non-serious reactions such as headache, local reaction, pyrexia and dizziness. Serious and rare adverse events include anaphylactoid reactions (one per 500,000 doses) and purpura.[6]

In Australia, surveillance of meningococcal disease, including detailed serogroup analysis, will continue so that the effect of the vaccination program can be monitored. This surveillance is carried out by the National Neisseria Network and the NNDSS systems and will need to be sensitive enough to detect changes in the epidemiology of meningococcal disease, in particular, increases in the incidence of other serogroups.

For further information about the program visit the Immunise Australia website at http://immunise.health.gov.au.

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Annual report of the National Influenza Surveillance Scheme, 2002

Keflemariam Yohannes,[1] Paul Roche,[1] Jenean Spencer,[1] Alan Hampson[2]

Abstract

Surveillance for influenza in Australia in 2002 was based on notifications to the National Notifiable Diseases Surveillance system from all states and territories, national and state-based sentinel practice consultations for influenza-like illness and reports of influenza virus isolations from a laboratory network. The impact of influenza was assessed by absenteeism data from a major national employer. Influenza A was the dominant type, 99 per cent of which were subtype H3N2 with only a single H1 isolate, which was identified as H1N2.The H3N2 isolates were closely related to the vaccine strain A/Moscow/10/99 and the A/Panama/2007/99, with less than one per cent showing genetic variation. Influenza B made up 21 per cent of circulating influenza and the majority of B strains were of the B/Victoria lineage, but had a haemagglutinin closely related to the B/Hong Kong/330/2001 strain.This strain was associated with two outbreaks but a proportion of vaccinees with the 2002 vaccine showed protective antibody titres.The 2002 influenza vaccine was given to 77 per cent of Australians over 65 years. *Commun Dis Intell* 2003;27:162–172.

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

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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 2001, pneumonia and influenza were underlying causes of 2,702 deaths (ICD–10 codes J10–J18; 2.1% of all deaths).[2] Influenza pandemics occur every 10 to 30 years. During these pandemics a quarter or more of the global population may be infected 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 an intermediate host 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 a rapid spread of influenza with dramatically increased rates of morbidity and mortality. After the pandemic of 1918 the H1N1 virus circulated widely in the human population. The Asian and Hong Kong pandemics in 1957 and 1968 introduced the H2N2 and H3N2 subtypes. There have been no major 'antigenic shifts' causing pandemics of influenza since 1968, however, the H1N1 subtype reappeared in the human population in 1977. Since 1977, influenza A (H1N1), A (H3N2) and influenza B viruses have been widespread globally, varying in frequency temporally and geographically.[3]

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.[4]

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 to provide new vaccine strains; 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 2002.

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);
2. laboratory diagnosis including virus isolation and serology by laboratories participating in the Laboratory Virology and Serology Reporting Scheme (LabVISE);
3. subtype data of influenza virus isolates forwarded by LabVISE laboratories provided by the WHO Collaborating Centre for Reference and Research on Influenza;
4. consultation rates for influenza-like illness diagnosed by sentinel general practitioners;
5. absenteeism data of workers from a national employer; and
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.[5] 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. In 2002 all states and territories reported influenza to NNDSS.

Laboratory surveillance

LabVISE is a national scheme of sentinel laboratories. In 2002, 12 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 is now in use for diagnosis.[3]

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 characteristics 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 antigenic match between circulating strains and the current vaccine) and to determine the composition of vaccine for the following influenza season.

Standard nomenclature for influenza viruses is based on type, the place where they were first identified, sequential number and year of isolation. 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, and laboratories throughout Oceania and South East Asia, using conventional serological techniques. A geographically and temporally representative sample of isolates, together with any strains demonstrating uncharacteristic reactions during antigenic characterisation are 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). Participation is voluntary in all sentinel general practice surveillance systems, leading to variation in the number of contributors. 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. The case definition for a clinical diagnosis of ILI varies between sentinel surveillance schemes (Table).

Table. Case definitions of influenza-like illness used in Australian sentinel practice schemes, 2002[6]

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 symptoms with sudden onset (<12 hours previously): cough, rigours or chills, fever, prostration and weakness, myalgia, redness of mucous membranes, influenza in close contacts

Sentinel general practices contributing to the ASPREN scheme are mostly located in capital cities and larger regional centres on the east coast of Australia. In 2002, the average number of contributing practices was 51 (range 32–65) each week. These practices together reported on an average of 5,674 (range 2,081–7,476) consultations per week.

The Northern Territory Tropical Influenza Surveillance reported cases of ILI throughout the year from between 8 and 12 centres in the tropical northern regions, reporting on between 469 and 1,092 consultations per week. Data were reported as the rate per 1,000 consultations on a weekly basis.

The New South Wales Influenza Surveillance program collects clinical reports from New South Wales practitioners who are part of ASPREN and from seven Public Health Units, four rural and three metropolitan (Southern New South Wales, New England, Mid North Coast, Macquarie, Illawarra, Central Coast, Northern Sydney, Western Sydney and South Eastern Sydney). The total number of participating practices varied from 14 to 58 per reporting period with a mean of 3,513 consultations across participating practices per week. Reports were published weekly in 2002 between 4 May and 28 September.

The Victorian Infectious Diseases Reference Laboratory, the WHO Collaborating Centre for Reference and Research on Influenza and the Department of Human Services contributed to the Victorian Influenza Surveillance in 2002. Reports were published fortnightly between 5 May and 29 September. The sentinel practices reporting to the scheme in 2002 varied between 16 and 23 metropolitan and 7 to 15 rural practices, which reported on between 5,348 and 8,509 consultations each fortnight. ILI was reported per 100 patients and converted to a rate per 1,000 consultations to allow comparisons with other sentinel schemes.

In Western Australia, between 4 and 13 metropolitan and 1 to 2 rural practices reported ILI from 3 June to 28 October 2002. The number of consultations in the sentinel practices was not recorded and the data were presented as the number of cases of ILI per practice for each week.

Absenteeism surveillance

Australia Post, a major nationwide employer, continued to provide sick leave absenteeism data during 2002 between March and September. Absenteeism, defined as an absence due to illness for at least three consecutive days, was reported as a rate per 100 employees per week.

Hospitalisation data

To assess the impact of influenza on hospitalisation, the Australian Institute of Health and Welfare made available 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 was identified (J10) and cases where the virus was not identified (J11). Data for the 2000–01 financial year was the most recent available at the time of writing this report.

Results

The influenza surveillance data presented here are limited and should be interpreted with caution. Laboratory-confirmed influenza represents 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 ILI vary between sentinel practices (Table) which make comparisons of influenza prevalence difficult. In addition, definitions of ILI have varied from year to year, so comparisons of data across years are complex.

National Notifiable Diseases Surveillance System

In 2002, 3,780 laboratory-confirmed cases of influenza were reported to the NNDSS. All jurisdictions submitted reports although few reports of confirmed infections were received from Tasmania due to limited access to laboratories for testing for influenza. The number of notification received in 2002 were threefold greater than those received in 2001. This was in part due to incomplete reporting in 2001, the first year in which laboratory-confirmed influenza was a nationally notifiable disease.[7]

The notifications to NNDSS by month of report are shown in Figure 1. Notifications showed a peak in August (1,188 notifications). The breakdown of laboratory-confirmed influenza cases reported to NNDSS by age and sex is shown in Figure 2. The age specific rates were highest among children aged less than five years (119 cases per 100,000 population), and among persons 85 years or older (31 cases per 100,000 population). Although the overall male to female ratio for influenza in 2002 was 1.1:1, in children aged less than five years, there was a higher rate of influenza among males (130 cases per 100,000 population) than females (100 cases per 100,000 population).

Figure 1. Notifications of laboratory-confirmed influenza to the National Notifiable Diseases Surveillance System, Australia, 2002, by month of onset

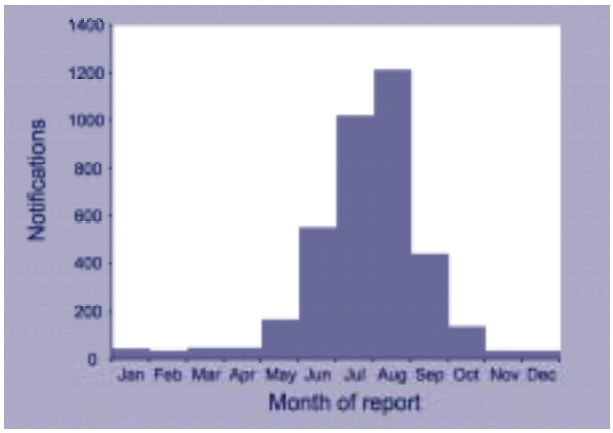
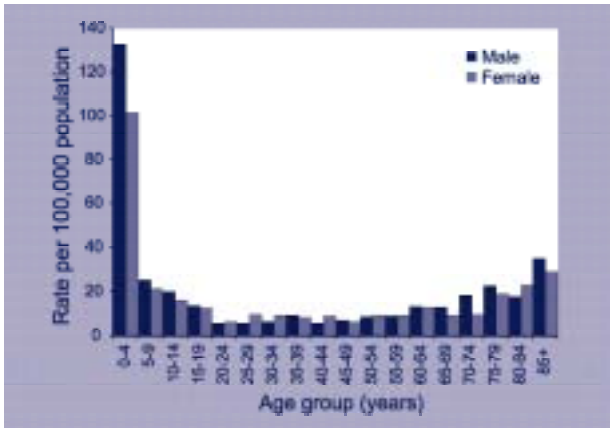


Figure 2. Notification rates of laboratory-confirmed influenza received by the National Notifiable Diseases Surveillance System, Australia, 2002, by age and sex



Laboratory surveillance

In 2002, a total of 2,345 laboratory diagnoses of influenza were made in participating laboratories of the LabVISE scheme. These were 1,798 influenza A and 547 influenza B diagnoses. The ratio of influenza A to B in 2002 was 3:1, which was lower than the 2001 ratio of 4:1. The overall influenza report showed a low level of activity until week 22 (3 June), when there was an increase in reports from 14 per week to approximately 45 per week. This was followed by a major peak of 169 reports per week in week 30 (29 July), then a decline to baseline (14 reports per week) by week 43 (28 October, Figure 3). The peak of influenza activity occurred earlier than in 2001 (Figure 4). In 2002, the peak in influenza B virus activity (week 22) preceded the peak of influenza A activity (week 30, Figure 3).

The seasonal pattern of influenza A and B activity between 1996 and 2002 is shown in Figure 5. The seasonal pattern in 2002 closely resembled that in 1997 with a relatively higher numbers of influenza B isolates, peaking earlier in the season than influenza A.

Figure 3. Laboratory reports of influenza, Australia, 2002, by type and week of specimen collection

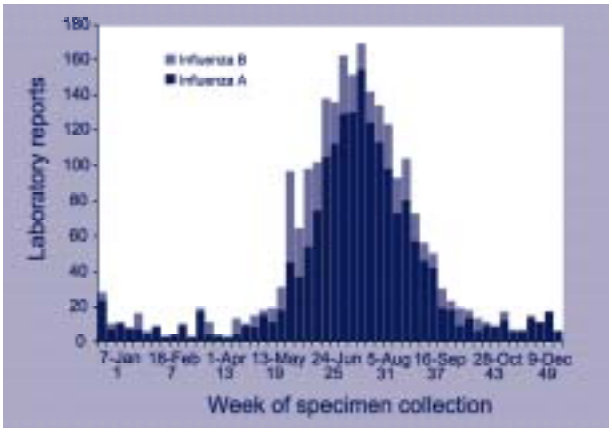


Figure 4. Laboratory reports of influenza, Australia, 2001 and 2002, by month of specimen collection

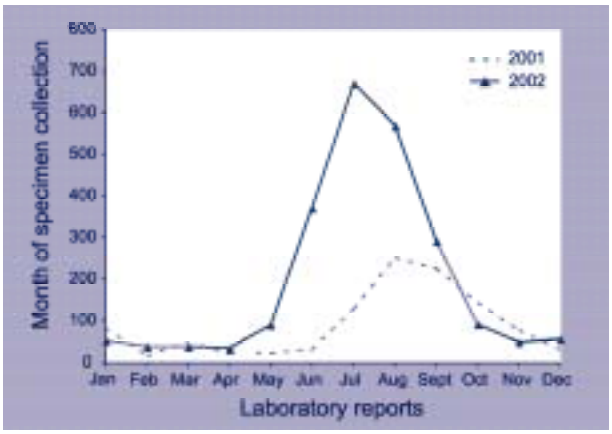
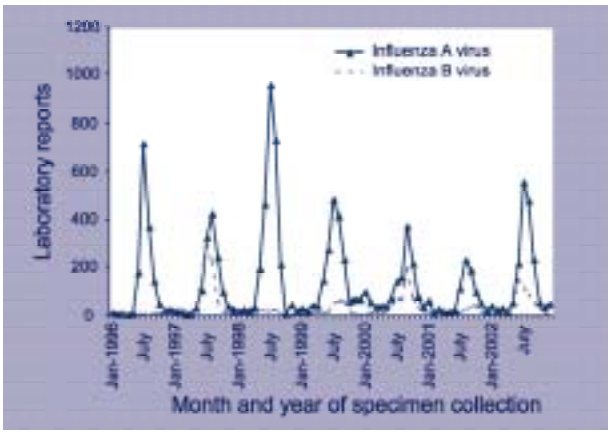


Figure 5. Laboratory reports of influenza, Australia, 1996 to 2002, by type and month of specimen collection



WHO Collaborating Centre for Reference and Research on Influenza

The WHO Collaborating Centre for Reference and Research on Influenza received 1,412 isolates and specimens that yielded viable viruses for antigenic analysis, 800 more than in 2001. Of these viruses 1,110 (78.4%) were influenza A(H3), 302 (21.5%) were influenza B and there was a single A(H1) strain. The variable region of the haemagglutinin was sequenced for 95 strains (1H1, 66H3, 28B) strains and the neuraminidase in 44 strains (27A and 17B) strains. The majority of A(H3) viruses were antigenically closely related to the A/Moscow/10/99 reference strain and the A/Panama/2007/99 vaccine strain with eight (<1%) strains showing some evidence of genetic variation. The neuraminidase was confirmed as N2 in all of 26 H3 viruses for which sequencing was undertaken. Sequencing of H3 haemagglutinin demonstrated continued genetic heterogeneity but no clear line of evolution at the moment (Figure 6). The single A(H1) strain was demonstrated to have a haemagglutinin that was antigenically and genetically closely related to A/New Caledonia/20/99 but an N2 neuraminidase genetically similar to those found on recent A(H3N2) viruses. Hence this virus belongs to the genetic reassortant lineage A(H1N2) which was first reported during the 2001–02 Northern Hemisphere season.[8] The A(H1N1), A/New Caledonia/20/99-like viruses continued to circulate in some countries while viruses of the A/Bayern/7/97 lineage have not been seen recently.

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

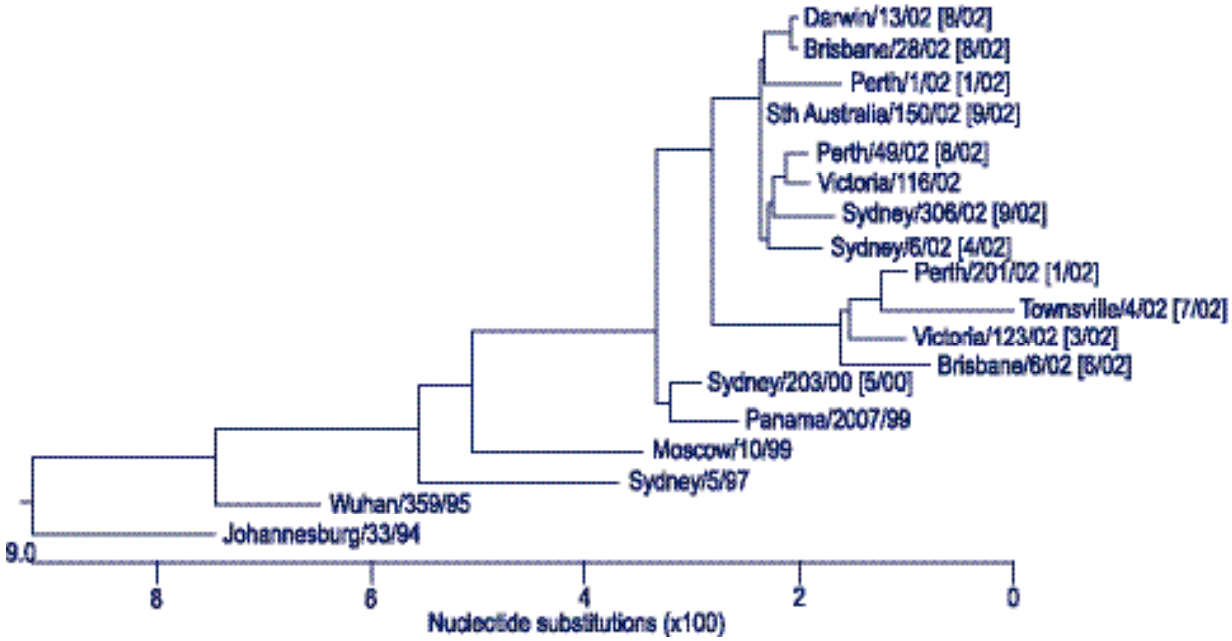
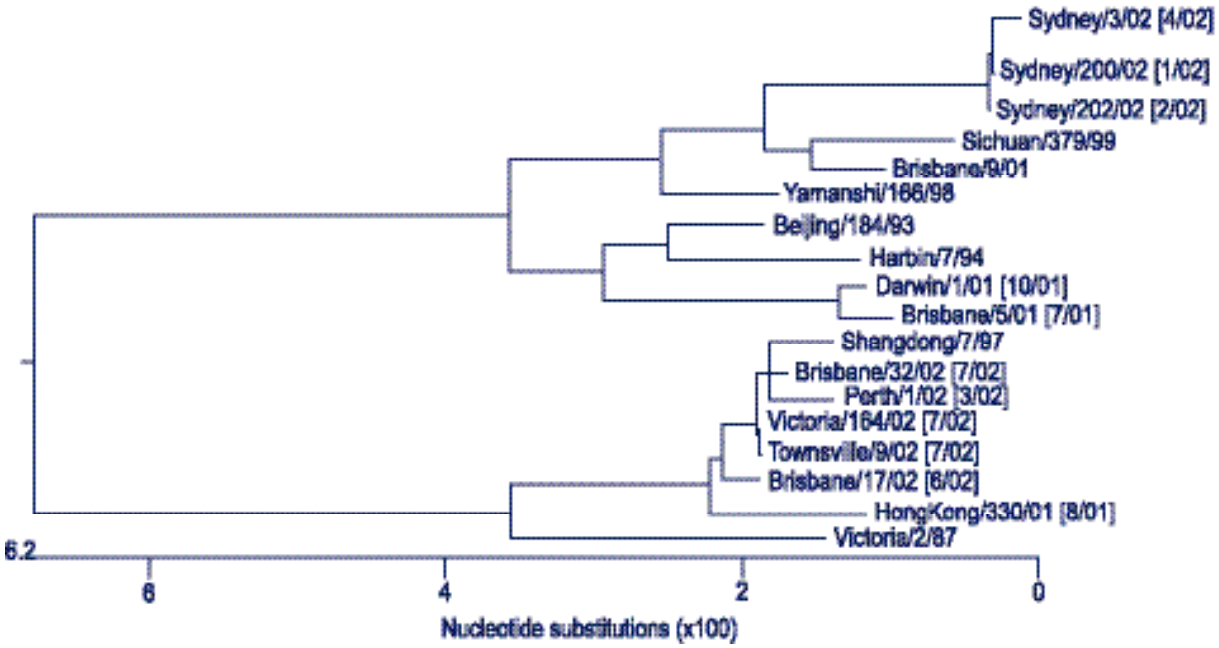


Figure 7. Evolutionary relationships between influenza B haemagglutinins (HA1 region)



The Australian influenza B viruses isolated during 2002 mainly (291 of 302 isolates) belonged to the lineage often referred to as the B/Victoria lineage which has not been seen in Australia for a decade. While this lineage of viruses had continued to circulate in Asia it had been absent from other regions for many years, re-emerging in some areas of Europe and North America during the 2001–02 winter.[9] Viruses isolated in Australia had a haemagglutinin that was antigenically and genetically closely related to the B/Hong Kong/330/2001 reference strain (Figure 7). However, by sequence analysis the neuraminidase of the isolates was found to be similar to that of the B/Sichuan/379/99-like viruses of the alternate lineage indicating that a genetic reassortment event had also occurred for these viruses. Some viruses isolated in the Northern Hemisphere 2001–02 winter were similarly found to be genetic reassortants.[9] The remaining 11 influenza B strains analysed belonged to the previously circulating lineage, nine were antigenically close to B/Sichuan/379/99 while two were more closely related to the older B/Harbin/7/94 reference strain.

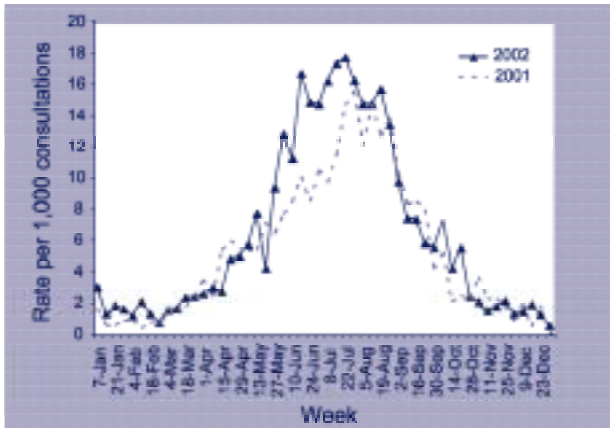
Based on the antigenic and genetic analyses and post-vaccination human serology studies conducted at the WHO Collaborating Centre for Reference and Research on Influenza the 2002 Australian influenza vaccine represented a good antigenic match for the circulating influenza A viruses but only for a minority of the influenza B strains.

After a decade of absence B/Hong Kong/330/2001-like viruses began to spread from Asia into other regions in 2001. However, apart from a small outbreak in Hawaii in May, this spread only became apparent some time after the WHO Consultation on Influenza Vaccine Formulation in September 2001 and the Australian Influenza Vaccine Committee meeting on 11 October 2001 which both recommended a B/Sichuan/379/99-like vaccine strain for the 2002 vaccine. Because there is a substantial antigenic difference between viruses of these two lineages, as expected, the 2002 vaccine produced reduced responses against the new B/Hong Kong/330/2001-like viruses. Nevertheless, around 40 per cent of younger adults and 25 per cent of older adults achieved antibody titres in the protective range against recent strains compared with 95 per cent and 80 per cent against the homologous virus, in younger and older adults respectively.

Sentinel general practice (GP) surveillance

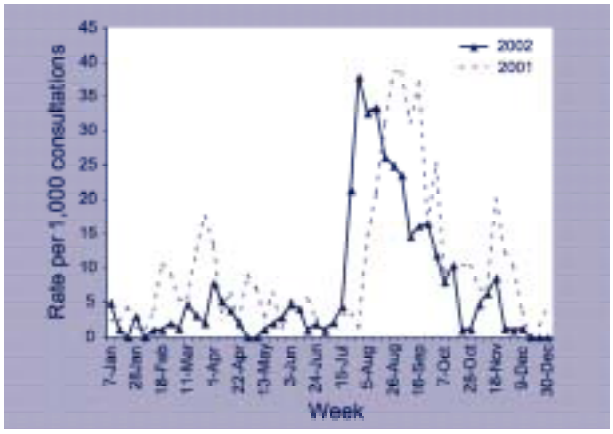
Reports of influenza-like illness to ASPREN practice sites showed a rapid rise starting in week 20 (27 May) and peaking in week 24 (17 June) when reports reached a rate of 16.6 cases per 1,000 consultations, and remained at that level for nine weeks. The peak in rates of ILI was earlier and slightly higher than in 2001 (Figure 8).

Figure 8. ASPREN consultation rates for influenza-like illness, Australia, 2001 and 2002, by week of report



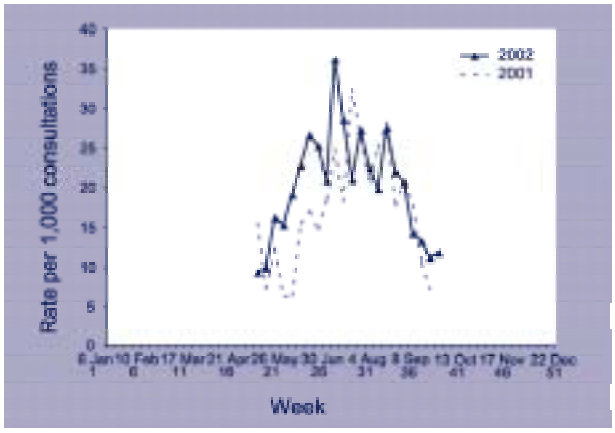
The Northern Territory Tropical Influenza Surveillance Scheme data showed one major peak (38.7 cases per 1,000 consultations) of influenza activity in week 30 (28 July) which was of the same magnitude but four weeks earlier than the peak influenza activity reported in 2001 (Figure 9).

Figure 9. Consultation rates for influenza-like illness,Northern Territory, 2001 and 2002, by week of report



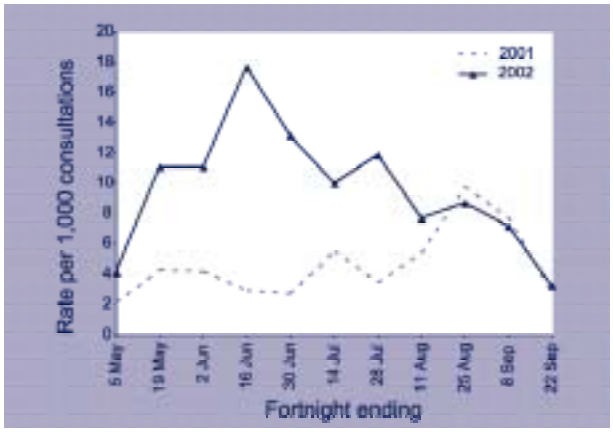
In New South Wales, influenza-like illness reports peaked in week 27 (7 July) at 36.2 cases per 1,000 consultations (Figure 10). In contrast with the peak consultation rate in 2001, the peak activity of influenza in New South Wales in 2002 was earlier and marginally higher.

Figure 10. Consultation rates for influenza-like illness, New South Wales, 2001 and 2002, by week of report



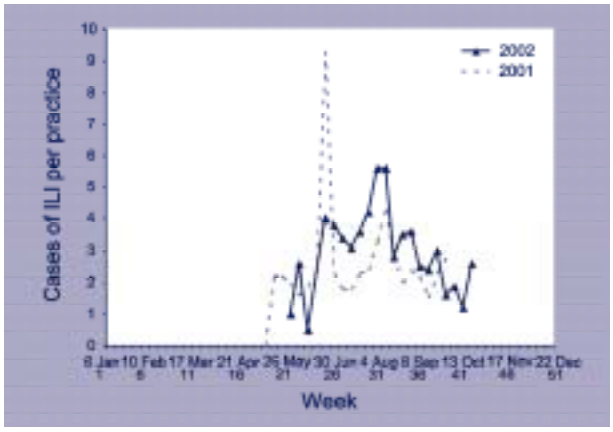
In Victoria, the reporting rate of influenza-like illness in 2002 peaked at 17.6 cases per 1,000 consultations in the fortnight ending 16 June (Figure 11). In comparison to the previous year (peak consultation rate on 16 August at 9.7 cases per 1,000 consultations), the peak consultation rate in 2002 was higher and occurred earlier in the year.

Figure 11. Consultation rates for influenza-like illness, Victoria, 2001 and 2002, by fortnight of report



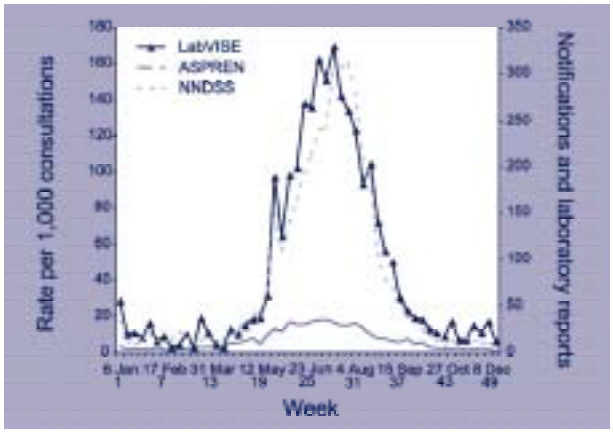
In Western Australia, the peak of reporting of influenza-like illness occurred later than in the eastern states, in week 32 to 33 (11–18 August) at 5.6 cases per practice (Figure 12).

Figure 12. Consultation rates for influenza-like illness, Western Australia,2001 and 2002, by week of report



A comparison of the NNDSS, ASPREN and LabVISE reports is shown in Figure 13. The peak in confirmed influenza notifications received by NNDSS, in reports of influenza-like illness to ASPREN, and in laboratory reports of influenza to LabVISE, overlapped in the period between week 29 to 32 (21 July to 4 August).

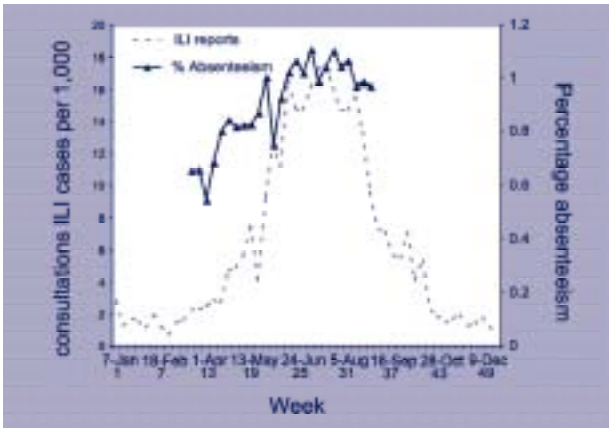
Figure 13. Influenza laboratory reports to LabVISE,notifications to NNDSS and consultation rates in ASPREN, Australia, 2002, by week of report



Absenteeism surveillance

Data supplied by Australia Post suggested an association between the peak in influenza activity and absenteeism. National absenteeism rates were highest at 1.1 per cent in weeks 29 and 30, which coincided with the peak in ASPREN reports of consultation of influenza-like illness (Figure 14).

Figure 14. Rates of absenteeism and consultation rates for influenza-like illness, Australia, 2002, by week of report



Hospitalisation due to influenza

In 2000–01 there were a total of 2,380 separations in Australian hospitals for influenza. Six hundred and fifty-five of these were cases in which the influenza virus was identified. Altogether influenza was responsible for 9,825 hospital days in 2000–01.

Discussion

In 2002, influenza activity in Australia was moderately increased compared with 2001, as assessed by all surveillance systems. This increase was more evident in temperate regions of Australia than in tropical regions. Although influenza A was predominant, there was increased activity of influenza B compared with 2001. Influenza A and B peaked at different periods, with influenza B peaking early in the flu season (early June) and influenza A peaking later in the season (late July).

Surveillance of influenza in Australia is based on a number of different systems. Although there are continuing problems in comparing data from different surveillance systems, there were some improvements in 2002. The addition of laboratory-confirmed influenza to the NNDSS has lead to a marked increase in the numbers of laboratory isolates of the influenza virus available for analysis. The need for timely data has been addressed by the fortnightly publication of data during the influenza season on the Communicable Diseases Australia Website (http://www.cda.gov.au). This allows the wide dissemination of information on the evolving dimensions of the annual influenza epidemic and provides data for public health action as required. The need to assess circulating influenza strains in a timely manner was demonstrated by the WHO Collaborating Centre for Reference and Research on Influenza during 2002, when the emergence of the B/Hong Kong strain was identified early in the season and assessments of the protective efficacy of the 2002 vaccine were undertaken rapidly.

In Australia in 2002, influenza A isolates were almost entirely H3N2 strains with only a small number of strains showing evidence of genetic variation and were closely related to the A/Panama/2007/99 vaccine strain. The virtual absence of H1N1 strains was in sharp contrast with the predominance of this strain in 2001.[7] Similar low levels of A/H1N1 were observed between 1996 and 1998.[10] The sequence of the single A/H1N1 was shown to have a haemagglutinin related to the vaccine strain, but a neuraminidase related to A/H3N2. This reassortment lineage (H1N2) was detected for the first time in the United Kingdom during the 2001–02 winter, when it made up 54 per cent of all influenza A viruses isolated.[11] The H1N2 virus was isolated more frequently from children aged less than 15 years of age but there was no evidence of the strain causing a pandemic or more severe disease. Preliminary data from the UK for the 2002–03 influenza season, indicates that H1N2 viruses comprised a small proportion of influenza viruses identified to date.[12] In the United States of America, in the 2002–03 season, preliminary data indicated that H1N2 influenza comprised 24 per cent of the strains characterised.[13]

Influenza B isolates showed evidence of a genetic reassortment with haemagglutinin closely related to B/Hong Kong/330/2001 and neuriminidase sequences related to the B/Sichuan/379/99. Similar viruses were identified in the Northern Hemisphere during the 2001–02 winter. Despite differences between the B/Hong Kong/220/2001 viruses and the B/Sichuan/379/99 vaccine strain used in the 2002 Australian vaccine, there was evidence that protective antibody titres were induced in a proportion of vaccinees. There were two reports of outbreaks of influenza due to the B/Hong Kong virus in Victoria, affecting approximately 136 individuals (Graham Tallis and Kerry-Ann O'Grady, Department of Human Services, personal communication).

Reports of the B/Hong Kong strain were also made in 2002 in New Zealand,[14] and in 2002–03 in the UK and the USA.[12,13]

Influenza vaccination of vulnerable populations such as the elderly is important to reduce the morbidity and mortality of annual influenza epidemics. The National Health and Medical Research Council recommends annual vaccination for influenza for all Australians aged over 65 years. A national telephone survey in October and November 2002 showed 76.9 per cent of Australian aged over 65 years received influenza vaccination in 2002.[15] This is a similar vaccination rate in this age group to that in 2001. A recent analysis of the protective efficacy of the influenza vaccine in Americans aged 65 years and older over two influenza seasons, demonstrated a reduction in hospitalisation for pneumonia or influenza of 29–33 per cent and a reduction in deaths from all causes of 48–50 per cent. The same study also noted a significant reduction in hospitalisations for cardiac disease and stroke in the same population.[16] An analysis of mortality associated with influenza and respiratory viruses in the USA showed an increase in influenza-associated death, probably the result of an ageing population.[17] These studies reinforce the need for continued annual influenza vaccination of the elderly.

In response to the emergence of this new influenza B, the recommended Australian influenza vaccine for 2003 has been changed to incorporate B/Hong Kong/330/2001 in place of B/Sichuan/379/99. The influenza A strains in the 2003 vaccine remain unchanged (A/New Caledonia/20/99(H1N1) and A/Moscow/10/99 (H3N2)).

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Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2001

A report of the Australian Mycobacterium Reference Laboratory Network

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Abstract

The Australian Mycobacterium Reference Laboratory Network collected and analysed laboratory data on new cases of disease caused by *Mycobacterium tuberculosis* complex in the year 2001. A total of 771 cases were identified, representing an annual reporting rate of 4.0 cases of laboratory-confirmed tuberculosis per 100,000 population. The predominant specimen type was sputum, (n=369) and a further 111 were collected at bronchoscopy. Smears were positive for 214 of 369 (58.0%) sputum and 42 of 111 (37.8%) bronchoscopy specimens respectively. Seven children (male n=5, female n=2) under 10 years of age had bacteriologically confirmed tuberculosis. A total of 69 isolates (8.9%), comprising 67 *M. tuberculosis*, one *M. africanum*, and one *M. bovis*, were resistant to at least one of the anti-tuberculosis agents. Excluding the *M. bovis* isolate, 61 of 64 (93.5%) were classified as having initial resistance, three had acquired resistance, and no data were available on the presence or absence of previous treatment for four patients. Resistance to at least isoniazid and/or rifampicin was noted for 67 isolates (8.7%), with resistance to both isoniazid and rifampicin (i.e. defined as multidrug-resistant disease) observed in 12 (1.6%) isolates. All of the multidrug-resistant isolates were *M. tuberculosis*, 10 were from the respiratory tract. The country of birth was known for 63 of 68 (92.6%) patients with a drug-resistant strain of *M. tuberculosis* or *M. africanum*; five were Australian-born and 58 (92.1%) had migrated from a total of 22 countries. One hundred and seven respiratory specimens had a nucleic acid amplification testing performed; 89 of 90 (98.9%) smear positives were nucleic acid amplification testing positive, whilst only 13 of 17 (76.5%) smear negative specimens were nucleic acid amplification testing positive. The 2001 laboratory data reveals a stable incidence rate and level of drug resistance in isolates from Australian patients with tuberculosis. *Commun Dis Intell* 2003;27:173–180.

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

Introduction

Since 1991, the National Notifiable Diseases Surveillance System (NNDSS) has provided statistics on cases of tuberculosis (TB) reported to public health authorities in Australia's States and Territories.[1] The Australian Tuberculosis Reporting Scheme has been conducted by the Australian Mycobacterium Reference Laboratory Network (AMRLN) since 1986.[2] Statistics compiled by the AMRLN relate to cases of bacteriologically confirmed tuberculosis whereas NNDSS data will have a proportion of cases that are identified on the basis of clinical and epidemiological information, or on non-bacteriological laboratory investigations.[3] This report describes the bacteriologically confirmed TB diagnoses for the year 2001.

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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 Guérin (BCG) strain of *M. bovis* is a member of the MTBC, no information on this organism is included in the present report. In 2001, nearly 60 laboratories performed culture for mycobacteria.[4] Almost all isolates of MTBC were referred to one of the five laboratories comprising the AMRLN for specific identification and drug susceptibility testing. Comparable methodologies are used in the reference laboratories. Relapse cases, as defined by the *National Strategic Plan for TB Control in Australia beyond 2000* prepared by the National TB Advisory Committee,[5] were included in the laboratory data as laboratories are generally unable to differentiate relapse cases from new cases. Temporary visitors to Australia were included as were illegal aliens 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 (where available):

- demography: patient identifier, age, sex, HIV status and state of residence;
- specimen: type, site of collection, date of collection and microscopy result;
- isolate: species of mycobacterium and results of drug susceptibility testing;
- nucleic acid amplification test (NAAT): results of testing; and
- if the isolate was drug resistant: patient country of origin, 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 collation and analysis. Duplicate entries (indicated by identical patient identifier and date of birth) were deleted prior to analysis. Rates were calculated using mid-year estimates of the population for the year 2001 supplied by the Australian Bureau of Statistics.[6]

For each case, 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 to indicate pulmonary disease. Cases with multi-site isolations, provided a sputum or bronchoscopy specimen was culture-positive, were listed as having pulmonary disease, the most important category for public health purposes. Cases for which there were multiple-site isolations were not categorised as having miliary or disseminated disease as differentiation is based on clinical findings that are generally not available to the reporting laboratories. Initial drug resistance was defined as the presence of drug-resistant strains of *M. tuberculosis* in cases of tuberculosis in which there was no known history of anti-tuberculosis treatment. Patients who had begun anti-TB treatment and had developed resistance to one or more of the drugs used during treatment were recorded as having acquired drug resistance.[7]

Results

Total reports and distribution by state or territory

There were 771 bacteriologically confirmed cases of tuberculosis in 2001 (Figure 1), representing an annual rate of 4.0 cases per 100,000 population. State-specific reporting rates varied from 2.2 cases (Queensland) to 11.6 cases per 100,000 population (Northern Territory) (Table 1). There were five patients from Papua New Guinea who were diagnosed in Australia (included in the Queensland data), five persons identified as asylum seekers from Afghanistan (South Australia n=4, Western Australia n=1), and two persons with temporary protection visas from East Timor.

Figure 1. Comparison between tuberculosis notifications and laboratory data, Australia, 1990 to 2001

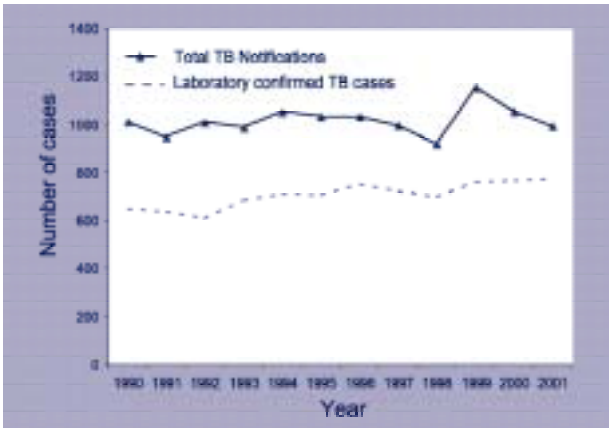


Table 1. Bacteriologically confirmed cases of tuberculosis in Australia, 1991 and 1998 to 2001, cases and rate per 100,000 population by state or territory*

State or territory	2001[8]		2000[9]		1999[9]		1998[10]		1991[10]	
	n	%	n	%	n	%	n	%	n	%
New South Wales†	327	4.8	307	4.5	291	4.3	289	4.4	246	4.0
Victoria	222	4.6	231	4.8	261	5.5	192	4.1	201	4.5
Queensland	81	2.2	76	2.1	75	2.1	85	2.5	79	2.7
Western Australia	68	3.6	63	3.3	64	3.4	66	3.6	46	2.8
South Australia	38	2.5	41	2.7	46	3.1	40	2.7	31	2.1
Tasmania	12	2.8	2	0.4	2	0.4	6	1.3	9	1.9
Northern Territory	23	11.6	45	23.0	21	10.9	22	11.6	21	12.4
Total	**771**	**4.0**	**765**	**4.0**	**760**	**4.0**	**700**	**3.7**	**633**	**3.6**

* Data from previous reports of the AMRLN.

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

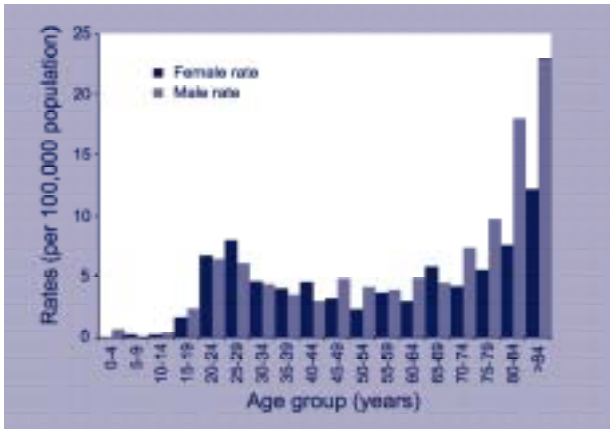
Causative organism

Almost all isolates were identified as *M. tuberculosis* (768) with only two isolates of *M. africanum* and one of *M. bovis*.

Distribution by gender, age and site of disease

Complete information for gender and age were submitted for 767 of the 771 cases. Seven children (male n=5, female n=2) under 10 years of age had bacteriologically confirmed tuberculosis (gastric aspirate n=5, sputum n=1, lymph node n=1). The relationship of tuberculosis to age and gender are shown in Figure 2. The overall male:female ratio was 1.1:1. Age and gender rates varied depending on the site of infection. The male:female ratio for pulmonary disease was 1.3:1. The predominant specimen type was sputum, including eight gastric aspirates (n=369, 47.9%); a further 111 (14.3%) and 14 were bronchoscopy or lung tissue/biopsy samples, respectively. The median age for both males and females with pulmonary disease was 25–29 years. Forty-seven (6.1%) isolates were of pleural origin. There were 139 (18.0%) isolates from lymph node with a male:female ratio of 1:1.6, the median age was 35–39 and 25–29 years respectively for males and females with lymph node disease. There were 16 isolates from other sites including usually sterile fluids (pericardial n=2, blood n=2), abscess (psoas n=2), and tissue (sternal n=2, parathyroid n=1, tonsil n=1).

Figure 2. Laboratory diagnosis of for *Mycobacterium tuberculosis* complex disease, Australia 2001, by age and sex



Association with HIV

The AMRLN database recorded the HIV status for only 81 (10.5%) patients. Two patients were identified as HIV seropositive; both were from South East Asia and both isolates were fully drug susceptible strains of *M. tuberculosis*.

Microscopy

Results of microscopy were available for 712 of 771 (92.3%) specimens; microscopy was not performed on five specimens. Results for a further 54 samples were unknown. Smears were positive for 214 of 369 (58.0%) sputum and 42 of 111 (37.8%) bronchoscopy specimens respectively (Table 2). A total of 47 pleural specimens were culture positive for M. tuberculosis with eight (17.0%) smear-positive for acid fast bacilli (AFB). Thirty-six (11.4%) specimens of pleural fluid and 11 (40.0%) pleural biopsies were smear positive for AFB. Of the 139 specimens of lymph node, microscopy results were available for 125; and 24 (19.2%) were smear-positive for AFB.

Drug susceptibility testing

Results of in vitro drug susceptibility testing were available for all 771 isolates for isoniazid (H), rifampicin (R), ethambutol (E), and pyrazinamide (Z). A total of 69 isolates (8.9%), comprising 67 *M. tuberculosis*, one *M. africanum*, and one *M. bovis*, were resistant to at least one of the above anti-tuberculosis agents. *M. bovis* is inherently resistant to pyrazinamide. Results of testing for streptomycin (S) were available for 228 of 771 (29.5%) isolates with eight (3.5%) demonstrating mono-resistance, and a further 11 isolates resistant to both S+H. Resistance to at least H and/or R was noted for 67 isolates (8.7%), with resistance to both H and R [i.e. defined as multidrug-resistance (MDR)] observed in 12 (1.6%) isolates. All of the MDR isolates were *M. tuberculosis* (MDRTB). Of the 12 MDRTB isolates, 10 were from the respiratory tract (sputum n=6, bronchoscopy n=4); the remaining isolates were from lymph node and pleural fluid (Table 3). One of the four bronchoscopy specimens, and two sputums were smear positive.

Of the 67 *M. tuberculosis* and one *M. africanum*, 43 (5.6%), 1 (0.1%) and one (0.1%) demonstrated mono-resistance to H, R, and E respectively. There was no mono-resistance to pyrazinamide. There were 66 strains that demonstrated resistance to H at a concentration of 0.1 mg/L in the radiometric BACTEC system. One isolate was not tested at the higher concentration of 0.4 mg/L. Of the remaining 65 strains, 43 (66.2%) demonstrated resistance at the higher level.

Thirty-four of 67 (50.7%) specimens culture-positive for drug-resistant *M. tuberculosis* were also smear-positive for AFB.

Initial or acquired resistance and country of origin

There were 67 *M. tuberculosis* and one *M. africanum* isolates resistant to at least one of H, R, E or Z. Of these, 61 of 64 (95.3%) were classified as having initial resistance, three had acquired resistance, and no data were available on the presence or absence of previous treatment for four patients. The country of birth was known for 63 of 68 (92.6%) patients; five were Australian-born, and 58 (92.1%) had migrated from a total of 22 countries.

Of the 58 migrants with drug-resistant disease, 37 (63.8%) had migrated from one of six countries: Vietnam (n=11), Philippines (n=10), India (n=7), Indonesia (n=6), and Papua New Guinea (n=3).

Use of nucleic acid amplification tests

Nucleic acid amplification tests (NAAT) were performed on 136 of 771 (17.6%) specimens which subsequently grew MTBC on culture. Sputum (n=89), bronchoscopy (n=18), and lymph node (n=12) were the most frequently tested. Of the 136 specimens, 110 were NAAT positive and 25 were negative. One specimen of bronchial washings (smear negative) produced a non-interpretable result due to the presence of inhibitors.

Smear positive specimens were more likely to have NAAT performed, (Table 4). Excluding the specimen with a non-interpretable result, 107 culture-positive respiratory specimens had NAAT performed; 89 of 90 (98.9%) smear positive specimens were NAAT positive, whilst only 13 of 17 (76.5%) smear negative specimens were NAAT positive. Importantly, four (2.9%) smear positive specimens (one each of bronchial washings and pleural fluid, two lymph nodes) that were culture positive for *M. tuberculosis*, were NAAT negative.

Table 2. Site of specimens smear– and culture-positive for *Mycobacterium tuberculosis* complex, Australia,2001

	Number*	Smear positive (%)
Sputum	369	58.0
Bronchoscopy	111	39.6
Lymph node	139	19.2
Pleural	47	17.8
Bone/joint	26	19.2
Genito-urinary	20	ND†
Peritoneal	13	ND
Skin	7	ND
CSF	5	ND

* Specimens not tabulated: 14 pulmonary tissue samples, 16 specimens from miscellaneous sites, and 4 of unknown site.

† Percentage of specimens smear positive not calculated due to small numbers.

Table 3. Drug resistance patterns in multidrug-resistant strains, Australia, 1996 to 2001

Resistance pattern (standard drugs)[1]	2001	2000	1999[9]	1998[9]	1997[10]	1996[10]
H+R only	8	3	2	2	6	10
H+R+E	1	1	1	1	1	1
H+R+Z	3	3	1	2	5	4
H+R+E+Z		1	0	1		0
Total (%)	**12 (1.6)**	**8 (1.0)**	**4 (0.5)**	**6 (0.9)**	**14 (1.9)**	**15 (2.0)**

H = Isoniazid, R = rifampicin, E = ethambutol, Z = pyrazinamide

Table 4. Results for nucleic acid amplification tests performed on respiratory specimens, Australia,2001

NAAT result*	Culture positive respiratory specimens	
	Smear positive	Smear negative
Positive	89	4
Negative	1	13
Total (110)	**90**	**17**

* A variety of nucleic acid amplification tests methods were used, depending upon laboratory

Discussion

The isolation of 768 *M. tuberculosis*, two *M. africanum*, and a single *M. bovis* from clinical specimens for the calendar year 2001, yielded a rate of four bacteriologically confirmed cases of tuberculosis per 100,000 population, an almost identical rate as in 2000, and consistent with data reported for the past decade.[8] The NNDSS reported 997 tuberculosis notifications in 2001, the second lowest notification rate on record.[11] The NNDSS has consistently reported higher notifications than the AMRLN laboratory data (range 24–40%) and for 2001, the 22.7 per cent difference between the two datasets was at the lowest end of the range. In 2001, the NNDSS dataset recorded 558 cases from the respiratory tract, 74 pleural, 210 lymphatic, and 43 bone/joint.[11] If the two databases are compared, 88.5 per cent, 66.2 per cent, 63.5 per cent and 60.5 per cent of respiratory, pleural, lymphatic and bone/joint cases respectively, were bacteriologically confirmed. Comparison of two unlinked databases is problematic. However, the data suggests that, with almost 90 per cent of pulmonary cases reported to NNDSS having a bacteriological confirmation of disease, respiratory TB is well investigated in Australia. In contrast, the lower proportion of extra-pulmonary disease confirmed by culture suggests that too much reliance is placed on clinical, histological or radiological diagnoses for these forms of TB.

As expected, the respiratory tract accounted for the majority of culture positive specimens. Of the 494 respiratory specimens, 111 were obtained at bronchoscopy, and of those that reported a microscopy result, 42 of 108 (38.9%) recorded a positive smear. In four cases, MDRTB was isolated from bronchoscopy specimens, including a single specimen that was also smear positive. Bronchoscopy is especially useful for the diagnosis of pulmonary tuberculosis in suspect, sputum smear negative patients and in non-sputum producers.[12] It is of interest that almost 40 per cent of bronchoscopy specimens were reported as microscopy smear positive which might suggest that bronchoscopies are being undertaken in persons who may well have been sputum smear positive but were not tested prior to bronchoscopy. Some bronchoscopies performed may not have been necessary had sputums been submitted,—or smear results retrieved—prior to bronchoscopy. The performance of bronchoscopies on smear-positive cases exposes the patients to a needless invasive procedure and represents an infection control hazard for the bronchoscopist and their support staff. A cost effective alternative to bronchoscopy is to perform three induced sputum tests on consecutive days.[13]

A total of 67 isolates (9.8%), comprising 66 *M. tuberculosis* and one *M. africanum* were resistant to at least one of H, R, E, or Z. One patient had *in vitro* resistance to R only, the 12 other isolates with resistance to R were MDRTB. Molecular tests that can determine presumptive rifampicin resistance directly from specimens are now available and may provide rapid presumptive evidence of MDRTB with a test sensitivity approaching 95 per cent.[14,15] There were 12 (1.6%) isolates with *in vitro* resistance to at least H+R (i.e. multidrug–resistant TB). The rate of MDRTB in Australia has remained steady over the past decade.

The country of birth was known for 63 of 68 (92.6%) patients with a drug-resistant strain of *M. tuberculosis* or *M. africanum*; five were Australian-born and 58 had migrated from a total of 22 countries. Drug-resistant cases among migrants reflect the performance of TB control programs in the countries from which the patients migrated. Determining a history of previous TB treatment among migrants is also problematic and confounds accurate classification of drug resistance as 'initial' or 'acquired'. Drug resistance data is therefore a poor indicator of the performance of Australian TB services. A far more meaningful performance indicator would be the proportion of patients who relapse or fail to respond to treatment within Australia in circumstances where the drug susceptibility profile of the original isolate is known.

For the first time, data were collected on nucleic acid amplification testing performed as a diagnostic test. The data were incomplete with at least one AMRLN member being unable to collect NAAT results for isolates submitted by referring laboratories. All of the 136 specimens where NAAT was performed were cultured for mycobacteria. The different performance of NAAT on smear-positive and smear-negative pulmonary specimens is demonstrated by the data. For smear positive specimens, there was high concordance of NAAT positivity with culture positivity. In contrast, smear negative respiratory specimens had a lower concordance, with

slightly over 75 per cent of culture confirmed specimens also NAAT positive, a finding higher than previous evaluations on NAAT performed in settings with a low incidence of tuberculosis.[16,17,18,19]

The use of NAAT for respiratory specimens from patients with suspected TB should be limited to respiratory smear positive specimens where the result is likely to influence clinical and/or public health decisions, and respiratory smear negative specimens from a patient with a high probability of TB and prompt management and public health decisions are required. The use of NAAT is inappropriate when a patient is respiratory smear positive and has a very high probability of TB, when a patient is respiratory smear negative and has a low probability of TB, and for monitoring treatment.

A further 14 specimens were NAAT positive but culture negative or culture was not performed, and were not included in the 2001 data. In several instances, a NAAT was performed on formalin-fixed tissue after the opportunity to culture diagnostic material had been precluded by the sample having been placed in formalin or another fixative. Whilst this approach may provide a retrospective diagnosis of tuberculosis,[20] it is not practical to identify the organism to species level (within MTBC), perform drug susceptibility testing, or undertake genotyping. When specimens need to be obtained by an invasive technique, the importance of considering mycobacterial disease in the differential diagnosis, especially prior to the procedure, cannot be over-emphasised. Unfortunately, anecdotal evidence suggests that requests for NAAT on fixed specimens are rising, a demanding technique that is time consuming, expensive, and insensitive.

The AMRLN database makes an important contribution to understanding the epidemiology of tuberculosis in Australia. The release of the *National Strategic Plan for TB Control in Australia Beyond 2000* by the Communicable Diseases Network Australia highlights the need to merge the AMRLN and NNDSS databases in order to provide a complete dataset to assess Australia's TB services against agreed performance indicators.

Acknowledgements

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

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, Victoria.

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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National atypical mycobacteria survey, 2000

Compiled by Frank Haverkort
from information supplied by the Australian Mycobacterium Reference Laboratory Network and the Special Interest Group in Mycobacteria within the Australian Society for Microbiology

Abstract

Infections with atypical mycobacteria in Australia during 2000 occurred at a rate of 1.8 cases per 100,000 population. The main sites of disease were the respiratory tract, soft tissue, and the lymphatics. The *Mycobacterium avium* complex was the most common group of mycobacteria isolated from respiratory, lymphatic sites, and blood.The rapidly growing mycobacteria, predominantly the *M. fortuitum–M. abscessus–M. chelonae* group were the most common soft tissue infections. Atypical mycobacteria were isolated from significant numbers of sputum 'smear positive' patients, requiring further tests to exclude *M. tuberculosis*. Geographical differences were observed for some *Mycobacterium* species, notably the isolation of *M. haemophilum* from Western Australia, and *M. ulcerans* from Victoria and Queensland. Newer molecular techniques, while improving precision and accuracy of identification, raise additional questions about the ecology of the atypical mycobacteria and their role in disease. *Commun Dis Intell* 2003;27:180–189.

Keywords: atypical mycobacteria, nontuberculous mycobacteria, atypical mycobacteriosis, laboratory diagnosis, Mycobacterium

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Introduction

Mycobacteria other than the *Mycobacterium tuberculosis* complex (commonly referred to as 'atypical', 'nontuberculous', 'environmental' mycobacteria, etc) have been implicated in a variety of clinical conditions including tuberculosis-like pulmonary disease, lymphadenitis, superficial and soft tissue infections, and severe disseminated disease. In addition to humans, a variety of animals, birds, reptiles and fish are susceptible to infection with atypical mycobacteria (AM). Modern taxonomic tools have identified many new species of AM, the majority of which are considered potential pathogens, given appropriate host conditions. Unlike tuberculosis, atypical mycobacteriosis is rarely a disease of public health importance, although nosocomial and iatrogenic outbreaks have been recorded. On the other hand, clinical management of atypical mycobacteriosis can be difficult, depending on the species involved, the site of disease, and co-existing host factors. Many AM infections are refractory to treatment with standard anti-tuberculosis agents.

In Australia, as in Europe and the United States of America, increased reporting of atypical mycobacterial disease can be attributed to a growing pool of immunosuppressed patients (particularly HIV+), and greater awareness on the part of clinical and laboratory personnel. A substantial proportion of laboratory resources in mycobacterial reference laboratories in industrialised countries is expended in the isolation, identification and susceptibility testing of AM. Many of these isolates are not associated with disease, but represent colonisation or environmental contamination of the patient.

Although the AM are environmental organisms, much remains to be elucidated with regard to their ecology and epidemiology. While disease due to *M. avium* complex (MAC) is apparently common throughout the developed world, species such as *M. xenopi* and *M. malmoense* are encountered more commonly in Europe than in the United States of America or Australia. Data for atypical mycobacteriosis in Australia are limited, primarily because cases are not notifiable in every state or territory, cases are not included in the National Mycobacterial Surveillance Scheme, and not all recovered AM are fully identified.

The results of a collaborative study undertaken by the Australian Mycobacterium Reference Laboratory Network (AMRLN) on patients whose specimens were culture-positive for AM in 2000 are reported here.

Methods

The data in this report are based on clinical specimens that were culture-positive for AM during the calendar year 2000. Almost 80 laboratories performed culture for mycobacteria in 2000 (Royal College of Pathologists of Australasia Quality Assurance Program) and mycobacterial isolates from Australian patients were forwarded to one of the five laboratories that comprise the AMRLN. The reference laboratories identify isolates to species level using standard procedures such as evaluation of phenotypic characteristics, commercial probes, 16S rDNA sequencing, and high performance liquid chromatography.

Reference laboratories provide specific identification of isolates from patients who are likely to have mycobacterial disease, e.g. smear-positive specimens from the respiratory or urinary tract, tissues and biopsies, usually sterile sites, and wound swabs. Single isolates of AM from the respiratory or urinary tract were less likely to be identified to species level as their clinical significance was often doubtful, and the laboratory techniques were expensive and time-consuming. Indeed, these isolates may never be referred to a reference laboratory. For these reasons, the total figure for AM isolations in 2000 (n=1,447) must be regarded as conservative.

For each patient, the nature of the first clinical specimen that yielded an isolate was used to record the nominal site of disease. Patients who had been culture positive for AM in previous years were excluded from the study. For each patient, the following data were collected:

- a unique identifier (usually a laboratory accession number);
- age and sex;
- specimen source of isolate;
- result of acid-fast microscopy; and
- species (or species complex) of isolate.

The reference laboratories liaised with the responsible medical practitioner in an attempt to ascribe clinical significance to each isolate. In cases where HIV-sero-positivity was known, such information was also recorded.

Results were categorised as:

- clinically significant (defined as 'associated with disease');
- not clinically significant (defined as 'colonising' or 'environmental contaminant'); or
- undetermined (including unknown and uncertain).

Data were forwarded to the study co-ordinators in a standard Excel spreadsheet.

Results

Isolates from 1,447 patients were included in the study. The AM isolates identified in 2000 by the patient's state or territory of residence and the specimen type are shown in Table 1. The majority of isolates (78.7%) were from pulmonary sources. When all AM isolates were considered, the incidence was 7.5 cases per 100,000 population. There were wide variations between the jurisdictions, from a low of 5.5 cases (New South Wales) to 71.3 cases (Northern Territory) per 100,000 population. The isolate was considered associated with disease, for 341 of 1,447 (23.6%) patients, an incidence of 1.8 cases per 100,000 of population (Table 1). Incidence rates for disease in jurisdictions ranged from 0.78 cases (New South Wales) to 4.1 cases (Northern Territory) cases per 100,000 population.

Although almost all isolates from lymph node (98.4%) and soft tissue (91.0%) were clinically significant, only 108 (9.5%) of 1,139 pulmonary isolates were associated with disease. Incidence rates for each of the four categories of site of disease were less than one isolate per 100,000 population. These data suggest that soft tissue infections are more prevalent in Queensland, and lymph node infections are more common in Western Australia and Victoria (Table 2), but these data need to be interpreted with caution.

Table 1. Specimen source of new isolates of atypical mycobacteria by state or territory, Australia, 2000

	Specimen source of isolate				Total	Cases per 100,000 population
	Pulmonary	Lymphatic	Soft tissue	Other		
NSW	290 (10)	7 (7)	10 (9)	53 (25)	**360 (51)**	**5.5 (0.78)**
NT	135 (4)	1 (1)	3 (2)	1 (1)	**140 (8)**	**71.3 (4.1)**
Qld	181 (43)	10 (9)	88 (78)	8 (6)	**287 (136)**	**8.0 (3.8)**
SA	73 (12)	3 (3)	4 (4)	4 (2)	**84 (21)**	**5.6 (1.4)**
Vic.	259 (20)	20 (20)	21 (20)	22 (4)	**322 (64)**	**6.7 (1.3)**
WA	201 (19)	20 (20)	18 (18)	15 (4)	**254 (61)**	**13.4 (3.2)**
Total	**1,139 (108)**	**61 (60)**	**144 (131)**	**103 (42)**	**1,447 (341)**	**7.5 (1.77)**
Cases per 100,000 population	**5.91 (0.56)**	**0.32 (0.31)**	**0.75 (0.68)**	**0.53 (0.22)**	**7.5 (1.77)**	

Numbers in parenthesis refer to cases in which the mycobacterium isolated was judged to be a pathogen.

'Pulmonary' refers to any tissue derived from the lungs (sputum, bronchoscopic collections, biopsies, etc) but not pleurae.

'Lymphatic' refers to any tissue, pus or aspirate clearly associated with a lymph node.

'Soft tissue' refers to skin-associated tissue or pus; bone and joint.

'Other' includes pleural fluid, blood, urine and any isolate from an undefined site.

Since there were few isolates from the Australian Capital Territory, isolations were included with those for New South Wales, and Tasmania was included into that for the testing state (mostly Western Australia).

Table 2. Predominant species identified among disease-associated isolates of atypical mycobacteria

	Specimen source of isolate			
	Pulmonary	**Lymphatic**	**Soft tissue**	**Other**
NSW	*n=10* *M. kansasii* (6) *M. avium* complex (4)	*n=7* *M. avium* complex (6) *M. xenopi* (1)	*n=9* *M. fortuitum* comp (4) *M. chelonae* (2) *M. marinum* (2) *M. abscessus* (1)	*n=25* *M. avium* (18) *M. intracellulare* (6)
NT	*n=4* *M. avium* complex (4)	*n=1* *M. avium* complex (1)	*n=2* *M. fortuitum* (2)	*n=1* *M. avium* complex (1)
Qld	*n=43* *M. avium* complex (24) *M. kansasii* (9) *M. abscessus* (7)	*n=9* *M. avium* complex (8) *M.* species (1)	*n=78* *M. fortuitum* comp (28) *M. marinum* (14) *M. chelonae* (10) *M. abscessus* (8) *M. ulcerans* (7)	*n=6* *M. avium* (4) *M. abscessus* (2)
Vic	*n=20* *M. avium* complex (17) *M. kansasii* (1)	*n=20* *M. avium* complex (18) *M. scrofulaceum* (2)	*n=20* *M. chelonae* (5) *M. haemophilum* (3) *M. ulcerans* (2) *M. abscessus* (2) *M. marinum* (2)	*n=4* *M. avium* (2)
SA	*n=12* *M. avium* complex (12)	*n=3* *M. avium* complex (3)	*n=4* *M. marinum* (2) *M. abscessus* (1) *M. fortuitum* (1)	*n=2* *M. avium* complex (2)
WA	*n=19* *M. avium* complex (12) *M. kansasii* (5)	*n=20* *M. avium* complex (12) *M. haemophilum* (6) *M. malmoense* (1) *M.* species (1)	*n=18* *M. haemophilum* (6) *M. marinum* (4) *M. abscessus* (2) *M. fortuitum* (2)	*n=4* *M. avium* (2) *M. abscessus* (2)

Atypical mycobacteria and site of disease

The *M. avium* complex was the predominant disease-associated atypical mycobacteriosis in pulmonary (73/108, 67.6%) and lymphatic (48/60, 80%) sites. *M. kansasii* was found in 21 of 108, (19.4%) pulmonary samples. The *M. fortuitum* complex, the *M. chelonae* – *M. abscessus* group and *M. marinum* were common isolates from soft tissue (37/131, 28.2%, 31/131, 23.7% and 24/131, 18.3% respectively). *M. ulcerans* was only recovered from soft tissue in Queensland (n=7) and Victoria (n=2) (Table 2).

Age and site of disease for clinically relevant atypical mycobacteria

The age distribution of mycobacterial disease for the categories of pulmonary, lymphatic and soft tissues are presented in Table 3.

Lymphatic disease occurred primarily in children, (50/60, 83%), although 7 of 60 (11.7%) cases were diagnosed in adults greater than 50 years of age. In contrast, no pulmonary disease was found in persons less than 10 years of age. Disease of soft tissue occurred less commonly (9%) in the under 10 years age group.

Isolation of atypical mycobacteria from the respiratory tract

There were 1,139 isolates cultured from the respiratory tract representing 78.7 per cent of all isolates (Table 1). Culture-positive specimens collected at bronchoscopy or by gastric lavage were considered to be of pulmonary origin. Only 108 of 1,139 (9.5%) isolates were considered to be disease associated, implying that over 90 per cent of AM cultured from respiratory specimens were not clinically significant. Members of the *Mycobacterium avium* complex (MAC) were the most common organism isolated accounting for 586 of 1,139 (51.4%) pulmonary isolates (Table 4). Seventy-two of 108 (67.3%) clinically relevant isolates were MAC. There were a small number (n=4) of MAC identified as MAC–X's, a group closely related to, but genetically distinct from *M. avium* and *M. intracellulare*[1] (Table 5).

The Queensland, New South Wales and Western Australian reference laboratories use techniques to identify MAC to species level (*M. avium, M. intracellulare* or MAC–X). The other MRL's use a commercial probe to report the three entities as *Mycobacterium avium* complex and these identifications are described as 'MAC' in the tables. *Mycobacterium intracellulare* was recovered almost twice as often as *M. avium* (ratio of 1.8:1) from pulmonary specimens (data not shown). For clinically relevant isolates identified to species level the ratio was slightly lower (1.5:1) (Table 5). Of the other organisms that were fully identified from pulmonary samples, the 'rapidly growing' mycobacteria and *M. gordonae* appear to be the next most commonly isolated organisms. Only 2 of 164 (1.2%) mycobacteria described as unidentified slowly (or rapidly) growing mycobacteria were disease associated (Table 4).

Smear-positivity and association with pulmonary disease

Smear-positivity was not a reliable indicator of disease in pulmonary samples. Approximately 10 per cent of samples in which disease was not established were smear positive for acid fast bacilli. In contrast, some 50 per cent of cases where the recovered AM was deemed to be associated with disease were smear positive (data not shown).

Table 3. Age distribution for new patients from whom a 'pathogenic'atypical mycobacterium was isolated

Age range	Pulmonary %	Lymphatic %	Soft tissue %
<10 years	0	83	8
10 to 50	23	5	50
>50 years	77	12	42

Table 4. Species or species complexes identified among atypical mycobacteria from pulmonary sources

	State or territory						Total
	NSW	NT	Qld	SA	Vic.	WA	
M. avium complex	164 (4)	47 (4)	106 (24)	37 (12)	119 (16)	113 (12)	**586 (72)**
M. gordonae	28 (0)		1 (0)	1 (0)	48 (0)	18 (0)	**96 (0)**
M. fortuitum complex	26 (0)	5 (0)	8 (1)	1 (0)	25 (0)	8 (0)	**73 (1)**
M. abscessus	13 (0)	1 (0)	20 (7)	1 (0)	5 (0)		**40 (7)**
M. kansasii	6 (6)	1 (0)	10 (9)		8 (1)	6 (5)	**31 (21)**
M. chelonae	8 (0)		4 (0)	1 (0)	8 (0)	1 (0)	**22 (0)**
M. xenopi	5 (0)		1 (0)		3 (0)		**9 (0)**
M. simiae	6 (0)					1 (0)	**7 (0)**
M. asiaticum		6 (0)					**6 (0)**
M. interjectum		1 (0)				3 (0)	**4 (0)**
M. shimoidei	1 (0)	1 (0)			1 (1)	1 (1)	**4 (2)**
M. lentiflavum		1 (0)			1 (0)	1 (0)	**3 (0)**
M. malmoense		1 (0)	1 (0)			1 (1)	**3 (1)**
M. szulgai		1 (0)			1 (1)		**2 (1)**
M. heckeshornense					1 (0)		**1 (0)**
Other *Mycobacterium* sp.	33 (0)	18 (0)	3 (0)		26 (0)	8 (0)	**88 (0)**
Slow-growers (unidentified)		52 (0)	21 (1)	32 (0)	12 (0)	40 (0)	**157 (1)**
Rapid-growers (unidentified)			6 (1)		1 (0)		**7 (1)**
Total	**290 (10)**	**135 (4)**	**181 (43)**	**73 (12)**	**259 (19)**	**201 (19)**	**1,139 (107)**

Numbers in parenthesis refer to cases in which the mycobacterium isolated was judged to be a pathogen

Table 5. Analysis of disease-associated isolates of MAC

	M. intracellulare	*M. avium*	MAC–x
Pulmonary	19	13	4
Lymphatic	15	7	1

Isolation of atypical mycobacteria from soft tissue and other sites

Soft tissue comprises skin, connective tissue, bone and joint but not blood or lymph node. 'Other sites' include pleural fluid, blood, urine and any isolate from an undefined site.

Of the 144 soft tissue isolates, 131 (91.0%) were considered disease associated. For 'other sites', 42 of 103 (40.8%) isolates were considered clinically relevant (Tables 1 and 2).

Queensland reported 88 of all 144 (61.1%) AM isolates from soft tissue, and of these, 78 of 88 (88.6%) were disease associated. *Mycobacterium marinum, M. ulcerans* and the rapidly growing mycobacteria were all isolated more frequently compared with the other jurisdictions. The Northern Territory and South Australia reported the fewest cases of soft tissue disease.

In 2000, *Mycobacterium haemophilum* was isolated only in Western Australia and Victoria, although it has been isolated from the other states and territories previously.

Almost all isolates of rapid growing mycobacteria (e.g. *M. fortuitum complex/ M. chelonae–M. abscessus*) were considered pathogenic. The significance of MAC isolations from other sites was frequently uncertain. *Mycobacterium heckeshornense* was recovered from synovial fluid, blood, and sputum from three different patients in Victoria. It was considered a significant finding in the two sterile sites, but of uncertain significance in sputum.

Isolation of atypical mycobacteria from lymphatic tissue

Most lymphatic disease occurred in children. Almost all (98.4%) AM isolated from lymphatic tissue were considered disease associated, representing an incidence rate of 0.31 isolates per 100,000 population (Table 1). Forty-eight of 60 (80.0%) AM were MAC, and of those that were identified to species level, 15 of 23 (65.2%) were *M. avium*, 7 of 23 (30.4%) were *M. intracellulare*, and there was a single MAC–X strain isolated (Table 5). In Western Australia, *M. haemophilum* was recovered from lymphatic tissue on six occasions (Table 2).

Isolation of atypical mycobacteria from blood

Twenty-nine AM were cultured from blood, and of these 25 of 29 (86.2%) isolates were MAC. Almost half the isolations were from New South Wales. Of the MAC identified to species level, 19 of 20 (95%) isolates were *M. avium* (Table 6). The recently described *M. heckeshornense*[2] was recovered from the blood of a Victorian patient. The HIV status of 12 of 29 (41.4%) patients were known and are shown in bold (Table 6).

Table 6. Isolation of atypical mycobacteria from blood, 2000

	State or territory						
	NSW	NT	Qld	SA	Vic.	WA	Total
M. avium	13		**4**			**2**	**19**
M. intracellulare	1						**1**
MAC		**1**		**2**	**2**		**5**
M. chelonae	1				1		**2**
M. heckeshornense					1		**1**
Rapid grower			1				**1**
Total	**15**	**1**	**5**	**2**	**4**	**2**	**29**

Known HIV positive cases are in bold.

Discussion

The present report, based on laboratory data obtained for the year 2000, is an attempt to describe the association of AM with clinical specimens and clinical disease. Conservatively, in the year 2000, there were 1.8 bacteriologically confirmed cases of disease caused by AM per 100,000 population, compared with 4.0 cases of bacteriologically confirmed tuberculosis per 100,000 population.[3]

In contrast to bacteriological confirmation of tuberculosis, where isolation of the causative agent almost always represents disease, the association between the isolation of AM and clinical disease is less frequent. Australian laboratories undertaking mycobacteriological investigations are increasingly using a broth-based culture system as their primary medium rather than the traditional solid media. The newer systems offer the advantage of reduced time to detection of positive cultures, but they also recover a greater number of AM.[4,5,6,7] Rapid techniques for the preliminary identification of mycobacteria are increasingly available to non-reference laboratories. These tests distinguish between members of the *Mycobacterium tuberculosis* complex and the AM (AccuProbe; Gen-Probe Inc, San Diego, California). A single, AM isolate from a smear negative sputum or urine specimen is, initially of doubtful clinical relevance and as further identification of the AM isolate is not usually warranted, it may not be forwarded to an AMRLN laboratory for additional testing. For this reason the total numbers of recovered AM in this report do not represent all isolations of AM and the calculated rates of AM disease are likely to be under-estimates.

Most AM are ubiquitous, free-living organisms that may be found in a wide variety of environments including soil, dust, air and water. Water appears to be a particularly attractive environment for AM, and they have been recovered from water distribution systems worldwide.[6,7] It is assumed that most people are infected from environmental sources, but isolation of AM from clinical specimens does not necessarily imply disease. The present report found that less than 10 per cent of AM recovered from the respiratory tract was associated with disease and that sputum smear positivity was not a reliable indicator for disease. The American Thoracic Society have developed diagnostic criteria, based on a combination of clinical, radiological and laboratory findings for pulmonary disease caused by AM.[8]

The last AM Survey conducted by the Special Interest Group in 1988 (David Dawson, unpublished data) produced data similar to the current survey. In 1988, there were 334 significant AM cases, with an incidence rate of two cases per 100,000 population. There were 81 pulmonary cases, 88 lymphatic cases but only 51 soft tissue cases. The figures for MAC were also similar in the 1988 survey (70 cases). However, there was a threefold increase in *M. kansasii* in 2000 (21 cases compared to only seven cases in 1988), and twice the number of *M. fortuitum, M. marinum and M. ulcerans* were recovered from soft tissue in 2000. This is probably a reflection of improved technology and skill in recovering AM, rather than an increased infection rate (Table 2).

In an attempt to see if pathogenicity (or lack thereof) could be linked to a particular organism recovered from a pulmonary specimen, data for all slowly growing AM were reviewed (Table 4). *M. gordonae*, recovered from 96 patients, was never associated with pulmonary disease. *M. fortuitum* complex organisms (recovered from 73 patients) were rarely associated with disease. *M. abscessus* (7/40) was more frequently associated with disease than *M. chelonae* (0/22) although the incidence was still low. *M. kansasii*, was usually considered pathogenic (21/31). *M. asiaticum* was isolated only in the Northern Territory and does not appear to be disease associated. Unfortunately the figures are too small to draw conclusions for the other species. As molecular identification methods become more accessible it may be possible to identify species in the many isolates (more than 14% in this survey) that conventional methods cannot identify and to clarify associations between AM species and clinical disease.

Organisms recovered from soft tissue were almost always considered to be clinically significant. There was a higher recovery rate noted from Queensland (61.1% of all isolations) than from other states. *M. haemophilum* was only isolated in Western Australia (from 6 lymph nodes and from 6 soft tissues) and Victoria (3 soft tissue). This is unlikely to be a reflection of geographical differences but differences in

laboratory testing of appropriate tissues for *M. haemophilum*. While reference laboratories routinely culture lymph nodes, bone/joint and skin samples for this pathogen, which has special growth requirements, this however, may not be the case in routine clinical laboratories. Knowledge of *M. haemophilum* ecology is still incomplete but it has been isolated from pulmonary sites in immuno-compromised patients. In a detailed analysis of Australian data covering the period 1977–2000 (presented at the Australian Society for Microbiology's Annual Scientific Meeting in Perth, Western Australia in 2001, by this author) it was shown that *M. haemophilum* was recovered as often from immuno-competent persons as from immuno-compromised (non-HIV) ones, and more often than in HIV+ persons.

The most common isolate from lymphatic tissue was MAC, (48/60, 80%). Not all MAC are fully identified to species level, so complete data for *M. avium* and *M. intracellulare* are unavailable. Where data were available, there were twice as many *M. avium* isolations as *M. intracellulare* (Table 5). *M. avium* accounted for 57 per cent of MAIS complex isolates in 1988, a similar figure (19/29, 65%) to that in 2000.

Atypical mycobacteria cause disease in Australia, with an incidence conservatively estimated at 1.8 cases per 100,000 population. The main sites of disease are in soft tissue, pulmonary and in the lymphatics. Pulmonary AM infections may also present as sputum 'smear positive' requiring further tests to exclude *Mycobacterium tuberculosis*. The *Mycobacterium avium* complex is the most common encountered mycobacteria, isolated from blood, pulmonary and lymphatic sites in both disease and non-disease. The pathogenic rapid growing mycobacteria (*M. fortuitum—M. abscessus—M. chelonae* group) were common in the soft tissue infections.

A better understanding of the ecology and etiology of AM in light of the emerging diversity of the genus (as demonstrated by the number of new species described in the past 10 years) will require reference laboratories to continually improve their laboratory practices and identify clinically relevant isolates to species level.

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Victorian Infectious Diseases Reference Laboratory, North Melbourne, Victoria.

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Annual report of the Australian Gonococcal Surveillance Programme, 2002

The Australian Gonococcal Surveillance Programme

Abstract

The Australian Gonococcal Surveillance Programme (AGSP) monitors the antibiotic susceptibility of *Neisseria gonorrhoeae* isolated in all states and territories. In 2002 the *in vitro* susceptibility of 3,861 isolates of gonococci from public and private sector sources was determined by standardised methods. Antibiotic susceptibility patterns again varied considerably between jurisdictions and regions. Resistance to the penicillins nationally was at 18 per cent but ranged up to 22 per cent in larger urban centres. Quinolone resistance in gonococci (QRNG) remained widespread. Nationally 10 per cent of all isolates were QRNG, and most of this resistance was at high MIC levels. All isolates remained sensitive to spectinomycin. A small number of isolates demonstrated some decreased susceptibility to ceftriaxone (MIC 0.06 mg/L or more) and were concentrated in New South Wales. Patterns of infection were unaltered from previous years. 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* 2003;27:189–195.

Keywords: surveillance, Neiserria gonorrhoeae, *antimicrobial resistance, gonorrhoea, antibiotics, quinolone, penicillin, spectinomycin, cephalosporin*

Introduction

Laboratory analyses can materially assist the control and treatment of gonorrhoea by confirmation of the diagnosis and in provision of antibiotic susceptibility data. The latter activity is crucial as antimicrobial resistance in *Neisseria gonorrhoeae* continues to spread to the detriment of treatment of the individual patient and public health management of gonococcal disease. The public health management of gonorrhoea is based on the use of standardised single dose treatment regimens, the efficacy of which is determined by in vitro resistance monitoring.[1] Since 1979, the Australian Gonococcal Surveillance Programme (AGSP) has monitored the susceptibility to antibiotics of gonococci isolated throughout the country. The AGSP is a collaborative program conducted by reference laboratories in each state and territory and data analysed by the program have been published quarterly from 1981 and annual reports have appeared in Communicable Diseases Intelligence since 1996. This report is based on data obtained during the 2002 calendar year.

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Methods

The AGSP is a component of the National Neisseria Network (NNN) of Australia and comprises participating laboratories in each state and territory (see acknowledgements). 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 gonococci remained unchanged in 2002, continuing efforts are needed to obtain cultures as the increasing use of non-culture-based methods of diagnosis reduces the number of isolates available for testing. Details of the numbers of organisms examined are provided in order to indicate the AGSP 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.[2] The AGSP also conducted a program-specific quality assurance (QA) program.[3] 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.

Results

Numbers of isolates

There were 3,951 gonococcal isolates referred to or else isolated in AGSP laboratories in 2002. The source and site of infection with these isolates are shown in the Table. One-thousand six-hundred and twenty-five gonococci (41% of the Australian total) were isolated in New South Wales, 694 (17.5%) in Victoria, 588 (14.9%) in Queensland, 565 (14.3%) in the Northern Territory, 347 (8.8%) in Western Australia, and 132 (3.3%) in South Australia with small numbers in Tasmania and the Australian Capital Territory. Of these, 3,861 remained viable for susceptibility testing.

Nationally 226 (6%) more isolates were received in 2002 than in 2001. The number of isolates rose by 120 in New South Wales, 105 in the Northern Territory and 50 in Western Australia. In Victoria and South Australia, numbers were stable in 2002. Queensland was the only state with more than a small decrease in numbers tested (5%). Numbers in other centres were low.

Table. Source and number of gonococcal isolates, Australia, 2002, by sex, anatomical site and state or territory*

	Site	NSW	Vic.	Qld	SA	WA	NT	Aust.
Male	Urethra	1,061	477	416	62	245	344	**2,605**
	Rectal	270	96	14	24	8	1	**413**
	Pharynx	145	50	11	17	2	1	**226**
	Other/NS	39	11	17	12	6	6	**91**
	Total	**1,515**	**634**	**458**	**115**	**261**	**352**	**3,335**
Female	Cervix	84	48	121	17	82	191	**543**
	Other/NS	15	12	9	0	4	10	**50**
	Total	**99**	**60**	**130**	**17**	**86**	**201**	**593**
Unknown	**Total**	**11**	**0**	**0**	**0**	**0**	**12**	**23**
Total		**1,625**	**694**	**588**	**132**	**347**	**565**	**3,951**

* Excluding those from the Australian Capital Territory and Tasmania.

The site of isolation and sex of some infected patients was not known.

Source of isolates

There were 3,335 strains from men and 593 from women, with a male to female (M:F) ratio of 5.6:1, similar to that for 2001. The number of strains from men increased by 161 and from women by 86 strains. The M:F ratio was again high in New South Wales (15.3:1) and Victoria (10.5:1) where strains were more often obtained from urban populations. The lower ratios in Queensland (3.5:1), Western Australia (3:1), and the Northern Territory (1.7:1), reflected the large non-urban component of gonococcal disease in those regions. Male rectal and pharyngeal isolates were most frequently found in South Australia (35% of isolates from men), New South Wales (27%) and Victoria (23%). These percentages are higher than in 2001 but also may reflect clinical sampling practices in those states. About four per cent of isolates are shown as being isolated from 'other' or unknown sites. These included nine cases of disseminated gonococcal infection in men in New South Wales, also noted in 2001. Although 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.

Antibiotic susceptibility patterns

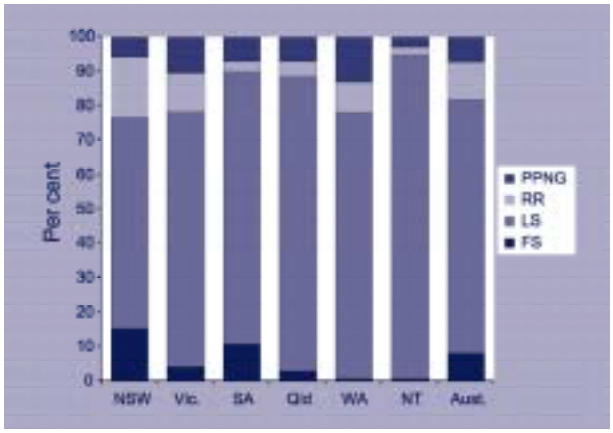
In 2002 the AGSP reference laboratories examined 3,861 gonococcal isolates for sensitivity to penicillin (representing this group of antibiotics), ceftriaxone (representing later generation cephalosporins), ciprofloxacin (representing quinolone antibiotics) and spectinomycin and for high level resistance to tetracycline. As in past years the patterns of gonococcal antibiotic susceptibility differed between the various states and territories. For this reason data are presented by region 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 2002 by penicillin MIC is shown in Figure 1. The MIC reflects the expression of multiple and different chromosomal changes present in an organism.[4] 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 1. Penicillin resistance of gonococcal isolates, Australia, 2002, by region



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

The number (421) and proportion (10.9%) of isolates resistant to penicillin by chromosomal mechanisms in 2002 was lower than the 558 (15.3%) recorded in 2001 but were similar to numbers and proportions seen in 2000.

Strains of this type were concentrated in New South Wales (275 CMRNG, 17 per cent of all isolates) and Victoria (76 CMRNG, 11%). A further increase in CMRNG was noted in Western Australia to 28 (8.5%) from 20 (6.9%) in 2001 and 6 (2%) in 2000. In contrast, the number and proportion of CMRNG in

Queensland (26, 4.6%) decreased markedly from 101 (17.3%) in 2001. In the Northern Territory, 12 strains represented 2.2 per cent of all isolates, similar to the number and proportion seen in 2001. In South Australia, about 3 per cent of isolates were CMRNG.

The number of PPNG isolated in 2002 (274) was identical to that seen in 2001, although as a proportion of all isolates, the 7.1 per cent was a little less than the 7.5 per cent in the previous year. Again the distribution of PPNG differed significantly by region. Western Australia had the highest proportion of PPNG; the 44 isolates representing 13.3 per cent of all gonococci. New South Wales had 94 PPNG (5.8%), Victoria 72 (10.5%), Queensland 39 (6.9%), and South Australia 9 (7.4%). Sixteen PPNG were found in the Northern Territory (3%). Information on the geographic location of the acquisition of PPNG was available in only 98 of the 274 infections and most data were from New South Wales and Western Australia. In both centres local acquisition was prominent. Indonesia, the Philippines, Thailand, Vietnam and China were the most frequently identified countries of probable overseas gonococcal infection. PPNG was also reported from infections acquired in Greece, Hong Kong and Papua New Guinea.

Ceftriaxone

Resistance leading to the third generation injectable agent ceftriaxone that is associated with treatment failure with the 250 mg dose recommended in Australia has yet to be described. In 2001, a small but increasing number of strains in a number of states showed a small increase in ceftriaxone MICs. In 2002, there were 21 gonococci with ceftriaxone MICs > 0.03 mg/L isolated in Australia. Nineteen of these were in New South Wales and there was one each in Victoria and Queensland. Isolates were sporadic of multiple phenotypes and usually resistant to quinolones and penicillins, but spectinomycin sensitive. Isolates with MICs of 0.06 mg/L and above, as determined by the AGSP methodology, are included in this category.

Spectinomycin

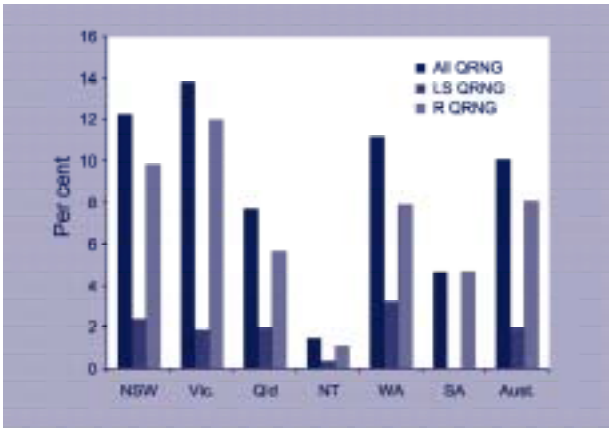
All isolates were susceptible. Resistance most often occurs as a result a single step ribosomal change.

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 lower level of resistance in about 90 per cent of cases, but lower doses of the antibiotic will more often result in treatment failure. At higher levels of resistance i.e. an MIC of 1 mg/L, treatment failure occurs in about 60 per cent of cases. The proportion of treatment failures increases exponentially as MICs rise even if higher dose regimens are used. Currently, gonococci with MICs up to 16 and 32 mg/L are being seen in Australia.

In 2002 a total of 389 (10%) gonococci had some level of resistance to quinolones (Figure 2). This is substantially less than the 638 (17.5%) gonococci QRNG seen in 2001. There has been considerable volatility in QRNG numbers in different patient populations in Australia over recent years. Until 1999, QRNG was particularly concentrated in homosexually active men (HAM) in New South Wales and Victoria. These QRNG seen in HAM were predominantly in the lower MIC range, namely, 0.06–0.5 mg/L. In 2001, QRNG were more widely dispersed through all centres in Australia, had higher MICs, and heterosexual spread was more pronounced. This pattern continued in 2002 and about 80 per cent of all QRNG, equivalent to eight per cent of all gonococci in Australia, had MICs in the higher range (1–32 mg/L) The highest proportion of QRNG was seen in Victoria where 96 QRNG were 14 per cent of the total number of isolates examined. In New South Wales there were 199 QRNG (12.3%), in Western Australia 37 (11%) and in Queensland 43 (7.7%). In other jurisdictions less than five per cent of isolates were QRNG.

Figure 2. Percentage of gonococcal isolates which were less sensitive to ciprofloxacin or with higher level ciprofloxacin resistance and all strains with altered quinolone susceptibility, Australia, 2002, by region



LS QRNG MIC 0.06 – 0.5 mg/L.
R QRNG MIC 1 mg/L or more.

Information on acquisition of QRNG was available in 125 of the 389 cases. In New South Wales 44 infections were acquired locally and 34 were acquired overseas, but in Western Australia only a quarter of cases were acquired locally. Overseas acquisition was from many sources. In addition to those listed above for PPNG acquisition, QRNG were acquired from Bangladesh, Malaysia, Fiji, New Zealand, the United Kingdom and Singapore.

High level tetracycline resistance

The spread of high level tetracycline resistance in *N. gonorrhoeae* (TRNG) is examined as an epidemiological marker even though tetracyclines are not a recommended treatment for gonorrhoea. There was an increase in TRNG isolation in 2002 when 442 (11.4%) strains of this type were detected. In 2001, 343 (9.4 %) TRNG were detected throughout Australia and a similar number and proportion were detected in 2000. Most TRNG were found in New South Wales (209, 12.9% of isolates) where local spread was noted. Western Australia (46, 13.8%) had the highest proportion of TRNG, closely followed by Queensland (77 isolates, 13.6%) and Victoria (91 isolates, 13.2%). Lower numbers and proportions were found in South Australia and the Northern Territory.

Discussion

Treatments for gonorrhoea are based on patterns of susceptibility of prevalent gonococci to recommended antibiotics. The World Health Organization states that once resistance to an antibiotic has reached a level of five per cent, then use of that agent should be discontinued. It is important to ensure that a sufficient number of samples are examined to reliably detect this level of resistance. Although the non-random distribution of antibiotic resistant gonococci makes it more difficult to estimate of the size of the sample required for surveillance purposes, the number of isolates available in Australia in 2002 remained sufficient for the purpose of detecting resistance at the five per cent level. However the sample size needed to detect low numbers of resistant gonococci distributed in a non-random fashion in a gonococcal population is extremely large. The limitations of culture-based diagnosis, especially in remote settings, decreases the number of gonococci available for testing. The AGSP examines all isolates available to its members. The use of non-culture-based methods for the diagnosis of gonorrhoea decreases this sample of gonococcal isolates for testing. A continuing commitment to maintenance of culture-based systems is required, while molecular methods for determining gonococcal antibiotic susceptibility remain problematic.[5]

The wider introduction of non-culture-based diagnostic methods has also meant that analysis of comparative rates and trends in gonorrhoea is now more difficult. However, some important inferences can be drawn from ancillary information obtained by the AGSP, most notably that on sites of infection and the ratio of disease in men and women. In 2002, as in previous years, considerable regional variation in susceptibility of gonococci to antibiotics was observed in Australia. The AGSP has been able to show that a considerable proportion of the gonorrhoea contracted in larger urban centres remains in homosexually active men where antibiotic resistant gonococci are also most often encountered. In contrast, gonorrhoea in rural settings is more often heterosexually transmitted and antibiotic resistance is a lesser current concern[6] while still requiring close attention as resistance patterns

shift. These differences in patterns of gonorrhoea in Australia mean that programmatic and standard treatment regimens are best derived from a consideration of local patterns of susceptibility rather than aggregated national data.

Penicillin resistance continues at a high rate in urban centres in 2002 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 11 and 23 per cent. CMRNG predominated in New South Wales. In Victoria, CMRNG and PPNG were in similar proportions whereas in Western Australia, Queensland and South Australia PPNG was prominant. The proportion of CMRNG in the Northern Territory remains low, but there has been a continuing shift upwards in MICs so that close surveillance was continued to ensure the efficacy of penicillin-based regimens.

Quinolone resistance also continued at higher than acceptable rates in 2002. Despite a continuing decrease in the number and proportion of QRNG in Australia, the percentage remains high at 10 per cent nationally. QRNG are widely dispersed and the majority of QRNG (8% of all gonococci) have MICs in the range 1–32 mg/L. Sustained domestic transmission continued together with the continued importation of QRNG from many sources. The widespread distribution of QRNG in neighbouring countries and noted in WHO based surveillance,[7] is relevant to treatment of individuals who acquire gonorrhoea overseas but present for treatment locally. 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.[8]

Of increasing concern in recent AGSP reports, and reinforced by findings in 2002, has been the appearance of gonococci with decreased susceptibility to third generation cephalosporin antibiotics. While most gonococcal isolates remained fully susceptible to ceftriaxone, the injectable third generation cephalosporin recommended for treatment in Australia, reports from Japan confirmed treatment failures with other oral third generation agents including cefixime.[9] This treatment failure with oral third generation cephalosporins in Japan coincided with the appearance of an increasing number of strains with increased MICs to these agents.[10] The increased cephalosporin MIC was subsequently shown to be associated with an altered *penA* gene in the gonococci.[11] The AGSP obtained examples of the Japanese isolates for comparative purposes and for use in the AGSP QA program. The gonococci encountered in Australia have characteristics similar to those obtained from Japan. It is emphasised in the Japanese reports that, although ceftriaxone is not used for gonococcal treatment in that country, it was believed that if used in appropriate doses, ceftriaxone would effect a cure with infections caused by these strains. 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 NNN has reviewed its methodology for the detection of gonococci with altered susceptibility to third generation cephalosporins and will continue to monitor the situation in Australia closely. Currently, these isolates are concentrated in New South Wales, but experience would suggest that spread to other regions will inevitably follow. If treatment failure of any type of gonorrhoea with any cephalosporin antibiotic is suspected, intense efforts should be made to obtain cultures of the organism for formal susceptibility testing in an NNN laboratory. All gonococci tested in Australia, including those with altered cephalosporin susceptibility, were susceptible to spectinomycin.

It is now well established that both the rates of gonorrhoea and the incidence of antibiotic resistance in *N. gonorrhoeae* continue to increase. One important element of control of gonorrhoea is the programmatic use of optimal antibiotic treatment and these regimens are best determined by use of data from surveillance of resistance patterns such as those derived by the AGSP.

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Paul Southwell, Susan Bradbury and Peter Collignon. Microbiology Department, Canberra Hospital, Garran, Australian Capital Territory.

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Annual report of the Australian Meningococcal Surveillance Programme, 2002

The Australian Meningococcal Surveillance Programme

Abstract

Since 1994,The National Neisseria Network, a nationwide collaborative laboratory program, has examined and analysed isolates of *Neisseria meningitidis* from cases of invasive meningococcal disease in Australia. The phenotypes (serogroup, serotype and serosubtype) and antibiotic susceptibility of 393 isolates of *N. meningitidis* from invasive cases of meningococcal disease were determined in 2002. Most disease was caused by serogroup B (210 isolates, 63%) or serogroup C (162 isolates, 41%) meningococci. An increased number of isolates in Victoria (129 from 78 in 2001) accounted for most of the increased national total. A diversity of phenotypes circulated in the different states and territories. Serogroup B strains predominated in all jurisdictions except Victoria, Tasmania and the Australian Capital Territory and were isolated from sporadic cases of invasive disease. Serogroup B phenotypes B:4:P1.4(7) and B:15:P1.7 were the most common and widely distributed. The common serogroup C phenotype in Victoria, C:2a:P1.4(7), was also common in Tasmania. Elsewhere in Australia it was detected only in low numbers. Other C:2a serosubtypes were prominent in other jurisdictions. About two-thirds of all isolates showed decreased susceptibility to the penicillin group of antibiotics (MIC 0.06 to 0.5 mg/L). Two isolates, one each from Darwin and Sydney, had MICs of 1 mg/L. From 1999, reports have also included diagnoses made by non-culture-based methods in these analyses. Data relating to 187 laboratory-confirmed but culture-negative cases supplemented information on culture confirmed cases in this report. *Commun Dis Intell* 2003;27:196–208.

Keywords: antibiotic resistance, meningococcal disease, Neiserria meningitidis

Introduction

A national laboratory-based program for the examination of isolates of *Neisseria meningitidis* from cases of invasive meningococcal disease (IMD), the National Neisseria Network (NNN), has operated since 1994 through the collaboration of reference laboratories in each jurisdiction. The NNN supplies information on the phenotype (serogroup, serotype and subserotype), and increasingly on the genotype, and the antibiotic susceptibility of meningococci from cases of IMD. Additional data from non-culture-based laboratory testing, derived from nucleic acid amplification assays (NAA) and serological examination, are included in the analyses. These data supplement those from clinical notification schemes. The characteristics of the meningococci responsible for IMD are important both for individual patient management and to tailor the public health response. The prospect of additional vaccines e.g. porin-based vaccines for serogroup B meningococcal disease, increases the need for precise data on circulating meningococcal subtypes.

Annual reports summarising data gathered since the inception of the program were published in *Communicable Diseases Intelligence*.[1–8] The following report analyses the characteristics of meningococci isolated in the calendar year 2002.

Methods

The NNN is a long term collaborative program for the laboratory surveillance of the pathogenic *Neisseria, N. meningitidis and N. gonorrhoeae.*[1–9] A network of reference laboratories in each state and territory (see acknowledgements) undertakes meningococcal isolate surveillance throughout Australia.

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Isolate-based surveillance

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

Phenotyping of invasive isolates of meningococci by serotyping and serosubtyping was based on the detection of outer membrane protein antigens using a standard set of monoclonal antibodies obtained from the National Institute for Public Health, the Netherlands. Increasingly, sequencing of products derived from amplification of the porin genes *porA* and *porB* has been used to supplement and supplant serotyping analyses based on the use of monoclonal antibodies. For the purposes of continuity and comparability, the typing data from both approaches have been unified in the accompanying tables by converting sequence data to the more familiar serotyping/serosubtyping nomenclature.

Antibiotic susceptibility was assessed by determining the minimal inhibitory concentration (MIC) to antibiotics used for therapeutic and prophylactic purposes. This program uses the following parameters to define the various levels of penicillin susceptibility/resistance when determined by a standardised agar plate dilution technique:[9]

sensitive, MIC ≤ 0.03 mg/L;

less sensitive, MIC 0.06 – 0.5 mg/L;

relatively resistant MIC ≥ 1 mg/L.

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

Non-culture-based laboratory-confirmed cases

Additional laboratory confirmation of suspected cases of IMD is increasingly available by means of non-culture-based methods including NAA and serological techniques. NAA testing is essentially by polymerase chain reaction (PCR) techniques[10] and has been progressively introduced in the different jurisdictions. Data from the results of these investigations were included for the first time in the 1999 report. The serological results are based on results of tests performed using the methods and test criteria of the Manchester PHLS reference laboratory, United Kingdom, as assessed for Australian conditions.[11,12,13] Where age, sex and outcome data for patients with non-culture-based diagnoses were available these were also recorded. The site of a sample of a positive NAA is also used to define the clinical syndrome. This separation is not possible for cases diagnosed serologically.

Results

Numbers of isolates from culture-confirmed cases

A total of 393 invasive isolates of meningococci were examined in 2002, 55 more than the 338 isolates examined in 2001, but closely approximating the 388 isolates seen in 2000. There were 129 isolates from patients whose infections were acquired in Victoria (33% of all isolates), 110 in New South Wales (28%), 76 (19%) from Queensland, 35 (9%) from Western Australia, 20 (5%) from Tasmania, 13 (3%) from South Australia, and 5 (1%) each from the Northern Territory and the Australian Capital Territory (Table 1). The increase in the number of isolates in 2002 was principally due to increases in the number of culture positive cases in Victoria, from 77 culture positive isolates in 2001. Slight increases in numbers of isolates compared to 2001 were noted in New South Wales (10 more) Tasmania (4 more) and Western Australia (3 more). In South Australia numbers decreased by 9 to 13 cases in 2002 with slight decreases in the Northern Territory and Queensland (3 and 2 less respectively). Numbers in the Australian Capital Territory remained unchanged from 2001.

Table 1. *Neisseria meningitidis* isolates, Australia, 2002, by state or territory and serogroup

State/territory	Serogroup										Total	
	B		C		A	Y		W135		NG*		
	n	%	n	%	n	n	%	n	%	n	n	%
ACT	1	20.0	4	80.0	0	0	0.0	0	0.0	0	5	1.3
NSW	71	64.6	34	30.9	0	2	1.8	2	1.8	1	110	28.0
NT	4	80.0	1	20.0	0	0	0.0	0	0.0	0	5	1.3
Qld	41	53.9	29	38.2	0	4	5.3	1	1.3	1	76	19.3
SA	9	69.2	4	30.8	0	0	0.0	0	0.0	0	13	3.3
Tas.	6	30.0	14	70.0	0	0	0.0	0	0.0	0	20	5.1
Vic.	47	36.4	72	55.8	0	4	3.1	6	4.7	0	129	32.8
WA	31	88.6	4	11.4	0	0	0.0	0	0.0	0	35	8.9
Total	**210**	**53.4**	**162**	**41.2**	**0**	**10**	**2.5**	**9**	**2.3**	**2**	**393**	**100.0**

* Not viable for serogrouping or not serogroupable

Seasonality

Sixty-six (17%) cases occurred between 1 January and 31 March, 84 (21%) between 1 April and 30 June, 131 (33%) between 1 July and 30 September and 112 (28%) between October and 31 December 2002. A winter peak of meningococcal disease is usual.

Age group

The age distribution of patients infected with invasive isolates in each state or territory is shown in Table 2. Nationally, the peak incidence of meningococcal disease traditionally occurred in those four years and under. Those aged less than one year or in the 1–4 age group accounted for 43 (10.9%) and 65 (16.5%) cases respectively. These numbers are essentially unchanged from 2001 although as a proportion of all cases in these two age groups, is lower than last year. A secondary peak in the 1–19 year age range was substantially increased to 95 (24.2%) from the 54 cases (16%) recorded in 2001. A further 45 cases (11.5%) occurred in those aged 20–24 years. The number (140) and proportion (35.7%) of culture positive cases in the 15–24 year age range in 2002 was considerably greater than the 89 (26%) cases in 2001 but similar to the 126 (32%) cases recorded in 2000.

Serogroup, serotype and serosubtype (phenotype) distribution

The distribution of the isolates by serogroup is shown in Tables 1 and 2. Nationally, 210 serogroup B isolates represented 53.5 per cent of all strains, similar in number, but a smaller proportion compared with culture positive cases in 2001.The 162 serogroup C strains (41.2%) was an increase in the number (122) and proportion (36%) detected in 2001 and also in 2000 (128, 33%). The number of serogroup W135 and serogroup Y strains both increased, compared to 2001 but accounted for a small proportion of cases. No serogroup A isolates were identified.

Some important differences in the distribution of serogroups were evident when data were disaggregated by region. Serogroup B meningococci predominated in national data (53%) and in all jurisdictions except Victoria, Tasmania and the Australian Capital Territory. When examined regionally, Western Australia (89% of isolates), South Australia (69%), the Northern Territory (80%), Queensland (54%) and New South Wales (65%) had high proportions of serogroup B strains. However in Victoria, serogroup B isolates were 36 per cent of the total and in Tasmania 30 per cent. Group B disease comprised mainly unlinked and apparently sporadic cases.

Table 2. *Neisseria meningitidis* isolates, Australia, 2002, by state or territory, serogroup and age*

State/territory		Age group (years)										Total
		<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	NS	
ACT	B	0	0	0	0	1	0	0	0	0	0	**1**
	C	0	0	0	0	1	0	1	2	0	0	**4**
	Total	**0**	**0**	**0**	**0**	**2**	**0**	**1**	**2**	**0**	**0**	**5**
NSW	B	15	15	11	4	9	4	2	6	5	0	**71**
	C	1	4	2	0	9	6	5	4	3	0	**34**
	Total	**16**	**19**	**13**	**4**	**19**	**11**	**8**	**11**	**9**	**0**	**110**
NT	B	1	2	0	0	0	0	1	0	0	0	**4**
	C	0	0	0	0	0	1	0	0	0	0	**1**
	Total	**1**	**2**	**0**	**0**	**0**	**1**	**1**	**0**	**0**	**0**	**5**
Qld	B	7	9	3	2	7	5	6	1	1	0	**41**
	C	0	3	6	2	10	2	5	0	1	0	**29**
	Total	**9**	**12**	**9**	**4**	**18**	**8**	**12**	**2**	**2**	**0**	**76**
SA	B	3	1	0	0	2	0	1	1	1	0	**9**
	C	0	0	0	0	1	2	1	0	0	0	**4**
	Total	**3**	**1**	**0**	**0**	**3**	**2**	**2**	**1**	**1**	**0**	**13**
Tas.	B	0	2	0	0	3	1	0	0	0	0	**6**
	C	0	2	1	0	4	0	4	2	1	0	**14**
	Total	**0**	**4**	**1**	**0**	**7**	**1**	**4**	**2**	**1**	**0**	**20**
Vic.	B	8	10	3	2	14	4	2	3	1	0	**47**
	C	1	8	5	3	22	13	11	5	4	0	**72**
	Total	**10**	**19**	**8**	**5**	**38**	**18**	**14**	**11**	**6**	**0**	**129**
WA	B	4	7	3	2	7	3	3	1	1	0	**31**
	C	0	1	0	1	1	1	0	0	0	0	**4**
	Total	**4**	**8**	**3**	**3**	**8**	**4**	**3**	**1**	**1**	**0**	**35**
Australia	n	43	65	34	16	95	45	45	30	20	0	**393**
	%	10.90	16.50	8.60	4.10	24.20	11.50	11.50	7.60	5.10	0.00	
Serogroup B	n	38	46	20	10	43	17	15	12	9	0	**210**
Australia	%	18.1	21.9	9.5	4.8	20.5	8.1	7.1	5.7	4.3	0.0	**53.5**
Serogroup C	n	2	18	14	6	48	25	27	13	9	0	**162**
Australia	%	1.2	11.1	8.6	3.7	29.6	15.4	16.7	8.0	5.6	0.0	**41.2**
Other	n	3	1	0	0	4	3	3	5	2	0	**21**
Australia	%											**5.3**

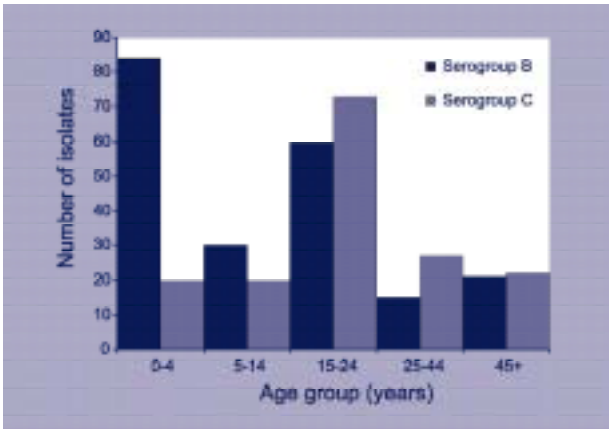
* Includes serogroup B and C data and totals only.

NS Age not stated.

Serogroup C strains were most prominent in the Australian Capital Territory and Tasmania where four of five isolates and 14 of 20 isolates, respectively, were serogroup C. The proportion of serogroup C infections in Victoria increased to 56 per cent and their number almost doubled to 72 from the 38 isolated in 2001. The number (34) and proportion (31%) of serogroup C isolates remained essentially unchanged in New South Wales in 2002. There were 29 group C isolates (38%) in Queensland, four (31%) in South Australia, four (11%) in Western Australia, and one in the Northern Territory.

Serogroup distribution has been typically age-associated, with serogroup B disease concentrated in younger age groups and serogroup C infections predominating in adolescents and young adults. In 2002, 84 (78%) of all isolates in those aged less than four years were serogroup B compared with 20 serogroup C isolates (18%) (Table 2, Figure 1). In those aged 5–14 years, serogroup B meningococcal cultures represented 60 per cent of isolates and serogroup C strains represented 40 per cent. Nationally, serogroup C isolates were more common in all age groups over 14 years (Figure 1) and represented 52 per cent of cases aged 15 years or above.

Figure 1. Number of serogroup B and C isolates, Australia, 2002, by age



However, some jurisdictional differences in the distribution of serogroup B and C meningococcal isolates were again evident in 2002 (Table 2, Figures 2, 3 and 4). In Western Australia and the Northern Territory, serogroup B isolates predominated in all age groups and in all centres serogroup B was more commonly encountered in those four years of age and under. New South Wales, Queensland and South Australia followed the national pattern with regard to age-associated serogroup distribution. In Victoria, serogroup C isolates were especially prominent in older, i.e. adolescent and young adult, age groups but were also seen more often in younger age groups than in other jurisdictions. In Tasmania, 11 of the 14 serogroup C isolates were in patients aged 15 years or more.

Figure 2. Number of serogroup B and C isolates, Victoria, 2002, by age

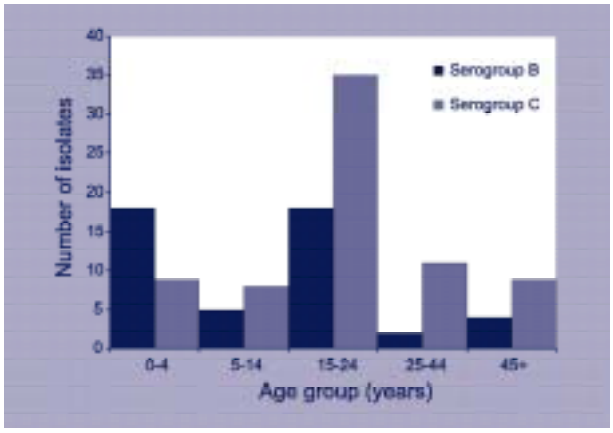


Figure 3. Number of serogroup B and C isolates, New South Wales, 2002, by age

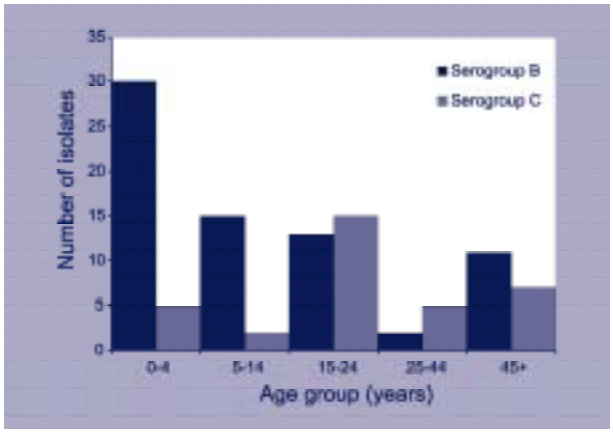
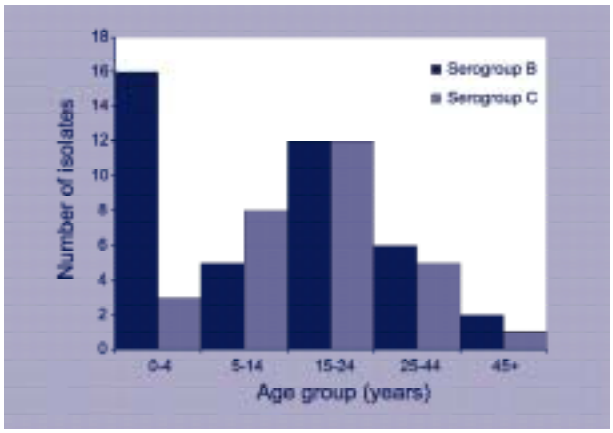


Figure 4. Number of serogroup B and C isolates, Queensland, 2002, by age



There was again considerable phenotypic heterogeneity amongst invasive isolates as determined by serotyping and serosubtyping. The predominant serotypes/serosubtypes in each state and territory are shown in Table 3. Serogroup B meningococci are in general quite heterogeneous, but also more difficult to characterise by serological methods and a number could not be phenotyped. Two main phenotypes circulated in Australia in 2002. The B:4:P1.4(7) phenotype was prominent in New South Wales and Victoria and was also present in Queensland, Western Australia and the Northern Territory. The other main phenotype circulating was B:15:P1.7 and strains were present in Victoria, New South Wales, Queensland, South Australia, and Western Australia. This distribution of serogroup B strains was similar to that in 2001.

Serogroup C meningococci are less diverse that serogroup B strains and nearly all strains were of serotype 2a. Phenotype C:2a:P1.4(7), which appeared in Victoria in 1999, continues to require special comment. There were 10 such isolates in Victoria in 1999, 24 in 2000, 19 in 2001 and 55 in 2002. This phenotype had hitherto been uncommon elsewhere in Australia, but was seen in all other jurisdictions in 2002. Numbers were low however, except for Tasmania. The other common serogroup C phenotypes were C:2a:P1.5 and C:2a:P1.5,2, the former being common in Queensland and New South Wales and present also in Victoria, Tasmania and Western Australia. Victoria and Tasmania also encountered phenotype C:2a:P1.5.10. Serotype 2b strains were not detected.

Site of isolation

There were 71 isolates from CSF either alone or with a blood culture isolate and 311 from blood cultures alone. There were 10 isolates from synovial fluid and one from skin. Trends in relative rates of isolation have been followed in these reports (Figure 2). The ratio of CSF isolates to blood culture isolates was 0.23:1, lower than that recorded in recent years.

Outcome data for cases with sterile site isolates

Outcome data (survived or died) were available for only 147 patients (37%). Twenty-six deaths were recorded in this group (17.6% Table 4). Outcomes were available for 82 (39%) serogroup B infections and 61 (37%) serogroup C infections. There were 9 (11%) deaths in serogroup B infections and 17 (27.8%) in serogroup C infections.

Where outcomes were known, there were four deaths in 25 patients (16%) with meningitis. One of these patients was infected with a serogroup B, and three with a serogroup C strain. Twenty-two deaths were recorded in 122 bacteraemic patients (18%). There were 71 cases of serogroup B meningococcal bacteraemia with eight deaths (11%) and another 47 cases were caused by serogroup C strains among whom 14 fatalities were recorded (29.8%). No fatalities were recorded in serogroup Y (1 case) and W135 (3 cases) bacteraemias.

Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillin

Three hundred and ninety-one isolates were available for determination of their susceptibility to penicillin. Using defined criteria, 127 strains (33%) were fully sensitive to penicillin and 262 (67%) less sensitive (MIC 0.06 to 0.5 mg/L). These proportions are similar to those observed in recent years. Nine isolates had MICs of 0.5 mg/L and two (one each from Darwin and Sydney) had MICs of 1 mg/L. An increase in geometric mean penicillin MICs of *N. meningitidis* has been reported by the AMSP in an examination of strains isolated between 1994 and 1999.[14]

Other antibiotics

All isolates were susceptible to ceftriaxone (and by extrapolation to other third generation cephalosporins) and to the prophylactic antibiotics rifampicin and ciprofloxacin.

Table 3. Commonly isolated serotypes and serosubtypes and phenotypes of *Neisseria meningitidis* of interest, Australia, 2002, by state or territory

State/territory	Serogroup B				Serogroup C			
	Serotype	n	Serosubtype	n	Serotype	n	Serosubtype	n
ACT	4	1	1.4	1	2a	4	1.4	1
NSW	4	32	1.4	22	2a	28	1.5	16
			1.5	2			1.5,2	3
			1.63	1			1.4	3
			1.7	2			1.2	1
			1.14	2			nst	5
			nst	3	nt	6	1.15	1
	15	7	1.7	4			1.16	1
	nt	23	1.14	5			1.2	1
			1.15	3			1.5,2	2
			1.4	3			nst	1
			nst	9				
	1	4	nst	3				
			1.14	1				
NT	4	1	1.7,4	1	2a	1	1.4	1
	14	1	nt	1				
	nt	2	nt	2				
Qld	15	9	1.7	5	2a	26	1.4	4
	4	3	1.14	2			1.5	18
			1.4	1			nst	4
	1	3	1.14	1	26	1	nst	1
			nst	2	nt	1	1.15	1
	nt	26	1.4	12				
			1.6	3				
			1.7	1				
			nst	8				
SA	15	3	1.7	3	2a	3	nst	3
	14	1	nst	1	1	1	1.14	1
	1	1	1.14	1				
	nt	4	1.7	1				
		nst	3					
Tas.	ND	6	ND	6	2a	14	1.7–2,4	9
							1.5,2	4
							1.5–1,10–8	1
Vic.	4	19	1.4	15	2a	70	1.4	55
			1.12,13	1			1.5	2
			1.14	1			1.5,2	5
			1.15	1			1.5,10	7
			1.15,10	1				
	15	12	1.7	11				
			nst	1				
	1	5	1.14	3				
			nst	2				
	2b	2	nst	2				
	nt	8	1.2	5				
WA	15	4	1.7	4	2a	2	1.5	2
	14	3	1.2	1			1.15	1
	4	2	1.4	1			1.4	1
	nt	20	1.4	10				
			1.14	2				
			nst	6				

nt Not typeable.

nst Not serosubtypeable.

ND Not determined.

Numbers and sources of non-culture diagnoses of invasive meningococcal disease in 2002

One hundred and eighty-seven additional cases of IMD were diagnosed by non-culture methods in 2002 (Table 5). In five instances where both serology and PCR testing were performed, both tests were positive. However it was more usual to have samples suitable for testing by only one of the above techniques.

With PCR testing it was also possible to categorise the disease type by source of specimen in a manner similar to that used for culture positive cases (Table 5). Of the 142 cases positive by PCR, 61 were from CSF or CSF and blood, 75 from blood only and 6 from other sites. This is a different distribution from that obtained with culture-based diagnosis. Diagnoses based on blood cultures alone yielded four times the number of isolates derived from culture of CSF. With PCR-based diagnosis, the ratio of diagnoses from blood to CSF positive was 1.2:1. The sources of positive PCR examination other than CSF or blood samples were synovial fluid (5) and pericardial fluid (1). In one case with a positive PCR from joint fluid, a CSF sample from the same patient was also positive and is recorded in the Table 5 under that heading. The pericardial fluid which was positive by PCR was also positive on serology.

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

In addition to diagnosis, PCR can also be used to ascertain the serogroup involved in the disease process. In most centres this is still restricted to serogroup B and C determinations. There were 142 cases where a PCR-based diagnosis was made and in 124 of these the serogroup was also determined (Table 6).

For those 45 cases diagnosed by serology alone (Table 7) age distribution was different with most diagnoses—(40/46)—in those aged 10 years or more. This reflects in part the difficulty in obtaining serum samples from young children. The categorisation of IMD by site of infection cannot be determined by serology. In 2002, an additional serological test to identify serogroup C infections was introduced.[13] This test was requested for 20 patients and was positive, i.e. a serogroup C infection was confirmed, in eight of these.

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

For IMD diagnosed by PCR based tests, the outcome was known in 46 cases, with nine deaths recorded. There were five deaths where blood PCR alone was positive (three of serogroup C and two where the serogroup was not determined). There were four instances of deaths where PCR was positive on a CSF sample, two each with serogroups B and C. Of the 37 cases where survival was recorded, the diagnosis was made on CSF samples in 11 cases due to serogroup B infections and in one case due to serogroup C organisms. For the 24 cases diagnosed as having IMD on PCR of a blood sample, 10 were with serogroup B and six were with serogroup C meningococci. The serogroup was not determined in the remainder. The other case where survival was recorded was in a case of septic arthritis, but the serogroup was not determined.

Table 4. Outcome of meningitic and septicaemic cases of meningococcal infection, culture positive cases, Australia, 2002, by serogroup

Disease type	Outcome	Serogroup					Total
		B	C	Y	W135	NG*	
Meningitis	Survived	10	11	0	0	0	21
	Died	1	3	0	0	0	4
	Total	11	14	0	0	0	25
Septicaemia	Survived	63	33	1	3	0	100
	Died	8	14	0	0	0	22
	Total	71	47	1	3	0	122
All cases	Total	82	61	1	3	0	147
	Died	9	17	0	0	0	26

* NG: Not viable for serogrouping or not serogroupable.

Table 5. Source of non-culture-based diagnosis of invasive meningococcal disease, Australia, 2002

Diagnostic method	Number
All non-culture-based diagnoses	187
PCR positive*	142
CSF PCR positive	53
CSF and blood PCR both positive	8
Blood PCR positive	75
Other†	6
Serology positive in the absence of positive PCR	45

* Including those with positive serology.

† Five joint fluids and one pericardial fluid.

Table 6.Serogroup and age distribution of invasive meningococcal disease diagnosed by polymerase chain reaction, Australia, 2002

Serogroup	Age group (years)										Total
	<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	Unknown	
B	10	14	5	2	15	8	12	6	0	0	72
C	0	1	5	3	13	9	12	6	2	0	51
Y	0	0	0	0	0	0	0	1	0	0	1
ND	0	7	0	1	1	0	1	1	1	6	18
All	10	22	10	6	29	17	25	14	3	6	142

ND serogroup not determined.

Table 7. Age distribution of serologically diagnosed cases of invasive meningococcal disease, Australia, 2002

Serologically diagnosed	Age group (years)										Total
	<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	Unknown	
Cases	1	5	0	4	12	12	8	4	0	0	46

Discussion

The total of 393 isolates examined by NNN laboratories in the Australian Meningococcal Surveillance Programme in 2002 was the highest number recorded since the program commenced in 1994. In 2001, 338 isolates were recovered and the annual numbers prior to this year have ranged between 323 and 388 cases. Disaggregation of the data by jurisdiction provides additional insights into case distribution. The number of isolates available in Victoria increased from 41 in 1998 to 94 in 1999 and 108 in 2000, declined to 77 in 2001 and increased to 129 in 2002. Changes in numbers in other jurisdictions were not marked so that the increase in the number of isolates nationally in 2002, was principally due to increases in Victoria.

Care must be taken before these data on isolation rates are applied to determine trends in disease rates. The number of isolates available for examination will always be less than the number of clinically notified cases because clinical surveillance case definitions allow for inclusion of culture negative cases under certain criteria. The number of culture negative cases will also vary according to the implementation of and adherence to the 'early treatment' practices now advocated for management of IMD. The increasing use of non-culture-based methods has closed this gap between laboratory confirmed and clinically diagnosed cases, but the introduction and use of non-culture-based diagnostic methods has varied in different jurisdictions over time. For these reasons, there should be care taken in comparing trend data from different states and territories in recent years on the basis of laboratory confirmation. The 187 cases confirmed by non-culture-based methods, when added to the 393 positive cultures, saw a total of 580 laboratory confirmed cases in 2002. Provisional data from the National Notifiable Diseases Surveillance System recorded a total of 675 cases of IMD in Australia in 2002.

The ratio of cases of meningitis to bacteraemia in culture confirmed cases (0.23:1) maintained an existing trend (Figure 2) remarked on in previous reports. Differences in meningitis/septicaemia rates were previously noted when these were derived from culture-based and non-culture-based methods. It was observed that the initial introduction of PCR based diagnosis saw positive CSF samples representing 2.5 times the number of diagnoses from blood. In 2002, this ratio was reversed with PCR examinations on blood yielding 1.3 times the rate of diagnoses from CSF. Since 2000, there has been an increasing trend towards positive diagnosis by NAA using blood samples.

The predominant disease pattern throughout the country remained sporadic infection with serogroup B meningococci. However, the proportion of serogroup C cases in aggregated data (41%) increased from 36 per cent in 2001. In 1998 this proportion was 25 per cent. The increase in recent years has been noted particularly in Victoria and Tasmania, but also in Queensland. There was little change in the number and proportion of serogroup C cases in New South Wales, South Australia or Western Australia.

No serogroup A meningococci were isolated and the proportion of serogroup Y and W135 strains increased only slightly.

In recent reports, it has been noted that the age distribution of IMD showed a primary peak in those aged four years or less with a secondary peak in adolescents and young adults. It was further observed that the primary peak (0–4 years) was predominantly with serogroup B infections and that serogroup C infection were more common among the young adults. In 2000, those aged 15–24 years had more IMD infections than those aged less than five years in aggregated data. Larger numbers of cases in young adults in New South Wales and Victoria influenced this pattern. In 2001, the age distribution of IMD was more typical with children aged less than five years the most frequently infected, although in Queensland and Tasmania the highest proportion of cases was still in young adults. In 2002, the proportion of cases among young adults was again higher than among infants. Infections in Victoria accounted for 40 per cent of infections in this age group nationally, most of these (45 of 56) were serogroup C strains. Serogroup B infections accounted for nearly 80 per cent of IMD infections in infants (Figures 1, 2, 3 and 4).

The subtypes of meningococci circulating within Australia have been determined mainly by phenotypic methods, by genotyping systems in Victoria. These data illustrate the similarities and differences in meningococci present in different jurisdictions and trend data show changes in

distributions of these subtypes. Patterns vary with time and influence decisions of public health relevance.[15] One particular phenotype, B:4:P1.4(7), has been associated with hyperendemic disease in New Zealand for many years. A monovalent porin vaccine, specific for this strain, has been developed and is undergoing clinical trials. This phenotype, or those closely related to it, have been present in Australia for some time and in New South Wales represented at least 20 per cent of all isolates. Use of *por* gene sequencing techniques may be required to establish the real incidence of infection due to this subtype throughout Australia. Phenotype B:15:P1.7 continues to be widely distributed. The frequency of C:2a:P1.4(7) strains in Victoria has continued to expand, accounting for much of the increase in serogroup C disease in that state and in 2002 more than 40 per cent of all isolates were of this type. In 2002, the 'Victorian' phenotype was increasingly identified elsewhere in Australia, albeit in low numbers. In Tasmania, it became the dominant phenotype. The phenotypes C:2a:P1.5 and C.2a:P1.5,2 continued to be frequently identified in New South Wales, Queensland and Victoria. Genetic recombination events in meningococci are frequent may manifest themselves as subtypes causing epidemic or hyperendemic IMD. Responses to these events are based on an intimate knowledge of relevant aspects of the organism. If the porin vaccine directed against the New Zealand hyperendemic strain proves to be effective, there may be a place for it in Australia also, given the high rate of infection with both serogroup B and C meningococci with this porin subtype.

Mortality data from this surveillance system needs to be interpreted with caution. Information on outcome was assessable in only 147 (37%) of culture positive cases and the 26 deaths recorded giving a mortality rate of 17.6 per cent, may not accurately represent the true rate. Although a higher mortality for serogroup C infections has been consistently recorded in NNN data, other factors, such as age, and time from onset to presentation and treatment, may also explain the difference in outcomes due to infection with different serogroups.

Penicillin MICs of 1 mg/L were detected in two strains in 2002. Penicillin MICs at or about 1 mg/L would still be expected to respond clinically to the currently recommended dose of penicillin,[16] although correlations between MIC and clinical outcome are difficult to establish because of the fulminant nature of fatal cases of meningococcal disease.

All isolates were susceptible to the third generation cephalosporins and the prophylactic agents rifampicin and ciprofloxacin.

Since 1994, the NNN has examined about 3,000 strains of invasive meningococcal isolates from all states and territories. The continuing evolution and development of laboratory techniques over this period mean that it is not always possible to make comparisons of data gathered in different years. The NNN data are used to supplement information collected separately by clinically based surveillance of IMD. The NNN data remain an essential component of IMD surveillance in Australia as decisions on public health management including vaccine policies are developed. These issues are likely to become increasingly complex. For further details the relevant NNN member in each jurisdiction should be contacted.

Acknowledgments

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

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Foodborne disease in Australia: incidence, notifications and outbreaks. Annual report of the OzFoodNet network, 2002

The OzFoodNet Working Group

Abstract

In 2002, OzFoodNet continued to enhance surveillance of foodborne diseases across Australia. The OzFoodNet network expanded to cover all Australian states and territories in 2002. The National Centre for Epidemiology and Population Health together with OzFoodNet concluded a national survey of gastroenteritis, which found that there were 17.2 (95% C.I. 14.5–19.9) million cases of gastroenteritis each year in Australia.The credible range of gastroenteritis that may be due to food each year is between 4.0–6.9 million cases with a mid-point of 5.4 million. During 2002, there were 23,434 notifications of eight bacterial diseases that may have been foodborne, which was a 7.7 per cent increase over the mean of the previous four years. There were 14,716 cases of campylobacteriosis, 7,917 cases of salmonellosis, 505 cases of shigellosis, 99 cases of yersiniosis, 64 cases of typhoid, 62 cases of listeriosis, 58 cases of shiga toxin producing *E. coli* and 13 cases of haemolytic uraemic syndrome. OzFoodNet sites reported 92 foodborne disease outbreaks affecting 1,819 persons, of whom 5.6 per cent (103/1,819) were hospitalised and two people died. There was a wide range of foods implicated in these outbreaks and the most common agent was *Salmonella* Typhimurium. Sites reported two outbreaks with potential for international spread involving contaminated tahini from Egypt resulting in an outbreak of *Salmonella* Montevideo infection and an outbreak of suspected norovirus infection associated with imported Japanese oysters. In addition, there were three outbreaks associated with animal petting zoos or poultry hatching programs and 318 outbreaks of suspected person-to-person transmission. Sites conducted 100 investigations into clusters of gastrointestinal illness where a source could not be identified, including three multi-state outbreaks of salmonellosis. OzFoodNet identified important risk factors for foodborne disease infection, including: *Salmonella* infections due to chicken and egg consumption, bakeries as a source of *Salmonella* infection, and problems associated with spit roast meals served by mobile caterers.There were marked improvements in surveillance during 2002, with all jurisdictions contributing to national cluster reports, increasing use of analytical studies to investigate outbreaks and 96.9 per cent of *Salmonella* notifications on state and territory surveillance databases recording complete information about serotype and phage type. During 2002, there were several investigations that showed the benefits of national collaboration to control foodborne disease. Sharing surveillance data from animals, humans and foods and rapid sharing of molecular typing information for human isolates of potentially foodborne organisms could further improve surveillance of foodborne disease in Australia. *Commun Dis Intell* 2003;27:209–243.

Keywords: surveillance, foodborne disease, disease outbreak, Salmonella, Campylobacter, Listeria, Yersinia, Shigella, *typhoid*

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Introduction

The World Health Organization recently developed a strategy to address the global issue of food safety.[1] The strategy highlighted that, 'surveillance is the basis for the formulation of national strategies to reduce food-related risks'. Many countries recognise the importance of improving foodborne disease surveillance due to high incidence and increasing spread of foodborne diseases, particularly in outbreaks.[2] While outbreaks may attract media attention and cause community concern, sporadic cases of foodborne disease far outweigh the number associated with outbreaks.[3] In addition, foodborne diseases have a major impact on communities and are increasingly affecting trade.[4]

In 2000, the Commonwealth Department of Health and Ageing (DoHA) established the OzFoodNet to enhance surveillance for foodborne disease.[5] OzFoodNet built upon an 18-month trial of active surveillance in the Hunter region of New South Wales and was modelled on the Centers for Disease Control and Prevention's FoodNet surveillance system (see http://www.cdc.gov/foodnet/).[6,7] The purpose of enhancing surveillance for foodborne disease in Australia was to investigate, describe and understand foodborne disease at the national level to provide better evidence of how to prevent foodborne illness.

The OzFoodNet network consists of epidemiologists specifically employed by each state and territory health department to conduct investigations and applied research into foodborne disease. The Network involves many different organisations, including the National Centre for Epidemiology and Population Health, and the Public Health Laboratory Network. OzFoodNet is a member of the Communicable Diseases Network Australia (CDNA), which is Australia's peak body for communicable disease control. The Commonwealth Department of Health and Ageing funds OzFoodNet and convenes a committee to manage the Network.

This is the second annual report of OzFoodNet and covers data and activities for 2002.

Methods

Population under surveillance

In 2002, the coverage of the network included the entire Australian population, which was estimated to be 19,662,781 persons.[8]

During 2002, OzFoodNet coverage expanded to include the Northern Territory and all of New South Wales. Prior to this, New South Wales had enhanced surveillance only in the Hunter region.

In 2002, the Hunter site continued to operate as a sentinel for foodborne disease occurrence in New South Wales. The Hunter site conducts thorough local investigation and provides a baseline for foodborne disease incidence in New South Wales. In 2002, the population covered by the Hunter site was estimated to be 544,623 persons.

Data sources

Incidence of gastroenteritis

To determine the burden of gastroenteritis in Australia, the National Centre for Epidemiology and Population Health (NCEPH) conducted a cross-sectional survey between September 2001 and August 2002 on behalf of OzFoodNet. A research company used Computer Assisted Telephone Interviews to interview randomly selected individuals from each state and the Northern Territory. The Australian Capital Territory was included in the sample for New South Wales and there was an over sample in the Hunter region. Respondents were asked whether they had diarrhoea or vomiting in the past four weeks, and about the symptoms related to that episode. Interviewers asked people reporting gastroenteritis in the previous month whether they sought medical care, provided a specimen of faeces for testing, were unable to carry out normal daily activities, or missed paid work.

People were considered to have had 'infectious gastroenteritis' if they:

- experienced three or more loose stools and/or two or more vomits in a 24 hour period;
- experienced four or more loose stools and/or three or more vomits in a 24 hour period where they had concomitant respiratory symptoms of respiratory illness; and

- did not have any non-infectious causes, such as pregnancy, medications, chronic illness, or alcohol consumption as a cause for their illness.

The results were analysed using a generalised regression estimator method and jackknife approach to estimation of standard errors (P Bell, Household Surveys Facilities, Australian Bureau of Statistics). Data were weighted by state, age, sex, the number of phone lines in the house and household size.

Estimating the burden of foodborne disease

To estimate the burden of foodborne disease we used Australian data from various sources and adopted the approach taken by Mead, *et al*.[3] OzFoodNet considered 28 'known' bacterial, viral and parasitic pathogens that can cause infectious gastroenteritis. To estimate the community incidence of these pathogens in Australia, data from the National Notifiable Diseases Surveillance System and state surveillance systems, from outbreak investigations in Victoria (Joy Gregory, personal communication, November 2002), from laboratories and from published results of a longitudinal study of gastroenteritis in Australia were used.[9,10,11]

Using these data, the literature and a Delphi assessment of Australian foodborne disease specialists, OzFoodNet estimated the proportion of gastroenteritis that was foodborne for each pathogen.[12] It was assumed that the proportion of gastroenteritis due to foodborne transmission among the 'unknown' agents was the same as for 'known' agents. The estimate of the proportion of foodborne among all these known pathogens was then used as proxy for estimating the proportion of all infectious gastroenteritis that was foodborne.

To account for inherent uncertainty in the data the potential distribution of the estimates were simulated to give credible intervals, similar to Bayesian inferential techniques.[13] OzFoodNet calculated the credible interval of foodborne disease for a 'typical year in Australia–2000'.

Rates of notified infections

All Australian states and territories require doctors and/or pathology laboratories to notify patients with infectious diseases that are important to public health. Western Australia is the only jurisdiction where laboratory notification is not mandatory under legislation, although most laboratories still notify the health department. OzFoodNet aggregated and analysed data on patients notified with the following diseases or conditions, a proportion of which may be acquired from food:

- *Campylobacter* infections;
- *Salmonella* infections;
- *Listeria* infections;
- *Yersinia* infections;
- shiga toxin producing *E. coli* infections and haemolytic uraemic syndrome;
- typhoid; and
- *Shigella* infections.

To compare disease to historical totals, OzFoodNet compared crude numbers and rates of notification to the mean of the previous four years. Where available, numbers and rates of notifications for specific sub-types of infecting organisms were compared to notifications for the previous year.

To calculate rates of notification the estimated resident populations for each jurisdiction for June 2002, or the specified year, were used.[8] Age specific rates for notified infections in each jurisdiction were calculated.

The date that notifications were received was used throughout this report to analyse notification data. These data are similar to those reported to the National Notifiable Diseases Surveillance System, but individual totals may vary with time and due to different approaches to analysis.

Gastrointestinal and foodborne disease outbreaks

OzFoodNet collected information on gastrointestinal and foodborne disease outbreaks that occurred in Australia during 2002. The reports collate summary information about the setting where the outbreak occurred, the month the outbreak occurred, the aetiological agent, the number of persons affected, the type of investigation conducted, the level of evidence obtained and the food vehicle responsible. To summarise the data, OzFoodNet categorised the outbreaks by aetiological agents, food vehicles and settings where the outbreak occurred. Data on outbreaks due to transmission from animals and cluster investigations were also summarised.

Risk factors for infection

To identify risk factors for foodborne infection in Australia, OzFoodNet reviewed summary data from outbreaks that occurred in 2002 and compared them to previous years. Data from several complementary OzFoodNet studies of foodborne illness in Australia were also examined.

Surveillance evaluation and enhancement

To identify areas where improvements to surveillance are critical, OzFoodNet compared the results of surveillance across different sites, including rates of reporting outbreaks, and investigation of clusters of *Salmonella*. To measure how well jurisdictions conducted surveillance for *Salmonella* OzFoodNet examined the completeness of information contained on state and territory databases in 2002. The proportion of notifications with serotype and phage type information were compared with results for the previous two years.

Results

Incidence of gastroenteritis

During the 12 months between September 2001 and August 2002, 11.2 per cent (683/6,096) of respondents reported gastroenteritis in the previous month. The overall weighted incidence of gastroenteritis was 0.92 (95% C.I. 0.77–1.06) cases per person per year. This equated to 17.2 (95% C.I. 14.5–19.9) million cases each year. About a third of cases resulted in either the person with gastroenteritis, or a carer of the sick person missing some work. After weighting, this equates to approximately 6.5 million lost days of work due to gastroenteritis annually.

The crude incidence of gastroenteritis was similar in all jurisdictions, except for the Northern Territory where it was markedly higher (Table 1). The survey identified that children reported the highest incidence followed by 20–40-year-old adults. Older persons and teenagers reported less gastroenteritis. The median duration of an episode of illness was two days. Gastroenteritis accounted for about 45 million days of illness each year in Australia.

People with more severe gastroenteritis were more likely to seek treatment. Over 20 per cent of persons with gastroenteritis visited a doctor for treatment and 19 per cent of these persons provided a faecal specimen. After weighting, there were an estimated total of 4.6 million visits to a health facility and 3.7 million visits to a doctor in Australia in one year. About 40 per cent of cases reported taking at least one medication for their illness. Pain killers were the most common medication taken during illness. After weighting, OzFoodNet estimates that 7.0 million persons take at least one medication each year for gastroenteritis, which includes prescription and medications purchased over the counter at pharmacies.

Table 1.Crude incidence of gastroenteritis in Australia,September 2001 to August 2002, by state or territory

Jurisdiction	Number surveyed	Number with gastroenteritis	Crude incidence (%)
New South Wales	1,031	111	10.3
Northern Territory	862	137	16.3
Queensland	825	81	9.6
South Australia	781	91	11.3
Tasmania	843	88	10.2
Victoria	895	91	9.9
Western Australia	859	84	9.8
Total	**6,096**	**683**	**11.2**

Burden of foodborne disease

Of the 28 potentially foodborne pathogens considered only 20 were considered relevant in Australia. The other eight pathogens either did not cause gastroenteritis, or were not locally acquired or transmitted by food. OzFoodNet estimated that 'known' enteric pathogens cause approximately 5 million cases of gastroenteritis each year in Australia. After considering data for these 'known' pathogens from the literature, outbreaks and expert opinion, it was estimated that the credible interval for the proportion of episodes caused by enteric pathogens in food was between 24 per cent and 40 per cent (mid point 32%). From this, it is conservatively estimated that the number of cases of foodborne illness in Australia in a typical year is between 4–6.9 million cases (mid point 5.4 million cases). Among the 'known' pathogens, pathogenic *Escherichia coli*, noroviruses, *Campylobacter* and *Salmonella* contributed the largest number of cases of foodborne gastroenteritis each year.

Rates of notified infections

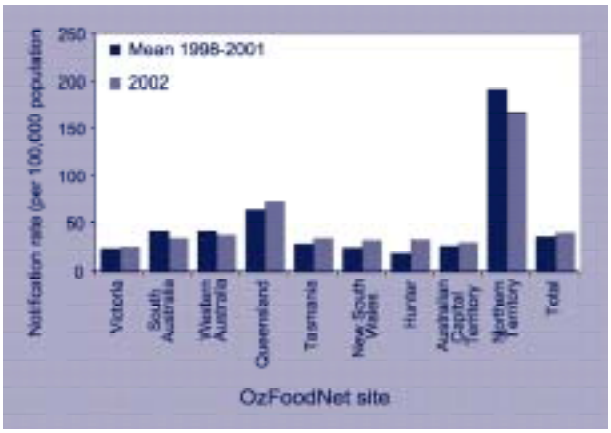
In 2002, OzFoodNet sites reported 23,434 notifications of eight diseases that were potentially foodborne. This was a 7.7 per cent increase from the mean of 21,761 notifications for the previous four years. Reports for these eight diseases make up almost a quarter of notifications to the National Notifiable Diseases Surveillance System.[14] A summary of the number and rates of notifications by OzFoodNet sites is shown in Appendix 1.

Salmonella *infections*

In 2002, OzFoodNet sites reported 7,917 cases of *Salmonella* infection, which equated to a rate of 40.3 cases per 100,000 population. This rate of notification represented an increase of 9.7 per cent over the mean rate for the previous four years (Figure 1). The rate of *Salmonella* notification in OzFoodNet sites ranged from 24.8 cases per 100,000 population in Victoria to 166.7 cases per 100,000 population in the Northern Territory.

Overall, notification rates of salmonellosis for 2002 were increased in the Hunter (62.2%), New South Wales (32.7%), Tasmania (21.5%), the Australian Capital Territory (15.1%), Queensland (12.4%) and Victoria (6.3%) compared to historical means. There were moderate declines in the notification rate of *Salmonella* in South Australia (–19.5%), the Northern Territory (–14.1%), and Western Australia (–10.3%).

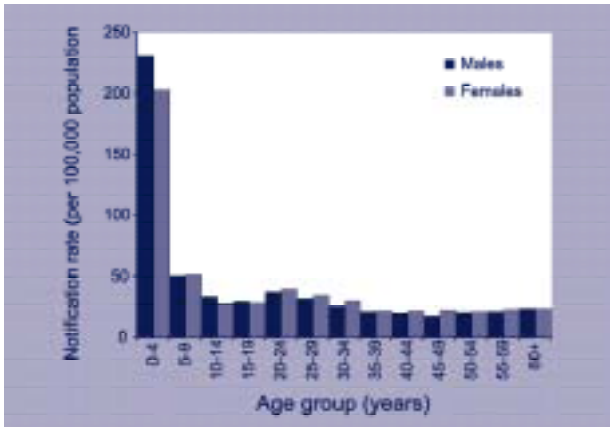
Figure 1. Notification rates of *Salmonella* infections for 2002 compared to mean rates for 1998–2001, by OzFoodNet site



OzFoodNet sites reported that the ratio of males to females was approximately 1:1, and ranged from 1.3:1 in the Northern Territory to 0.8:1 in the Hunter. The median age of cases ranged between 17and 26 years at all OzFoodNet sites, except for the Northern Territory and Queensland where the median ages were 1 and 7 years respectively. There were no major changes in the median ages of salmonellosis cases from 2001 to 2002.

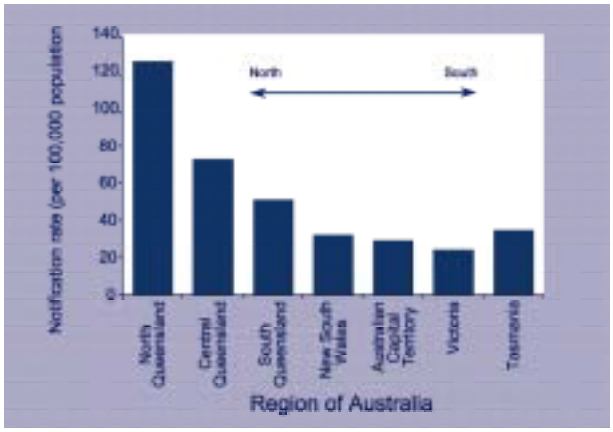
The highest rate of *Salmonella* infection was 230.4 cases per 100,000 population in 0–4-year-old males (Figure 2). The rate was highest in this age group for all sites and ranged from 83.4 cases per 100,000 population in Victoria to 1,421.8 cases per 100,000 population in the Northern Territory. Notification rates were elevated in the 5–9 year age group in all jurisdictions. In all jurisdictions there was also a secondary peak in notification rates in the 20–29 year age range for males and females, which was particularly noticeable in Tasmania.

Figure 2. Age specific notification rates of salmonellosis, Australia, 2002



Rates of salmonellosis were highest in northern areas of Australia, with the highest rate in the Kimberley region.[9,14] Western Australia reported that the Kimberley region had a rate of 332 cases per 100,000 population, which was a 40 per cent decline from the rate reported in 2001. Thirty-nine per cent (128/330) of *Salmonella* notifications in the Northern Territory were in persons of Aboriginal or Torres Strait Island origin. OzFoodNet sites reported that notification rates of salmonellosis increased from south to north along the eastern seaboard of Australia (Figure 3).

Figure 3. Rates of *Salmonella* notifications in selected regions of eastern Australia, 2002, by date of notification



Notifications were analysed by date of receipt at the health department. Rates were directly standardised to the Australian Bureau of Statistics estimated resident population for Australia in 2002. Estimated resident populations for Queensland were from the Australian Bureau of Statistics 2001 Australian Census.

During 2002, there were 704 notifications of *Salmonella* Typhimurium 135 (including 135a) to OzFoodNet sites making it the most common infection (Table 2). This compared to 636 notifications of this phage type last year. There were 604 notifications of *S.* Typhimurium 9, which has been a common phage type for many years. In 2002, Western Australia experienced a significant increase of *S.* Typhimurium 9 and had the highest total number of notifications for this phage type for all jurisdictions. *S.* Typhimurium 126 continued to emerge as a significant new phage type around Australia, which followed a large outbreak in South Australia in 2001. *S.* Typhimurium 170 also continued to increase in Queensland, New South Wales and Victoria. In 2002, there was a significant decrease in numbers of *S.* Typhimurium 64 from previous years. There were 382 cases of *S.* Saintpaul, making it the most common *Salmonella* serovar following *S.* Typhimurium.

Certain *Salmonella* serovars traditionally occupy localised niches in specific geographical areas in Australia. During 2002, *Salmonella* Birkenhead was the third and fourth most common serovar in Queensland and New South Wales respectively. This elevated notification rate reflects an endemic focus of *Salmonella* Birkenhead in northern New South Wales and south-eastern Queensland. In Tasmania, *S.* Mississippi, which is rarely reported elsewhere in Australia, made up 48 per cent (79/165) of *Salmonella* notifications. The notification rate for *S.* Mississippi in Tasmania was 16.7 notifications per 100,000 population. Similarly, in the Northern Territory, S. Ball made up 14.8 per cent of *Salmonella* notifications with a rate of 24.7 cases per 100,000 population. This was the highest specific rate for a *Salmonella* subtype in any OzFoodNet site.

In total, OzFoodNet sites conducted 75 investigations into clusters and point source outbreaks of salmonellosis during 2002. A source of infection was identified for 40 per cent (30/75) of these investigations.

Table 2. Numbers, rates and proportions of the top 10 *Salmonella* infections, 2001 to 2002, by OzFoodNet site*

OzFoodNet site	*Salmonella* type (serovar/phage type)	Top 10 infections					
		2002 n	2002 rate†	Proportion %‡	2001 n	2001 rate	Ratio§
Australian Capital Territory	Typhimurium 9	17	5.3	17.7	10	3.2	1.7
	Typhimurium 135	9	2.8	9.4	2	0.6	4.5
	Typhimurium 197	7	2.2	7.3	0	0.0	–
	Bovismorbificans 24	6	1.9	6.3	0	0.0	–
	Potsdam	4	1.2	4.2	0	0.0	–
	Typhimurium 170	4	1.2	4.2	0	0.0	–
	Typhimurium U290	3	0.9	3.1	0	0.0	–
	Typhimurium 64	3	0.9	3.1	2	0.6	1.5
	Stanley	3	0.9	3.1	5	1.6	0.6
	Adelaide	3	0.9	3.1	0	0.0	–
Hunter	Montevideo	22	4.0	12.3	1	0.2	22.0
	Typhimurium 9	14	2.6	7.8	3	0.6	4.7
	Typhimurium 135	13	2.4	7.3	15	2.8	0.9
	Agona	9	1.7	5.0	1	0.2	9.0
	Potsdam	9	1.7	5.0	2	0.4	4.5
	Typhimurium U290	8	1.5	4.5	3	0.6	2.7
	Typhimurium 170	7	1.3	3.9	6	1.1	1.2
	Virchow 34	5	0.9	2.8	0	0.0	–
	Typhimurium 64	5	0.9	2.8	9	1.7	0.6
	Chester	4	0.7	2.2	1	0.2	4.0
	Javiana	4	0.7	2.2	0	0.0	–
	Singapore	4	0.7	2.2	0	0.0	–
	Typhimurium 195	4	0.7	2.2	0	0.0	–
	Typhimurium 197	4	0.7	2.2	0	0.0	–
	Typhimurium U307	4	0.7	2.2	0	0	–
New South Wales	Typhimurium 9	262	3.9	12.2	132	2.0	2.0
	Typhimurium 135	196	3.0	9.1	201	3.1	1.0
	Typhimurium 170	151	2.3	7.0	35	0.5	4.3
	Birkenhead	89	1.3	4.1	89	1.4	1.0
	Typhimurium 126	64	1.0	3.0	97	1.5	0.7
	Typhimurium 197	61	0.9	2.8	1	0.0	61.0
	Montevideo	59	0.9	2.7	4	0.1	14.8
	Bovismorbificans 24	55	0.8	2.6	1	0.0	55.0
	Typhimurium 135a	50	0.8	2.3	41	0.6	1.2
	Potsdam	44	0.7	2.0	10	0.2	4.4

Table 2 continued. Numbers, rates and proportions of the top 10 *Salmonella* infections, 2001 to 2002, by OzFoodNet site*

OzFoodNet site	*Salmonella* type (serovar/phage type)	Top 10 infections					
		2002 n	2002 rate†	Proportion %‡	2001 n	2001 rate	Ratio§
Northern Territory	Ball	49	24.7	14.9	30	15.2	1.6
	Saintpaul	18	9.1	5.5	17	8.6	1.1
	Chester	17	8.6	5.2	12	6.1	1.4
	Litchfield	16	8.1	4.9	8	4.0	2.0
	Anatum	13	6.6	4.0	9	4.6	1.4
	Muenchen	12	6.1	3.6	19	9.6	0.6
	Typhimurium 135	9	4.5	2.7	9	4.6	1.0
	Agona	6	3.0	1.8	0	0.0	–
	Hvittingfoss	6	3.0	1.8	1	0.5	6.0
	Reading	6	3.0	1.8	6	3.0	1.0
Queensland	Virchow 8	279	7.5	10.2	183	5.0	1.5
	Saintpaul	227	6.1	8.3	173	4.8	1.3
	Birkenhead	136	3.7	5.0	134	3.7	1.0
	Typhimurium 170	138	3.7	5.1	20	0.6	6.9
	Hvittingfoss	114	3.1	4.2	53	1.5	2.2
	Aberdeen	112	3.0	4.1	81	2.2	1.4
	Typhimurium 135	110	3.0	4.0	143	3.9	0.8
	Chester	84	2.3	3.1	68	1.9	1.2
	Typhimurium 9	80	2.2	2.9	50	1.4	1.6
	Waycross	68	1.8	2.5	34	0.9	2.0
South Australia	Typhimurium 8	56	3.7	13.6	3	0.2	18.7
	Typhimurium 126	40	2.6	9.7	110	7.3	0.4
	Typhimurium 99	26	1.7	6.3	4	0.3	6.5
	Typhimurium 108	25	1.6	6.1	31	2.1	0.8
	Typhimurium 9	24	1.6	5.8	49	3.3	0.5
	Typhimurium 145	19	1.2	4.6	0	0.0	–
	Typhimurium 126	17	1.1	4.1	15	1.0	1.1
	Typhimurium 12a	15	1.0	3.6	12	0.8	1.3
	Typhimurium 135a	15	1.0	3.6	13	0.9	1.2
	Typhimurium 135	13	0.9	3.2	24	1.6	0.5

Table 2 continued. Numbers, rates and proportions of the top 10 *Salmonella* infections, 2001 to 2002, by OzFoodNet site*

OzFoodNet site	*Salmonella* type (serovar/phage type)	Top 10 infections					
		2002 n	2002 rate†	Proportion %‡	2001 n	2001 rate	Ratio§
Tasmania	Mississippi	79	16.7	47.9	98	20.8	0.8
	Typhimurium 135	20	4.2	12.1	5	1.1	4.0
	Potsdam	14	3.0	8.5	0	0.0	–
	Typhimurium 9	11	2.3	6.7	11	2.3	1.0
	Typhimurium 126	4	0.8	2.4	1	0.2	4.0
	SaintPaul	3	0.6	1.8	2	0.4	1.5
	Newport	3	0.6	1.8	1	0.2	3.0
	Muenchen	3	0.6	1.8	1	0.2	3.0
	Agona	2	0.4	1.2	2	0.4	1.0
	Niarembe	2	0.4	1.2	0	0.0	–
	Typhimurium 197	2	0.4	1.2	0	0.0	–
	Typhimurium U290	2	0.4	1.2	1	0.2	2.0
Victoria	Typhimurium 135	177	3.6	21.2	92	1.9	1.9
	Typhimurium 170	162	3.3	19.4	72	1.5	2.3
	Typhimurium 9	152	3.1	18.2	127	2.6	1.2
	Typhimurium 126	61	1.3	7.3	16	0.3	3.8
	Saintpaul	43	0.9	5.2	10	0.2	4.3
	Typhimurium U290	39	0.8	4.7	4	0.1	9.8
	Typhimurium 4	21	0.4	2.5	80	1.7	0.3
	Infantis	21	0.4	2.5	27	0.6	0.8
	Potsdam	19	0.4	2.3	8	0.2	2.4
	Aberdeen	15	0.3	1.8	3	0.1	5.0
	Enteritidis 4b	15	0.3	1.2	2	0.0	7.5
Western Australia	Typhimurium 135	65	3.4	8.9	89	4.7	0.7
	Typhimurium 9	45	2.3	6.2	18	0.9	2.5
	Saintpaul	42	2.2	5.8	45	2.4	0.9
	Chester	34	1.8	4.7	31	1.6	1.1
	Enteritidis 4b	28	1.5	3.8	3	0.2	9.3
	Muenchen	27	1.4	3.7	26	1.4	1.0
	Typhimurium 135a	27	1.4	3.7	17	0.9	1.6
	Typhimurium 141	20	1.0	2.7	9	0.5	2.2
	Anatum	18	0.9	2.5	15	0.8	1.2
	Typhimurium U290	14	0.7	1.9	4	0.2	3.5
	Senftenberg	14	0.7	1.9	15	0.8	0.9

* Where there were multiple tenth ranking *Salmonella* types all data have been shown, giving more than 10 categories for some sites.

† Rate per 100,000 population.

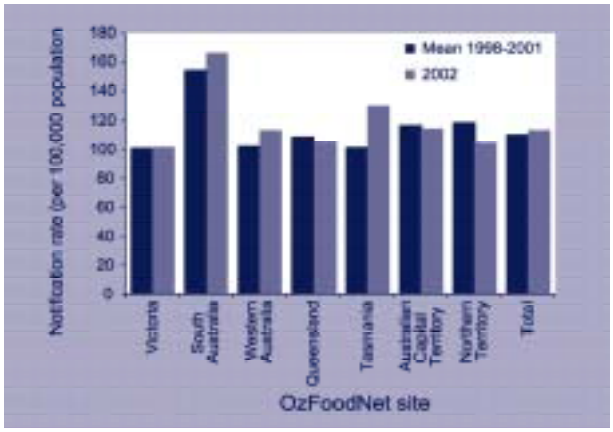
‡ Proportion of total *Salmonella* notified for this jurisdiction in 2002.

§ Ratio of the number of reported cases in 2002 compared to the number reported in 2001.

Campylobacter *infections*

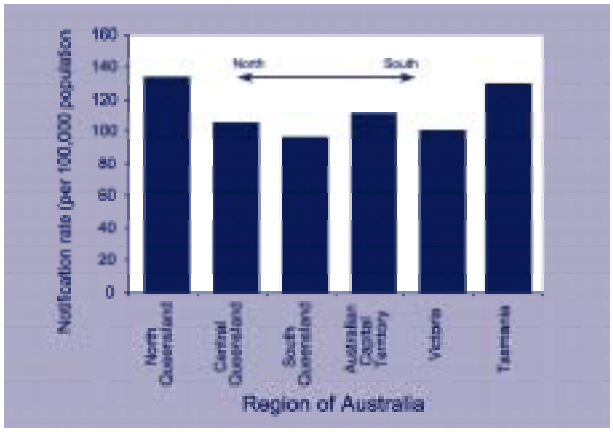
Data for campylobacteriosis were not available for New South Wales, including the Hunter Health Area. With this exception, in 2002 OzFoodNet sites reported 14,716 cases of *Campylobacter* infection, which equated to a rate of 113 cases per 100,000 population.[2] This rate represented a 5.8 per cent increase over the mean for the previous four years (Figure 4). The increase was consistently observed in each quarter of 2002, with the highest rates in spring.

Figure 4. Notification rates of *Campylobacter* infections for 2002 compared to mean rates for 1998-2001, by site excluding New South Wales



Rates of campylobacteriosis increased in Tasmania (27.1%), Western Australia (10.3%), and South Australia (7.5%). Rates were similar to historical means for Victoria, the Australian Capital Territory and Queensland. The Northern Territory experienced a 9.5 per cent decline from historical reports. Geographically, there was no trend in increasing or decreasing rates of notification of *Campylobacter* infection with latitude along the eastern seaboard, in contrast to the pattern observed for *Salmonella* infections (Figure 5). The highest rate of *Campylobacter* infection was 165.7 notifications per 100,000 population in South Australia and the lowest rate was 101.2 notifications per 100,000 population in Victoria.

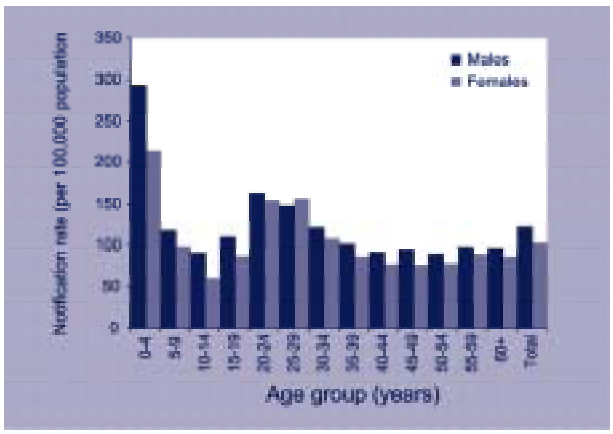
Figure 5. Rates of *Campylobacter* notifications in selected regions of eastern Australia, 2002, by date of notification



Notifications were analysed by date of receipt at the health department. Rates were directly standardised to the Australian Bureau of Statistics estimated resident population for Australia in 2002. Estimated resident populations for Queensland were from the Australian Bureau of Statistics 2001 Australian Census.

The overall ratio of male to females was 1.2:1. All sites, except Tasmania, reported a slight predominance of males amongst notified cases, with male to female ratios ranging from 1.1:1 in Queensland to 1.5:1 in the Northern Territory. The median ages of cases ranged from 17 to 30 years. The highest age specific rates were in male children in the 0–4 year age group, with a secondary peak in the 20–29 year age range for males and females (Figure 6). The highest age specific rates were in males in the 0–4 year age group in the Northern Territory (518 cases per 100,000 population) and South Australia (473 cases per 100,000 population). The lowest rates in the 0–4 year age group was in Tasmanian female children (128 cases per 100,000 population).

Figure 6. Age specific notification rates of campylobacteriosis, Australia, 2002

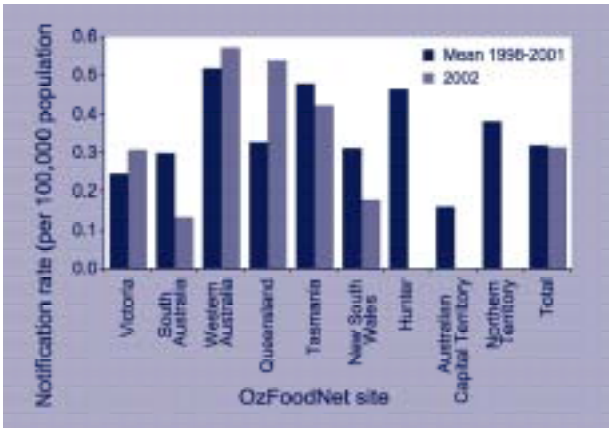


There was only one investigation of *Campylobacter* during 2002 where a source was identified, which occurred in a community-wide increase in Far North Queensland. Thirty-three per cent (68/208) of notified cases in the Northern Territory were in persons of Aboriginal or Torres Strait Island descent.

Listeria

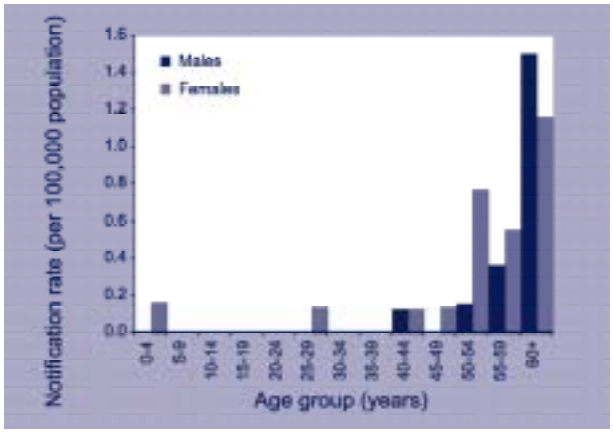
OzFoodNet sites reported 62 cases of listeriosis in 2002, which represents a notification rate of 0.3 cases per 100,000 population (Figure 7). This was a slight increase of 1.2 per cent in the same number of notifications compared to the historical mean. Western Australia (0.6 cases per 100,000 population) had the highest notification rate amongst OzFoodNet sites, which was followed by Queensland (0.5 cases per 100,000 population). There were no common source outbreaks of listeriosis detected during the period, although sites investigated several instances of temporal clustering of cases identified using Pulsed Field Gel Electrophoresis (PFGE).

Figure 7. Notification rates of *Listeria* infections for 2002 compared to mean rates for 1998–2001, by OzFoodNet site



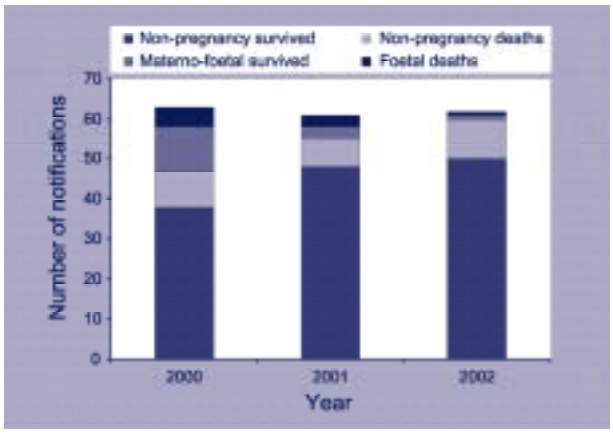
Ninety-seven per cent (60/62) of infections during 2002 were reported in persons who were either elderly and/or immunocompromised. More cases among females were notified during 2002, with the male to female ratio being 0.8:1. OzFoodNet sites reported that the median ages of non-pregnancy associated cases were between 60–86 years. The highest age specific rate of 1.5 cases per 100,000 population was in males over the age of 60 years (Figure 8). There was one notification of listeriosis in a 20-day-old female in Victoria and environmental transmission was suspected. Seventeen per cent (10/60) of non-pregnancy associated cases died.

Figure 8. Age specific notification rates of non-pregnancy associated listeriosis, Australia,2002



Sites reported two maternal foetal *Listeria* infections during 2002, which equated to a rate of 0.8 cases per 100,000 births.* The foetus or neonate died in one of these cases. There was a substantial decline in the number of materno-foetal infections in the three years between 2000 and 2002 (Figure 9).

Figure 9. Notifications of *Listeria* showing non-pregnancy related infections and deaths and materno-foetal infections and deaths, Australia, 2000 to 2002



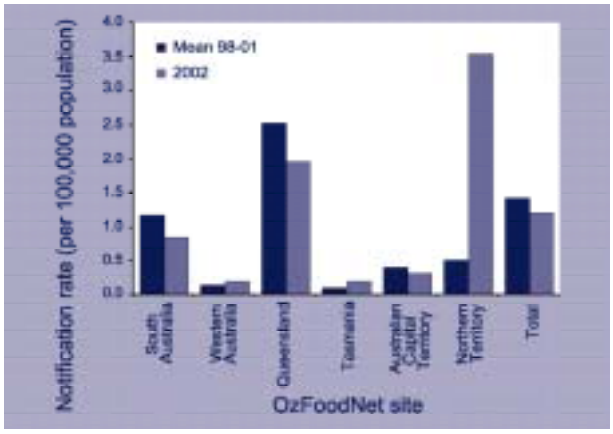
* Births data from the Australian Institute of Health and Welfare National Perinatal Statistics Unit for 1999 and includes live births and foetal deaths.[15]

Yersinia

The CDNA agreed to stop reporting notifications of *Yersinia* infections to the National Notifiable Diseases Surveillance System, as of January 2001. The main reason for this was the apparent decline in incidence and lack of identified outbreaks. In May 2001, the Victorian Government revised regulations governing reporting of infectious diseases, at which time they removed yersiniosis from the list of reportable conditions. *Yersinia* is also not notifiable in New South Wales. No other Australian jurisdictions have amended their legislation to remove yersiniosis from lists of reportable conditions.

In 2002, OzFoodNet sites reported 99 cases of yersiniosis, which equated to a rate of 1.2 notifications per 100,000 population (Figure 10). The overall rate declined 15.1 per cent from previous years, when adjusted for the absence of reporting from Victoria and New South Wales. The Northern Territory recorded seven cases of yersiniosis during 2002, giving a rate of 3.0 cases per 100,000 population. This was the highest rate nationally and considerably higher than historical levels in this jurisdiction. The reasons for the increase were unclear, although laboratory practices in the Territory did not change during 2002 (personal communication, Gary Lum, Royal Darwin Hospital, 21 May 2003).

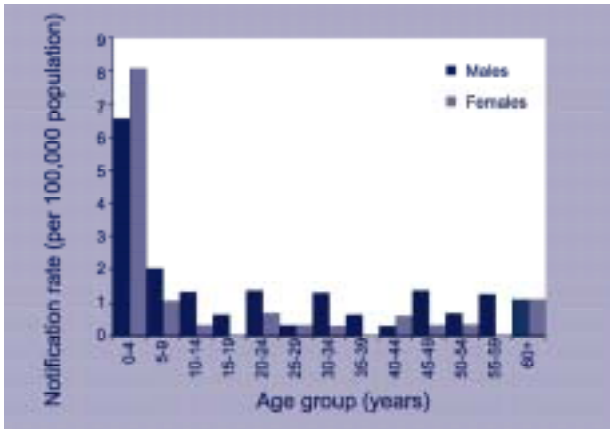
Figure 10. Notification rates of *Yersinia* infections for 2002 compared to mean rates for 1998–2001, Australia excluding Victoria and New South Wales, by OzFoodNet site



Queensland reported 74 per cent (73/99) of all cases, which equated to a rate of 2.0 cases per 100,000 population. The rates of yersiniosis were similar in all three Queensland health zones, and ranged from 1.6 to 2.5 notified cases per 100,000 population.

Overall there was a predominance of notifications in males, with the male to female ratio being 1.4:1. In the two jurisdictions with the majority of cases—South Australia and Queensland—infections in males were more common than in females, with male to female ratios of 2.3:1 and 1.4:1 respectively. Despite this, the highest age specific rate of notification (8.1 cases per 100,000 population) was in females in the 0–4 year age group (Figure 11). The Northern Territory (35.0 cases per 100,000 population) and Queensland (12.5 cases per 100,000 population) reported the highest rates in this age group of females.

Figure 11. Age specific notification rates of *Yersinia* infections, Australia excluding Victoria and New South Wales, 2002

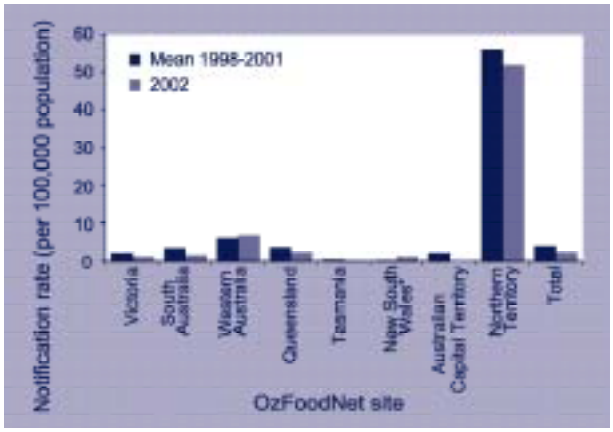


The decrease in *Yersinia* notifications has been occurring for several years and has been observed in other countries. They may be due to changes in laboratory testing practices or a true decline in incidence. Despite the low rates of this disease, it is important for health agencies to continue surveillance for yersiniosis due to its potential for foodborne spread and to monitor the effect of zoonotic control programs.

Shigella

OzFoodNet sites reported 505 cases of shigellosis during 2002, which equated to a notification rate of 2.6 cases per 100,000 population (Figure 12). This was a 38 per cent decrease in the rate of notification compared with historical averages, after adjusting for the introduction of notifications from New South Wales in January 2001.

Figure 12. Notification rates of *Shigella* infections for 2002 compared to mean rates for 1998–2001, by OzFoodNetsite

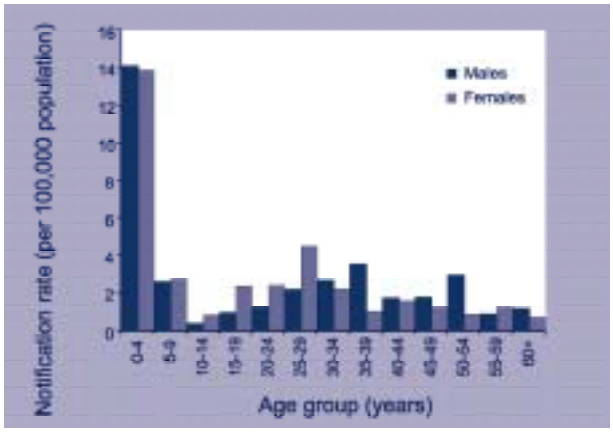


* Shigellosis became notifiable in New South Wales from 2001 onwards.

The highest rate of notification was in the Northern Territory (52 cases per 100,000 population), which was 20 times higher than the overall Australian rate. Eighty-seven per cent (90/103) of notifications in the Northern Territory were in persons of Aboriginal or Torres Strait Island origin. Only Western Australia observed an increased rate compared to the four years mean, the majority of which was related to an increase in cases in the fourth quarter of 2002 from remote areas of the state.

The male to female ratio of shigellosis cases was 1:1. The highest age specific rates were in males (14.1 cases per 100,000 population) and females (13.9 cases per 100,000 population) in the 0–4 year age group, with secondary smaller peaks in the 25–29 year age group for females and the 35–39 year age group for males (Figure 13). There were no reported outbreaks of shigellosis or confirmed links with food. In Australia, the majority of shigellosis infections are thought to be due to person-to-person transmission, or are acquired overseas.

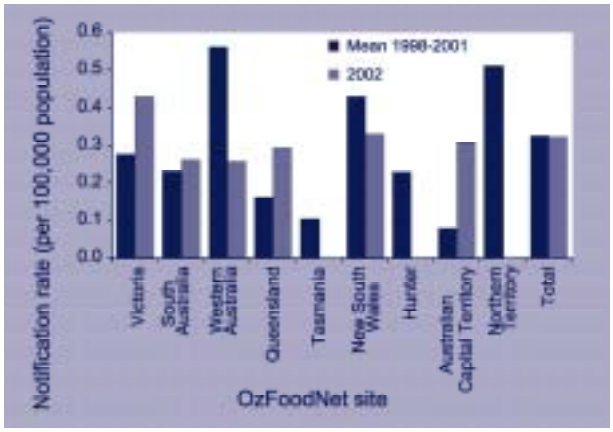
Figure 13. Age specific notification rates of shigellosis, Australia, 2002



Typhoid

OzFoodNet sites reported 64 cases of typhoid infection during 2001, equating to an overall notification rate of 0.3 cases per 100,000 population (Figure 14). The number of notifications was similar to previous years. The highest rate was reported in Victoria (0.4 cases per 100,000 population). Tasmania, the Northern Territory and the Hunter sites did not report any cases.

Figure 14. Notification rates of typhoid infections for 2002 compared to mean rates for 1998–2001, by OzFoodNet site



Where travel status was known, sites reported that 95 per cent (54/57) of cases of typhoid had recently travelled overseas (Table 3). Thirty-seven per cent (20/54) of these cases had recently travelled from Indonesia or Bali and the predominant phage types were D2 (6 cases) and E2 (4 cases). Nineteen cases had travelled to India or the subcontinent and the predominant phage type of *S. Typhi* was E1a (12 cases). The three non-travelling cases were either long-term carriers or infected by close contact with a known carrier. Travel status was unknown for seven cases. Information on phage type was reported for 66 per cent (42/64) of isolates.

Table 3. Travel status for typhoid cases, Australia, 2002

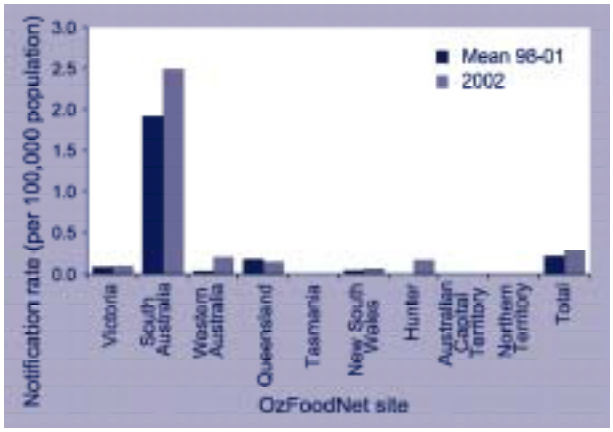
Country	Number of cases	Predominant phage types*
Indonesia	18	E2 (4), D2 (5)
Syria/Lebanon	2	
India	9	E1a (7)
Papua New Guinea	6	E4 (1), D2 (4)
Bangladesh	5	E1a (2)
Pakistan	5	E1a (3)
Kenya/Sudan	2	A (2)
Bali	2	D2 (1)
Samoa	1	E1a (1)
Philippines	1	
Malaysia	1	E4 (1)
Italy	1	
Carrier	1	
Infected by carrier	2	
Travel on ship to high risk areas	1	
Unknown	7	
Total	**64**	

* Numbers in parentheses represent number of cases infected by the phage type. Note that other phage types may have caused disease in returned travellers but are not shown here.

Shiga toxin producing E. coli *infections*

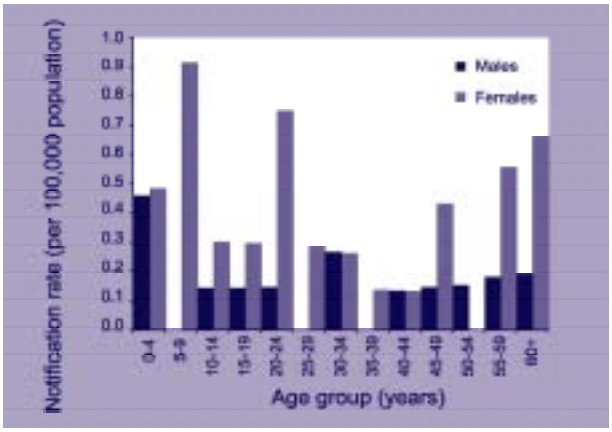
OzFoodNet sites reported 58 cases of shiga toxin producing *E. coli* (STEC) infection during 2002 (Figure 15). This number does not include cases of haemolytic uraemic syndrome where a toxigenic *E. coli* was isolated. The notification rate of 0.3 cases per 100,000 population was a 28 per cent increase over the mean rate for previous years. South Australia (38 cases) reported the majority of cases. All sites reporting cases had an increase in the number of cases notified, except for Queensland where there was a 14 per cent decrease from previous years. There were no cases reported from Tasmania, the Australian Capital Territory or the Northern Territory during 2002.

Figure 15. Notification rates of shiga toxin producing *E. coli* infections for 2002 compared to mean rates for 1998–2001, by OzFoodNet site



The male to female ratio of cases was 0.3:1 and the highest rates were in 4–9 and 20–24-year-old females (Figure 16). The reason for the strong predominance of females amongst notified cases is unknown. The highest rate was in South Australia, which reported 2.5 notifications per 100,000 population.

Figure 16. Age specific notification rates of shiga toxin producing *E. coli* infections, Australia, 2002



The majority of cases in South Australia were detected by polymerase chain reaction (PCR) and no typing details were available (Table 4). *E. coli* O157 was the most common serotype, making up 34 per cent of notifications. This represented an 82 per cent increase in reports of this serotype from the previous year. None of these *E. coli* O157 isolates were the H7 subtype, although H type was rarely reported. There were six notifications of *E. coli* O26 making it the second most common serotype. There were no cases of *E. coli* O111 notified during 2002.

The marked difference in notification rates between states and territories is a result of the practices that pathology laboratories use to screen faecal specimens for toxin producing *E. coli*. The different tests employed in reference laboratories account for the distribution of *E. coli* serotypes. Some laboratories predominantly use PCR testing and never culture, which means that a high proportion of notified cases are not definitively identified to the serotype level. South Australia has the most intensive testing regime and test bloody stool (both microscopic and macroscopic) for the presence of the genes coding for production of shiga toxin. Faecal specimens testing positive are then tested using specific PCR assays for virulence characteristics and specific *E. coli* serotypes. Queensland tests bloody faecal specimens using an enzyme linked immunosorbent assay test kit to detect the presence of shiga toxin. Positive faecal specimens are then tested for STEC using specific PCR tests. Laboratories in most other Australian jurisdictions only test for STEC on request from a doctor or in outbreak settings.

All of the cases appeared to be sporadic, except for one outbreak of *E. coli* O26 associated with animal contact in South Australia.

Table 4. Infecting subtypes of shiga toxin producing *E. coli* causing diarrhoea, Australia, 2001 to 2002

Organism type	Total 2002	Total 2001
O157	16	8
O26	6	5
O157:H–	3	1
Other *E. coli* serotype*	3	4
O113	2	1
O157 other H type	1	1
O157:H7	0	1
Untypable	1	2
Not typed†	23	20
Unspecified	3	6
Total	**58**	**49**

* Includes positive reports obtained by PCR that designated specimens as “non-O157 non-O111”.

† South Australia reported 96% (22/23) of not typed E. coli, which were PCR positive where no culture was obtained or serotyped.

Haemolytic uraemic syndrome

There were 13 cases of haemolytic uraemic syndrome reported during 2002, corresponding to an overall rate of 0.1 case per 100,000 population. New South Wales reported seven of these cases, three of which were notified in the Hunter. Victoria reported four cases, and Queensland and the Northern Territory both reported one case each (Figure 17).

The male to female ratio of cases was 0.7:1 and the highest rate of infection was in females in the 30–34 year age group (0.3 cases per 100,000 population). Sites reported that STEC were isolated for 46 per cent (5/13) of cases (Table 5). Three cases were due to the O157 serotype, making it the most common. There was one case of haemolytic uraemic syndrome due to *E. coli* O157:H7 during 2002.

Figure 17. Numbers of notifications of haemolytic uraemic syndrome, Australia, 1998 to 2002, by month of notification and OzFoodNet site

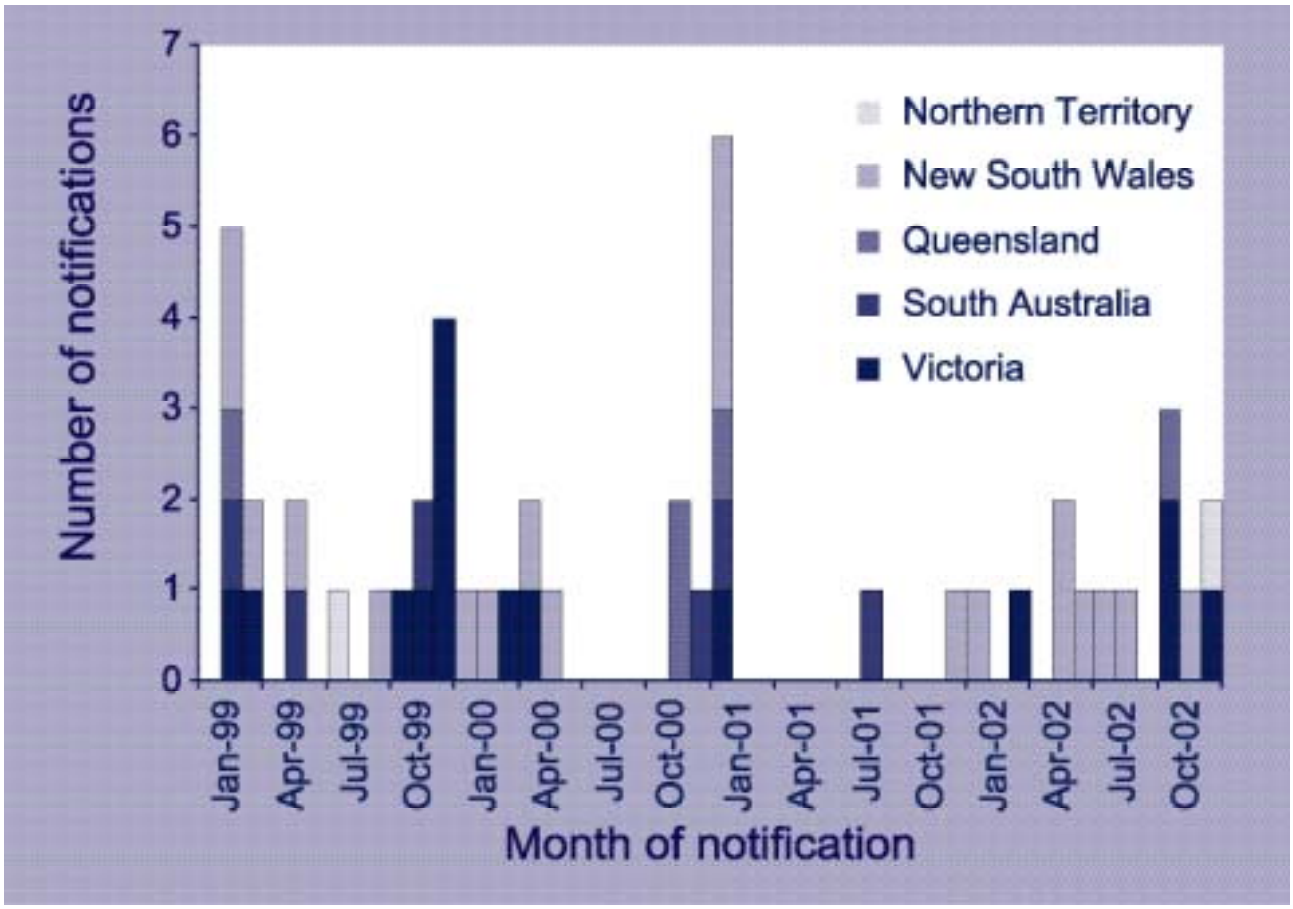


Table 5. Notifications of haemolytic uraemic syndrome and infecting subtypes of shiga toxin producing *E. coli*, Australia, 2001 to 2002

Organism type	Total 2002	Total 2001
O157	1	0
O157:H–	1	0
O157:H7	1	0
O157 other serotype	0	1
Other *E. coli* serotype	2	0
Untypable	0	2
Unspecified	7	5
No toxigenic *E. coli* – clinical diagnosis only	1	0
Total	**13**	**8**

Gastrointestinal and foodborne disease outbreaks

During 2002, OzFoodNet sites reported 513 outbreaks of gastrointestinal illness affecting 11,791 persons. Ninety-two of the outbreaks were due to transmission from contaminated food or water giving an overall rate of 4.7 outbreaks per million population. Three outbreaks were due to contact with infected animals.

The aetiology of the remaining outbreaks was either difficult to determine or were likely person-to-person transmission. Sites conducted 100 investigations into clusters where the mode of transmission was not determined, or a foodborne source was not identified. Person-to-person transmission was suspected as the cause of 318 outbreaks affecting 8,203 persons. The majority of these outbreaks occurred in aged care facilities and hospitals, and were due to norovirus.

Foodborne disease outbreaks

In 2002, 92 foodborne disease outbreaks affected 1,819 persons, hospitalised 103 persons and caused two deaths (Table 6). Appendix 2 shows a summary description of each outbreak.

Victoria reported the largest number of outbreaks (26/92, 28.3%), followed by New South Wales (23/92, 25%). The reporting rates of foodborne outbreaks for different OzFoodNet sites ranged from 1.5 per million persons in Western Australia to 31.2 per million persons in the Hunter. The Australian Capital Territory and the Northern Territory did not report any outbreaks with a foodborne mode of transmission during 2002. The majority of outbreaks occurred in summer and autumn (Figure 18). There was a peak in December relating to pre-Christmas functions, which was also observed in 2001.

Figure 18. Outbreaks of foodborne disease, Australia, 2001 to 2002

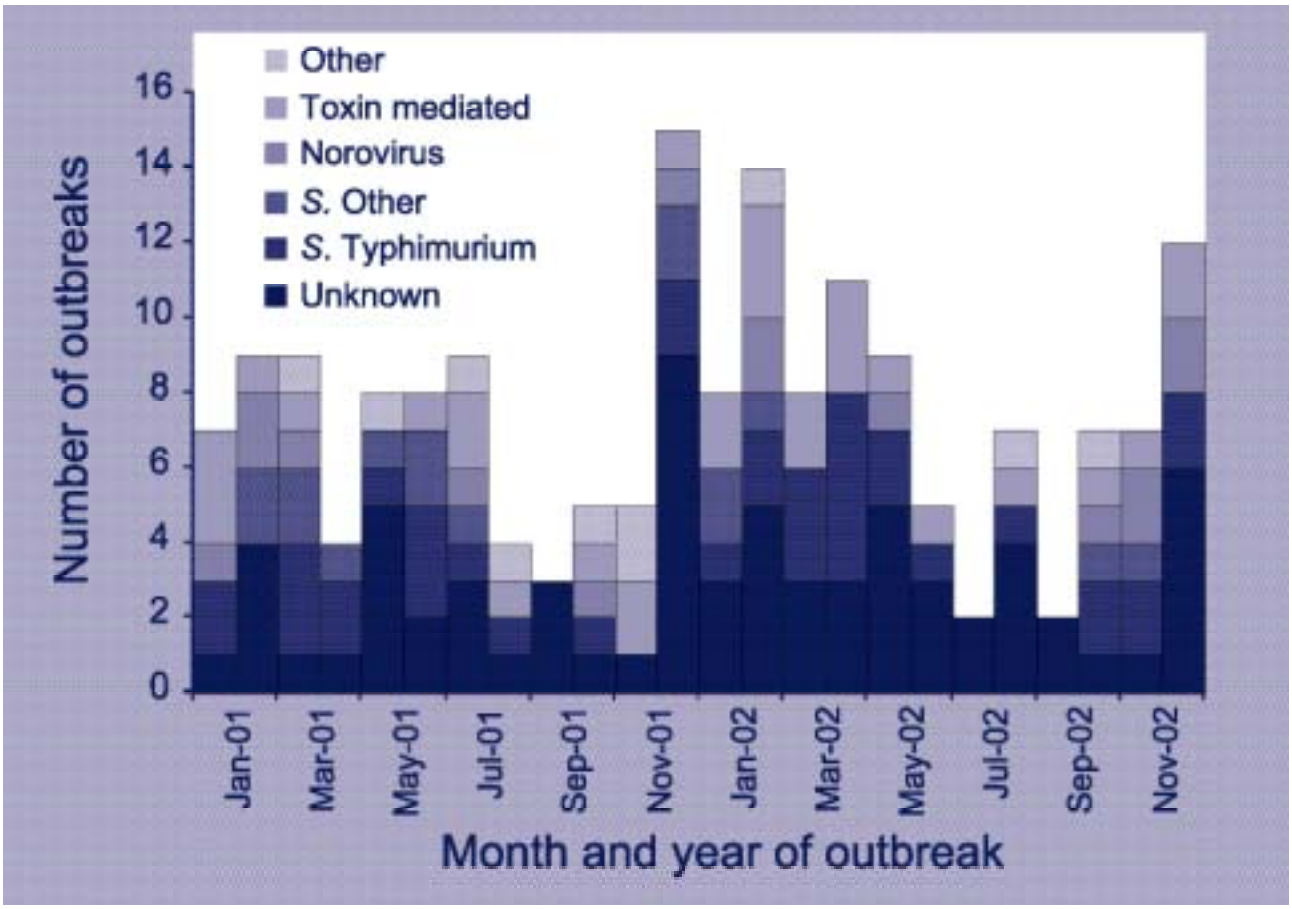


Table 6. Outbreaks of foodborne disease in Australia, 2002, by OzFoodNet site

State	Number of outbreaks	Number affected	Hospitalised	Deaths	Mean number of cases per outbreak
Hunter	17	143	8	0	8.4
New South Wales	23	404	21	0	17.6
Queensland	18	205	18	1	11.4
South Australia	4	113	22	1	28.3
Tasmania	1	5	3	0	5.0
Victoria	26	859	29	0	33.0
Western Australia	3	90	2	0	45.0
Total	**92**	**1,819**	**103**	**2**	**20.0**

Table 7. Aetiological agents responsible for foodborne disease outbreaks showing number of outbreaks and numbers of persons affected, Australia, 2002

Agent category	Number of outbreaks	Number affected	Hospitalised	Deaths
S. Typhimurium	21	471	61	2
C. perfringens	8	155	1	0
Norovirus	8	378	3	0
Salmonella other	5	72	7	0
Ciguatera	4	14	7	0
S. aureus	2	15	1	0
B. cereus	1	37	0	0
Campylobacter	1	24	6	0
Hepatitis A	1	8	0	0
Mixed toxins	1	272	13	0
Suspected wax esters	1	10	0	0
V. parahaemolyticus	1	2	0	0
Unknown	38	361	4	0
Total	**92**	**1,819**	**103**	**2**

Aetiological agents

The most common agent responsible for foodborne disease outbreaks was *Salmonella*, which was responsible for 28 per cent (26/92) of outbreaks (Table 7). These outbreaks affected a total of 543 persons with a hospitalisation rate of 13 per cent (68/543). *S.* Typhimurium was responsible for 81 per cent (21/26) of *Salmonella* outbreaks. Two fatalities were reported from two separate outbreaks of *S.* Typhimurium. There was only one outbreak of campylobacteriosis which affected 24 persons, and one small outbreak of *Vibrio parahaemolyticus*.

There were 16 outbreaks of toxin related illness during 2002. The most common was due to *Clostridium perfringens* (8 outbreaks). There were four outbreaks due to ciguatera fish poisoning, all of which were small (median of 3 persons). Ciguatera fish poisoning had the highest hospitalisation rate of 50 per cent (7/14). There were three outbreaks due to *Staphylococcus aureus*, one of which was a large outbreak in which *Bacillus cereus* was also identified. *B. cereus* was responsible for two outbreaks both involving rice meals.

There were nine outbreaks of known viral aetiology, eight of which were due to norovirus. These outbreaks of norovirus affected 378 persons, but only 0.8 per cent (3/378) were hospitalised. The other outbreak of viral illness was due to hepatitis A and affected eight persons.

There was one outbreak of gastroenteritis suspected to be due to wax esters from escolar or oilfish marketed under the name of rudderfish. Thirty-eight (41%) outbreaks were of unknown aetiology; these affected 361 persons and four cases were hospitalised.

Food vehicles

There was a wide variety of foods implicated in outbreaks of foodborne disease during 2002 (Table 8), although investigators could not identify a source for 34 per cent (31/92) of outbreaks. Fish, poultry and mixed foods were implicated in six outbreaks each. There were six outbreaks associated with red meat and a further six outbreaks associated with seafood. Five outbreaks were associated with eggs. There were two outbreaks associated with Vietnamese pork/beef rolls and two associated with kebabs.

Outbreaks involving cream filled cakes, egg dishes and fish had hospitalisation rates of 20 per cent or higher. Two outbreaks of salmonellosis, one associated with cream cakes and the other with a raw egg dish, resulted in two fatalities.

Table 8. Categories of food vehicles implicated in foodborne disease outbreaks, Australia, 2002

Vehicle category	Number of outbreaks	Number affected	Hospitalised	Deaths
Fish	6	26	7	0
Mixed foods	6	345	22	0
Poultry	6	57	8	0
Red meat/meat products	5	101	0	0
Dessert	4	71	2	0
Cream filled cake	3	61	12	1
Pizza	3	17	0	0
Rice dishes	3	46	1	0
Seafood	3	68	2	0
Suspected egg dishes	3	27	2	1
Suspected seafood	3	12	0	0
Egg dishes	2	23	8	0
Salad dishes	2	99	15	0
Sauces	2	38	0	0
Soup	2	23	0	0
Kebabs	2	49	5	0
Vietnamese rolls	2	52	8	0
Asian foods	1	12	1	0
Bean dish	1	132	1	0
Sandwiches	1	12	0	0
Suspected red meat/meat products	1	4	0	0
Unknown	31	544	9	0
Total	**92**	**1,819**	**103**	**2**

Outbreak settings

The most common setting for the occurrence of outbreaks was at restaurants (43%), followed by the home (13%), takeaway venues (11%), and events catered for by professional companies (8%) (Table 9). There were two outbreaks in community settings. Five outbreaks were due to foods purchased from bakeries, two of which were Asian bakeries. There were four small outbreaks (median size: 5 persons) associated with national franchised fast food outlets. There were two outbreaks each at schools, childcare centres, cruises and community fairs.

Investigative methods and levels of evidence

States and territories investigated 28 outbreaks using retrospective cohort studies and nine outbreaks using case control studies. Fifty per cent (14/28) of outbreak investigations using cohort studies were of unknown aetiology. Twenty-one per cent (6/28) of investigations using cohort studies were *Salmonella* outbreaks. Fifty per cent of *C. perfringens* outbreak investigations used cohort studies. Sixty-seven per cent (6/9) of outbreak investigations using case control studies were due to *Salmonella*. The remaining 55 outbreaks relied on descriptive information to attribute a foodborne cause or identify a food vehicle.

To attribute the cause of the outbreak to a specific food vehicle, investigators obtained analytical evidence from epidemiological studies for 12 outbreaks. Microbiological evidence of contaminated food was found in eight outbreaks, with a further eight outbreaks investigations obtaining both microbiological and analytical evidence. Investigators obtained analytical and/or microbiological evidence for 52 per cent (14/27) of *Salmonella* outbreaks. Seventy-two per cent (66/92) of outbreaks relied on descriptive evidence to implicate a food or foodborne transmission.

Table 9. Categories of settings for foodborne disease outbreaks, Australia, 2002

Setting category	Number of outbreaks	Number affected	Hospitalised	Deaths
Restaurant	40	736	23	0
Home	12	120	23	0
Takeaway	8	66	5	0
Commercial caterer	7	154	4	0
Bakery	5	113	20	1
Aged care facility	4	68	4	1
National franchised fast food	4	20	0	0
Fair/festival/mobile service	2	278	14	0
Child care	2	19	1	0
Community	2	29	6	0
Cruise/airline	2	21	1	0
School	2	180	2	0
Hospital	1	13	0	0
Institution	1	2	0	0
Total	**92**	**1,819**	**103**	**2**

Significant outbreaks

Six outbreaks affected 50 persons or more in 2002. Two were due to norovirus, two due to bacterial toxins, and two due to *Salmonella* Typhimurium. Four of these outbreaks occurred at restaurants, one at a school and one at a community festival. A variety of foods were implicated in these outbreaks, including: a bean dish, Caesar salad, seafood salad, lamb curry and a mixed meal of rice and meats.

The outbreak associated with the Caesar salad was due to *S*. Typhimurium 8, and occurred in South Australia. Seventy-eight cases were associated with this outbreak and 15 persons were hospitalised. Fifty-eight per cent (45/78) of cases were laboratory confirmed as *S*. Typhimurium 8. Several salad ingredients tested positive for *S*. Typhimurium 8 including the dressing, anchovies and parmesan cheese, with a very high organism count detected in the salad dressing (830,000 per gram). A meal of lamb, rice and potatoes contaminated with *S. aureus* and *B. cereus* caused an outbreak of gastrointestinal illness at a religious festival. Approximately 45 per cent of 600 persons attending the event became violently ill after eating food that was prepared with inadequate facilities for cold storage and preparation.

There were 20 outbreaks affecting between 20 and 50 persons. Cakes were implicated in four of these, two of which were caused by *Salmonella*. In one outbreak of *S*. Typhimurium 99 in South Australia, the bakery used the same piping bag to dispense both sausage meat, and cream for cakes. Two outbreaks were due to Vietnamese rolls containing pork and/or beef contaminated by *S*. Typhimurium 135 and *S*. Typhimurium 126.

Queensland reported an outbreak of *Campylobacter* infections from northern Queensland in August 2002. The public health unit, in conjunction with the Queensland site, interviewed 24 cases who identified chicken as a likely source and no other common exposures. The public health unit investigated a local poultry abattoir that was the main supplier of chickens for the region. Investigators collected samples of raw fresh chicken from the abattoir and from retail outlets representing three different chicken producers in Queensland. Sixty-seven per cent (29/43) of raw chicken samples were positive for the presence of *Campylobacter*. A specific *Campylobacter* subtype (Fla type 7) was the predominant subtype among human cases in northern Queensland and in chicken from two Queensland poultry manufacturers. PFGE typing of Fla type 7 isolates found that strains from human cases (Fla type 7; PFGE type P1) were indistinguishable from those obtained from the local abattoir. Fla type 7 *Campylobacter* isolates obtained from the other southern Queensland chicken manufacturers were distinct from these isolates by PFGE typing.

During February, the Hunter site investigated an outbreak of *C. perfringens* intoxication affecting 33 persons following a spit roast meal. The company had transported the meats to Newcastle from Sydney without proper temperature controls. At a national surveillance teleconference, it was reported that this outbreak was similar to four others in the Australian Capital Territory prior to Christmas. After investigation, it was identified that the same company supplied all five meals. All Australian jurisdictions reviewed their records to identify other similar incidents. The survey identified that the company had caused a total of 12 separate outbreaks affecting 332 persons in four jurisdictions in the previous five years.

There were two outbreaks associated with imported foods that could have international implications. One outbreak of *S*. Montevideo in the Hunter affected 47 persons and was linked to a local takeaway kebab shop. A further six associated cases were notified in 2003, which are not included in the outbreak total reported here. The investigation found several products in the kebab shop positive for *S*. Montevideo including tahini and hommus. Unopened jars of tahini originating from Egypt subsequently tested positive for *S*. Montevideo and *S*. Tennessee. This outbreak resulted in nationwide consumer and trade recalls, and an international alert to electronic list servers. Despite the potential for wider spread, New South Wales was the only site to report infections, although there were three cases in interstate visitors. There were no human infections reported overseas.

The other outbreak with potential for international spread occurred in Western Australia in August 2002. Delegates of a mining conference in Kalgoorlie became ill after consuming 'oyster shooters' served at a cocktail party. Over 1,000 persons attended the conference and the attack rate from a cohort study of 700 participants was

23 per cent. The oyster shooters were prepared using bulk oyster meat imported from Japan, and tomato juice. The label on the packet of oysters clearly stated, 'cook before consumption'. Norovirus was suspected as the cause of illness, although no virus was detected in faeces, or in a different batch of the same brand of oysters. There were three outbreaks of confirmed norovirus associated with Korean imported bulk oyster meat in New Zealand at the same time (Gail Greening, Institute of Environmental Science and Research, New Zealand, personal communication, April 2002).

Animal-to-person outbreaks

Sites reported three outbreaks that were transmitted from animal-to-person during 2002. Two of these were *Salmonella* outbreaks associated with poultry hatching programs in childcare centres. One was an outbreak of *S*. Agona affecting seven children in the Hunter region associated with ducklings. The other was an outbreak of *S*. Typhimurium 170 affecting six children in Queensland following hatching of chickens. A trace-back investigation in Queensland identified *S*. Typhimurium 170 and *S*. Typhimurium 12 in environmental samples from two poultry breeder sheds operated by the hatchery, which supplied eggs for the hatching program.

The other outbreak of animal-to-person gastroenteritis was due to shiga toxin producing *E. coli* in South Australia. Six persons were affected after either visiting or having contact with persons visiting a petting zoo located at a regional fair. The predominant *E. coli* serotype was O26, although some later cases were non-O26. Investigation of the petting zoo revealed a pig with same multiplex polymerase chain pattern for STEC which was negative for *E. coli* O26.

Cluster investigations

A cluster is defined as an increase in infections that are epidemiologically related in time, place or person where investigators are unable to implicate a vehicle or determine a mode of transmission for the increase. An example is a temporal or geographic increase in the number of cases of a certain type of *Salmonella* serovar or phage type. Another example is a community-wide increase of cryptosporidiosis that extends over some weeks or months. In this report, there are a small number of point source outbreaks where the mode of transmission is indeterminate that have been classified as a cluster.

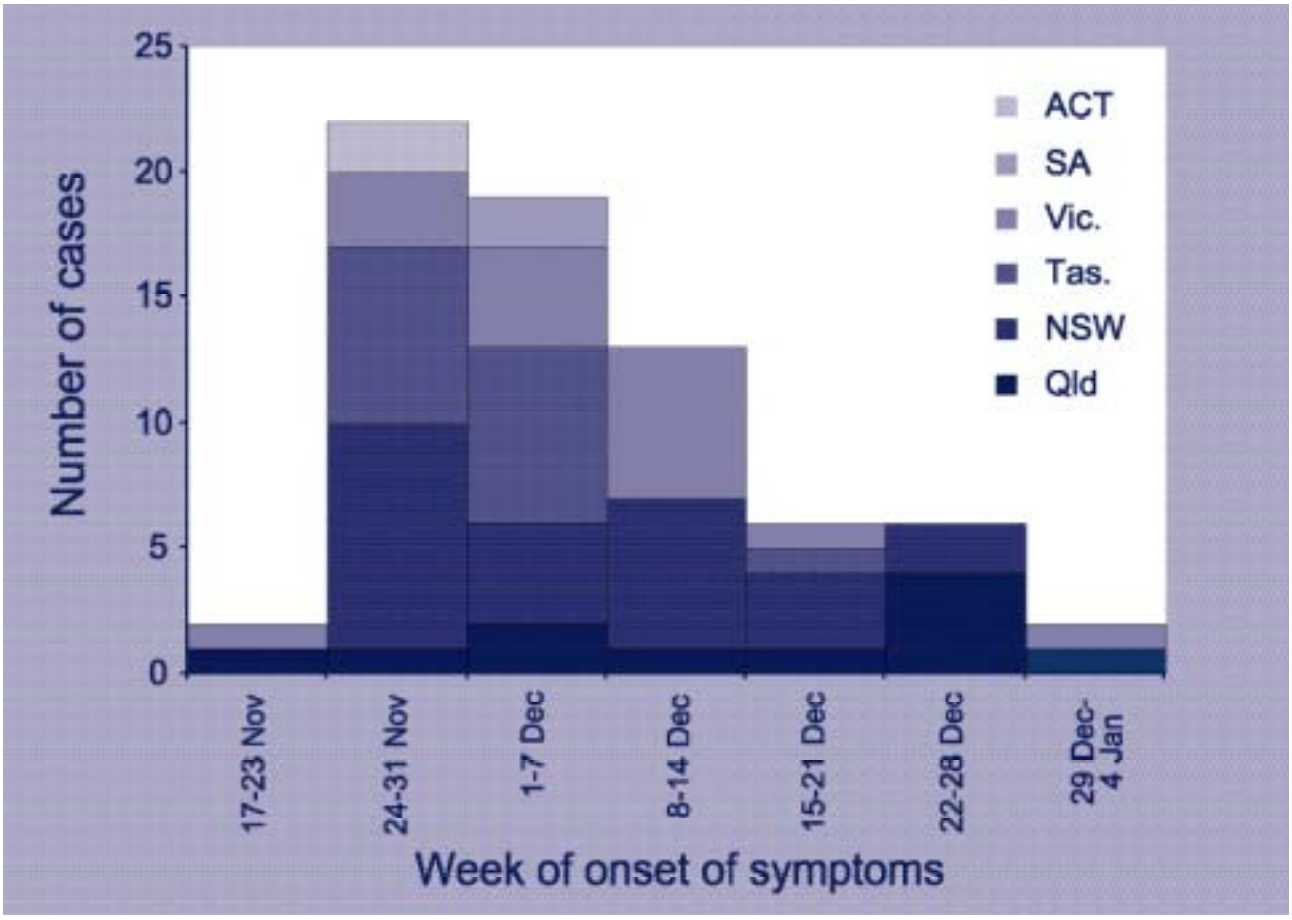
During 2002, states and territories conducted 100 cluster investigations, including three multi-state investigations. These clusters affected 1,751 persons with 65 cases hospitalised and one death. Forty-five per cent (45/100) of these investigations related to clusters of *Salmonella*. *Salmonella* clusters affected 601 persons with 53 cases hospitalised and one death. *S*. Typhimurium was responsible for 38 per cent (17/45) of cluster investigations. Of the remaining 28 investigations, there were 25 other different *Salmonella* serovars involved. Fifty-three per cent (53/100) of cluster investigations were of unknown aetiology.

There was one investigation of norovirus in a restaurant where the mode of transmission was unable to be determined. The Northern Territory reported a cluster of *Cryptosporidium* infections in the first six months of 2002. This community-wide increase was linked to infections acquired in a childcare centre and a local pool.

The first multi-state cluster investigation occurred in January 2002 and was related to *S*. Typhimurium 170.[16] Queensland, New South Wales and Victoria jointly investigated cases to generate hypotheses. Many cases were interviewed, although the source of infections was not identified.

The other two cluster investigations in November 2002 were of *S*. Kottbus and *S*. Potsdam. States and territories investigated less than 20 cases of *S*. Kottbus. The *S*. Kottbus cluster was spread across Australia and no common exposure was identified. The *S*. Potsdam cluster investigation involved New South Wales, the Australian Capital Territory, Victoria, South Australia and Tasmania. Thirty-four per cent of S. Potsdam cases were New South Wales residents, although the rate in Tasmania (3.2 cases per 100,000 population) was tenfold higher than any other jurisdiction (Figure 19).

Figure 19. Cases of *Salmonella* Potsdam, Australia, November to December 2002, by date of onset



Sites interviewed 50 cases of *S.* Potsdam using hypothesis-generating questionnaires. Reliable food histories were available for 25 of these cases. The most commonly consumed foods in the three days prior to illness were fresh tomatoes (68%) and chicken (68%). Fifty-two per cent of cases ate tomatoes on the day before onset of illness. Investigators suspected that the source of infection was a type of fresh salad produce, although comparison with food histories from population-based controls indicated that it would be difficult to show this epidemiologically.

Collecting reliable food histories during the Christmas period complicated epidemiological investigations. Food safety agencies were involved in a complicated traceback investigation for produce and other foods. Despite these intensive efforts, no source of infection for the outbreak was identified.

The true number of clusters investigated is difficult to determine, as the figures do not include all cluster investigations conducted in Public Health Units or local government areas. Jurisdictions have different definitions of 'cluster' and triggers for investigating clusters to fit with staff resources and local priorities.

Risk factors for infection

Food

During 2002, OzFoodNet identified several important risk factors for foodborne illness as a result of outbreak investigations and from preliminary results of case control studies. These included risks due to the following foods and settings for foodborne disease.

Eggs

Sites continue to report outbreaks associated with the consumption of egg-based products, such as mayonnaise and salad dressings. These outbreaks can be large and serious, as highlighted by the outbreak of *S.* Typhimurium 8 in South Australia. There is a need to review the circumstances of egg-associated outbreaks in detail to identify potential interventions, and whether there is a need for better quality assurance in the industry. The restaurant and catering industries need to be made aware of the risks of using raw unpasteurised eggs in sauces and dressings.

Chicken and poultry

During 2002, OzFoodNet finalised a multi-state case control study into risk factors for *Campylobacter* infection. Preliminary results indicate that one of the major risk factors for illness is consumption of chicken. Chicken was the only vehicle implicated in *Campylobacter* outbreaks in 2002, despite it being the most common gastrointestinal disease notified to health departments in Australia. In 2002, outbreaks of poultry-associated salmonellosis continue to occur, including two animal-to-person outbreaks. Poultry is consumed by approximately 80 per cent of people each week. To make our food supply safer, Australia needs to consider ways to reduce the burden of infections due to this source of infection in the community.

Vietnamese pork rolls

There were two outbreaks of *Salmonella* infection associated with these ethnic specialty dishes during 2002. Health authorities have been aware of the health risks associated with Vietnamese pork rolls for several years. Large outbreaks associated with these rolls have occurred in at least three states due to poor preparation and handling.[17,18] The occurrence of two more outbreaks in 2002 show that they are a particularly high-risk food. Regulatory agencies and restaurants need to urgently improve the safety of these popular foods.

Red meats and meat products

There were several outbreaks associated with red meats during 2002. These were due to a mixture of pathogens and in a variety of settings. The investigation into the multi-state outbreak of *Salmonella* Typhimurium 170 did not identify a specific food vehicle, but suggested potential links to red meat and poultry consumption. From this investigation is clear that there is a need for better and more timely sharing of data from human, animal and food surveillance systems.

Imported foods

The two outbreaks associated with imported foods during 2002 showed the potential for the spread of foodborne disease internationally. Oysters are known to be at high risk of norovirus contamination. New Zealand reported similar outbreaks at the same time as outbreaks in Australia, although the source of oysters was different. Caterers should follow cooking instructions where provided to prevent foodborne disease. The outbreak of *S.* Montevideo associated with Egyptian tahini in the Hunter highlighted the potential for contamination of sesame-based products with *Salmonella*. In this outbreak, the level of contamination was very low (1–2 organisms per gram). However, *Salmonella* were able to rapidly multiply when the tahini was used to make hommus. Agencies investigating outbreaks of salmonellosis should consider tahini, helva and other sesame-based products as potential sources of contamination. It may help to increase the risk classification of these products and frequency of testing on importation into Australia

Settings

There were several settings where food was prepared or consumed that were identified as high risk for foodborne disease, which included:

Bakeries

The five outbreaks occurring in bakeries in 2002 revealed the need for better assessment of food safety issues in these premises. Two of the outbreaks were related to Vietnamese pork rolls and the remaining three were associated with cakes filled with cream and/or custard. All outbreaks have been caused by *Salmonella* sp. Epidemiological investigation of these outbreaks often does not uncover the real source of contamination, as there is a time lag between food consumption and the recognition of the outbreak. There is a need for research to determine what are the critical food safety problems in these facilities that result in foodborne disease.

Restaurants and catered events

Outbreaks due to this sector constituted 57 per cent (47/92) of outbreaks. A variety of pathogens caused these outbreaks, including *Salmonella, C. perfringens*, norovirus and *V. parahaemolyticus*. Outbreaks involving restaurants and commercial caterers are more readily recognised, as the meals are often served to large numbers of persons. Clearly there is a need to continue to monitor the causes of outbreaks in this sector to identify gaps in food safety practices. The outbreak of *C. perfringens* poisoning due to spit roasts highlighted the problems for regulatory agencies operating at the state level dealing with food businesses operating in more than one jurisdiction with poor food practices for preparing food.

Hospitals and aged care

People resident in aged care settings and hospital patients are at particular risk for foodborne disease, which is shown by the five outbreaks that occurred during 2002. Two were due to *C. perfringens* indicating problems with preparation and handling of foods for residents. The outcomes for patients in these settings are often more adverse, as these sub-populations more susceptible to serious gastrointestinal disease. During 2002, OzFoodNet reviewed listeriosis notifications in Australia, which identified that three out of five listeriosis outbreaks in the years between 1995 and 2000 had occurred in hospital settings. The food supplied to hospital patients and persons in institutions should be readily monitored. There is also a high potential for foodborne transmission of norovirus when food handlers become infected during the many person-to-person outbreaks that occur each year in these facilities.

Surveillance evaluation and enhancement

Continuous improvement of surveillance is important to ensure that foodborne illness is investigated rapidly and effectively. To improve surveillance it is necessary to evaluate and compare surveillance conducted at different sites.

National information sharing

In 2002, all jurisdictions contributed to a fortnightly national cluster report to identify foodborne illness that was occurring across state and territory boundaries. The cluster report was useful for identifying common events affecting different parts of Australia. The cluster report is useful for tracking the investigation of multi-state clusters, such as *S.* Typhimurium 170, and *S.* Potsdam. The cluster report was also important in identifying a single spit roast company as the cause of several outbreaks spread over time and several jurisdictions. The cluster report supplemented information sharing on a closed list server, teleconferences and at quarterly face-to-face meetings.

Outbreak reporting and investigation

During 2002, the Hunter site reported the highest reporting rate of outbreaks of foodborne disease (31.2 per 100,000 population) and foodborne salmonellosis (7.3 per 100,000 population). The rates of other sites reporting foodborne *Salmonella* outbreaks ranged between 1.3–2.1 outbreaks per 100,000 population. Victoria investigated the largest number of foodborne disease outbreaks (26 outbreaks; 5.4 per 100,000 population) and *Salmonella* clusters (26 clusters; 5.3 per 100,000 population).

States and territories conducted 52 analytical studies (cohort or case control studies) to investigate foodborne disease outbreaks or clusters of suspected foodborne illness. Investigators used analytical studies for 40 per cent (37/92) of foodborne disease outbreaks, which was similar to 2001. The Hunter had the highest rate for investigations of foodborne disease or potentially foodborne clusters using analytical studies, followed by South Australia. Queensland had one of the lowest rate of analytical investigation despite a high rate of reporting for foodborne outbreaks. This was mainly due to several outbreaks of ciguatera where only descriptive investigation was necessary.

Completeness of Salmonella *serotype and phage type reports*

There was considerable improvement in the completeness of *Salmonella* available on state and territory surveillance databases between the years 2000 to 2002 (Table 10). Overall 96.2 per cent (6,994/7,267) of *Salmonella* notification on databases contained either serotype or phage type, which was an increase of 4.3 per cent from 2000 and 1.7 per cent from 2001.

Only 89.1 per cent (49/55) of phage type information was reported for *S.* Hadar and 92.7 per cent (114/123) *S.* Heidelberg. Phage typing information was available for 95.0 per cent (307/323) of reports for *S.* Enteritidis in 2002. The largest increase in completeness between 2000 and 2002 was reported for *S.* Heidelberg (25.4%) and *S.* Bovismorbificans (16%).

South Australia had the highest proportion of complete *Salmonella* notification (100%), while four sites reported 98 per cent or higher. New South Wales reported the lowest rate of completeness, but recorded a 10.8 per cent improvement when compared to 2000 figures. Western Australia also reported an increase of 10.7 per cent, as a result of improved case notification to the health department.

Table 10. Number of *Salmonella* infections notified and proportion of notifications with serotype and phage type information available, Australia, 2000 to 2002, by OzFoodNet site

Information required	Year	*Salmonella* notifications n	*Salmonella* with serotype %	*Salmonella* infections with phage typing information						*Salmonella* infections with information
				S. Bovis-morbificans %	*S.* Enteritidis %	*S.* Hadar %	*S.* Heidelberg %	*S.* Typhimurium %	*S.* Virchow %	
ACT	2000	102	96.1	–	100.0	0.0	0.0	100.0	100.0	95.1
	2001	78	98.7	100.0	100.0	100.0	–	100.0	100.0	98.7
	2002	96	97.9	100.0	100.0	–	–	91.1	100.0	93.8
Hunter	2000	86	94.2	100.0	50.0	66.7	100.0	97.4	0.0	87.2
	2001	117	97.4	85.7	50.0	–	50.0	97.1	100.0	92.3
	2002	179	95.5	100.0	100.0	–	0.0	95.1	100.0	92.7
NSW	2000	1,334	92.2	50.0	77.8	52.9	18.2	88.1	38.2	80.5
	2001	1,668	94.2	63.6	80.8	35.3	84.6	95.8	67.2	87.9
	2002	2,147	95.5	92.2	81.5	58.3	75.9	95.7	89.2	91.3
NT	2000	323	91.6	33.3	20.0	50.0	100.0	100.0	50.0	88.9
	2001	390	90.8	100.0	33.3	100.0	–	100.0	100.0	90.3
	2002	329	96.0	100.0	66.7	–	–	100.0	–	95.7
Qld	2000	1,818	97.2	100.0	94.8	100.0	90.6	93.1	97.4	97.7
	2001	2,169	97.0	100.0	91.8	73.3	91.8	95.8	95.0	97.8
	2002	2,722	97.5	100.0	100.0	100.0	97.6	98.1	98.9	99.4
SA	2000	452	99.6	100.0	100.0	100.0	–	100.0	100.0	99.6
	2001	613	99.8	100.0	100.0	100.0	–	100.0	100.0	99.8
	2002	520	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

Table 10 continued. Number of *Salmonella* infections notified and proportion of notifications with serotype and phage type information available, Australia, 2000 to 2002, by OzFoodNet site

Information required	Year	*Salmonella* notifications n	*Salmonella* with serotype %	*Salmonella* infections with phage typing information						*Salmonella* infections with information
				S. Bovis-morbificans %	*S.* Enteritidis %	*S.* Hadar %	*S.* Heidelberg %	*S.* Typhimurium %	*S.* Virchow %	
Tas.	2000	127	96.9	100.0	100.0	–	0.0	100.0	100.0	96.1
	2001	159	98.7	100.0	100.0	–	–	96.3	–	98.1
	2002	165	99.4	100.0	100.0	100.0	–	100.0	100.0	99.4
Vic.	2000	1,005	97.9	96.3	100.0	90.0	75.0	99.8	99.1	97.4
	2001	1,090	97.7	100.0	100.0	100.0	100.0	99.8	100.0	97.6
	2002	1,207	99.2	90.9	100.0	88.9	100.0	100.0	100.0	99.0
WA	2000	936	92.4	100.0	93.1	85.0	0.0	91.4	80.0	87.7
	2001	858	95.8	85.7	95.7	85.7	0.0	97.0	66.7	93.8
	2002	730	98.6	100.0	100.0	100.0	–	99.2	100.0	98.4
OzFoodNet	2000	6,097	95.4	78.3	90.5	77.8	67.3	94.1	90.4	92.0
	2001	7,025	96.2	87.3	91.4	78.8	88.5	97.5	92.4	94.7
	2002	7,916	97.4	94.3	95.0	89.1	92.7	98.0	97.6	96.9

Discussion

Each year in Australia, it is estimated that 17.2 million persons experience infectious gastroenteritis and approximately 5.4 million (credible interval 4.0–6.9 million) of these may be due to contaminated food. These estimates are comparable to previous Australian and other international reports and clearly demonstrate the burden that foodborne disease has on Australian society.[3,19,20] The large burden justifies the attention given to foodborne disease surveillance and enhancing the safety of our food supply.

Gastrointestinal infections notified to health departments represent only a small proportion of cases occurring in the community, as most are mild and do not require medical attention. The gastroenteritis survey provides insight into the health seeking behaviour of Australians, with one in five persons with gastroenteritis visiting a doctor and one in 20 providing a faecal specimen. An intervention trial in Melbourne found that persons with acute gastroenteritis who submitted faecal specimens had pathogens identified in only 25 per cent of stools despite intensive testing, which demonstrates that there are many other gastrointestinal pathogens that are unrecognised.[21] Despite this, notifications of gastrointestinal infections to health departments provide a picture of illness that may potentially be due to food.

In 2002, notifications of selected gastrointestinal infections in Australia were 7.7 per cent higher than the historical mean, which may reflect a true increase in incidence, changing laboratory practices or improving surveillance. Certainly, the increased rates of STEC notification reflected changing patterns of testing faeces and diagnostic tests. This is likely to increase in future, as laboratories screen more specimens and diagnostic tests improve. The crude notification rate of *Salmonella* infections also increased, while *Campylobacter* and *Listeria* infection rates were stable. There were decreases observed for yersiniosis, shigellosis and haemolytic uraemic syndrome.

Australia has similar rates of notified gastrointestinal infections to some other developed countries including Canada, Norway, and the United Kingdom.[22,23,24] Australian rates are lower than rates in neighbouring New Zealand and higher than active surveillance data for salmonellosis in the United States of America (USA).[25,26] Notified *Salmonella* in the USA affects 16.1 cases per 100,000 population compared to 40.3 cases per 100,000 population in Australia. Even more startling is the difference in *Campylobacter* notification rates in the USA at 13.4 cases per 100,000 population compared to 110.1 cases per 100,000 population in Australia. The lower rates in the USA may be due to differences in access to healthcare, stool submission rates and testing regimes in laboratories.

The USA reports higher rates of toxigenic *E. coli* O157:H7 (1.7 cases per 100,000 population) than Australia.[26] This organism is easily isolated on routine pathology media and may reflect changes in testing procedures or a true difference in incidence. In 2002, Australian states and territories reported a doubling in the number of *E. coli* O157 infections, although the total numbers and rates remain small. H typing was not available for the majority of these, but it is likely that the majority are not the H7 subtype as laboratories have rarely isolated it in previous years. The increasing use of molecular detection methods often means that organisms are not cultured for faecal specimens for subsequent serotyping or profiling.

South Australia has conducted enhanced surveillance for STEC for several years, but had not identified any outbreaks until the outbreak at a petting zoo at a regional fair in 2002. Petting zoos have been commonly associated with outbreaks of STEC and other gastrointestinal diseases.[27,28,29] There were two other outbreaks of salmonellosis following poultry hatching programs in two different states. While some Australian states have prepared guidelines for petting zoos it is important that these cover poultry hatching programs and that all zoo operators are aware of the requirements to prevent infections.[30]

Salmonella caused the most foodborne outbreaks of any agent during 2002. Like many other countries, *Salmonella* infections are a serious problem for Australia.[31] Not only do they cause considerable morbidity, but investigations consume much public health effort and resources. For every two *Salmonella* outbreaks that are attributed to food, there are another three cluster investigations where no source is identified. In addition, there may be as many as 14 cases in the community for every case

reported to Australian surveillance systems (OzFoodNet unpublished data). To identify causes of more of these outbreaks, we may need to critically evaluate our current methods of investigation. Investigations are becoming more complicated due to the increasing use of molecular methods for comparing isolates and regular trace back of foods consumed by cases to the source of food supply.[31,32]

OzFoodNet identified several risk factors for foodborne infections in 2002 based on the surveillance data and epidemiological studies. Many of these risk factors have been previously recognised, but need to be considered again. The risk posed by raw eggs used in dressings could be easily addressed by the use of pasteurised eggs. While Australia does not have endemic *S*. Enteritidis 4 that contaminates the internal contents of eggs, there are clearly other subtypes that are associated with eggs. Infections due to poultry, red meats and imported foods continued to occur in 2002. Food handling and preparation practices in bakeries need to be addressed to prevent outbreaks of salmonellosis, which are an increasing problem.[33,34,35]

It is important to recognise some of the many limitations of the data that OzFoodNet report. Surveillance data are inherently biased and require careful interpretation. These biases include: the higher likelihood that certain population groups will be tested, and different testing regimes in different states and territories, resulting in different rates of disease. Some of the numbers of notifications are small, as are populations in some jurisdictions. This can make rates of notification unstable and meaningful interpretation difficult. Importantly, some of the most common enteric pathogens are not notifiable, particularly norovirus and enteropathogenic *E. coli*. These organisms may be notified as the cause of outbreaks, but not individual cases of disease. There can also be considerable variation in assigning causes to outbreaks depending on investigators and circumstances.

There have been consistent improvements to surveillance in recent years, which is shown by the large number of analytical studies used in investigations of outbreaks. The success of national communication through OzFoodNet was highlighted by the identification of 12 separate incidents associated with a single spit roast company operating in several states. We observed a difference in the rate of reporting foodborne disease outbreaks that probably reflects sensitivity of surveillance and differing thresholds for investigation in different jurisdictions.[36] There was a significant improvement in the completeness of *Salmonella* typing information on state and territory databases, which reflects better quality surveillance data. In the future, OzFoodNet aims to regularly compare the timeliness of *Salmonella* typing reporting to review the effectiveness of data transmission for surveillance systems.

Despite these improvements to surveillance, we need to critically evaluate our efforts in order to prevent foodborne infections. In particular, there is a need to strengthen laboratory-based surveillance using standardised molecular methods for profiling organisms, such as *Listeria*, outbreak-associated *Salmonella* and shiga toxin producing *E. coli*. This information needs to be rapidly communicated to public health investigators to enable more timely investigation of widely spread clusters and prevention of outbreaks. Countries such as the United Kingdom routinely use PFGE as a successful adjunct to traditional *Salmonella* typing to assist with outbreak investigations.[37] Other potential improvements could include: standardised approaches to cluster investigation, and sharing surveillance information from animal and food testing data. None of these new initiatives will occur without appropriate resources, but the burden of diseases clearly requires that we improve to acquire better surveillance data to support control activities.

The burden of foodborne disease is a major concern to the community, industry and government. It is important that foodborne disease surveillance is able to assess whether food safety policies and campaigns are working. National surveillance of foodborne diseases has many benefits and provides long-term data to assist with this task. OzFoodNet needs to consider what kinds of foodborne disease data are useful to evaluate the effect of interventions to make food safer. This will require closer working relationships with food safety professionals, microbiologists, veterinarians and industry. To ensure that control of foodborne disease remains a focus for government, it may be pertinent to set target goals for foodborne diseases similar to other national health priority areas.[26,38]

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Appendix 1. Summary of gastrointestinal infections notified to OzFoodNet sites potentially due to food, 2002

		Australian Capital Territory	Hunter	New South Wales	Northern Territory	Queensland	South Australia	Tasmania	Victoria	Western	Total
Campylobacter	cases	367	nn	nn	208	3,905	2,519	610	4,932	2,175	14,716
	rate	114.0	nn	nn	105.0	105.3	165.7	129.0	101.2	112.9	113.0
Salmonella	cases	96	179	2,147	330	2,722	520	165	1,207	730	7,917
	rate	29.8	32.9	32.3	166.7	73.4	34.2	34.9	24.8	37.9	40.3
Yersinia	cases	1	nn	nn	7	73	13	1	nn	4	99
	rate	0.3	nn	nn	3.5	2.0	0.9	0.2	nn	0.2	1.2
STEC	cases	0	0	5	0	6	38	0	5	4	58
	rate	0.0	0.0	0.1	0.0	0.2	2.5	0.0	0.1	0.2	0.3
HUS	cases	0	3	7	1	1	0	0	4	0	13
	rate	0.0	0.6	0.1	0.5	0.0	0.0	0.0	0.1	0.0	0.1
Typhoid	cases	1	0	22	0	11	4	0	21	5	64
	rate	0.3	0.0	0.3	0.0	0.3	0.3	0.0	0.4	0.3	0.3
Shigella	cases	0	2	84	103	95	26	1	66	130	505
	rate	0.0	0.4	1.3	52.0	2.6	1.7	0.2	1.4	6.7	2.6
Listeria	cases	0	0	12	0	20	2	2	15	11	62
	rate	0.0	0.0	0.2	0.0	0.5	0.1	0.4	0.3	0.6	0.3

nn not notifiable

Appendix 2. Outbreak summary for OzFoodNet sites, 2002

State	Month of outbreak	Setting category	Agent responsible	Number exposed	Number affected	Evidence*	Epidemiological study†	Responsible vehicles
Hunter	Jan	Restaurant	*S.* Potsdam	Unknown	17	AM	C	Egg based dressings
	Feb	Restaurant	*V. parahaemolyticus*	Unknown	2	D	D	Unknown
	Feb	Commercial caterer	*C. perfringens*	33	16	AM	C	Spit roasted beef and/or pork
	Apr	National franchised fast food	Unknown	Unknown	5	D	C	Suspected seafood pizza
	May	Restaurant	Unknown	Unknown	3	D	D	Suspected seafood
	May	Restaurant	Unknown	Unknown	5	D	C	Unknown
	Jun	Restaurant	Unknown	Unknown	4	D	C	Suspected seafood
	Aug	Restaurant	Unknown	19	5	D	C	Unknown
	Aug	Takeaway	Unknown	Unknown	2	D	D	Suspected kebabs
	Nov	Home	Norovirus	Unknown	6	D	D	Unknown
	Nov	Restaurant	Unknown	Unknown	5	D	D	Unknown
	Nov	Takeaway	*S.* Montevideo	Unknown	47	M	D	Imported tahini
	Dec	Restaurant	Norovirus	Unknown	3	D	D	Unknown
	Dec	Restaurant	Unknown	Unknown	4	D	D	Unknown
	Dec	Restaurant	Unknown	Unknown	4	D	D	Suspected beef dish
	Dec	Restaurant	Unknown	Unknown	11	D	C	Unknown
	Dec	Restaurant	Unknown	Unknown	4	D	D	Unknown
NSW	Jan	National franchised fast food	*S.* Virchow	4	3	A	CCS	Chicken
	Jan	Takeaway	Unknown	Unknown	4	D	D	Mixed foods
	Jan	Takeaway	Unknown	2	2	D	D	Barbecue chicken
	Jan	Takeaway	Unknown	2	2	D	D	Special fried rice
	Feb	Restaurant	*S.* Typhimurium 9	Unknown	8	A	C	Deep fried ice cream
	Feb	School	*S.* Typhimurium 9	900	132	A	CCS	Baked beans/chilli con carne
	Mar	National franchised fast food	Unknown	4	4	D	D	Pizza
	Mar	Home	Ciguatera poisoning	Unknown	7	M	D	Spanish mackerel
	Apr	Home	Unknown	56	20	D	C	Suspected pasta
	Apr	Takeaway	Unknown	2	2	D	D	Fish
	Apr	Bakery	*S.* Typhimurium 126	Unknown	32	M	D	Vietnamese pork/chicken rolls
	May	Restaurant	Unknown	10	2	D	D	Beef curry
	Jun	Fair/festival/ mobile service	Unknown	Unknown	6	D	D	Cake
	Jun	Takeaway	Unknown	5	5	D	C	Pizza

Appendix 2 continued. Outbreak summary for OzFoodNet sites, 2002

State	Month of outbreak	Setting category	Agent responsible	Number exposed	Number affected	Evidence*	Epidemiological study†	Responsible vehicles
	Jul	Restaurant	Unknown	Unknown	4	D	D	Unknown
	Jul	Restaurant	Unknown	130	30	D	CCS	Unknown
	Sep	Cruise/airline	Unknown	3	3	D	D	Chicken casserole
	Sep	Restaurant	Unknown	5	4	D	D	Mixed foods
	Oct	Bakery	*S.* Typhimurium 135	Unknown	29	D	D	Cream filled cake
	Oct	Restaurant	Hepatitis A	Unknown	8	D	D	Yum cha
	Nov	Restaurant	*S.* Typhimurium 170 var	Unknown	6	D	C	Not identified
	Dec	Restaurant	*C. perfringens*	261	70	A	C	Lamb curry
	Dec	Restaurant	*S.* Typhimurium 126	1,200	21	A	CCS	Thai salad
Qld	Jan	Takeaway	Ciguatera	Unknown	2	D	D	Spanish mackerel
	Jan	Restaurant	*C. perfringens*	Unknown	2	D	D	Unknown
	Jan	Aged care facility	*S.* Typhimurium 102	53	12	D	C	Suspected egg-white dish
	Feb	Home	Ciguatera	2	2	D	D	Striped perch
	Feb	Institution	*S.* Potsdam	Unknown	2	D	D	Unknown
	Feb	National franchised fast food	*Staphylococcus aureus*	9	8	D	D	Pizza
	Feb	Restaurant	Unknown	7	6	D	D	Unknown
	Mar	Home	*S.* Typhimurium 135a	10	10	D	D	Salmon/egg/onion/rice patties
	Apr	Home	Ciguatera	5	3	D	D	Grunter bream
	May	Restaurant	Unknown	7	7	D	D	Unknown
	Jun	Commercial caterer	*Bacillus cereus*	250	37	AM	CCS	Rice
	Aug	Restaurant	Unknown	23	16	D	D	Unknown
	Aug	Community	*Campylobacter jejuni*	Unknown	24	M	D	Chicken
	Oct	Restaurant	*S.* Hadar 22	Unknown	3	D	D	Suspected egg dish
	Nov	Restaurant	*S.* Typhimurium 197	24	8	D	D	Unknown
	Dec	Home	*C. perfringens*	3	3	D	D	Unknown
	Dec	School	Norovirus	200	48	D	D	Unknown
	Dec	Child care	S. Typhimurium 135a	Unknown	12	D	D	Suspected egg sandwiches
SA	Apr	Restaurant	*S.* Typhimurium 8	Unknown	78	AM	CCS	Caesar salad
	May	Community	*S.* Typhimurium 43	Unknown	5	A	CCS	Sliced ham
	May	Home	*C. perfringens*	12	8	D	C	Potato and meat pie
	Oct	Bakery	*S.* Typhimurium 99	Unknown	22	AM	C	Cream and custard cakes

State	Month of outbreak	Setting category	Agent responsible	Number exposed	Number affected	Evidence*	Epidemiological study†	Responsible vehicles
Tas.	Mar	Restaurant	*S.* Typhimurium 135	Unknown	5	D	D	Unknown
Vic.	Feb	Commercial caterer	Unknown	90	32	A	C	Chocolate mud cake
	Feb	Restaurant	Norovirus	30	12	D	D	Unknown
	Feb	Restaurant	Unknown	23	18	D	C	Unknown
	Feb	Hospital	Unknown	13	13	D	D	Soup
	Mar	Restaurant	Unknown	15	8	D	C	Unknown
	Mar	Home	*S.* Typhimurium 135	67	19	A	C	Roast chicken
	Mar	Commercial caterer	Unknown	16	12	D	C	Suspected sandwiches
	Mar	Fair/festival/mobile service	*S. aureus, B. cereus*	600	272	M	D	Mixed foods
	Apr	Home	*S.* Typhimurium 135	9	6	M	D	Home barbequed chicken
	Apr	Aged care facility	*S.* Typhimurium 9	302	18	D	D	Unknown
	Apr	Home	*S.* Typhimurium 170	Unknown	6	M	D	Hedgehog — possibly eggs
	Apr	Child care	*S.* aureus	Unknown	7	M	D	Rice
	Apr	Cruise/airline	*C. perfringens*	34	18	D	C	Unknown
	May	Restaurant	Unknown	33	10	A	C	Pea and ham soup
	May	Restaurant	Norovirus	650	192	D	C	Unknown
	May	Bakery	*S.* Typhimurium U290	Unknown	10	A	CCS	Cream filled cakes/pastries
	Jun	Bakery	*S.* Typhimurium 135	Unknown	20	D	D	Vietnamese pork rolls
	Aug	Aged care facility	*C. perfringens*	69	15	D	D	Suspected gravy
	Aug	Restaurant	*S.* Typhimurium 135	Unknown	12	D	D	Suspected spring rolls
	Oct	Aged care facility	*C. perfringens*	64	23	D	D	Suspected gravy
	Oct	Commercial caterer	Norovirus	unknown	25	A	C	Suspected cheesecake
	Oct	Restaurant	Suspected viral	296	23	D	C	Mixed foods.
	Nov	Commercial caterer	Norovirus	140	32	D	C	Mixed foods.
	Nov	Restaurant	Suspected wax ester	15	10	D	D	Suspected rudderfish
	Dec	Restaurant	Unknown	20	5	D	D	Steak or sauce
	Dec	Restaurant	Unknown	77	41	D	C	Unknown
WA	Feb	Home	Unknown	100	30	D	D	Unknown
	Feb	Restaurant	Norovirus	230	60	AM	C	Seafood salad
	Aug	Commercial caterer	Unknown	1,100	Unknown	A	CCS	Oyster shooters
			7,234	1,819				

* A = analytical epidemiological evidence; D=descriptive evidence: M=microbiological evidence.

† C = cohort study; CCS=case control study; D=descriptive study.

An outbreak of cryptosporidiosis associated with an animal nursery at a regional fair

Rosie H Ashbolt,[1] David J Coleman,[1] Avner Misrachi,[1] Joe M Conti,[1] Martyn D Kirk[2]

Abstract

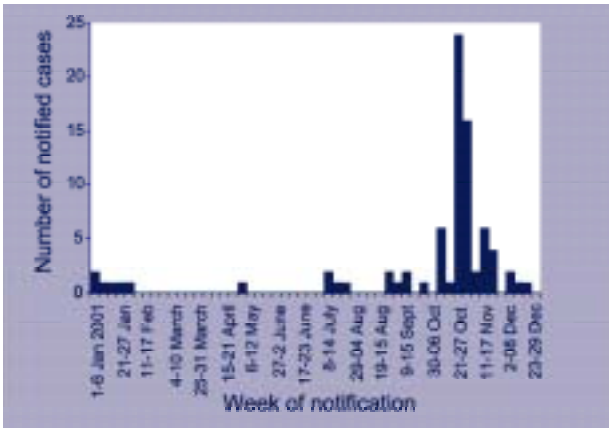
Cryptosporidiosis is a common gastrointestinal illness that is transmitted from infected persons, animals, or contaminated water or food.This article reports on an outbreak of cryptosporidiosis associated with an animal nursery at an agricultural show held in northern Tasmania during October 2001. Eighty-one per cent of cases (38/47) notified to the Tasmanian Department of Health and Human Services over a 35 day period were interviewed to determine potential sources of infection. Eighty-one per cent of interviewed cases (29/36) reported that they had attended the agricultural show, and 75 per cent (27/36) reported contact with animals in the animal nursery. Cases occurring more than one incubation period after the agricultural show were significantly more likely to have had contact with someone else with diarrhoea (p<0.01). This is the first reported outbreak of cryptosporidiosis associated with an animal nursery in Australia. The outbreak demonstrates the importance of infection control policies and hygiene measures in the animal nursery setting. *Commun Dis Intell* 2003;27:244–249.

Introduction

Cryptosporidium is an important and widespread cause of enteric infection in humans and animals. The incubation period for cryptosporidiosis is 1–12 days[1] and the main symptoms are watery diarrhoea and stomach cramps. Infection is usually spread through contaminated drinking or recreational water, contact with infected animals, and contact with infected persons.[2,3,4] In Australia, reported outbreaks of cryptosporidiosis have generally been associated with swimming pools.[5,6] The association between dairy farming and cryptosporidiosis is well-documented, although it is not commonly described in Australia.[7,8]

In Tasmania, cryptosporidiosis is a notifiable disease reported by laboratories. In 2001 the rate of cryptosporidiosis in Tasmania was 16.5 cases per 100,000 population compared with background rates of 10.8 cases per 100,000 population and 9.3 cases per 100,000 population in the years 2000 and 2002 respectively. Over this three year period, 98 per cent of all notified *Cryptosporidium* infections occurred in the north of the state and 61 per cent of all cases were reported in spring. The seasonal distribution of cases for 2001 is presented in Figure 1.

Figure 1. Notified cases of laboratory-confirmed cryptosporidiosis, Tasmania, 2001



On 22 October 2001, a laboratory in northern Tasmania reported a cluster of 10 cases of cryptosporidiosis. A further 38 cases were notified over the following 34 days.

Public health officers from the Tasmanian Department of Health and Human Services investigated this cluster of cryptosporidiosis cases to determine a source for the infections and to implement appropriate public health action.

Methods

Cases notified to the department from 22 October 2001 were considered part of the outbreak investigation. Investigation reports for cases prior to 22 October 2001 were reviewed and the cases were not considered to be part of the outbreak. A standard hypothesis-generating questionnaire was used. The questionnaire contained information on basic demographics, symptoms, and exposure to potential risk factors. Information on contacts, household members and others that may have been ill at the same time was also sought. Staff from public and environmental health and local council environmental health officers administered the questionnaire using telephone interviews.

Once the outbreak was identified, surveillance was enhanced for diarrhoeal disease in the local area by increasing the timeliness and reporting of laboratory tests. General practitioners were alerted to the outbreak and requested to lower their threshold for the investigation of gastrointestinal illness.

The regional Environmental Health Officer investigated potential environmental sources for the outbreak. Public health officers also made inquiries with the government veterinary laboratory about the incidence of diarrhoeal disease in animals.

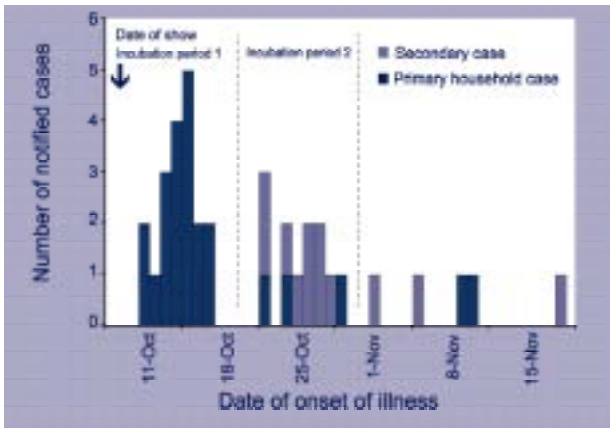
Results

There were 48 cases of cryptosporidiosis notified during the outbreak period, 22 October to 24 November 2001. Laboratories serving the northern part of Tasmania notified 98 per cent (47/48) of these cases. Eighty-one per cent (38/47) of these northern laboratory-confirmed cases were interviewed. Seven cases were unable to be contacted, and two were not interviewed for other reasons.

The ages of case patients had a bimodal distribution with peaks in children aged 1–9 years and adults aged 20–34 years. Forty per cent of cases were male. Cases were resident across seven local council areas, with the majority of cases resident within the Greater Launceston Statistical Subdivision. The symptoms most frequently reported were: diarrhoea (94%), vomiting (88%), abdominal cramps (69%), and nausea (65%). Eleven per cent (4/38) of cases were hospitalised. The duration of symptoms was difficult to determine as most cases were still symptomatic when interviewed.

The reported date of onset of symptoms for two cases was greater than three months prior to collection of the faecal specimen and therefore these cases were not included in analyses of potential exposures. The epidemic curve (Figure 2) depicts the distribution of cases by onset date during the outbreak period.

Figure 2. Cases of cryptosporidiosis among persons living in northern Tasmania, 14 October to 21 November 2001, by date of onset of patient's symptoms



The shape of the epidemic curve suggested that cases separated into two subgroups: cases within an initial wave, and cases in a second wave. As the route of infection may have differed for the two sub-groups, the prevalence of risk factors was described for each sub-group (Table 1).

Table 1. Prevalence of risk factors for infection in cases of cryptosporidiosis among persons living in northern Tasmania, 14 October to 21 November 2001, by date of onset of patient's symptoms

	Cases in first wave (n=19)		Cases in second wave (n=17)	
	n	%	n	%
Male	6	32	10	59
Attended childcare centre	3	16	3	18
Overseas travel	0	0	1	6
Contact with pets	15	79	14	93
Attendance at agricultural show A	16	84	13	76
Attendance at the agricultural show on 11 October 2001	15	79	13	76
Contact with animals in the animal nursery (at the agricultural show)	14	74	13	76
Contact with animals on a farm	4	21	4	24
Untreated water consumed	1	5	1	6
Swimming at pool A	4	21	3	18
Contact with an ill person prior to illness	0	0	12	71

The shape of the epidemic curve and the prevalence of risk factors were consistent with an initial point source infection followed by secondary transmission.

The time period for the initial wave was 12 days (equivalent to the incubation period for cryptosporidiosis), commencing on the opening date for the agricultural show. Eighty-four per cent of cases in the initial wave (16/19) attended the local agricultural show. Of those who attended the show, 88 per cent (14/16) also reported attending the animal nursery at the show with most cases reporting that they touched the animals.

While a similar proportion of cases in the second wave attended the agricultural show, secondary transmission was the probable route of infection. Cases in the second wave were significantly more likely to have had contact with someone else who was ill compared with cases in the initial wave ($p<0.01$). All cases reporting a family member ill prior to their own illness were in the second wave of cases.

Cases who reported that other family members were also ill also reported that 80 per cent of these ill family members attended the agricultural show with the case. Typically, cases report family groups attended the agricultural show with one member contacting cryptosporidiosis (either confirmed or un-confirmed) and transmitting the infection to other family members.

It is also possible to interpret the epidemic curve as a point source infection followed by an extended tail, which is plausible if these later cases were considered to have a longer than usual incubation period of 14–28 days, rather than the expected 1–12 days. There was no difference in the age distribution of cases in the two waves.

The number of confirmed cases in the latter part of the outbreak may have been influenced by enhanced surveillance and lower testing thresholds.

Overall, 81 per cent (29/36) of all cases had attended the local agricultural show, and 75 per cent (27/36) of cases had contact with animals at the animal nursery. Four cases reported attending swimming lessons at a single venue prior to becoming ill, with one case reporting attendance after the onset of illness. The five children that attended child care went to four different child-care centres. In this outbreak, drinking water or contact with recreational water was not likely to be the source of infection as these exposures did not account for a high proportion of cases.

Public health response

Investigation of the animal nursery

The three-day agricultural show had ended by the time the investigation commenced. The regional Environmental Health Officer interviewed organisers of the agricultural show and reported that animals were obtained from various sources within the community and included goats, lambs, sheep, calves, pet rats, dog and puppies, rabbits, chickens, some poultry and native animals. All animals remained in the animal nursery for the duration of the show except for some pet rabbits. An organiser indicated that some of the calves showed symptoms of diarrhoea, but could not provide information on the date or duration of symptoms. Toilet facilities and hand basins were provided nearby for attendees of the agricultural show.

Swimming pools

Four cases reported attending swimming lessons at one swimming pool and one case reported swimming after the onset of symptoms. This swimming pool was widely used for learn-to-swim and toddler classes. As a precautionary measure, the local council advised the swimming pool to hyper-chlorinate and backwash the filters. During the outbreak, there was no evidence of further spread of *Cryptosporidium* infection through swimming pool usage.

Veterinary investigations

As the agricultural show had ended prior to the investigation, animal faecal samples were not collected. Thus no specific animal could be microbiologically linked with the outbreak, however calves at the animal nursery were reported as having diarrhoea. The government veterinary laboratory also reported the presence of *Cryptosporidium* in scours from cattle tested at the time of the outbreak (personal communication, K Formiatti, veterinary microbiologist, Mt Pleasant Laboratories, 9 November 2001).

Communication

A newspaper article provided the community with information on the outbreak and how to avoid further transmission. The laboratories and local council environmental health officers were given a summary report at the conclusion of the outbreak.

Following a review of the outbreak, a public health advisory letter was sent to relevant organisations such as the education department, agricultural show societies, and wildlife parks for further dissemination. The advice given was based on the most recently published Centers for Disease Control and Prevention Recommendations: Farm Animal Contact, September 2001.[9]

Discussion

This article reports on a localised outbreak of cryptosporidiosis associated with an animal nursery at an agricultural show. While cryptosporidiosis has been linked to farm visits by children elsewhere,[10,11] this is the first reported outbreak of cryptosporidiosis in Australia in which an animal nursery in the agricultural show setting has been implicated.

The show was held over a three-day period, however all but one case reported attending the show on the first day. No ruminants were removed from the animal nursery during the three-day event, but the spoilt hay was removed each day. It is feasible that the mode of transmission was infected faeces in the hay.

This investigation was limited to laboratory confirmed cases, however it is expected that many more were ill as evidenced by 42 per cent of interviewed cases reporting other household members ill with similar symptoms. Many of the cases reported later in this outbreak could be attributed to person-to-person transmission as reflected in the epidemic curve and exposure histories.

Among cases occurring over the outbreak period, the other principal source of infection reported was exposure to scouring calves in the

farm setting. Two of the three cases who did not attend the show reported exposure to scouring calves at home. Additionally, a review of seven cases notified to public and environmental health staff prior to the outbreak indicated that four of these cases were exposed to cattle prior to illness.

In future outbreaks of cryptosporidiosis, the genotyping of *Cryptosporidium* could be used to confirm epidemiological findings. *Cryptosporidium parvum* is the most commonly identified etiologic agent of human cryptosporidiosis and can be divided into two distinct sub-populations: genotype 1, found almost exclusively in humans, and genotype 2 which is found in both ruminants and humans.[12] In this outbreak we did not determine the species or genotype of *Cryptosporidium* present. Clearly such typing would have helped in the identification of the pathway to *Cryptosporidium* infection.

In springtime, agricultural shows are very much a part of the fabric of regional Australia. In this outbreak, close contact with young animals in an animal nursery at an agricultural show was linked to cryptosporidiosis. This finding highlights the infectious hazard posed by contact with animals in this setting and brings to our attention the potential for other more dangerous infections to be acquired. Elsewhere, outbreaks of Shiga-like toxin producing *Escherichia coli* O157 infection have been reported in the animal nursery setting.[13,14]

It is important that both staff and visitors are educated about the risks associated with animal contact and are alerted to the simple precautions that can prevent the transmission of infections. These include the mandatory provision of dedicated hand-washing or disinfecting facilities, obvious and prominent warning signage and the separation of animal contact from food sale and eating areas.

The South Australian Department of Human Services has prepared comprehensive guidelines for infection control for petting zoos which could provide a useful model for national guidelines.[15]

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A study of the foodborne pathogens: *Campylobacter*, *Listeria* and *Yersinia*, in faeces from slaughter-age cattle and sheep in Australia

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Abstract

In a study of faeces from 475 slaughter-age cattle and sheep from 19 herds or flocks, *Campylobacter* species (*C. jejuni* and *C. coli*) were cultured from all production systems studied and from 73.7 per cent (14/19) of herds or flocks.Within individual properties there was a higher prevalence in cattle than in sheep, with *Campylobacter* being most commonly isolated from feedlot cattle. The median prevalences and ranges were: for dairy cattle, six per cent (0–24%), feedlot beef cattle, 58 per cent (12–92%) pasture beef cattle, two per cent (0–52%), mutton sheep, 0 per cent (0–4%) and prime lambs eight per cent. *Listeria ivanovii* was cultured from one dairy cow but *Yersinia enterocolitica* was not cultured from any animal. *Campylobacter* is the leading bacterial causative agent of acute diarrhoea in humans in many industrialised countries. While the role of cattle and sheep in producing human campylobacteriosis either directly or via contaminated food, remains to be epidemiologically clarified, this study suggests that the production system, particularly for cattle, may be an important consideration. *Commun Dis Intell* 2003;27:249–257.

Keywords: foodborne pathogens Campylobacter, Listeria, Yersinia

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Introduction

Bacterial pathogens associated with human food poisoning may be present in the production animal and therefore be potential sources of contamination. The chain of events from slaughter, through processing, storage and food preparation can allow multiplication of these contaminating organisms.

As part of a larger project investigating Shiga-like toxin producing *E. coli* and *Salmonella* in cattle and sheep, a 'snapshot' study of 19 properties was undertaken to ascertain the prevalence of *Campylobacter* (*C. jejuni* and *C. coli*), *Listeria* (*L. monocytogenes* and *L. ivanovii*) and *Yersinia* (*Y. enterocolitica*) in faeces from slaughter-age animals in New South Wales and Queensland. The aim was to test animals that were about to be slaughtered but had not yet left the farm.

Campylobacter is the leading bacterial cause of acute diarrhoea in man in many industrialised countries including Australia.[1,2,3] *C. jejuni* and *C. coli*, are the causative agents, with *C. jejuni* most commonly isolated. *C. jejuni* and *C. coli* are almost identical in behaviour and epidemiology[4] and therefore discussions in this report relating to *C. jejuni*, apply to both organisms. *C. jejuni* is part of the natural intestinal flora of a wide range of birds and animals[3] and can be pathogenic in these species. Transmission to humans is usually via faecal contamination of food and water,[5] and common sources are poultry, unpasteurised milk, untreated water and contact with domestic pets. Campylobacteriosis is more frequently associated with the consumption of poultry than red meat.[5,6] At present the roles of red meat and production animals in producing this human illness, remain to be epidemiologically clarified.[2,6]

Listeria is widely distributed in the environment (particularly in soil and vegetation) and in many animal species, with *L. monocytogenes* being pathogenic for humans, animals and birds.[7] Listeriosis in humans is primarily via contamination of food during production and processing, particularly dairy products.[8,9] The organism also has been frequently isolated from both red and white meat.[10] Intestinal contents from cattle[11] and dirty hides[12] are considered major contributors to carcass contamination. On-farm sources of *Listeria* include poorly made silage,[13] soil, straw, faeces, and sewage, both raw and treated.[8] The level and impact of listerial carriage in the intestine and faeces of cattle and sheep as a contributor to the contamination of meat in Australia is not currently known.

Yersinia enterocolitica is recognised worldwide as a foodborne pathogen. From human disease studies, pig meat features more prominently than red meat as the likely source.[14,15] Pigs, dogs and cats appear to be the main animal reservoirs for the strains of *Y. enterocolitica* associated with human disease. In contrast, the reported bovine and ovine isolation rates are lower.[15,16]

Materials and methods

Property selection

Nineteen commercial cattle and sheep properties in New South Wales and Queensland were selected to cover all production systems producing red meat: six dairy cattle properties, four feedlot beef cattle properties, four pasture beef cattle properties, two prime-lamb properties and three mutton-sheep properties (Table). These properties were a subset of 215 properties in a larger research study, in which properties were selected with and without a history of *Salmonella* in the preceding two years. Of the 19 properties in this study, nine had a history of *Salmonella*. There was no selection in relation to a history of the three pathogens in this study.

Animal selection and sampling

Animal Care and Ethics approval was given in New South Wales and Queensland. From each property, 25 animals were selected at random from those meeting the following criteria: animals were to be within one month of the expected slaughter date or equivalent age; grazing animals were to be fresh off pasture and to be sampled within four hours of yarding and not yarded overnight; feedlot cattle were to have been on feed for a minimum of 60 days; dairy cattle were to be greater than four years old and in the last 100 days of a lactation cycle. Twenty-five animals were selected because this represented the available number of animals that were ready for slaughter from the properties and was considered a large enough sample to demonstrate potential significant differences. At least 2 g of faeces was collected per rectum

(using a new sterile glove for each animal), and placed in an individually numbered sterile specimen container. Specimens were transported chilled to arrive at the laboratory within 24 hours of collection.

Data collection

Statistical analysis was done using MS Excel 97. Questionnaires for each production system were prepared and analysed in Epi Info 6.04 (Centers for Disease Control and Prevention, Atlanta, Georgia, USA). They were designed to identify possible risk factors associated with the excretion of the bacterial pathogens and included approximately 100 questions under the following headings: property management; environment; management of sampled animals; access to manures; nutrition and feed; water; health status—animal and human. The questionnaire was completed by the producer and veterinarian.

Laboratory testing

Campylobacter culture

Faeces (1.0 g) were inoculated into 10 mL Preston Selective Enrichment Broth (Oxoid), incubated at 42˚C for 48 hours in a microaerophilic atmosphere (CampyGen, Oxoid or a gas mixture consisting of 5% O_2, 10% CO_2, 85% N_2), then subcultured (10 µL) onto Preston *Campylobacter* Selective Agar (Oxoid) plates which were incubated as described above.

Identification of Campylobacter *spp*.

Up to three suspect colonies (based on characteristic morphological appearance) were subcultured. Isolates were considered to be *Campylobacter* spp. if they were oxidase positive, motile and Gram stained smears of suspect colonies revealed small tightly coiled spiral organisms. Isolates were identified as *C. jejuni* or *C. coli* as described by Barrow *et al*.[17]

Listeria *culture*

Faeces (0.1 g) were inoculated into 10 mL Listeria Primary Selective Enrichment Medium (UVM I, Oxoid), and incubated at 30˚C for 24 hours, then. 0.1 mL of the UVM I broth was inoculated into Listeria Secondary Selective Enrichment Medium (UVM II), incubated at 30˚C for 24 hours. This was then subcultured (10 µL) onto an Oxford (Oxoid) plate and a Palcam (Oxoid) plate which were incubated aerobically at 37˚C for 48 hours.

Identification of Listeria *spp*.

Up to five colonies were subcultured onto sheep blood agar. ß-haemolytic colonies that were Gram positive short rods with rounded ends were further tested and identified as *L. monocytogenes* or *L. ivanovii* according to Barrow *et al*.[17]

Yersinia *culture*

Faeces (1.0 g) were added to 10 mL phosphate buffered saline (pH 7.2), incubated at 4˚C for seven days, vortexed to allow large particles to settle, subcultured (10 µL) onto a Cefsulodin-Irgasan-Novobiocin (CIN) (Oxoid) plate and incubated at 32˚C for 48 hours.

Identification of Yersinia *spp*.

Up to three typical colonies were subcultured onto sheep blood agar. Colonies were considered typical of *Yersinia enterocolitica* if they appeared as 'bulls eye' colonies with deep red centres surrounded by a transparent periphery on CIN agar. Isolates were identified as *Yersinia enterocolitica* according to Barrow *et al*.[17]

Results

The results and prevalence rates of *Campylobacter, Listeria* and *Yersinia* in each of the 19 herds and flocks sampled are summarised in the Table.

Table. Prevalence of *Campylobacter, Listeria*, and *Yersinia* in cattle and sheep from 19 herds or flocks, based on testing 25 faecal samples per property

Product type	Property number	Date	District	Approximate stocking rate (DSE/ hectare)	Approximate stocking rate Number of head/m^2	Diarrhoea family/ workers	Campylobacter isolations (%)	Listeria isolations (%)	Yersinia isolations (%)
Dairy cattle	1	1/06/98	Northern NSW	24		Y	24	0	0
	2	26/08/98	Southern NSW	20		Y	24	0	0
	3	2/06/98	Northern NSW	23		N	8	0	0
	4	12/05/98	Central NSW	7		N	4	0	0
	5	26/05/98	Southern NSW	10		Y	4*	4†	0
	6	13/05/98	Southern NSW	20		N	0	0	0
							Median 6 Mean 10.7		
Feedlot beef	1	5/08/98	Southern Qld		0.09	N	92	0	0
	2	16/07/98	Central NSW		0.09	N	76	0	0
	3	21/07/98	South-East Qld		0.10	N	40	0	0
	4	22/04/98	Southern NSW		0.06	N	12	0	0
						Median 58 Mean 55			

Table continued. Prevalence of *Campylobacter, Listeria*, and *Yersinia* in cattle and sheep from 19 herds or flocks, based on testing 25 faecal samples per property

Product type	Property number	Date	District	Approximate stocking rate (DSE/ hectare)	Approximate stocking rate Number of head/m^2	Diarrhoea family/ workers	Campylobacter isolations (%)	Listeria isolations (%)	Yersinia isolations (%)
Pasture beef	1	28/10/98	Southern NSW	2		N	56	0	0
	2	4/08/98	Western NSW	1		N	4	0	0
	3	5/08/98	Western NSW	1		Y	0	0	0
	4	28/10/98	Southern NSW	3		N	0	0	0
							Median 2 Mean 15		
Mutton sheep	1	7/05/98	South-west NSW	1		N	4	0	0
	2	13/05/98	South-west NSW	1		N	0	0	0
	3	10/06/98	Central NSW	3		N	0	0	0
							Median 0 Mean 1.3		
Prime lambs	1	11/08/98	Southern NSW	3		N	8	0	0
	2	30/06/98	Central NSW	4		N	8	0	0
							Median 8 Mean 8		

DSE Dry sheep equivalent.

Y Yes.

N No.

* *Campylobacter coli*, all other *Campylobacter* isolates were *Campylobacter jejuni*.

† *Listeria ivanovii*.

Campylobacter

From the 19 herds or flocks, *Campylobacter* spp. were found in all production systems and 73.7 per cent (14/19) of all herds and flocks tested. *Campylobacter jejuni* was isolated from all production systems and one isolation of *C. coli* was made from one dairy cow. Within individual properties there was an apparent higher prevalence in cattle than in sheep, with *Campylobacter* being most commonly isolated from feedlot cattle. The median prevalences and ranges were: for dairy cattle, six per cent (0%–24%), feedlot beef cattle, 58 per cent (12%–92%) and pasture beef cattle, two per cent (0%–52%), mutton sheep, 0 per cent (0%–4%) and prime lambs eight per cent. No significant differences ($p<0.05$) were detected between pairs of means when the means for all cattle properties and all sheep properties were compared or when different production systems were compared using a 2 tailed t-test for two samples assuming equal variances

For the four feedlots sampled, two factors, stocking density and weather conditions, were identified from the questionnaires as possible contributors to the number of animals shedding *Campylobacter*. Feedlot beef property 4, which had the lowest prevalence of *Campylobacter*, also had the lowest cattle density and was the only feedlot where dry weather conditions prevailed. Of the pastured animals: dairy cattle, pasture beef cattle, mutton sheep and prime lambs; the dairy cattle had the highest stocking rates and also the highest prevalence of *Campylobacter*. Of the four pasture beef properties sampled, property 1 had higher levels of *Campylobacter* (56% of samples positive) than the other pasture beef properties (0%–4% of samples positive). From the questionnaire, a possible risk factor was identified: because of drought conditions, the cattle had been grazed near the house septic tank absorption trench. No other property reported that sampled animals had grazed near an absorption trench. Because of the limited nature of this survey, results from the questionnaires were not proven to be statistically significant associations, but have been reported as possible associations.

Listeria

L. monocytogenes was not isolated from any animal. One dairy cow (dairy cattle property 5) was positive for *L. ivanovii*. Of the 19 properties, two feedlots and three dairies included silage in the ration, but dairy cattle property 5 did not.

Yersinia

No *Yersinia* associated with human disease (*Y. enterocolitica*) was isolated from any of the properties.

Three of the six dairies and one of the four pasture beef properties reported diarrhoea in the family or workers at the property in the two months prior to collecting the cattle faecal samples. No causative agent was identified for any of these human cases. The three dairies, but not the pasture beef property were detected to have animals shedding *Campylobacter*, but there was no statistically significant association with the human illness reported.

Discussion

C. jejuni and *C. coli*, *L. monocytogenes* (to a lesser extent *L. ivanovii*) and *Y. enterocolitica* are bacterial pathogens that cause food poisoning in humans. In our study, *C. jejuni* was commonly isolated and there was a higher prevalence in cattle than in sheep. This study demonstrated a difference between cattle from different production systems, with feedlot cattle having a higher prevalence than either dairy cattle or pasture beef cattle. In contrast, *L. ivanovii* was isolated from only one bovine; *L. monocytogenes* and *Y. enterocolitica* were not isolated at all.

Studies from other countries report a wide variation of *campylobacter* carriage rate in domestic food-producing animals. This may reflect the different geographic/climatic conditions, and management practices.[2] New Zealand abattoir studies in both dairy cattle and sheep demonstrated higher prevalence rates than our study: New Zealand dairy cattle had isolation rates for *C. jejuni* or *C. coli* from rectal swabs of 24 per cent, 31 per cent and 12 per cent during summer, autumn and winter respectively,[18] and New Zealand sheep had prevalence rates of 2.4 per cent for lambs and 14 per cent for adult sheep.[19] In the study of New Zealand dairy cattle, approximately half of the isolates were *C. jejuni* and the other half, *C. coli*. Interestingly, we only isolated *C. coli* from one animal in our study. An abattoir study in Australia by Grau[20] found *C. jejuni* in 54 per cent of calf faecal samples and 12.5 per cent of cow faecal samples and also observed that lot-fed cattle were more likely to have *C. jejuni* in their intestinal tracts and on their carcasses than were pasture-fed cattle. The true proportion of animals carrying *Campylobacter* can only be

ascertained by field studies, as abattoir surveys may give false (higher) figures due to transport associated stress, cross-infection during transport and mixing of animals before slaughter.

The higher stocking rate in dairies, compared with other grazing cattle and sheep, was identified as a possible risk factor for *Campylobacter* prevalence. High stocking density and wet weather were identified as possible contributors to the number of feedlot animals shedding *Campylobacter*. Both these factors would increase the level of moisture in the pen and encourage the survival of the organisms. In addition to these possible contributing factors, feedlot rations are high in carbohydrate compared with pasture and therefore may provide a more suitable environment in the gastrointestinal tract for *Campylobacter* to survive and proliferate. Three pasture beef properties had no or low levels of *Campylobacter*, but one property had a prevalence of 56 per cent. From the questionnaire a possible risk factor was identified for this property: the cattle grazed near the house septic tank-absorption trench.

Campylobacter prevalence has been reported to be seasonal, with both humans and animals[3,18] having higher levels during the warmer months. Our study was conducted in the cooler months of the year (between May and October), so we could anticipate higher levels during warmer months. Nielsen *et al*[21] demonstrated an overlap between serotypes of *C. jejuni* found in humans, poultry, and cattle, indicating that poultry and cattle should be considered in the transmission via food to humans. This 'snapshot' study was too small to demonstrate statistically significant differences ($p<0.05$) between production systems but the trends observed indicate that cattle, and in particular feedlot cattle, must be considered a potential source of *Campylobacter* for humans. The animals in this study were healthy animals still on-farm and ready for slaughter, and as such would be carrying *C. jejuni* into the abattoir environment.

It is interesting to note that, from the results of the questionnaire, diarrhoea in humans was recorded from three of the six dairy properties, one of three pasture beef properties, but not from the other production systems. The causative agents for the diarrhoea in humans were unknown and there was no statistically significant association between diarrhoea in humans and any of the pathogens isolated from animals on the same property. Nonetheless, it was found that the three dairy properties with cases of human diarrhoea also had animals shedding *Campylobacter*. This organism is the most common bacterial cause of diarrhoea in humans and Thompson[22] found a strong association between human campylobacteriosis and living on a farm. Our report of diarrhoea in dairy workers warrants further investigation. The management of dairy cattle exposes the workers to cattle faeces much more commonly than in any other production system.

L. monocytogenes was not isolated from any property in this study. *L. ivanovii* was cultured from one dairy cow but was not isolated from any sheep.[23] The dairy cow was on pasture and fed supplementary pellets but no silage. It had been stressed by drought followed by recent rain. In Australian sheep, outbreaks of clinical listeriosis have been reported following both floods24 and drought.[25] *L. ivanovii* is a rare human pathogen[26] and is considered to be less virulent than *L. monocytogenes*.[27] Mutton is considered the most common food associated with the presence of *L. ivanovii*.[28]

Studies in other countries have found relatively high levels of *L. monocytogenes* in cattle and sheep. These levels may be a reflection of more intensive production systems, housing inside in winter and supplementary feeding. Housing animals is not a common practice in Australia. High prevalence rates in cattle faeces have been reported in Yugoslavia 19 per cent,[29,30] Germany 33 per cent,[31] Denmark 51 per cent,[32] Canada 14.5 per cent,[33] Scandinavia 3.1 per cent (spring to autumn on pasture) to 9.2 per cent (winter indoors).[34] One study in Hungary,[35] showed 90 per cent of 50 healthy sheep investigated during summer were excreting *Listeria* in their faeces, nasal mucus, vaginal mucus or milk. For the properties in our study, cattle and sheep production systems in Australia did not favour the carriage of *Listeria*, although silage was used in half the feedlots and dairies.

No *Yersinia* associated with human foodborne disease (*Y. enterocolitica*) was isolated from any of the properties. This result is in keeping with other studies that indicate that cattle and sheep are an unlikely source for human infection.[15,16]

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An outbreak of *Salmonella* Typhimurium phage type 135a in a child care centre

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On 12 December 2002, the Brisbane Southside Public Health Unit commenced a cluster investigation of four notifications of *Salmonella* Typhimurium infection amongst children under five years of age within a defined geographical area in south-west Brisbane. Initial investigations found that these children attended the same child-care centre (CCC). An Outbreak Control Team conducted investigations that included surveillance, microbiological testing of suspected cases, inspection and environmental sampling of food preparation facilities and other equipment within the CCC, sampling of food specimens, review of menus and audits of selected food suppliers.

The CCC had 30 full time staff and was licensed for 146 children (71 and 75 children in different wings). In all 350 children aged from six weeks to six years attended per week. Sixteen people associated with the CCC reported symptoms of gastroenteritis between 20 November and 7 December 2002, including four staff, one parent and 11 children attending the CCC. Ten cases of *S.* Typhimurium phage type 135a infection were identified including one parent of a symptomatic CCC attendee. Onset dates of confirmed cases ranged from 1 to 7 December 2002 (Figure). The age range of the CCC attendee cases was one to five years. The cases belonged to different age cohorts in both wings of the CCC.

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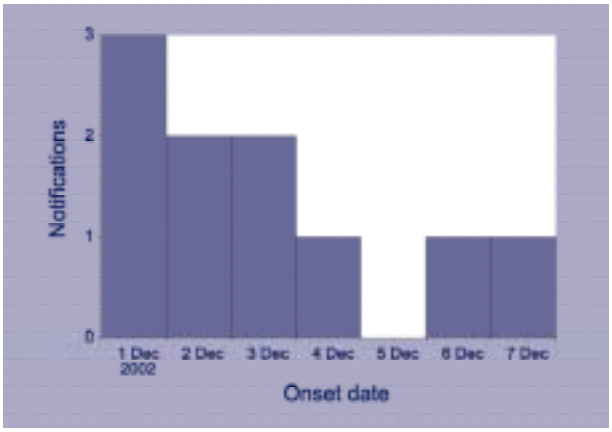
Figure. Onset date of *Salmonella* Typhimurium phage type 135a notifications associated with a south-west Brisbane child-care centre, December 2002



The child cases had attended the CCC on a number of occasions during the week preceding onset of their infection and all had attended the centre on 28 November 2002. All children had consumed lunch and snacks provided by the centre. Three adult staff had been unwell in the period 20 to 26 November 2002 with symptoms of gastrointestinal infection. The food handler who prepared the meals on 28 November 2002 subsequently developed gastroenteric illness on 29 November 2002. All four staff tested negative for *Salmonella* on specimens collected up to three weeks after their illness.

Inspection of the CCC food preparation facilities revealed a number of items of concern including poor temperature control, inadequate refrigeration capacity, poor cleaning procedures and inadequate pest management. Eggs used in the preparation of egg sandwiches on 28 November were purchased from a local butcher, supplied through a vendor who purchased the eggs from a local egg farm. The egg farm did not clean the eggs prior to distribution. Eggs obtained from the CCC tested negative for *Salmonella* spp. However, subsequent drag swabs of the egg farm were positive for *S*. Typhimurium phage type 135a from two of three sheds.

At the time of publication, 135a is not an internationally recognised phage type of *S*. Typhimurium. It is based on a variation of the standard phage pattern for phage type 135, first reported in connection with contaminated orange juice in 1999.[1] During 2001 to 2002, 149 cases of *S*. Typhimurium phage type 135a were detected in Queensland, an average of 6 cases per month. There were six unrelated cases of *S*. Typhimurium 135a reported in Queensland during December 2002. (personal communication, John Bates, Public Health Microbiology, Queensland Health). Subsequently, outbreaks of *S*. Typhimurium 135 have been reported in connection with the use of inadequately cleaned eggs in aged care and community settings.[2,3] These reports prompted the Outbreak Control Team to audit the egg supply of the CCC.

We suggest that this outbreak of *S*. Typhimurium phage type 135a in a child-care centre may be associated with the introduction of the organism on eggs purchased from a local supplier who did not comply with recommended cleaning procedures for eggs.[4] Cross contamination within the facility is suggested by the occurrence of enteric illness in the sole food handler for the centre. Person-to-person transmission cannot be excluded as a factor in this outbreak as it is documented in the parent of one CCC attendee who did not consume food from the CCC but was the parent of a child case.

Legislation and standards are in place for food safety in child-care centres which are monitored and regulated in Queensland by local government.[5] The Commonwealth Department of Health and Ageing is currently developing food safety program templates for child-care centres and family day care. As yet, these do not specifically mention egg quality. The issue of the safe production of eggs in Queensland is being addressed through the development of a Food Safety Scheme for Eggs.[6]

Eggs purchased for child-care centre use should have been produced under a quality assurance program that guarantees that the eggs have been cleaned and checked for cracks. There is no current legal requirement for eggs to be produced under a quality assurance program, which may make it difficult for people to determine the quality of the eggs they are buying. Food handlers should be aware of this issue when purchasing and preparing eggs, in particular of the importance of confirming that the eggs have come from a quality assured supplier and of washing their hands after handling eggs that may not have been produced under a quality assurance program. However, under no circumstances should uncleaned or cracked eggs be used for children attending child-care centres as this age group is particularly vulnerable to the serious consequences of *Salmonella* infection.

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Human infestation with bird mites in Wollongong

Charles R Watson

Abstract

This is a report of a case of bird mite infestation which occurred in Wollongong in mid-December 1996. The individual suffered hundreds of bites, most of which were marked by itchy red papules 3–4 mm in diameter. Tiny mobile parasites (< 1 mm) collected from the skin and adjacent bedroom wall were identified as bird mites from the family *Gamasidae*, most probably from the genus *Ornithonyssus*. The source of the infestation was a starling nest under the eaves adjacent to the bedroom. The report summarises the ways bird mite bites can be distinguished from other insect and arachnid bites. If bird mite infestation is not correctly diagnosed, families who attempt to repeatedly treat it as if it were lice or scabies may incur considerable expense until the source of infestation is eliminated. *Commun Dis Intell* 2003;27:259–261.

Keywords: bird mites, rickettsia

Introduction

Bites from insects and mites can cause individuals considerable discomfort, and if the infestation is not accurately identified and treated, the episode may prove very disruptive and expensive for a family. A case of bird mite infestation is presented in order to highlight the diagnostic issues surrounding this relatively uncommon cause of bites in humans. Bird mites are arachnids, and like spiders they have eight legs and a combined abdomen and thorax. They can easily be distinguished from spiders because the head and thorax-abdomen are fused to form an oval body, whereas in spiders the head is clearly separated from the thorax-abdomen. The most common bird mites found in Australia are the red poultry mite (*Dermanyssus*) and the northern fowl mite (*Ornithonyssus*).

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Case report

The infestation was experienced by the author, who was at the time a resident of East Corrimal, a suburb of Wollongong in New South Wales. On two successive nights in mid-December 1996, the author was repeatedly woken by very itchy bites around the axilla, trunk and groin, as well as formication (the sensation of ants crawling over the skin) on the face. An initial search did not reveal any visible cause of the bites, but on the morning following the second evening of bites, the author found a number of tiny animals (< 1 mm) crawling on the anterior thigh. These animals were collected with transparent adhesive tape and later examined with a microscope. At this time, 24 hours after the attack began, there were about 50 obvious bites on the body, most of which were marked by red papules 3–4 mm in diameter.

Examination of the specimens collected from the skin by the author showed that they were bird mites. Examination of illustrations in standard texts[1,2] indicated that they most probably belong to the genus *Ornithonyssus*.

After being alerted to the diagnosis of bird mite infestation the author recalled that there was a starlings' nest under the eave of the roof about 3 metres from the bed, and that the young chicks had recently left the nest (as judged by the cessation of squawking that had accompanied the morning feeding of the chicks in the previous week). When the wall near the site of the nest was examined carefully, it was found to be covered with hundreds of tiny crawling animals of the same size as those found on the skin. They were collected, examined and found to be the same as those collected from the skin.

Immediate treatment of the subject with permethrin cream ('Lyclear') and insecticide treatment of the infested room was sufficient to control the problem. However, it should be noted that removal of an infested nest and fumigation of the roof cavity and adjacent rooms would be recommended in order to eradicate the source of an infestation.

Discussion

The significance of bird mite infestation in humans is not so much the annoyance and discomfort caused by the bites, but the expense of repeated treatment if the problem is thought to be lice or scabies, and the source of infestation is therefore not eliminated.

Some bird mites have been found to carry viral or rickettsial pathogens, but their significance as a vector for human infestation has not been demonstrated.[2] However, it has been suggested that *Ornithonyssus bacoti* (the tropical rat mite) may be responsible for the transmission of *Rickettsia akari*,[1] so the potential for transmission of rickettsial disease should not be ignored.

Blood-sucking mites are ectoparasites of a wide range of domestic and wild birds, as well as small mammals and certain reptiles. Bird mites are an important cause of ill health in poultry, and infestations result in decreased egg production, weakness, and susceptibility to infection. Under unusual circumstances, such as when breeding birds and their nestlings desert a nest, mites may attack other vertebrate hosts, including humans.[3,4] Insecticide treatment of infested humans and temporary vacation of infested premises are not sufficient to eliminate the problem because adult mites can survive for weeks or months without feeding.[3,5] Identification of bird mites can be attempted with a microscope and illustrations from a standard parasitology text.[1,2] Because most bird mites are less than a millimetre long, a magnification of 40 to 100 times is necessary for accurate identification.

Once the parasite has been identified, the source of infestation must be found. This is usually a poultry yard or a bird's nest.

Anecdotal evidence indicates that bird mite bites are often misdiagnosed by general practitioners, and the bites are treated as if the problem was scabies or body lice. The problem with misdiagnosis is that treatment of the individual and their clothing and bedding will not eliminate the source of the infestation, and it may recur, requiring further treatment.

The issue was discussed with a number of pharmacists in the Wollongong area in 1996; all were aware of the bird mite problem because they sell insecticidal creams and washes to affected families during the bird mite season. The Wollongong pharmacists reported that the problem is most common in December. Veterinarians in the Wollongong area encounter bird mite infestations in domestic animals and are well aware of its seasonal incidence.

The differential diagnosis of itchy bites in humans is complicated by the fact that some of the causes are relatively rare. The major alternatives to be considered are scabies, fleas, body lice, mosquitoes, sand flies, horse flies, spiders, centipedes, bed bugs, ticks, midges, bird mites, and harvest mites.[4,1] Diagnosis requires information on the circumstances in which the bites occurred, and the nature and distribution of lesions. In difficult cases, an entomologist should be consulted. Goddard emphasises the importance of excluding imaginary insect or mite infestations ('delusory parasitosis').[1] The characteristics of a bird mite infestation are shown in the Box.

Box. Characteristics of bird mite infestation

- Commonly in late spring or early summer.
- Parasites are barely visible (< 1 mm long) but can be found crawling on the skin.
- The parasites do not burrow into the skin.
- The parasites have a characteristic appearance – they can be collected with transparent adhesive tape and recognised with the aid of an identification key and a low power microscope.
- The bites usually produce small itchy papules.
- Source of infestation is usually obvious – such as a bird nest or poultry yard.

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An assessment of the implementation of the pneumococcal conjugate vaccination program for Aboriginal and Torres Strait infants in North Queensland

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Abstract

A cohort of 199 Aboriginal and Torres Strait Islander infants, born in north Queensland in August and September 2002, were followed-up to ascertain the uptake of the 7-valent pneumococcal conjugate vaccine (7vPCV) by certain ages. Although 70 per cent of the cohort had received a dose of 7vPCV by three months, only 50 per cent had received three doses by seven months. Most (approximately 90%) of the children who received the vaccine by three and five months were given it at the same time as the other two scheduled injectable vaccines, and most (84%) of the children received three doses of 7vPCV by 12 months of age. However, 18 per cent of the cohort had not received any vaccines (other than those given at birth), another 10 per cent had received the other scheduled vaccines but no 7vPCV by three months of age, and 38 per cent had received the other vaccines but not two doses of 7vPCV by five months. A variety of measures are described that have been put in place to attain optimal coverage of Aboriginal and Torres Strait Islander infants in north Queensland with 7vPCV and to improve timeliness. *Commun Dis Intell* 2003;27:262–266.

Keywords: pneumococcal conjugate vaccinate, surveillance, vaccination, pneumonia

Introduction

The licensure of the 7-valent pneumococcal conjugate vaccine (7vPCV; Prevenar®) in Australia in early 2001 provided a means to prevent much of the pneumonia and perhaps some of the otitis media affecting young Aboriginal and Torres Strait Islander children. Draft recommendations on the use of 7vPCV in a National Childhood Pneumococcal Vaccination Program[1] gave priority to Aboriginal and Torres Strait Islander children as the main target population for the Program in recognition of the high burden of invasive pneumococcal disease in these children. The Commonwealth provided funding for the Program's vaccines, in particular 7vPCV, and Aboriginal infants in the Northern Territory began to receive 7vPCV in early June 2001.[2]

Because 7vPCV was not only a completely new vaccine, but also a very expensive one, a decision was made to roll-out the vaccine methodically in north Queensland over several months. Before any 7vPCV was distributed to a vaccine service provider, that provider had to attend a specific training session delivered by Tropical Public Health Unit (TPHU) staff, and had to be conversant with all aspects of 7vPCV including transportation, storage and administration. To support the training, an interim Standing Drug Order for the vaccine was prepared and distributed so that endorsed registered nurses could participate in the delivery of the vaccine. Various other provider and parent resources were also developed; the roll-out of 7vPCV took place in north Queensland from July to September 2001.

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This report details an assessment of the roll-out that was undertaken by determining the uptake of 7vPCV in a defined cohort of Aboriginal and Torres Strait Islander infants. The specific objectives were to determine:

- the percentages of the cohort that had received 7vPCV by three, five and seven months of age, as recommended;[1]
- the percentages of those children who were vaccinated with 7vPCV by three and five months of age that had also received the other two recommended injectable vaccines (i.e. DTPa-hepB and Hib) at the same time;
- the percentage of the cohort that had received three doses of 7vPCV by 12 months of age; and
- the percentages of the children attending each category of vaccine service provider that had received the vaccine by three, five and seven months of age.

Methods

All Aboriginal and Torres Strait Islander births that took place in August and September 2001 in four major public obstetric units in north Queensland were identified from hospital separation data held at the hospitals (Table 1). The uptake of a first dose of 7vPCV by three months of age was determined using the statewide computerised immunisation register, Vaccination Information and Vaccination Administration System (VIVAS).[3] Similarly, the uptake of the second and third doses (separated from the previous dose by at least a month) by five and seven months of age, respectively, were determined. The information on VIVAS also indicated whether the vaccine had been administered at the same time as the other two recommended vaccines.

The various vaccine service providers who had provided vaccines to the children in the cohort were categorised as 'community health/Royal Flying Doctor Service (RFDS)' (the latter provides much of the child health services in remote settings in north Queensland), 'Aboriginal and Torres Strait Islander health services', 'general practices' and 'other' (mainly hospitals that opportunistically vaccinate children). The percentages of the cohort children who had been given 7VPCV by each category of provider by three, five and seven months were determined.

If any child in the cohort had apparently not received 7vPCV by three months of age, the primary health care provider of the mother was contacted, where possible, to determine whether the vaccine had in fact been given. Similarly, if any child had apparently not received the vaccine by five (or seven) months of age, the vaccine provider who administered the vaccines by three (or five) months of age was contacted.

The details of any child in the cohort who had not received any vaccines (apart from the vaccines given at birth) by three months of age, and who did not appear to have a designated vaccine service provider, were forwarded to the local Community Health services for follow-up. Where possible, these services undertook catch-up vaccination with all overdue vaccines.

Results

The cohort consisted of 199 infants at three months of age (Table 1), but because two of the infants died (one from pertussis) before reaching five months, the cohort consisted of 197 children thereafter.

Although 70 per cent of the cohort had received a dose of 7vPCV by three months, only 50 per cent had received three doses by seven months (Table 2). Of the 60 children who had not received a dose of 7vPCV by three months, 36 (60%) had not received any vaccines other than those given at birth, and another 20 (33%) had received the other scheduled vaccines (i.e. DTPa-hepB, Hib and OPV) by three months. Of the 139 children who had received a dose of 7vPCV by three months, 127 (91%) had received it at the same time as the other two injectable vaccines.

Of the 78 children who had not received a second dose of 7vPCV by five months of age, two (3%) had not received any vaccines other than those given at birth, and 18 (23%) had received the other scheduled vaccines by five months of age but no 7vPCV. Another 57 (73%) had received the other scheduled vaccines but had received only one dose of 7vPCV by five months of age. Of the 119 children who had received two doses of 7vPCV by five months, 110 (92%) had received the second dose at the same time as the other two injectable vaccines.

Of the 99 children who had not received a third dose of 7vPCV by seven months of age, 60 (61%) had received only two doses, 28 (28%) had received only one dose, and 11 (11%) had not received any doses, respectively, by this age. Altogether, 166 (84%) of the cohort (197 children) received three doses of 7vPCV by 12 months of age; another 19 (10%) received two doses by 12 months.

With the exception of those who attended general practices, the percentage of children who were given 7vPCV by each category of vaccine service provider declined at each successive age milestone (Table 3). However, of those Aboriginal and Torres Strait Islander infants who consulted general practitioners, only about a quarter were given 7vPCV at each age milestone.

Table 1. Aboriginal and Torres Strait Islander births used for the assessment of the roll-out of 7vPCV in north Queensland (birth cohort August and September 2001)

Hospital	Aboriginal and Torres Strait Islander births	
	n	%
Cairns Base	90	45
Kirwan Womens (Townsville)	53	27
Thursday Island	29	15
Mt Isa Base	27	13
Total	**199**	**100**

Table 2. The number and percentage of the cohort children given the appropriate number of doses of 7vPCV, by each successive age

Outcome	First dose by 3 months		Two doses by 5 months		Three doses by 7 months	
	n	%	n	%	n	%
Vaccinated appropriately	139	70	119	60	98	50
Not vaccinated appropriately	60	30	78	40	99	50
Total	**199**	**100**	**197**	**100**	**197**	**100**

Table 3. The number and percentage of the total number of children given 7vPCV, by vaccine service provider category and vaccination age

Vaccine service provider category	First dose by 3 months		Two doses by 5 months		Three doses by 7 months	
	n/N	%	n/N	%	n/N	%
Community health/ RFDS	99/115	86	92/132	70	77/134	57
ATSIHS	26/30	87	22/35	63	16/34	47
General practice	6/22	27	5/20	25	5/19	26
Other	8/11	73	0/7	0	0/9	0
Total	**139/178**	**78**	**119/194**	**61**	**98/196**	**50**

n Number.

N Total number.

RFDS = Royal Flying Doctor Service; ATSIHS = Aboriginal and Torres Strait Island Health Service

Discussion

This assessment has revealed several encouraging outcomes. Most (70%) of the children received the first dose of 7vPCV by three months, most (approximately 90%) of the children who received the vaccine by three and five months were given it simultaneously with the other two scheduled injectable vaccines, and most (84%) of the children received three doses of 7vPCV by 12 months of age.

However, the assessment had also revealed several problems. Firstly, it is a concern that 18 per cent (36 children) of the cohort had not received any vaccines, other than those given at birth, by three months of age. Children who are late in starting their vaccinations are at high risk of not completing the recommended standard vaccination schedule by two years of age.[4]

Secondly, it is worrying that 20 children (10% of the cohort) had received the other scheduled vaccines but no 7vPCV by three months of age, and that 75 (38% of the cohort) had received other scheduled vaccines but not two doses of 7vPCV by five months. There are several possible reasons for this, but probably the most important overall is a reluctance to administer three injectable vaccines simultaneously. A consequence of not administering 7vPCV at the same time as the other two vaccines is a considerable risk that the child may not return for the 7vPCV; this is particularly likely if the child comes from a highly mobile and hard-to-reach population, such as some Aboriginal and Torres Strait Islander children.

It appears that in general, vaccine service providers have more concerns about administering multiple injectable vaccines simultaneously to children than parents do.[5,6] This has considerable implications because vaccine service providers' attitudes towards immunisation are powerful determinants of parental decision making about vaccination for their children. Nevertheless, if vaccine service providers can be convinced that multiple vaccinations are safe and effective, their concerns can be overcome and they can administer three (and even four) injectable vaccines simultaneously in a way that appears to be quite acceptable to parents.[7,8]

Thirdly, the poor uptake in those children who attended GPs is an on-going concern. There are two possible reasons for this problem. Because of the cost of the vaccine, it is not possible to have 7vPCV in stock in every general practice in Queensland and therefore GPs may not have had the vaccine available to administer to Aboriginal and Torres Strait Islander children at the same time as the other scheduled vaccines. (This would have contributed to the failure to deliver 7vPCV simultaneously, as outlined above, particularly by three months of age.) Since this assessment, those GPs who regularly see Aboriginal and Torres Strait Islander patients, and those who have already initiated a 7vPCV series, have received doses of 7vPCV as stock-in-hand.

However, a greater problem is that many GPs apparently did not ascertain whether a child is of Aboriginal and Torres Strait Islander descent, and therefore could not identify whether the child was eligible for funded 7vPCV. This problem is openly acknowledged by some GPs, and the Queensland Divisions of General Practice have offered to assist GPs to ask about a child's Indigenous status. Meanwhile, the Personal Health Record given to each mother of a newborn Aboriginal and Torres Strait Islander infant in north Queensland has been modified to indicate that the infant is of Aboriginal and Torres Strait Islander descent. The modified Personal Health Record indicates that the infant is eligible for 7vPCV as well as other recommended childhood vaccines.

The decline in the uptake of 7vPCV with each successive dose in the children who attended Community Health/RFDS vaccine service providers is probably an artefact. This is because the Community Health services were given the particular responsibility of following-up the children who had not received 7vPCV. Therefore the Community Health/RFDS vaccine service providers were given 17 extra children between who were late in starting their vaccinations, and they would have been very difficult to vaccinate appropriately by age.

Nevertheless, the decline in uptake with each successive milestone age in the children who attended the other vaccine service providers is of concern. Although only approximately 17 per cent of the cohort attended the five Aboriginal and Torres Strait Island Health Services in north Queensland, the uptake of 7vPCV in these children fell from 87 per cent by three months to 47 per cent by five months of age. Similarly, none of the approximately eight Aboriginal and Torres Strait Islander children opportunistically vaccinated in hospitals in north Queensland by five and seven months of age received 7vPCV.

The importance of 7vPCV for Aboriginal and Torres Strait Islander children has been reported in a recent edition of the TPHU newsletter, and TPHU staff have emphasised it in in-service training of practice staff and Aboriginal and Torres Strait Islander Health Workers.

Although most Aboriginal and Torres Strait Islander infants ultimately receive three doses of 7vPCV, for too many it is given late. This problem must be corrected; not only does severe pneumococcal disease, in particular pneumococcal meningitis, occur at a much earlier age in Aboriginal and Torres Strait Islander children,[9] but otitis media also begins very early in life in these infants.[10]

Acknowledgments

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Prevalence of markers of Q fever exposure in the Kimberley, Western Australia

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Abstract

Although a large pastoral industry exists in the Kimberley region of Western Australia, there is no previously published information about the prevalence of immune markers for Q fever exposure in this region's population. This paper identifies the prevalence of, and factors associated with, positive immune markers of Q fever, and reports the uptake of Q fever vaccination by eligible subjects in the Kimberley region of Western Australia. Data regarding Q fever risk-factors were obtained using a standard questionnaire. Immunity and previous exposure to Q fever were assessed using both serology and a skin test, in accordance with accepted protocol. Fifty-nine subjects underwent Q fever pre-vaccination testing.The prevalence of a positive skin and/or blood test,indicating past exposure was 66 per cent (95% CI 52% – 78%). After controlling for age and having lived on a farm at any time, employment in the pastoral industry was the only factor significantly associated with being skin and/or blood test positive (OR=24.6, 95% CI 3.0 – 204). Acceptance of vaccination was high, with 75 per cent of eligible subjects undergoing vaccination. The high prevalence of immune markers for Q fever in the Kimberley in this sample indicates that the disease is present in the region, despite the last recorded case being in 1986. Ensuring that Q fever vaccination is readily accessible to pastoralists, abattoir workers and other at-risk groups is a challenge that needs to be met by these industries and health services of the region. *Commun Dis Intell* 2003;27:267–271.

Keywords: Q fever, immunity, vaccination

Introduction

The Kimberley region in the far north of Western Australia, has supported a large pastoral industry for many years. This industry contributes significantly to the economy, history and the culture of Aboriginal and non-Aboriginal people of the region. High prevalences of immune markers to Q fever (indicating previous exposure) and/or high Q fever incidences, have been documented in other Australian regions with a large pastoral industry, such as central and south-west Queensland and northern New South Wales.[1,2,3,4] No published information about Q fever immunity exists for the Kimberley or any other regions of Western Australia.

In 2002, health services in Western Australia were informed that the National Q Fever Management Program would be extended to include free vaccination for pastoral workers until the end of the 2002–03 financial year. The Kimberley Public Health Unit, in partnership with the Kimberley branch of the Pastoralist and Graziers Association (PGA), Community Health Services throughout the region, and the Kimberley Division of General Practice conducted a Q fever vaccination program in the latter months of 2002.

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The Kimberley region is sparsely populated, with a population of 32,000 scattered over 420,000 square kilometres. There are pastoral stations throughout the region, in sub-tropical as well as semi-arid areas. A few members of the pastoral community have ready access to health services provided by nurses and Aboriginal health workers in remote area clinics, visiting primary health care staff employed by the Department of Health Western Australia, the Royal Flying Doctor Service and Aboriginal Community Controlled Organisations. However, most people living and working on pastoral properties, can only access primary health care by travelling to one of the six towns in the region. This may involve many hours driving on rough roads or flying by light aircraft at the individual's own expense. There is very little private general practice in the region, with almost all medical practitioners being employed by the Department of Health Western Australia or a non-government organisation.

The aims of this paper are:

- to identify the prevalence of immune markers to Q fever in the Kimberley region of Western Australia;
- to identify the factors associated with the presence of positive immune markers; and
- to report the uptake of vaccination by eligible subjects during the first four months of the program.

Methods

Subjects eligible for the National Q Fever Management Program were informed about the program by word-of-mouth, radio interviews and advertisements in local newspapers and professional newsletters. Pre-vaccination screening was conducted at a variety of workplace settings such as the 2002 PGA Annual General Meeting at Go Go Station and an abattoir (the only one in the region, a small enterprise that operates for about six months each year and employs less than 20 people), and at Community Health Centres in several towns throughout the region.

An employee questionnaire, pre-screening and vaccination, and consent forms were obtained from the National Q Fever Management Program's website, http://www.qfever.org.

All subjects completed these forms prior to Q fever pre-vaccination screening. Data regarding Q fever risk-factors were obtained using the employee questionnaire. Immunity and previous exposure to Q fever were assessed using both serology and a skin test, in accordance with the protocol described by Marmion.[5] Testing was conducted by doctors and community health nurses under the supervision of a medical practitioner experienced in this field. Skin tests were read after seven days. Serological testing for antibodies to *Coxiella burnetii* (immunofluorescent assay, followed by confirmatory enzyme immunoassay in borderline cases) was done at PathCentre, Perth.

The outcome of interest was the presence of immune marker(s) to Q fever indicating past exposure. Subjects with a positive skin test and/or a positive blood test were classified as immune 'positives', i.e. have been previously exposed to Q fever, and were not eligible for Q fever vaccination. Subjects with negative blood and skin tests were classified as non-immune 'Negatives' and were offered Q fever vaccination.

Data were entered into SPSS. The association between being 'positive' and Q fever risk factors, i.e. independent variables, such as occupation, length of time working in their current industry, length of time living on a farm, consumption of unpasteurised milk etc, was analysed using logistic regression.

In accordance with ethics guidelines, patient-identified information was restricted to two of the authors, who were responsible for interpretation of all test results.

Results

Fifty-nine subjects were tested between 23 August and 26 November 2002. None reported a past history of Q fever, or having been tested or vaccinated for Q fever. No-one was refused testing because of a medical contra-indication to being tested. Fourteen (24%) reported a past history of an influenza-like illness lasting more than seven days. The demographic characteristics of these subjects and their risk factors for Q fever are shown in Tables 1 and 2.

Table 1. Demographic characteristics of subjects, n=59

Characteristic	Frequency	
	n	%
Sex		
Male	47	80
Female	12	20
Age (years)		
Range	16–65	
Mean/median	38/35	
<20	5	8
20–29	17	29
30–39	13	22
40–49	11	19
50+	13	22
Occupation		
Pastoral industry	33	59
Abattoir worker	12	21
Other (includes vets, stock inspectors, nurses)	11	20

Table 2. Subjects' exposure to Q fever risk factors

Risk factor	Yes		No	
	n	%	n	%
Grew up/live on a farm,* n=54	43	80	11	20
Regularly visit(ed) a farm,* n=55	37	67	18	33
Feedlot work, n=55	11	20	44	80
Stock/farm* work, n=55	36	65	19	35
Tannery work, n=55	0	0	55	100
Animal transport, n=55	26	47	29	53
Shearing, n=55	6	11	49	89
Animal husbandry, n=55	17	31	38	69
Livestock buying, n=55	11	20	44	80
Milking cows/goats, n=55	12	22	43	78
Collecting cattle/sheep manure, n=55	16	29	39	71
Slaughtering livestock privately, n=55	32	58	23	42
Dressing kangaroo carcasses/pelts, n=55	16	29	39	71
Consuming unpasteurised cow/goat milk, n=55	23	42	32	58
Other activity associated with livestock production, n=55	16	29	39	71
	Range		**Mean/median**	
Work duration in current industry (years)	0–50		18/19	
Years lived on a farm*	0–48		18/18	

* Sheep, cattle, goat or diary farm

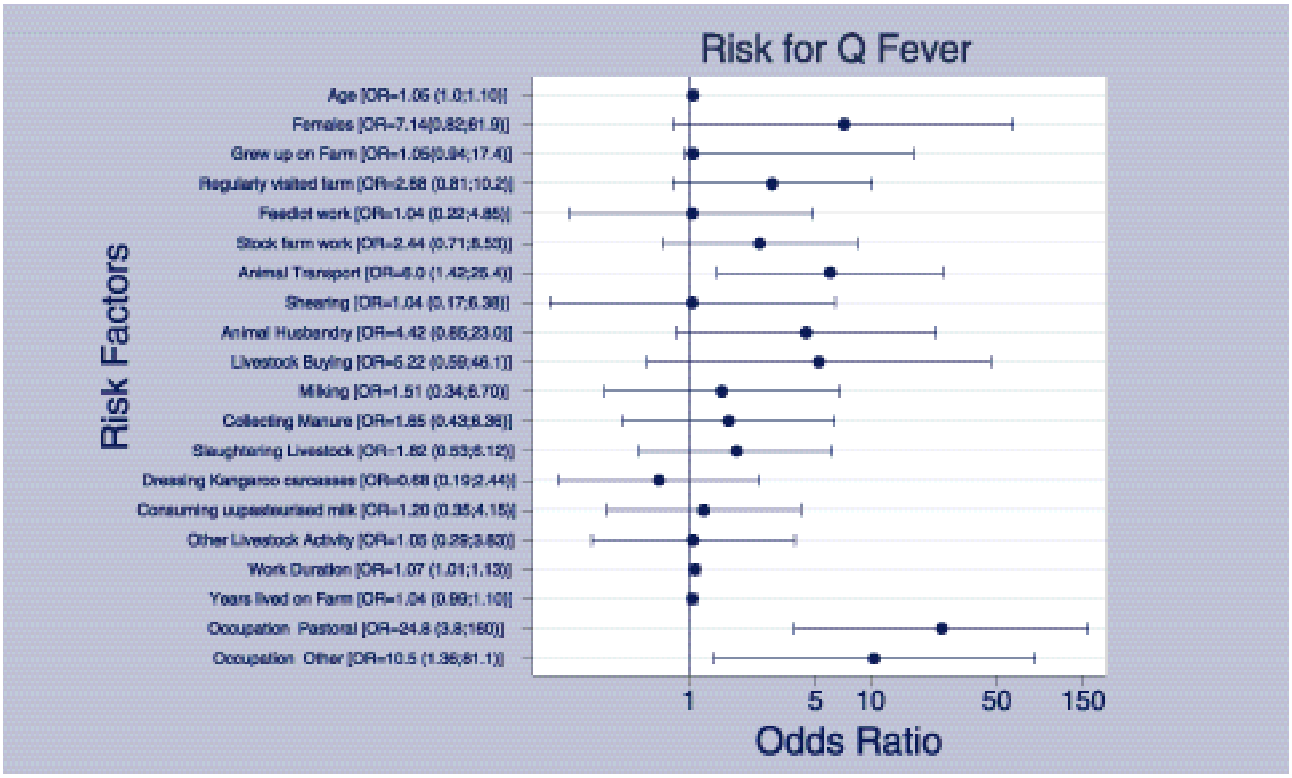
Of the 59 subjects tested, 12 did not present at the appropriate time for reading of the skin test. All but one of these 12 subjects had negative serology. Of the 59 subjects for whom blood test and the 47 for whom skin test results were available, three (5%) had positive serology and 30 (64%) had a positive skin test. Combining skin and serology results in the 47 patients for whom both tests were available, 31 subjects (66%, 95% CI 52%–78%) were 'positive' and 16 (34%, 95% CI 22%–48%) were 'negative'. At least seven (23%) of the 'positive' subjects were born, and had lived, in the Kimberley all or almost all their lives.

Logistic regression was used to explore the association between the outcome variable 'positive' and the independent variables listed in Tables 1 and 2. Univariate analysis indicated a significant association between 'positive' and the occupation groups; 'pastoral industry', and 'other', and 'ever having worked in animal transport' (p=0.001, p=0.024 and p=0.015, respectively), as shown in the Figure. The associations between 'positive' and age, sex and 'grew up/live on a farm' were of borderline significance (p=0.069, p=0.074 and p=0.06, respectively). The association between 'positive' and tannery work was not calculable as none of the subjects had worked in a tannery.

Multivariate analysis including age, occupation groups (abattoir worker, pastoral industry and other), 'grew up/live on a farm' and the interaction between occupation and 'grew up/live on a farm' showed that the occupation group 'pastoral industry' was the only variable consistently and significantly associated with 'positive' (p=0.003), i.e. after controlling for age and having grown up or lived on a farm, the odds of a pastoral worker being 'positive' was 25 times that of any other occupational group (OR=24.6, 95% CI 3.0 – 204).

Of the 16 subjects who were eligible for vaccination following testing, 12 were vaccinated, one refused and three were planning, but had not yet had the opportunity, to be vaccinated. Nine (75%) of the 12 subjects who were vaccinated were abattoir workers, two (17%) were pastoral workers and one (8%) was a vet.

Figure. Univariate analysis of risk factors for Q fever exposure



The ORs for age, work duration and years lived on a farm are for each year of increase.

Farm refers to a sheep, cattle, goat or diary farm.

Discussion

This first report of the prevalence of immune markers of Q fever exposure in the Kimberley region shows a high prevalence of positive immune markers despite the absence of any recorded Q fever cases since 1986 (personal communication, Jag Atrie, Communicable Disease Control Program, Department of Health Western Australia) and lack of anecdotal reports of the disease from medical practitioners in the region. The prevalence of positive immune markers in our sample is higher than the 18.5 per cent reported from central Queensland where reported Q fever incidence is much higher than in the Kimberley.[2]

The reasons for the above findings may include the stoic nature of remote pastoralists who tend to avoid taking time off work and seeking health care for minor, self-limiting symptoms. For example, one of the subjects, a station manager, had clinical and serological findings consistent with Q fever infection, but said he did not intend seeking medical advice until the end of the mustering season. Relatively poor access to health care also plays a role in the remote area residents not presenting for health care unless symptoms are severe. Lack of awareness of Q fever among health staff may have contributed to lack of testing and notification of cases. In addition, pastoral practices in the Kimberley are different to those of south-west Queensland and northern New South Wales, with fewer cattle per unit of land area and the bulk of pastoral work being done in the 'dry season' only (personal communication, Nathan Webb-Smith, Beefwood Park Station). This may influence pastoralists' exposure to Q fever in such a way as to predispose to the development of sub-clinical infection followed by natural immunity, rather than clinical disease.

A large proportion of non-immune subjects were vaccinated indicating high community acceptance of Q fever vaccination in this region. However, achieving high vaccination coverage, especially among pastoral industry workers will be a challenge for both the health and pastoral industries. Reasons for this include remoteness of workplaces, the small number of staff at each station, the logistical difficulties inherent in Q fever vaccination (as a minimum of two visits seven days apart are required for adequate pre-vaccination screening) and the high staff turnover in both industries (for example, only five of the eight health staff trained in Q fever pre-vaccination screening in August 2002, were still working in the region in January 2003, and of the six individuals tested at one pastoral station, two had ceased employment prior to the reading of the skin test—the manager's reaction to this indicated that this level of staff turnover was not uncommon).

One of the limitations of this study is the small sample size. Further testing of at-risk groups in the Kimberley would be useful in confirming these findings. Testing of livestock and wildlife, and sampling of stockyard dust would also be useful as there is limited information about the environmental prevalence of the causative organism (*Coxiella burnetii*) in the Kimberley.

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Melioidosis in northern Australia, 2001–02

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Abstract

Melioidosis, caused by the Gram negative bacterium *Burkholderia pseudomallei*, is endemic in northern Australia. Using data collated from centres in Western Australia, the Northern Territory and Queensland, this report describes the epidemiology of this disease between 1 November, 2001 and 31 October, 2002.There were 47 cases seen during this period with an average annual incidence of 5.8 cases per 100,000 population. In Indigenous Australians, an incidence of 25.5 cases per 100,000 population was seen. The timing and location of cases was generally correlated with rainfall across northern Australia.A case-cluster in a Queensland community was associated with post-cyclonic flooding. Risk factors included diabetes, alcohol-related problems and renal disease. Pneumonia (51%) was the most common clinical diagnosis.The mortality rate attributable to melioidosis was 21 per cent, although a number of other patients died of underlying disease. Despite improvements in recognition and treatment, melioidosis is still associated with a high morbidity and mortality, particularly in Indigenous Australians. *Commun Dis Intell* 2003;27:272–277.

Keywords: melioidosis, Burkholderia pseudomallei, *epidemiology*

Introduction

Melioidosis is caused by the organism *Burkholderia pseudomallei*, a Gram negative bacterium present in soil and surface water. The disease is endemic in northern Australia and South East Asia.[1]

There is a spectrum of presentations from acute sepsis to more chronic disease; infection may involve any organ but primarily involves the lungs and intra-abdominal organs. Risk factors for infection include diabetes, hazardous alcohol intake and chronic renal disease.[2]

The majority of cases are associated with the wet season and exposure to surface water and mud, implying that acute infection most commonly occurs soon after exposure. The incubation period of acute disease is between 1 and 21 days.[3] However, latent infections with presentation delayed for months or years have also been described.[3] Two outbreaks in the endemic region of Australia have been attributed to contamination of the community water supply with *B. pseudomallei*.[4,5]

This report, using notification data, describes the epidemiology of melioidosis in the 2001–02 season in northern Australia.

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Methods

Data were collated data from the following sources: the Menzies School of Health Research, Darwin; the Tropical Public Health Unit, Queensland Health; and the Department of Health and PathCentre, Perth. Rainfall data were obtained from the Bureau of Meteorology.[6] Population statistics, derived from the 2001 national census, were obtained from the Australia Bureau of Statistics.[7] Locations of towns were taken from the Gazetteer of Australia, 2001.[8] Cases within the northern region of Australia were included. The endemic region is generally regarded as the area north of 20˚S. All cases were from within this region, except one north Queensland case from Mackay (21˚10'S) where autochthonous cases had been seen in previous years.

Melioidosis is a notifiable disease in Queensland, the Northern Territory and Western Australia. A case was included if cultures from any body site were positive for *B. pseudomallei* and the patient presented with a illness consistent with melioidosis during the period between 1 November 2001 and 31 October 2002. Location was taken from the patient's place of residence; for travellers, the place of presentation. Serological diagnoses were not included as previous work has suggested that positive serology is neither sensitive or specific in the diagnosis of melioidosis.[9,10]

The timing of the wet season varies in northern Australia; in Western Australia and the Northern Territory, it is defined as the six month period between 1 November and 30 April, in north Queensland it is defined as from 1 December to 31 May.

Results

Epidemiology

In the 12 months to 31 October 2002 there were 47 cases of melioidosis in the northern areas of Australia: Western Australia (1 case); Northern Territory (23 cases); and Queensland (23 cases). Epidemiological features of these cases are summarised in the Table.

Table. Cases of melioidosis, northern Australia, 1 November 2001 to 31 October 2002, by state or territory

	State or territory			
	NT	Qld	WA	Total
Number of cases	23	23	1	47
Mortality	4 (17%)	7 (30%)	1	12 (26)
Attributed to illness	3	6		10
Other cause	1	1	0	2
Wet season cases*	18 (78%)	21 (95%)	1	40 (87%)
Median age (range) years	51 (3–79)	56 (30–87)	22	52 (3–87)
Male	15 (65%)	17 (74%)	1	33 (70%)
Total population	148,641	596,498	41,969	809,334
Rate (per 100,000 population per year)	15.5	3.9	2.4	5.8
Indigenous	13 (57%)	12 (52%)	0	25 (53%)
Indigenous rate (per 100,000 population per year)	42.0	22.3	0	25.5
Paediatric (<15 years)	1	0	0	1

* Wet season: Northern Territory and Western Australia: November–April, Queensland: December–May; date of onset not evident for one Queensland case

In addition, a number of patients were notified but excluded; this included three patients who had been notified previously and re-presented with relapsed disease. *B. pseudomallei* was also isolated from the sputum of a 14-year-old boy with cystic fibrosis; because he was otherwise asymptomatic, it was considered that the isolation indicated colonisation, and the case was not included. One patient presented to a hospital in Perth (31˚S) with an exposure history from an overseas endemic area. There were also two cases elsewhere in southern Australia where culture-confirmed melioidosis was epidemiologically linked to travel to the Northern Territory.

The median age was 52 (range 3 to 87) years and 33 (70%) were male. There was one child (3 years of age) with melioidosis during this time. There were 25 infections involving Indigenous Australians. The rate of melioidosis was 5.8 cases per 100,000 population overall and 25.5 cases per 100,000 population in Indigenous Australians.

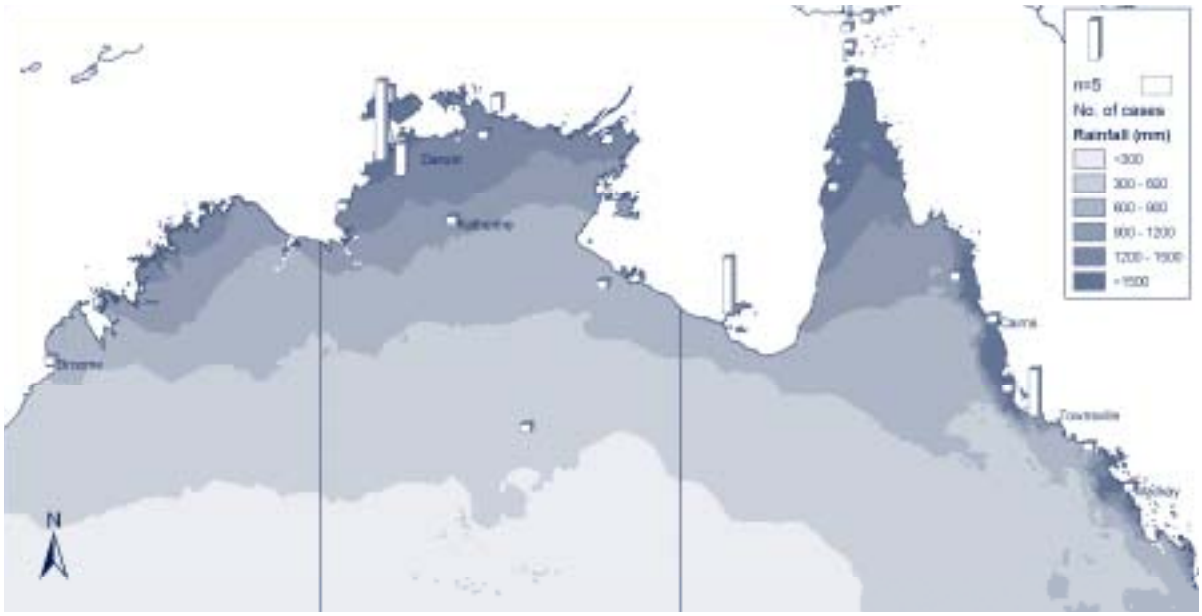
In the Northern Territory, most cases were seen around Darwin. Ten cases were from the Darwin urban region and four from the rural areas surrounding Darwin. One patient presented to Tennant Creek Hospital (19˚39'S); although locally acquired cases have been seen there previously, this patient had recently travelled from further north in the endemic area. Six patients developed their illness in remote Aboriginal communities and were transferred to Royal Darwin Hospital for further management.

In Queensland, there were three main geographical foci of cases. Seven cases (30% of the Queensland total) were from the one Gulf community, six (26%) were from Townsville and adjacent suburbs, four (17%) were from the Torres Strait and the Northern Peninsula Area, and the remaining six (26%) were from other areas. Of note, only one case was acquired in Cairns.

Rainfall and incident cases

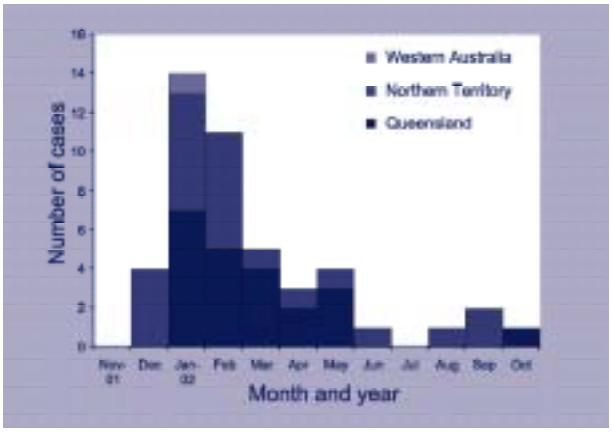
The majority (87%) of cases were seen during the wet season months (Table). During this time, rainfall across the Top End of the Northern Territory was between 150–600 mm less than the 1961–1990, 30-year median rainfall. On the western side of Cape York, rainfall was between 75–300 mm greater than the median rainfall.[6] The location of cases together with average annual rainfall is detailed in Figure 1.

Figure 1. Geographic distribution of cases of melioidosis and average annual rainfall in Australia



In the Northern Territory, the number of cases was low compared to previous years; in the preceding 12 months there were 33 cases and in the 1997–98 season there were 48 cases. In addition, there were no cases in November for the first time since 1989; this was attributed to the lower than average rainfall. Most cases occurred during the Northern Territory wet season (n=18), with 12 cases in January and February, coinciding with the relatively late onset of the monsoon rain (see Figure 2).

Figure 2.Monthly number of cases of melioidosis, northern Australia, November 2001 to October 2002*



* Date of onset not evident for one Queensland case

In Queensland, the seasonal peak started in January (see Figure 2). However, five of the seven cases in January, and seven (30%) of the total number of cases, were from the same community in the Gulf country north of Mt Isa. The first two cases at the community became unwell within three days of a tropical cyclone that passed over the community, leading to extensive flooding, in early January. Similarly, two of the three cases in Townsville in February occurred very soon after heavy rainfall in the city.

Clinical features

Risk factors for infection included diabetes (n=20; 43%), alcohol-related problems (n=14; 30%), renal disease (n=9: 19%), chronic obstructive airway disease (n=8, 17%), immunosuppression (n=4; 8.5%) and malignancy (n=2; 4.2%). Only seven patients (15%) did not have obvious medical risk factors; only two of these did not have a history of occupational or recreational exposure to mud/water.

Most cases had pneumonia (n=24; 51%) with other infections involving bone/joint (n=3; 6.3%), prostate (n=5; 11%), skin/soft tissue (n=3; 6.3%), gastrointestinal tract (n=1), spleen (n=1) and the central nervous system (n=1).

Overall, 12 patients (25%) died; 5 (41%) from the overwhelming acute infection. Two deaths were felt to be attributable to other causes (underlying end stage renal disease and malignancy). Two patients died prior to or on admission to hospital. The case fatality rate in Indigenous Australians was higher than in other patients, but this difference was not statistically significant (33% vs 17%, Fisher's exact: p=0.3).

Excluding two patients who died prior to or during admission in Queensland, and two patients with mild pneumonia who did not require admission, the remaining 43 patients spent a total of 1,182 days in hospital. The median duration of hospital stay was 18 days (range 1 to 114).

Discussion

Melioidosis is endemic in northern Australia. The average annual rate in the Top End of the Northern Territory is 16.5 cases per 100,000 population with a rate of 34.5 cases per 100,000 population in the 1997/98 season.[11] In the Torres Strait communities in northern Queensland between 1995 and 2000, the annual rate was 42.7 cases per 100,000 population.[12] These rates are much higher than those documented in northeast Thailand (3.5–5.5 cases per 100,000 population)[13] and Singapore (1.7 cases per 100,000 population).[14]

Rainfall during the 2001–02 wet season varied from previous seasons, with later and lower than average precipitation in the Top End of the Northern Territory. This was reflected in the lower number of cases in the Northern Territory, with no cases seen in November for the first time since records commenced in 1989. However, the higher than average rainfall around Cape York was not associated with increased numbers of cases in this area possibly indicating the influence of as yet undefined factors other than rainfall per se. Ongoing studies are examining the role of other environmental factors, such as rainfall rate, soil type and physical properties of drinking and surface water, in the epidemiology of melioidosis.

As previously noted,[9] Indigenous Australians are over-represented in the melioidosis cases. In the defined area of northern Australia, it was estimated that 12.4 per cent of the total 2001 population at risk were Aboriginal and Torres Strait Islander people, whereas 53 per cent of the cases in this report occurred in Indigenous people. Although this may partly be related to exposure, risk factors such as diabetes and renal disease are also more common in this population. Other important risk factors in this and the wider population include high alcohol intake, and occupational and recreational exposures.

The prevalence of risk factors, namely diabetes, alcohol-related problems, chronic lung disease and chronic renal disease is similar to that described previously in Australia.[11] Similarly, the clinical features of this disease, with pneumonia present at presentation in half the cases, with smaller percentages of patients with skin and soft tissue infections, osteomyelitis and genitourinary infection reflect patterns noted previously.[2] The clinical pattern of disease varies from the Thai series, where many of the cases have no obvious clinical focus.[15] In addition, paediatric disease is much less common in Australia in comparison to Thailand.[16]

The diversity of presentations with melioidosis is illustrated by a number of the cases during this year. A 51-year-old man, presented to his local medical officer with impotence following a flu-like illness, and a prostatic abscess was subsequently diagnosed. A 3-year-old child presented with ataxia and brainstem encephalitis following a culture-positive scalp boil. Two patients identified as having had previous mycobacterial infections, one with *M. leprae* and another with *M. terrae*; isolated case reports have noted this association that may reflect a common host susceptibility to these intracellular pathogens.[17,18,19] Additionally, the presentation of non-acute melioidosis may mimic that of tuberculosis. A number of patients in previous years had been treated for presumed tuberculosis, but subsequent cultures were negative for *M. tuberculosis* and positive for *B. pseudomallei* (unpublished data).

The only culture-confirmed case of melioidosis presenting in the endemic region of Western Australia during the 12 month period was a tourist, who presented following recent travel from the Northern Territory. Despite appropriate antibiotics and intensive supportive therapy he had a rapidly fatal septic course. The occurrence of melioidosis in this and other travellers, although uncommon, reinforces the need for clinicians throughout Australia to be mindful of this disease in patients that have been in endemic areas.

The mortality from melioidosis in the Northern Territory has halved over the past decade. Historically, most deaths have been attributable to the complications of severe sepsis due to overwhelming infection.[2] With improvements in recognition of melioidosis, the earlier commencement of therapy, and improved intensive care management of patients with severe sepsis, an increasing proportion of deaths are attributable to causes other than the sepsis syndrome, such as the complications of the prolonged treatment course and underlying disease.

There is considerable economic cost associated with melioidosis. Hospital admission, including the need for intensive care, is likely to represent only a fraction of the cost associated with this disease. Treatment of melioidosis often requires outpatient administration of expensive antibiotics and extensive follow-up, and may involve patients in remote settings.

Ongoing studies are aimed at determining the environmental factors important in the development of this disease, such as contamination of potable water supplies, and exploring better therapeutic strategies. Efforts are also continuing to improve the awareness of melioidosis in communities to reduce exposure to this organism in high-risk individuals during the wet season.

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The Australian Technical Advisory Group on Immunisation

History

The Australian Technical Advisory Group on Immunisation (ATAGI) was established in 1997 by the Commonwealth Minister for Health to advise and make recommendations on the technical and scientific elements of the National Immunisation Program. Since 1997, ATAGI recommendations have informed the development and implementation of every technical change to the Australian Standard Vaccination Schedule (ASVS) and the National Immunisation Program. To date these have included:

- a change to the timing of the second measles-mumps-rubella vaccination from 10–13 years to 4 years of age in support of the Measles Elimination Campaign;
- a change to acellular pertussis containing vaccines as a routine ASVS requirement;
- the introduction of routine hepatitis B vaccination for infants;
- the introduction of a high-risk infant and children's vaccination program against pneumococcal disease using conjugate and polysaccharide pneumococcal vaccines;
- a change to the recommendation for tetanus and diphtheria boosting; and
- the addition of meningococcal C conjugate vaccine at 12 months of age on the ASVS for all children.

The ATAGI has been operating in an environment where change to national immunisation requirements is becoming increasingly possible due to the availability of new and better vaccines. The ATAGI has developed procedures and practices to deliver timely evidence based advice to government on immunisation 'best buys'. The development of this advice has included consideration of the safety, public health impact and cost-effectiveness of any new vaccination intervention being considered for funding under the National Immunisation Program umbrella.

A major element of ATAGI's work program has been and will continue to be the revision and updating of the *Australian Immunisation Handbook* for the National Health and Medical Research Council (NHMRC). ATAGI produced the 7th edition of the handbook. This edition was published following NHMRC endorsement in March 2000. The 8th edition of the handbook has been developed and is expected to be published following NHMRC endorsement in late 2003.

Role

The ATAGI's current terms of reference were updated in early 2000. Its primary role is to:

- provide advice to the Minister for Health and Ageing on technical issues relating to the National Immunisation Program and other related issues;
- operate cooperatively with the NHMRC's Health Advisory Committee to:
  - provide technical advice to the NHMRC on issues relating to immunisation as required, and
  - to enable NHMRC endorsement of key documents such as the Australian Standard Vaccination Schedule;
- consider vaccines likely to be approved in Australia and provide advice on their use and to consider burden of disease issues related to immunisation;
- liaise with the National Immunisation Committee, the Communicable Diseases Network Australia and the Australian Drug Evaluation Committee on matters relating to the implementation of immunisation policies and procedures;
- liaise with the Therapeutic Goods Administration and the Department of Health and Ageing's Pharmaceutical Benefits Branch on matters relating to the availability and cost-effectiveness of vaccines intended for inclusion on the Australian Standard Vaccination Schedule;
- liaise with the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases on immunisation and related burden of disease issues; and
- provide advice to the Department of Health and Ageing on immunisation policy, including funding.

The ATAGI has convened specialist working parties to consider and report on new vaccination initiatives. These specialist groups may be jointly convened and managed with other peak advisory groups such as the Communicable Diseases Network Australia and/or the National Immunisation Committee, or be convened and managed by ATAGI itself. To date, these groups have developed new vaccination recommendations for poliomyelitis, influenza, pneumococcal disease, varicella, meningococcal disease and pertussis.

Membership

The membership of ATAGI currently comprises:

- a Chairperson, nominated and endorsed by the Minister for Health and Ageing who is a member of the NHMRC's Health Advisory Committee;
- the Chairperson of the National Immunisation Committee;
- three clinical paediatricians with particular experience in immunisation;
- two members with expert immunisation program delivery expertise;
- a public health physician;
- a clinical microbiologist;
- a representative from the National Centre for Immunisation Research and Surveillance;
- a representative from the Australian Divisions of General Practice;
- a representative from the Royal Australian College of General Practitioners;

- a representative from the Consumers Health Forum; and
- a representative from the Department of Health and Ageing.

In addition, the ATAGI may appoint expert working parties comprising members of the ATAGI and other persons nominated by the ATAGI or other peak advisory groups with specific expert knowledge, to address particular issues.

The Chair of the ATAGI is Professor George Rubin, Professor of Public Health and Community Medicine, University of Sydney, Director, Effective Healthcare Australia.

Current issues

Issues currently on ATAGI's agenda include:

- the review and revision of the National Health and Medical Research Council's 2000–2002 Australian Standard Vaccination Schedule;
- the review and revision of the National Health and Medical Research Council's 7th Edition of the *Australian Immunisation Handbook*;
- the development of an electronic version of the *Australian Immunisation Handbook* that will be available for access by the Internet or as a stand alone program distributed on a CD ROM;
- the development of recommendations to further develop and strengthen Australia's place as an immunisation leader in the region;
- the development of new processes to enhance the timely development of evidence based recommendations on immunisation for Australian governments;
- to continue to review and enhance recommendations on best practice immunisation service delivery; and
- the development of systems and processes to assist members to effectively contribute to ATAGI decision making outside of the formal meeting paradigm.

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Who reads what in *CDI?* The 2002 readership survey

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Background

Communicable Diseases Intelligence (*CDI*) has been published by the Commonwealth Department of Health and Ageing since 1976. The bulletin was published fortnightly until 1997, when publication changed to four-weekly and then monthly from 2000. In 2001, the publication schedule changed to quarterly. *CDI* has been available electronically since 1996 on the Communicable Diseases Australia Website: http://www.cda.gov.au/. *CDI* is made available to subscribers (both institutional and personal) in print and electronic formats free of charge.

CDI aims to provide information about the incidence of and risk factors for communicable diseases in Australia to inform and assist those with responsibility for communicable disease control in a wide variety of settings. In order to assess how useful subscribers find the bulletin, a survey of the readers of *CDI* was conducted late in 2002. This was the first readership survey since 1995, when 1,476 responses from 4,872 subscribers were analysed and published.[1]

Methods

A questionnaire was sent to all 2,167 subscribers on the mailing list as at October 2002, with a subscription renewal form in the third issue of *CDI* in 2002 (Appendix). A postage-paid envelope was supplied with each questionnaire. Four questions were asked: the respondents areas of interest and profession, how frequently the subscriber read the various sections of *CDI* and whether the subscriber would be willing to pay for *CDI* in the future.

The questionnaire was mailed to subscribers in early October. Responses received up to 24 December 2002 were included in the analysis.

Results

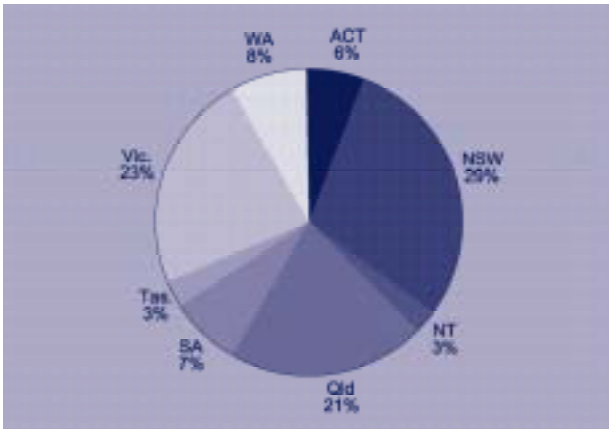
Response rate

In all 566 responses (26% of the total sent) were completed and returned. Of the 2,167 questionnaires posted, 901 were sent to institutions. No response was expected from these institutional nor from overseas subscribers. Therefore, the response rate for individual subscribers within Australia was 56 per cent.

Geographical distribution

There were 1,905 (88%) subscribers in Australia and 262 (12%) subscribers outside Australia who received the questionnaire. The breakdown of subscribers within Australia by state and territory is shown in Figure 1. The geographical distribution of *CDI* subscribers reflects the Australian population distribution.

Figure 1. The distribution of Australian subscribers to Communicable Diseases Intelligence, 2002, by state or territory of mailing address

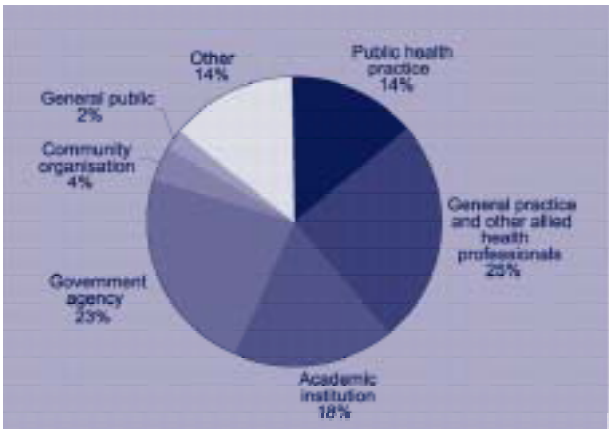


Subscriber's professions

Readers were asked to indicate which professional category best described their position. Readers were able to choose more than one option and all readers completed this section.

There were 595 responses from the 566 subscribers. The breakdown of subscribers by professional category is shown in Figure 2.

Figure 2. The self-described professional categories of respondents to the *Communicable Diseases Intelligence* readership survey, 2002



The 'other' category (n=84) included laboratory workers (n=33), infection control practitioners (n=9) and retired people (n=12). *CDI* subscribers are mostly in Australia (88%) although there is a significant proportion of subscriptions (262, 12%) overseas. Within Australia, subscriptions to individuals (1,004, 53%) only slightly outnumbers subscriptions from institutions (901, 47%). Institutions included libraries (n=307, 34%), laboratories (n=203, 23%), hospitals (n=189, 21%), government departments (n=148, 16%) and businesses (n=54, 6%).

Subscriber's interests

Readers were asked to indicate their major area of interest. Options were given according to the classification of diseases in the National Notifiable Diseases Surveillance System (NNDSS). More than one area could be chosen. All respondents completed this section. There were 1,818 responses from 566 subscribers giving an average of 3.2 responses per subscriber. The areas of interest of subscribers are shown in Table 1.

Subscribers reading habits

Readers were asked how often they read certain sections of *CDI*. For each section they were asked whether they read the section always, sometimes or never. The results are shown in Table 2.

Table 1.The major areas of interest in communicable diseases of respondents to the *Communicable Diseases Intelligence* readership survey, 2002

Area	Number of responses	% of subscribers
Bacterial infections*	339	60.0
Vaccine preventable diseases	324	57.0
Bloodborne infections	261	46.0
Foodborne disease	258	45.5
Vectorborne disease	228	40.0
Sexually transmitted infections	198	35.0
Quarantinable infections	139	24.5
Zoonoses	15	2.5
Other†	56	10.0
Total	**1,818**	

* Tuberculosis, legionellosis, meningococcal infection and leprosy

† Responses included: 'general overview' (19, 3%) or a specific disease (13, 2%).

Table 2.The frequency that respondents to the *Communicable Diseases Intelligence* readership survey, 2002 read various sections of *CDI*

Section	Frequency*		
	Always %	Sometimes %	Never %
Outbreak reports	64.0	33.0	2.0
Annual reports	61.0	34.0	5.0
Editorials	51.5	44.0	4.5
Short reports	51.5	45.0	3.5
Overseas briefs	50.5	45.0	4.5
Articles	50.0	48.0	2.0
Surveillance reports	48.5	48.5	3.0

Access to *Communicable Diseases Intelligence*

When asked if they would continue their subscription to *CDI* if a payment was required for subscription, 269 (47.5%) readers indicated that they would, 230 (40.5%) answered that they would not and 67 (12%) did not respond. As a subsidiary question, readers were whether they would want access to *CDI* in print or electronically. There were 509 responses to this question. The printed version was preferred by 215 (42%), 251 (49%) preferred the electronic version, and 19 (4%) would want access to both formats.

Discussion

This survey provides the editorial staff of *CDI* with valuable information for the future development of the bulletin.

CDI has a significant proportion of readers outside Australia. These include both individuals and institutions, mostly in the South East Asia and Western Pacific Region. *CDI* is therefore an important medium by which the international community gains information about communicable disease in Australia.

Within Australia, a large proportion of subscriptions (47%) go to institutions. These include hospital and laboratories (44%), libraries, (34%) and government departments (16%). Of interest were the 54 subscriptions held by businesses.

There is a greater diversity in the professional backgrounds of *CDI* subscribers than was expected. Subscribers in public health practice or in a government agency, who were assumed to be the majority of subscribers, comprised only 37 per cent of the *CDI* readership. Many of the institutional subscribers to *CDI*, who did not respond to the survey may be public health professionals.

One quarter of subscribers described themselves as in general practice or other allied health professionals, which suggests that *CDI* is read by primary care physicians and other health professionals outside of public health. The 1995 readership survey (Herceg, 1996) also noted that 37 per cent of the readership were medical practitioners. Clearly, *CDI* is an important and continuing resource for information on communicable disease in Australia for a wide group of health professionals.

The *CDI* subscribers have a broad range of interests across the spectrum of communicable disease reported on in *CDI*. A recent analysis of the content of *CDI* in 2002 indicates that the proportion of published articles reflects the interests of the readership, although no articles on quarantinable or zoonotic diseases were published in 2002 (*CDI* Editorial Advisory Board discussion paper, 2003 unpublished).

The editors were gratified to see the high proportion of responders to the survey who regularly read the various sections of *CDI*. Outbreak reports were the most often read despite the changes in publication frequency of *CDI*. It would appear that readers value all parts of the bulletin and these will be retained in the future. The ranking of 'most read' sections of *CDI* was similar to that seen in the 1995 readership survey.

In terms of access, a large proportion (41%) would prefer not to pay for *CDI* and, in contrast to the 1995 survey a larger proportion prefer to access *CDI* electronically (49%) than in the print format (42%). This no doubt reflects general changes in attitudes and practice with regard to electronic media and is important information for future planning of *CDI*.

We thank all who participated in this survey and welcome feedback from the readers of *CDI* at any time.

Reference

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Appendix: Communicable Diseases Intelligence *2002 readership survey questionnaire*

1. What are your major areas of interest?

☐ Bloodborne virus
☐ Bacterial infections (TB, legionella, leprosy, meningococcal)
☐ Foodborne disease
☐ Quarantinable disease
☐ Vectorborne disease
☐ Sexually transmissible infections
☐ Vaccine preventable diseases
☐ Other (specify) _____

2. What professional category best describes your current position?

☐ Public Health Practice (i.e. Public health physician or a public health nurse)
☐ General practice and other allied health professionals
☐ Academic institution
☐ Government agency (this includes local, State/Territory or Federal Government)
☐ Community organisation
☐ General public
☐ Other (specify) _____

3. Do you read the following sections of *CDI*?

	Always	Sometimes	Never
Annual reports (e.g. NNDSS, meningococcal, TB, etc.)	☐	☐	☐
Articles	☐	☐	☐
Short reports	☐	☐	☐
Outbreak reports	☐	☐	☐
Editorials	☐	☐	☐
Surveillance reports	☐	☐	☐
Overseas briefs	☐	☐	☐

4. If *CDI* were available to subscribers only on payment of a subscription.

Would you continue your subscription Y/N

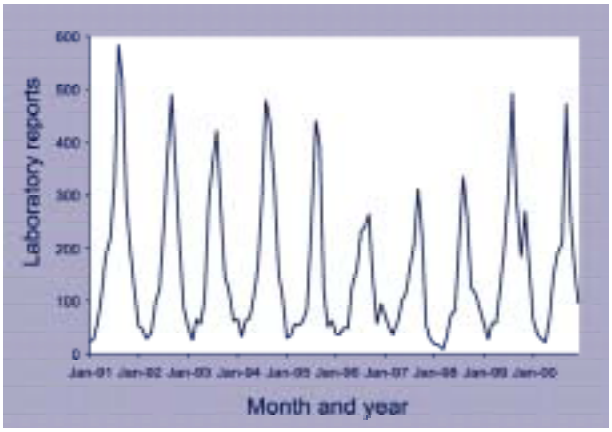
Would you want access to ☐ print version of *CDI* ☐ electronic version of *CDI* ☐ both

Errata

Australia Notifiable Diseases Status, 2001

In the Australia's notifiable diseases status, 2001: annual report of the National Notifiable Diseases Surveillance System (*Commun Dis Intell* 2003;27:71), the Laboratory reports to the Laboratory Virology and Serology Reporting Scheme of rotavirus infection, Australia, 1991 to 2000, by month of specimen collection shown in Figure 71 was incorrect. The correct figure is shown below.

Figure 71. Laboratory reports to the Laboratory Virology and Serology Reporting Scheme of rotavirus infection, Australia, 1991 to 2000, by month of specimen collection



Letter to the Editor: Varicella surveillance: simpler than you think?

In the Letter to the Editor on varicella surveillance, (*Commun Dis Intell* 2003;27:100) the heading for Figure 3 was incorrect. The correct figure heading should read 'Number of notified cases of chickenpox, South Australia, 1 January 2002 to 31 January 2003, by month of onset'.

Pneumococcal disease in Australia: current status and future challenges

In the article 'Pneumococcal disease in Australia: current status and future challenges' published in the last issue of *Communicable Diseases Intelligence* (*Commun Dis Intell* 2003;27:78-88). The last sentence on page 80 which reads, 'In Victoria, preliminary data from a National Health and Medical Research Council funded hospital-based case-cohort study suggest that 23vPPV has an effectiveness of 80 per cent when measured against an outcome of hospitalisation for pneumonia' is erroneous and should be ignored.

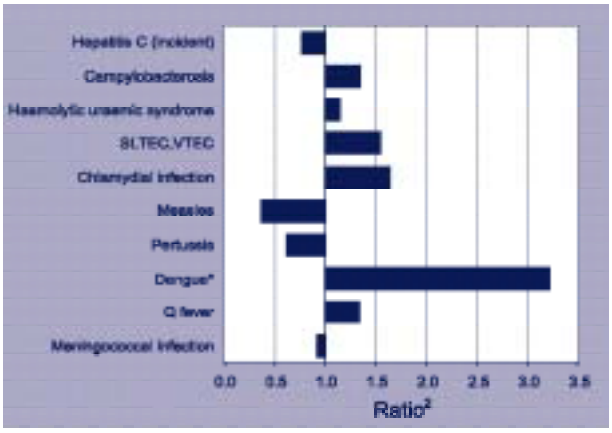
Communicable Diseases Surveillance

Highlights for 1st quarter, 2003

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

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia. NNDSS collates data on notifiable communicable diseases from State or Territory health departments. The Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme which collates information on laboratory diagnosis of communicable diseases. In this report, data from the NNDSS are referred to as 'notifications' or 'cases', and those from ASPREN are referred to as 'consultations' or 'encounters' while data from the LabVISE scheme are referred to as 'laboratory reports'.

Figure 1. Selected[1] diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 January to 31 March 2003 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

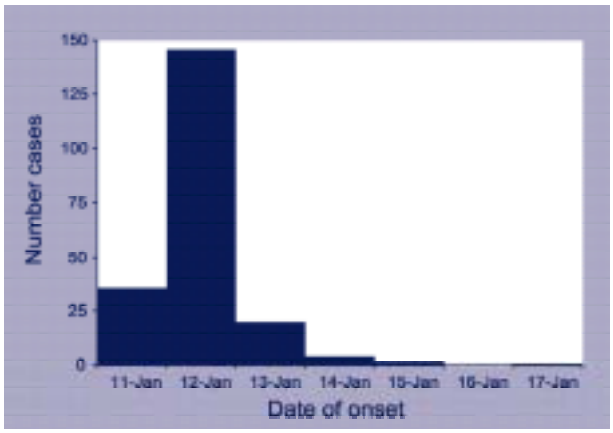
Salmonellosis

There were 2,747 notifications of salmonellosis (Table 2) in the first quarter of 2003—an increase of 6.7 per cent over the five year average for the same period. Most cases were sporadic.

A large outbreak of salmonellosis was associated with Vietnamese take-away food in Footscray, Victoria. Up to 213 cases were notified between 11 and 17 January 2003 (Figure 2), and 22 patients were hospitalised. One death, possibly associated with the outbreak occurred, in a 49-year-old male.

The causative agent, *S.* Typhimurium phage type 135, was isolated from egg-butter made in the restaurant which was used as an ingredient in pork rolls. As a result of the outbreak the premises were closed for a period and staff were trained in food handling procedures. Broader surveillance of premises selling similar products also occurred.

Figure 2. Notifications of cases of *S.* Typhimurium phage type 135, Fitzroy, Victoria, January 2003



Listeriosis

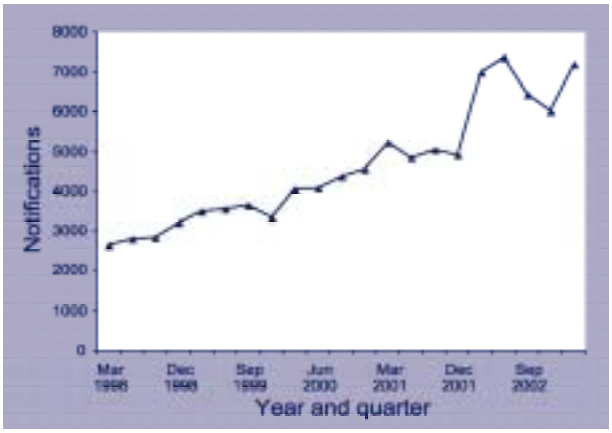
There were 20 cases of listeriosis reported during the first quarter 2003. Three materno-foetal infections were reported, two from Western Australia and one from Victoria. The remaining cases occurred in older (age range 58–82 years) or immunocompromised persons. No outbreaks were identified.

Sexually transmissible diseases

Chlamydial infections

Notifications of chlamydial infections have continued their increase of the last few years (Figure 3). There were 7,230 notifications for the first quarter of 2003, which represents an increase of three per cent over the number (7,215) for the same period in 2002. The national notification rate was 36.7 cases per 100,000 population.

Figure 3. Trends in notifications of chlamydial infections, Australia, January 1998 to March 2003, by quarter



Nationally, there was a 20 per cent increase from the fourth quarter of 2002 (8,160 notifications) to the first quarter 2003. Increases by jurisdictions ranged from 13 to 38 per cent. Notifications from the Northern Territory decreased by 10 per cent, from 436 to 393 notifications, however they also recorded the highest notification rate, 199 cases per 100,000 population.

Gonococcal infection

For the first quarter of 2003, 1,679 cases of gonorrhoea were notified, an increase of nine per cent over the five year average for the same period. The highest number of notifications were recorded in Western Australia (392) and the Northern Territory (327).

Eleven notifications were received from Tasmania. Ten of the cases were males, aged between 18 and 46 years, and nine of the 10 were living in the greater Hobart area. This epidemic follows similar outbreaks among men who have sex with men that occurred in Sydney during the late 1990s, and in Melbourne.[1,2]

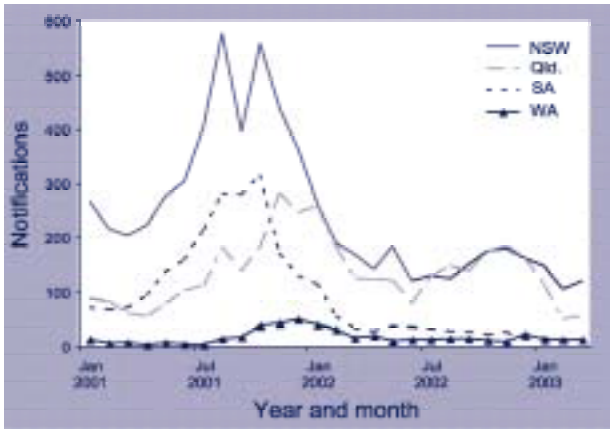
Vaccine preventable diseases

Pertussis

During the first quarter of 2003, 862 cases of pertussis were notified to the NNDSS. This number is 40 per cent less than the average number for the same period (1,421) over the previous five years. Decreases in notification numbers for all states and territories were recorded, and ranged between 23 and 73 per cent. Pertussis notifications by month (2000–2003) in selected jurisdictions are shown in Figure 4. Australia experiences periodic epidemics of pertussis, in three-to-four year cycles. With the last epidemic year in 2001, notification numbers for the present year represent an inter-epidemic year, with totals below the five year mean in all jurisdictions.

The largest number of notifications were reported in New South Wales (379 notifications, 64% of 5-year mean) and Queensland (226 notifications, 66% of 5 year mean). The highest rates in Queensland were in the southern Statistical Divisions outside Brisbane and the other South-East Queensland population centres.

Figure 4. Trends in notifications of pertussis, New South Wales, Queensland, South Australia and Western Australia,January 2001 to March 2003, by month of onset



The largest number of notifications were in the 10–14 year age group, which accounted for 15 per cent of notifications. Overall, 525 notifications (62%) were for adults aged 20 years or more. The number aged less than five years was 89 (10% of total) cases with 48 (7%) cases aged less than one year. For cases aged less than one year, the highest number of notifications were in Queensland (16), New South Wales (13) and Western Australia (11). No deaths from pertussis were reported in the quarter. The high proportion of adult and teenage cases indicates that these groups may be a significant source of the virus, from whom partially or unimmunised infants are contracting pertussis.

Vectorborne diseases

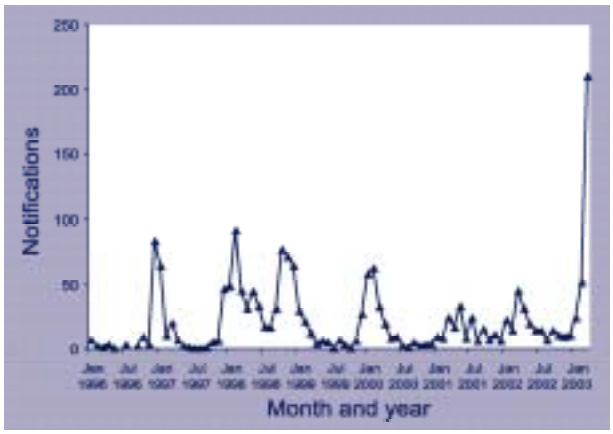
Dengue

There were 336 notifications of dengue reported to NNDSS for the first quarter of 2003. This represents more than three times the average number notified for the same period during the previous five years.

Twenty-eight imported cases were notified from New South Wales, but most of the remainder were from Queensland (287, 84%), where an outbreak of dengue serogroup 2 began in Cairns in mid-February. The index case in the outbreak was a woman who contracted the disease in New Guinea and became ill in Cairns on 22 January. Three secondary cases had an onset of disease in mid-February. The Queensland Dengue Area Response Team was then deployed to issue alerts and identify the source of the outbreak. During March, over 200 further cases were notified. Two cases were notified from Townsville but these infections were shown to be acquired in Cairns. The epidemic has continued into May, and over 400 cases have been reported to date.

The last outbreak of dengue in the Cairns region in 1997–1999, was caused by the dengue serogroup 3 virus.[3] Because the present outbreak of dengue is serotype 2, there has been concern of the possibility of cases of dengue haemorrhagic fever. However to date there has been no reports of this, or of deaths, arising from the Cairns outbreak. The relation of the present outbreak to the 1997 outbreak is illustrated in Figure 5.

Figure 5. Trends in notifications dengue fever, Australia, June 1996 to March 2003, by month of onset



Coincident with the outbreak, eight cases of imported dengue in Cairns have also been identified. These originated in Papua New Guinea and Bali. Major outbreaks of dengue (serotypes 1 and 3) in have occurred in the Western Pacific Region and are continuing to occur.

Kunjin

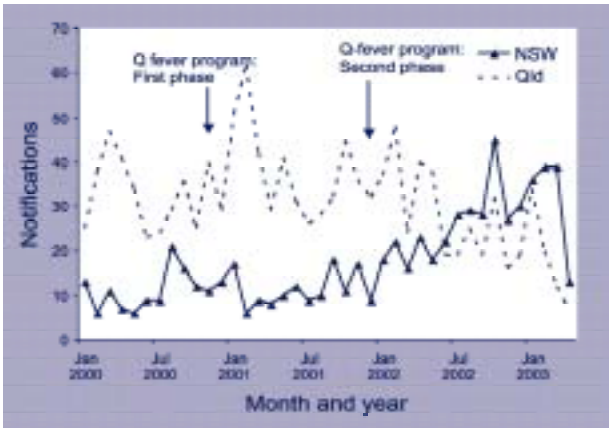
Four cases of Kunjin were notified during the first quarter of 2003. The cases, all male, were reported from Queensland, between 6 February and 13 March. The ages ranged between 34 and 56 years.

As flavivirus infections often occur in travellers in rural regions, and clinical symptoms may develop weeks after infection, the possibility exists that some of the cases may have been contracted in another jurisdiction.

Q fever

There were 199 notifications of Q fever reported in the first quarter of 2003. This is similar to the same period in 2002 (190), but is 33 per cent higher than the five-year average for the same period (149 notifications). The recent trends demonstrates a probable decrease in notifications in Queensland (72 notifications for the present quarter) (Figure 6). This is offset though by the increase observed for New South Wales (113 notifications). The lower rates recorded for Victoria and Western Australia have remained relatively consistent with six and four notifications for these states during the first quarter of 2003, respectively. One case each was notified from the Australian Capital Territory and the Northern Territory. The last notified case from Tasmania was in August 2001.

Figure 6. Notifications of Q fever, New South Wales and Queensland, January 2000 to March 2003, by month of onset



The Commonwealth Government's Q fever vaccination program commenced in October 2000.[4] As a result of this program notifications have initially increased in all jurisdictions due to identification of cases through screening.

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Avner Misrachi, Tasmania Department of Health and Human Services.

References

1. Donovan B, Bodsworth NJ, Rohrsheim R, McNulty A, Tapsall JW. Increasing gonorrhoea reports — not only in London. *Lancet* 2000;355:1908.
2. Victorian Department of Human Services. Surveillance report. *Victorian Infectious Diseases Bulletin* 2002;5:7–15.
3. Hanna JN, Ritchie SA, Phillips DA, Serafin IL, Hills SL, van den Hurk AF, *et al*. An epidemic of dengue 3 in far north Queensland, 1997–1999. *Med J Aust* 2001;174:178–182.
4. NSW Health Department. Q fever register developed to address health concern in the meat industry. *New South Wales Public Health Bulletin* 2002;13:113.

Tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 26,210 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 January and 31 March 2003 (Table 2). The notification rate of diseases per 100,000 population for each State or Territory is presented in Table 3.

There were 4,520 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 January to 31 March 2003 (Tables 4 and 5).

Table 1. Reporting of notifiable diseases by jurisdiction

Disease	Data received from:*
Bloodborne diseases	
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)	All jurisdiction, except NT
Hepatitis C (incident)	All jurisdictions except Qld
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Hepatitis (NEC)	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacterosis	All jurisdictions except NSW
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listerosis	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
SLTEC,VTEC	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible diseases	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis	All jurisdictions
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Laboratory-confirmed influenza	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease – invasive	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
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
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssaviruses (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Other bacterial infections	
Invasive meningococcal infection	All jurisdictions
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Tuberculosis	All jurisdictions

* Jurisdictions not yet reporting on diseases either because legislation has not yet made some diseases notifiable in that jurisdiction or data are not yet being reported to the Commonwealth

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

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 January to 31 March 2003, by date of notification*

Disease	State or Territory								Total 1st quarter 2003[1]	Total 4th quarter 2002[1]	Total 1st quarter 2002[1]	Last five years mean 1st quarter	Year to date 2003[3]	Last five years YTD mean	Ratio†
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis B (incident)	0	18	5	10	1	2	36	12	84	122	99	89	84	89	0.9
Hepatitis B (unspecified)	16	701	NN	177	41	19	435	100	1,489	2,221	1,874	1,773	1,489	1,773	0.8
Hepatitis C (incident)	2	11	NN	NN	13	1	13	41	81	116	130	108	81	108	0.8
Hepatitis C (unspecified)	60	1,586	43	664	138	111	867	321	3,790	5,152	4,545	4,964	3,790	4,964	0.8
Hepatitis D	0	1	0	0	0	0	3	0	4	4	3	3	4	3	1.3
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Campylobacterosis[2]	104	NN	94	1,189	850	161	1,571	513	4,482	5,554	3,810	3,337	4,482	3,337	1.3
Cryptosporidiosis	4	58	54	50	20	3	55	260	504	360	2,113	N/A	504	N/A	N/A
Haemolytic uraemic syndrome	0	2	1	0	0	0	2	0	5	6	3	4	5	4	0.8
Hepatitis A	2	38	18	20	2	2	25	8	115	107	145	379	115	379	0.3
Hepatitis E	0	2	0	0	0	0	0	0	2	1	1	0	2	0	0.0
Listerosis	0	6	0	4	0	1	8	1	20	19	14	18	20	18	1.1
Salmonellosis	31	746	106	851	145	63	574	231	2,747	2,252	2,880	2,573	2,747	2,573	1.1
Shigellosis	3	20	46	24	11	1	18	26	149	138	151	148	149	148	1.0
SLTEC,VTEC[3]	0	0	0	2	15	0	3	0	20	20	16	13	20	13	1.5
Typhoid	0	4	0	0	0	1	11	3	19	20	34	28	19	28	0.7
Quarantinable diseases												0		0	
Cholera	0	0	0	0	0	0	0	0	0	2	1	1	0	1	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0

Table 2 continued. Notifications of diseases received by State and Territory health authorities in the period 1 January to 31 March 2003, by date of notification*

Disease	State or Territory								Total 1st quarter 2003[1]	Total 4th quarter 2002[1]	Total 1st quarter 2002[1]	Last five years mean 1st quarter	Year to date 2003[3]	Last five years YTD mean	Ratio[†]
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible diseases															
Chlamydial infection	106	1,760	393	1,890	470	138	1,540	933	7,230	8,160	7,015	4,426	7,230	4,426	1.6
Donovanosis	0	0	4	3	0	0	0	0	7	4	9	6	7	6	1.1
Gonococcal infection[4]	7	300	327	261	75	11	306	392	1,679	2,025	1,769	1,538	1,679	1,538	1.1
Syphilis[5]	1	223	74	36	6	6	92	27	465	671	470	410	465	410	1.1
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Haemophilus influenzae type b	0	1	0	1	0	0	0	0	2	6	11	7	2	7	0.3
Laboratory-confirmed influenza	3	49	1	17	3	0	4	10	87	645	125	N/A	87	N/A	N/A
Measles	0	0	0	4	1	0	16	0	21	3	5	60	21	60	0.4
Mumps	0	12	0	2	2	0	0	3	19	20	17	36	19	36	0.5
Pertussis	12	379	4	226	43	21	128	49	862	1,823	1,817	1,421	862	1,421	0.6
Pneumococcal disease – invasive	8	87	11	44	31	6	73	26	286	802	295	N/A	286	N/A	N/A
Rubella[6]	0	11	0	23	0	0	2	2	38	96	56	82	38	82	0.5
Tetanus	0	1	0	0	0	0	0	0	1	0	2	2	1	2	0.6
Vectorborne diseases															
Arbovirus infection NEC	0	6	0	18	0	0	4	0	28	7	6	23	28	23	1.2
Barmah Forest virus infection	1	71	9	185	1	0	4	6	277	181	296	243	277	243	1.1
Dengue	2	28	4	287	2	0	3	10	336	47	85	104	336	104	3.2
Japanese encephalitis	0	0	0	0	0	0	0	0	0	0	0	N/A	0	N/A	N/A
Kunjin virus infection	-	0	0	4	0	0	0	0	4	0	0	N/A	4	N/A	N/A
Malaria	4	45	14	78	3	2	20	9	175	137	157	228	175	228	0.8
Murray Valley encephalitis	0	0	0	0	0	0	0	0	0	0	2	N/A	0	N/A	N/A
Ross River virus infection	0	54	101	360	8	2	3	43	571	150	667	1,558	571	1,558	0.4

Table 2 continued. Notifications of diseases received by State and Territory health authorities in the period 1 January to 31 March 2003, by date of notification*

Disease	State or Territory								Total 1st quarter 2003[1]	Total 4th quarter 2002[1]	Total 1st quarter 2002[1]	Last five years mean 1st quarter	Year to date 2003[2]	Last five years YTD mean	Ratio†
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	N/A		N/A	N/A
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	N/A		N/A	N/A
Brucellosis	0	1	0	3	0	0	0	0	4	13	12	9	4	9	0.5
Leptospirosis	0	19	0	27	0	0	0	0	46	28	73	72	46	72	0.6
Other lyssavirus	0	0	0	0	0	0	0	0	0	0	0	N/A		N/A	N/A
Ornithosis	0	7	0	1	0	0	16	0	24	45	19	18	24	18	1.3
Q fever	1	113	1	72	2	0	6	4	199	239	190	149	199	149	1.3
Other bacterial infections															
Legionellosis	0	19	2	7	10	1	30	14	83	120	61	71	83	71	1.2
Leprosy	0	0	0	0	0	0	1	0	1	0	3	1	1	1	1.0
Invasive meningococcal infection	1	28	1	16	6	2	24	8	86	224	110	95	86	95	0.9
Tuberculosis	6	67	5	8	10	1	60	11	168	321	289	253	168	253	0.7
Total	**374**	**6,474**	**1,318**	**6,564**	**1,909**	**555**	**5,953**	**3,063**	**26,210**	**31,861**	**29,380**	**24,229**	**26,210**	**24,229**	**1.1**

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 2003. (Rate per 100,000 population)

Disease[1]	State or Territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic[†]	WA	
Bloodborne diseases									
Hepatitis B (incident)	0.0	0.3	2.5	0.3	0.1	0.4	0.7	0.6	0.4
Hepatitis B (unspecified)	5.0	10.5	NN	4.7	2.7	4.0	8.9	5.2	7.6
Hepatitis C (incident)	0.6	0.2	NN	NN	0.9	0.2	0.3	2.1	0.5
Hepatitis C (unspecified)	18.6	23.8	21.8	17.8	9.1	23.4	17.7	16.6	19.2
Hepatitis D	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacterosis[2]	32.3	NN	47.5	31.9	55.8	34.0	32.1	26.5	34.3
Cryptosporidiosis	1.2	0.9	27.3	1.3	1.3	0.6	1.1	13.4	2.6
Haemolytic uraemic syndrome	0.0	0.0	0.5	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis A	0.6	0.6	9.1	0.5	0.1	0.4	0.5	0.4	0.6
Hepatitis E	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Listerosis	0.0	0.1	0.0	0.1	0.0	0.2	0.2	0.1	0.1
Salmonellosis	9.6	11.2	53.6	22.8	9.5	13.3	11.7	11.9	13.9
Shigellosis	0.9	0.3	23.3	0.6	0.7	0.2	0.4	1.3	0.8
SLTEC,VTEC[3]	0.0	0.0	0.0	0.1	1.0	0.0	0.1	0.0	0.1
Typhoid	0.0	0.1	0.0	0.0	0.0	0.2	0.2	0.2	0.1
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.
Sexually transmissible diseases									
Chlamydial infection	32.9	26.4	198.8	50.7	30.9	29.2	31.5	48.2	36.7
Donovanosis	0.0	0.0	2.0	0.1	0.0	0.0	0.0	0.0	0.0
Gonococcal infection[4]	2.2	4.5	165.4	7.0	4.9	2.3	6.3	20.3	8.5
Syphilis[5]	0.3	3.3	37.4	1.0	0.4	1.3	1.9	1.4	2.4
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Laboratory-confirmed influenza	0.9	0.7	0.5	0.5	0.2	0.0	0.1	0.5	0.4
Measles	0.0	0.0	0.0	0.1	0.1	0.0	0.3	0.0	0.1
Mumps	0.0	0.2	0.0	0.1	0.1	0.0	0.0	0.2	0.1
Pertussis	3.7	5.7	2.0	6.1	2.8	4.4	2.6	2.5	4.4
Pneumococcal disease – invasive	2.5	1.3	5.6	1.2	2.0	1.3	1.5	1.3	1.4
Rubella[6]	0.0	0.2	0.0	0.6	0.0	0.0	0.0	0.1	0.2
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3 continued. Notification rates of diseases by state or territory, 1 January to 31 March 2003. (Rate per 100,000 population).

Disease[1]	State or Territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic[†]	WA	
Vectorborne diseases									
Arbovirus infection NEC	0.0	0.1	0.0	0.5	0.0	0.0	0.1	0.0	0.1
Barmah Forest virus infection	0.3	1.1	4.6	5.0	0.1	0.0	0.1	0.3	1.4
Dengue	0.6	0.4	2.0	7.7	0.1	0.0	0.1	0.5	1.7
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	0.1	0.0	0.0	0.0	0.0	0.0
Malaria	1.2	0.7	7.1	2.1	0.2	0.4	0.4	0.5	0.9
Murray Valley encephalitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	0.0	0.8	51.1	9.7	0.5	0.4	0.1	2.2	2.9
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Leptospirosis	0.0	0.3	0.0	0.7	0.0	0.0	0.0	0.0	0.2
Other lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.1	0.0	0.0	0.0	0.0	0.3	0.0	0.1
Q fever	0.3	1.7	0.5	1.9	0.1	0.0	0.1	0.2	1.0
Other bacterial infections									
Legionellosis	0.0	0.3	1.0	0.2	0.7	0.2	0.6	0.7	0.4
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Invasive meningococcal infection	0.3	0.4	0.5	0.4	0.4	0.4	0.5	0.4	0.4
Tuberculosis	1.9	1.0	2.5	0.2	0.7	0.2	1.2	0.6	0.9

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 laboratories for the reporting period 1 January to 31 March 2003*

	Laboratory	January 2003	February 2003	March 2003	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	94	93	66	253
	New Children's Hospital, Westmead	18	19	25	62
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	22	–	–	22
	South West Area Pathology Service, Liverpool	59	60	34	153
Queensland	Queensland Medical Laboratory, West End	611	384	599	1594
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	384	451	401	1236
Tasmania	Northern Tasmanian Pathology Service, Launceston	10	9	1	20
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Monash Medical Centre, Melbourne	10	4		14
	Royal Children's Hospital, Melbourne	13	30	42	85
	Victorian Infectious Diseases Reference Laboratory, Fairfield	63	82	27	172
Western Australia	PathCentre Virology, Perth	260	275	354	889
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	–	20	–	20
Total		**1,544**	**1,427**	**1,549**	**4,520**

* The complete list of laboratories reporting for the 12 months, January to December 2003, 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 5.Virology and serology laboratory reports by state or territory[1] for the reporting period 1 January to 31 March 2003, and total reports for the year[2]

	State or Territory								This period 2003	This period 2002	Year to date 2003[3]	Year to date 2002
	ACT	NSW	NT	Qld	SA	Tas	Vic.	WA				
Measles, mumps, rubella												
Measles virus	–	–	–	2	2	–	8	–	12	5	12	5
Mumps virus	–	1	–	2	–	–	–	2	5	5	5	5
Rubella virus	–	1	–	7	–	–	–	–	8	20	8	20
Hepatitis viruses												
Hepatitis A virus	–	1	2	4	2	–	–	7	16	21	16	21
Hepatitis D virus	–	–	–	–	–	–	2	2	4	1	4	1
Arboviruses												
Ross River virus	–	3	42	135	7	–	–	21	208	202	208	202
Barmah Forest virus	–	3	7	63	–	–	–	4	77	66	77	66
Dengue type 1	–	–	–	–	–	–	–	2	2	–	2	–
Dengue type 2	–	–	–	–	–	–	–	1	1	1	1	1
Dengue type 3	–	1	–	–	–	–	–	1	2	–	2	–
Dengue not typed	–	1	1	–	1	–	–	11	14	110	14	110
Murray Valley encephalitis virus	–	–	1	–	–	–	–	–	1	3	1	3
Flavivirus (unspecified)	–	–	–	32	–	–	4	–	36	10	36	10
Adenoviruses												
Adenovirus type 1	–	1	–	–	–	–	–	–	1	–	1	–
Adenovirus type 40	–	–	–	–	–	–	–	11	11	9	11	9
Adenovirus not typed/ pending	–	27	3	13	104	1	5	31	184	165	184	165
Herpes viruses												
Herpes virus type 6	–	–	–	–	–	–	–	1	1	–	1	–
Cytomegalovirus	5	48	–	21	166	–	7	1	248	292	248	292
Varicella-zoster virus	2	36	14	169	46	–	9	128	404	492	404	492
Epstein-Barr virus	2	20	7	153	166	–	17	68	433	499	433	499
Other DNA viruses												
Molluscum contagiosum	–	1	–	–	–	–	–	7	8	5	8	5
Contagious pustular dermatitis (Orf virus)	–	–	–	–	–	–	–	1	1	–	1	–
Poxvirus group not typed	–	–	–	–	–	–	1	–	1	1	1	1
Parvovirus	–	2	–	10	4	–	18	14	48	98	48	98
Picornavirus family												
Coxsackievirus A16	–	2	–	–	–	–	–	–	2	–	2	–
Echovirus type 3	–	1	–	–	–	–	–	–	1	–	1	–
Echovirus type 6	–	4	–	–	–	–	–	–	4	27	4	27
Echovirus type 9	–	4	–	–	–	–	–	–	4	8	4	8
Echovirus type 11	–	1	–	–	–	–	–	–	1	–	1	–
Poliovirus type 1 (uncharacterised)	–	5	–	–	–	–	–	–	5	4	5	4
Rhinovirus (all types)	–	54	1	–	–	–	–	51	106	94	106	94
Enterovirus not typed/ pending	–	1	5	1	2	–	–	30	39	125	39	125

Table 5 continued. Virology and serology laboratory reports by state or territory[1] for the reporting period 1 January to 31 March 2003, and total reports for the year[2]

	State or Territory								This period 2003	This period 2002	Year to date 2003[3]	Year to date 2002
	ACT	NSW	NT	Qld	SA	Tas	Vic.	WA				
Ortho/paramyxoviruses												
Influenza A virus	–	5	1	6	81	–	–	4	97	101	97	101
Influenza A virus H3N2	–	–	–	–	–	–	–	1	1	–	1	–
Influenza B virus	–	–	–	–	17	–	1	3	21	26	21	26
Parainfluenza virus type 1	–	2	–	–	13	–	–	–	15	42	15	42
Parainfluenza virus type 2	–	3	–	–	15	–	–	1	19	9	19	9
Parainfluenza virus type 3	–	13	2	4	58	–	–	31	108	61	108	61
Respiratory syncytial virus	–	31	8	7	46	–	2	24	118	125	118	125
Other RNA viruses												
HTLV-1	–	–	1	–	1	–	–	2	4	–	4	–
Rotavirus	–	5	–	–	24	1	2	22	54	91	54	91
Reovirus (unspecified)	–	1	–	–	–	–	–	–	1	1	1	1
Calicivirus	–	–	3	–	–	–	–	31	34	8	34	8
Norwalk agent	–	–	–	–	–	–	26	–	26	51	26	51
Coronavirus	–	–	–	–	–	–	–	1	1	–	1	–
Other												
Chlamydia trachomatis not typed	8	161	40	407	259	12	1	312	1,200	923	1,200	923
Chlamydia pneumoniae	–	–	–	–	–	–	–	1	1	2	1	2
Chlamydia psittaci	–	–	–	–	1	–	14	1	16	12	16	12
Mycoplasma pneumoniae	1	27	–	38	50	7	45	8	176	282	176	282
Mycoplasma hominis	–	4	–	–	–	–	–	–	4	–	4	–
Coxiella burnetii (Q fever)	–	3	2	17	23	–	3	5	53	59	53	59
Rickettsia prowazeki	–	–	–	–	–	–	–	2	2	–	2	–
Streptococcus group A	–	5	6	71	–	–	41	–	123	94	123	94
Yersinia enterocolitica	–	1	–	1	–	–	–	–	2	2	2	2
Brucella abortus	–	1	–	–	–	–	–	–	1	–	1	–
Brucella species	–	1	–	–	–	–	–	–	1	2	1	2
Bordetella pertussis	–	14	1	35	41	–	30	8	129	371	129	371
Legionella pneumophila	–	1	–	–	4	–	29	1	35	16	35	16
Legionella longbeachae	–	–	1	–	2	–	–	7	10	7	10	7
Legionella species	–	–	–	–	–	–	3	–	3	2	3	2
Cryptococcus species	–	–	–	1	2	–	–	–	3	6	3	6
Leptospira species	–	–	–	2	4	–	–	–	6	11	6	11
Treponema pallidum	–	40	93	122	90	–	1	2	348	279	348	279
Entamoeba histolytica	–	–	–	1	–	–	1	1	3	5	3	5
Toxoplasma gondii	–	2	–	–	4	–	4	3	13	10	13	10
Echinococcus granulosus	–	–	–	–	3	–	1	–	4	10	4	10
Total	**18**	**538**	**241**	**1,324**	**1,238**	**21**	**275**	**865**	**4,520**	**4,871**	**4,520**	**4,871**

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.

Additional reports

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 network of general practitioners who report presentations of defined medical conditions each week. The aim of ASPREN is to provide an indicator of the burden of disease in the primary health setting and to detect trends in consultation rates.

There are currently about 50 general practitioners participating in the network from all states and territories. Seventy-five per cent of these are in metropolitan areas and the remainder are rural based. Between 4,000 and 6,000 consultations are recorded each week.

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

In 2003, 13 conditions are being monitored, five of which are related to communicable diseases. These include influenza, gastroenteritis, antibiotic prescription for acute cough, varicella and shingles. Definitions of these conditions were published in Commun Dis Intell *2003;27:125–126.*

Data from 1 January to 31 March 2003 are shown as the rate per 1,000 consultations in Figures 7, 8 and 9.

Australian Paediatric Surveillance Unit

The Australian Paediatric Surveillance Unit (APSU) conducts nationally based active surveillance of rare diseases of childhood, including specified communicable diseases and complications of rare communicable diseases in children. The primary objectives of the APSU are to document the number of Australian children under 15 years newly diagnosed with specified conditions, their geographic distribution, clinical features, current management and outcome. Contributors to the APSU are clinicians known to be working in paediatrics and child health in Australia. In 2001, over 1,000 clinicians participated in the surveillance of 15 conditions through the APSU, with an overall response rate of 98 per cent. The APSU can be contacted by telephone: +61 2 9845 2200, email: apsu@chw.edu.au. For more information see Commun Dis Intell *2003;27: 128–129.*

The results for 1 January to 31 December 2002 are shown in Table 6.

Figure 7. Consultation rates for influenza-like illness, ASPREN, 1 January to 31 March 2003, by week of report

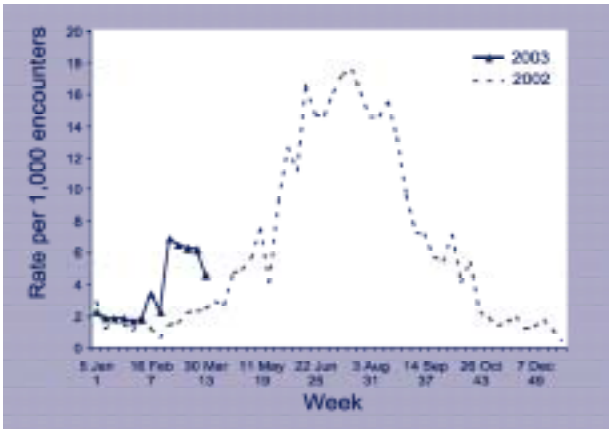


Figure 8. Consulatation rates for gastroenteritis, ASPREN, 1 January to 31 March 2003, by week of report

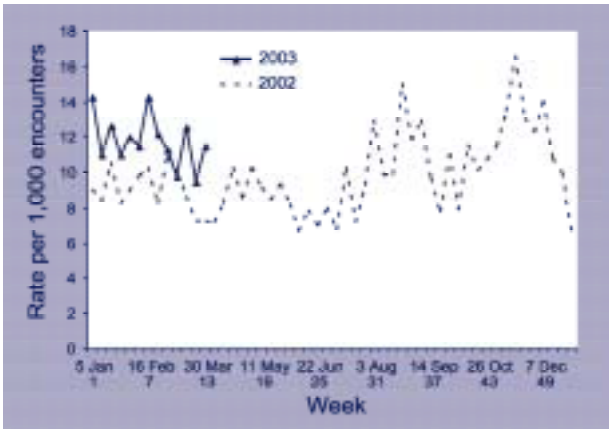


Figure 9. Consultation rates for chickenpox and shingles,ASPREN, 1 January to 31 March 2003, by week of report

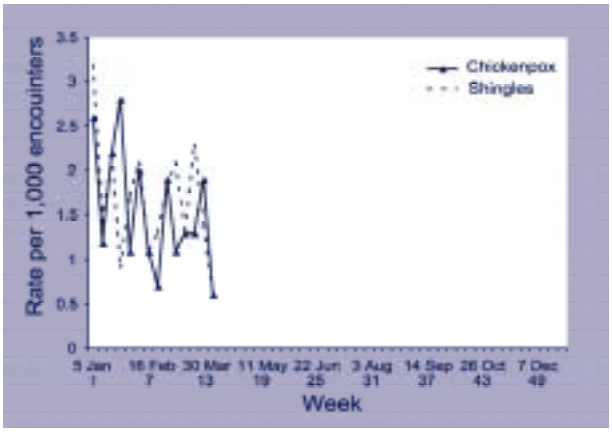


Table 6. Confirmed cases of communicable diseases reported to the Australian Paediatric Surveillance Unit between 1 January and 30 December 2002*

Condition	Previous reporting period 2001*	Current reporting period 2002
Acute flaccid paralysis	44	28
Congenital cytomegalovirus	16	9
Congenital rubella	0	2†
Perinatal exposure to HIV	24	25
Neonatal herpes simplex virus infection	11	11

* Surveillance data are provisional and subject to revision

† Both children born to mothers who had rubella in Indonesia. One child was born in Indonesia, one child born in Australia.

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. Internet: http://www.med.unsw.edu.au/nchecr. Telephone: +61 2 9332 4648. Facsimile: +61 2 9332 1837. For more information see Commun Dis Intell 2003;27:126.

HIV and AIDS diagnoses and deaths following AIDS reported for 1 October to 31 December 2002, as reported to 31 March 2003, are included in this issue of Communicable Diseases Intelligence *(Tables 7 and 8).*

Table 7.New diagnoses of HIV infection,new diagnoses of AIDS and deaths following AIDS occurring in the period 1 October to 31 December 2002, by sex and State or Territory of diagnosis

	Sex	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Totals for Australia			
										This period 2002	This period 2001	Year to date 2002	Year to date 2001
HIV diagnoses	Female	0	10	0	8	1	0	3	3	25	26	90	96
	Male	0	102	0	40	1	0	57	12	212	179	735	692
	Not reported	0	2	0	0	0	0	0	0	2	0	3	1
	Total[1]	0	114	0	48	2	0	60	15	239	205	833	789
AIDS diagnoses	Female	0	2	0	0	1	0	0	0	3	5	13	20
	Male	0	16	0	8	2	1	9	2	38	43	177	168
	Total[1]	0	18	0	8	3	1	9	2	41	48	191	189
AIDS deaths	Female	0	2	0	1	0	0	0	0	3	4	6	14
	Male	0	11	1	3	4	1	2	0	22	18	73	83
	Total[1]	0	13	1	4	4	1	2	0	25	22	79	97

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

Table 8. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 31 December 2002, by sex and State or Territory

	Sex	State or Territory								Australia
		ACT	NSW	NT	QLD	SA	TAS	VIC	WA	
HIV diagnoses	Female	28	693	14	193	77	7	273	147	**1,432**
	Male	237	11,955	116	2,261	749	85	4,398	1,020	**20,821**
	Not reported	0	236	0	0	0	0	24	0	**260**
	Total[1]	265	12,909	130	2,461	826	92	4,713	1,173	**22,569**
AIDS diagnoses	Female	9	212	0	54	30	4	85	32	**426**
	Male	90	4,914	38	923	376	47	1,780	394	**8,562**
	Total[1]	99	5,139	38	979	406	51	1,874	428	**9,014**
AIDS deaths	Female	4	124	0	37	18	2	57	21	**263**
	Male	71	3,390	26	607	251	31	1,327	271	**5,974**
	Total[1]	75	3,523	26	646	269	33	1,391	293	**6,256**

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

Childhood immunisation coverage

Tables 9, 10 and 11 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register.

The data show the percentage of children fully immunised at age 12 months for the cohort born between 1 October and 31 December 2001; at 24 months of age for the cohort born between 1 October and 31 December 2000; and at 6 years of age for the cohort born between 1 October and 31 December 1996, 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 the NCIRS at telephone: +61 2 9845 1256, Email: brynleyh@chw.edu.au.

Immunisation coverage for 'fully immunised' at 12 months of age for Australia has decreased marginally from the last quarter by 0.3 percentage points to 91.4 per cent (Table 9). The change in 'fully immunised' coverage varied by state and territory with most jurisdictions experiencing small decreases in coverage, the exception being South Australia with a significant 1.8 per cent decrease. South Australia was also the only jurisdiction with any important changes in coverage for individual vaccines. It experienced not insignificant decreases in coverage for diphtheria, tetanus, pertussis (DTP) (–1.4%), poliomyelitis (OPV) (–1.4%) and *Haemophilus influenzae* type b (Hib) (–1.1%) vaccines. One explanation for this decrease is that the Australian Childhood Immunisation Register (ACIR) field officer for South Australia was able to focus attention on data cleaning for the 12-month cohort during the months of June, July and August 2002 but then changed the focus to the 24-month group for the following three months (M. Watson, Department of Human Services, South Australia, personal communication, 30 April 2003). This is an important example that demonstrates the value of the ACIR field officers, and the value of the continuing role of educating providers to sustain quality in recording of immunisations.

Every jurisdiction still has coverage greater than 90 per cent for all individual vaccines at 12 months of age and five jurisdictions have greater than 95 per cent coverage for hepatitis B vaccine: New South Wales (95.1%), the Northern Territory (96.1%), Queensland (95.2%), South Australia (95.4%), and Tasmania (95.1%).

Coverage measured by 'fully immunised' at 24 months of age for Australia decreased marginally from the last quarter by 0.4 percentage points to 89.0 per cent (Table 10). Coverage for individual vaccines for Australia basically remained unchanged with DTP vaccine coverage still almost three percentage points lower than coverage for the other vaccines calculated for this age group. The most important jurisdictional changes in 'fully immunised' coverage at 24 months of age occurred in Western Australia (–1.5%) and the Northern Territory (+2.0). In fact, there were some significant increases in coverage for most vaccines in the Northern Territory with coverage for DTP increasing by 1.8 per cent to 88.2 per cent, and coverage for Hib increasing by 1.6 per cent to 94.8 per cent. Western Australia had the opposite experience with significant decreases in coverage for DTP (–1.6%) and measles, mumps, rubella (–1.1%).

Table 11 shows immunisation coverage estimates for 'fully immunised' and for individual vaccines at 6 years of age for Australia and by state or territory. 'Fully immunised' coverage at 6 years of age for Australia remained unchanged from the previous quarter at 82.2 per cent. There was also very little change for the jurisdictions, with the exception of Tasmania who experienced the only significant change in 'fully immunised' coverage at this age, a 2.2 per cent increase. Coverage for individual vaccines for Australia and the jurisdictions for this age group also showed little change.

Figure 10 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, 24 months and 6 years.

Table 9. Proportion of children immunised at 1 year of age, preliminary results by vaccine and state or territory for the birth cohort 1 October to 31 December 2001; assessment date 31 March 2003

Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	932	20,818	860	11,913	4,302	1,449	15,031	5,888	**61,193**
Diphtheria, tetanus, pertussis (%)	91.9	92.5	91.5	92.9	92.6	93.9	93.1	91.1	**92.6**
Poliomyelitis (%)	91.5	92.4	91.1	92.8	92.5	93.8	93.0	91.1	**92.5**
Haemophilus influenzae type b (%)	93.9	94.5	95.4	94.5	94.8	95.2	95.1	94.4	**94.7**
Hepatitis B (%)	94.1	95.1	96.1	95.2	95.4	95.1	94.9	94.1	**95.0**
Fully immunised (%)	90.1	91.3	90.8	91.8	91.4	92.8	91.9	90.1	**91.4**
Change in fully immunised since last quarter (%)	+0.8	–0.1	+0.4	–0.0	–1.8	–0.2	–0.5	+0.2	**–0.3**

Table 10. Proportion of children immunised at 2 years of age, preliminary results by vaccine and state or territory for the birth cohort 1 October to 31 December 2000; assessment date 31 March 2003[1]

Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	1,072	21,673	787	12,219	4,305	1,553	15,458	5,960	**63,027**
Diphtheria, tetanus, pertussis (%)	89.5	90.8	88.2	91.8	91.7	93.6	92.0	89.6	**91.2**
Poliomyelitis (%)	94.2	94.6	96.8	94.6	95.3	96.7	95.7	93.8	**94.9**
Haemophilus influenzae type b (%)	93.8	93.5	94.8	94.0	94.5	96.0	94.8	92.6	**94.0**
Measles, mumps, rubella (%)	93.8	93.7	95.4	94.0	94.7	95.8	95.2	92.7	**94.2**
Hepatitis B (%)	94.9	95.4	98.1	95.2	96.0	97.4	96.3	95.0	**95.7**
Fully immunised (%)[1]	86.8	88.0	87.0	89.7	90.0	92.9	90.0	87.3	**89.0**
Change in fully immunised since last quarter (%)	–0.7	–0.4	+2.0	–0.6	+0.5	+0.5	–0.1	–1.5	**–0.4**

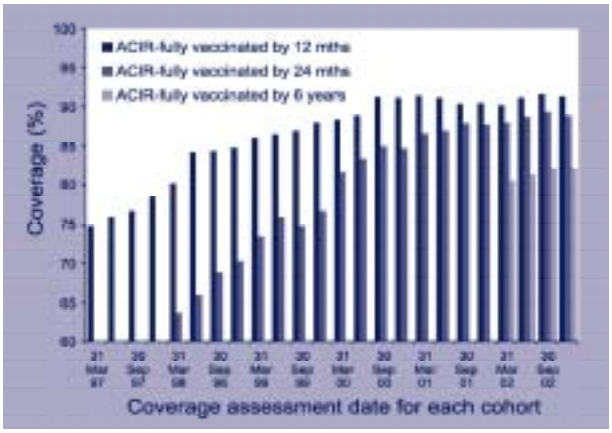
1. The 12 months age data for this cohort were published in Commun Dis Intell 2002;26:309.
2. These data relating to 2 year-old children should be considered as preliminary. The proportions shown as 'fully immunised' appear low when compared with the proportions for individual vaccines. This is at least partly due to poor identification of children on immunisation encounter forms.

Table 11. Proportion of children immunised at 6 years of age, preliminary results by vaccine and state or territory for the birth cohort 1 October to 31 December 1996; assessment date 31 March 2003

Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	1,122	22,333	16,393	12,531	4,738	6,525	1,632	765	**66,039**
Diphtheria, tetanus, pertussis (%)	83.5	83.6	86.7	83.8	83.0	81.9	85.7	83.5	**84.3**
Poliomyelitis (%)	83.6	83.7	87.0	84.0	83.3	82.2	86.1	85.4	**84.5**
Haemophilus influenzae type b (%)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Measles, mumps, rubella (%)	83.4	82.0	86.7	83.7	82.1	81.8	84.8	84.3	**83.6**
Hepatitis B(%)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**0.0**
Fully immunised (%)[1]	81.8	80.5	85.4	82.3	80.8	79.9	83.8	82.2	**82.2**
Change in fully immunised since last quarter (%)	–0.1	–0.3	–0.5	–0.6	–1.0	+2.2	+0.7	+0.4	**0.0**

1. These data relating to 6 year-old children should be considered as preliminary. The proportions shown as 'fully immunised' appear low when compared with the proportions for individual vaccines. This is at least partly due to poor identification of children on immunisation encounter forms.

Figure 10. Trends in vaccination coverage, Australia, 1 October to 31 December, by age cohorts



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 *quarterly reports include only* 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 counts should be interpreted with caution, and are affected by surveillance artefacts such as newly recognised (such as S. *Typhimurium 197 and* S. *Typhimurium U290) and incompletely typed* Salmonella.

Reported by Joan Powling (NEPSS Co-ordinator) and Mark Veitch (Public Health Physician), Microbiological Diagnostic Unit — Public Health Laboratory, Department of Microbiology and Immunology, University of Melbourne. NEPSS can be contacted at the above address or by telephone: +61 3 8344 5701, or facsimile: +61 3 9625 2689.

Reports to the National Enteric Pathogens Surveillance System of Salmonella infection for the period 1 January to 31 March 2003 are included in Tables 11 and 12. Data include cases reported and entered by 16 April 2003. Counts are preliminary, and subject to adjustment after completion of typing and reporting of further cases to NEPSS. For more information see Commun Dis Intell *2003;27:129.*

First quarter 2003

The total number of reports to the National Enteric Pathogens Surveillance System of human *Salmonella* infection increased to 2,461 in the first quarter of 2003, 59 per cent more than the fourth quarter of 2002, but a similar count to the first quarter of 2002. Each year, the highest number of cases is reported in the first quarter.

During the first quarter of 2003, the 25 most common *Salmonella* types in Australia accounted for 1,690 (69%) of all reported human *Salmonella* infections.

S. Typhimurium phage types 135, 170 and 9, *S.* Saintpaul and *S.* Chester were the most common salmonellae. With the exception of *S.* Chester, these have been among the five most common salmonellae each quarter since the first quarter of 2002; *S.* Typhimurium phage type 135 has been the most common *Salmonella* for each of the past four quarters.

The most notable recent increase in reports has been *S.* Typhimurium phage type 197. This phage type was first reported in 1990 and was rare until October 2002. There were 75 cases in the fourth quarter of 2002 and 78 cases have been reported in the first quarter of 2003. Cases have been reported predominantly from New South Wales and Queensland.

Table 11. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 January to 31 March 2003, as reported to 16 April 2003

Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total all *Salmonella* for quarter	35	673	68	770	120	63	537	195	**2,461**
Total contributing *Salmonella* types	19	111	29	116	50	15	92	61	**223**

Acknowledgement: Thanks to contributing laboratories and scientists.

Table 12. Top 25 *Salmonella* types identified in Australian States and Territories, 1 January to 31 March 2003

National rank	*Salmonella* type	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 1st quarter 2003	Last 10 years mean 1st quarter	Year to date 2003	Year to date 2002
1	*S.* Typhimurium 135	15	86	10	54	5	3	184	20	377	197	377	273
2	*S.* Typhimurium 170	1	85	0	38	0	0	68	2	194	40	194	135
3	*S.* Typhimurium 9	0	35	0	7	14	1	56	9	122	171	122	307
4	*S.* Saintpaul	0	14	4	72	3	3	8	13	117	119	117	159
5	*S.* Chester	1	33	2	42	11	0	5	10	104	57	104	58
6	*S.* Typhimurium 197	0	38	0	31	0	0	6	3	78	1	78	4
7	*S.* Birkenhead	0	33	0	40	0	0	0	2	75	90	75	115
8	*S.* Infantis	1	34	1	5	6	3	19	2	71	46	71	42
9	*S.* Virchow 8	0	5	0	56	0	0	2	0	63	64	63	156
10	*S.* Muenchen	2	6	1	29	3	0	7	9	57	55	57	47
11	*S.* Mississippi	0	2	0	1	0	37	1	0	41	33	41	37
12	*S.* Typhimurium 12	0	13	0	15	7	0	4	0	39	9	39	22
13	*S.* Hvittingfoss	0	7	0	25	1	0	1	0	34	23	34	64
14	*S.* Anatum	0	1	7	18	1	0	4	3	34	32	34	36
15	*S.* Aberdeen	0	1	0	32	0	0	0	0	33	37	33	70
16	*S.* Typhimurium U290	2	5	0	2	0	0	19	4	32	4	32	28
17	*S.* Singapore	0	17	0	3	3	0	1	4	28	19	28	23
18	*S.* Waycross	0	9	0	18	0	0	0	0	27	39	27	50
19	*S.* Typhimurium 44	0	12	0	9	2	0	3	0	26	31	26	13
20	*S.* Virchow 34	0	1	0	19	0	0	5	0	25	32	25	41
21	*S.* Typhimurium 4	0	18	0	0	1	3	2	0	24	18	24	33
22	*S.* Typhimurium 126	0	8	0	5	3	0	2	6	24	30	24	65
23	*S.* Montevideo	0	15	0	7	1	0	0	0	23	7	23	30
24	*S.* Potsdam	1	4	0	11	2	1	3	0	22	21	22	26
25	*S.* Agona	0	5	0	9	1	1	4	0	20	18	20	25