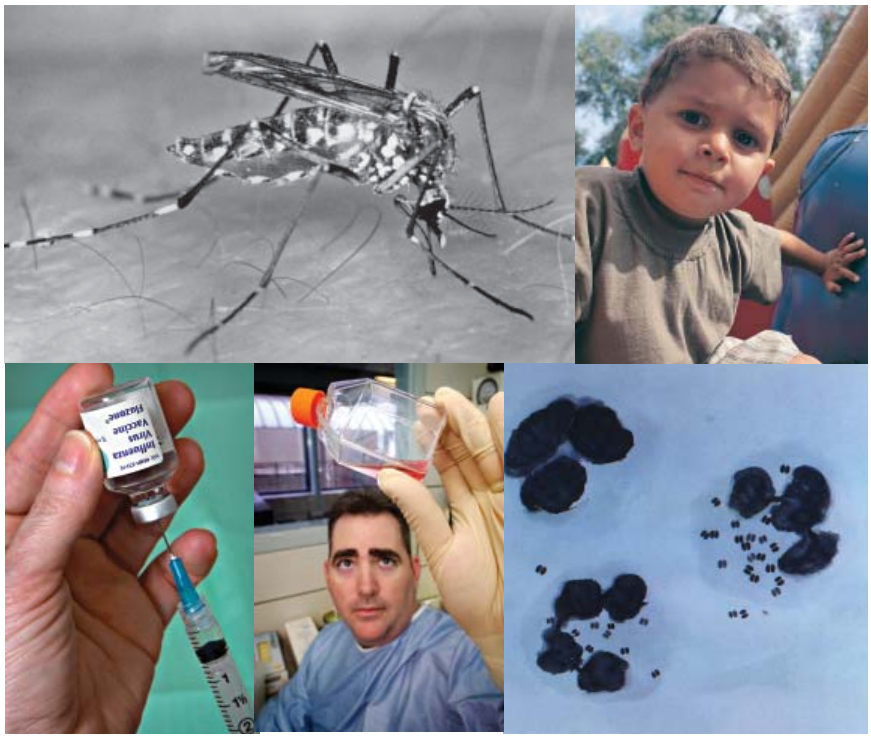
Australian Government
Department of Health and Ageing

Communicable Diseases Intelligence



Quarterly report

Volume 28
Issue no 2
2004

Communicable Diseases Intelligence

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Volume 28

Issue no 2

2004

ISBN 0725-3141

Communicable Diseases Intelligence aims to diseminate information on the epidemiology and control of communicable diseases in Australia. *Communicable Diseases Intelligence* invites contributions dealing with any aspect of communicable disease epidemiology, surveillance or prevention and control in Australia. Submissions can be in the form of original articles, short reports, surveillance summaries, reviews or correspondence. Instructions for authors can be found in *Commun Dis Intell* 2004;28:95–97.

Communicable Diseases Intelligence contributes to the work of the Communicable DIseases Network Australia (www.cda.gov.au/cdna/)

Website

http://www.cda.gov.au/index.htm

Subscriptions and contacts

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This journal is indexed by *Index Medicus*, Medline and the Australasian Medical Index

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Front cover: Surveillance and Epidemiology Section, Australian Government Department of Health and Ageing. Images sourced from the Centers for Disease Control and Prevention Public Health Image Library, courtesy of the Centers for Disease Control and Prevention, Atlanta, Georgia, and Pubilcations Unit, Australian Government Department of Health and Ageing. Clockwise from top left: Domestic *Aedes aegypti* mosquito, a vector for dengue and yellow fever (CDC); Australian Indigenous child, (provided by Publications Unit, Australian Government Department of Health and Ageing); Extracting influenza virus vaccine from a 5 ml vial (provided by Jim Gathany, CDC); Patrick Stockton of the Special Pathogens Branch examining a T-25 flask used in the SARS virus isolation, (CDC); Gram-stain of urethral exudate, diagnostic of gonococcal urethritis (CDC).

Printed by Union Offset, Canberra

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Contents, *cont.*

Vaccine preventable diseases and vaccination coverage in Aboriginal and Torres Strait Islanders people, Australia, 1999 to 2002

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National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

Abstract

This report complements the Vaccine Preventable Diseases and Vaccination Coverage reports produced biannually since 2000 by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases in association with the Australian Institute of Health and Welfare. It integrates the available sources of routinely collected data relevant to the current status of vaccine preventable diseases and vaccine coverage in Aboriginal and Torres Strait Islander people in Australia. It aims to better inform Indigenous communities, Indigenous health care providers and planners of immunisation services of the current status and future needs for vaccine prevention in Indigenous people. The data presented here demonstrate that vaccination programs have had a significant impact on the health of Aboriginal and Torres Strait Islander people. Several areas are highlighted for further development of vaccination policy recommendations, in particular high rates of preventable hepatitis A and B, influenza and pneumococcal disease. Areas where more research is needed include means to more accurately monitor vaccination status, the applicability of meningococcal serogroup B vaccines when available, and effective ways of increasing vaccination coverage and timeliness of vaccination. Such issues need to be considered and implemented in full cooperation with Aboriginal and Torres Strait Islander people. ***Commun Dis Intell*** **2004;28:127–159.**

Keywords: Aboriginal, indigenous people, Torres Strait Islander, vaccination coverage, vaccine preventable disease

Introduction

Rationale

Aboriginal and Torres Strait Islander people experience a greater burden of both communicable and non-communicable diseases compared to the non-Indigenous population.[1] Specific information on vaccination coverage and vaccine preventable diseases (VPDs) in Aboriginal and Torres Strait Islander people has been limited, with most published data coming from studies in certain regions or occasional jurisdictional reports. While national data on other Aboriginal and Torres Strait Islander health issues have been published utilising existing administrative data collections,[1] this report is the first comprehensive descriptive analysis of vaccination coverage and vaccine preventable diseases in Aboriginal and Torres Strait Islander people at the national level. The report and its rationale have the support of the National Aboriginal Community Controlled Health Organisation (NACCHO).

Social and economic context

The relationship between socioeconomic status and health is well documented. A gradient is observable with lower rates of death and most illnesses as socioeconomic position improves.[2] Aboriginal and Torres Strait Islander people are disadvantaged in terms of most socioeconomic indices (income, education, employment, housing) compared to other Australians.[1] In particular, the disproportionate exposure to overcrowded living conditions and inadequate essential infrastructure which Aboriginal and Torres Strait Islander people experience is known to facilitate the spread of many infectious diseases.[1]

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This report is also available separately as *Commun Dis Intell* 2004:28 Suppl 1 and as AIHW Cat. No. IHW 13.

Demographic context

The Aboriginal and Torres Strait Islander population has a significantly younger age profile than the Australian population as a whole. Comparisons between Indigenous and other populations must take age into consideration, either through age standardisation or stratification by age group.[1]

Approach

This report presents data on notifications of VPDs and on hospitalisations and deaths coded as being related to VPDs for the years 1999 to 2002. The completeness of identification of Aboriginal and Torres Strait Islander people varies between Australian jurisdictions. In accordance with previous practice,[3] the data for notifications, hospitalisations and deaths are presented in different combinations of states and territories, as outlined below in the methods section.

Methods

Vaccine preventable diseases data

Three sources of routinely collected data were used for this report. Notification data were obtained from the National Notifiable Diseases Surveillance System (NNDSS), hospitalisation data from the Australian Institute of Health and Welfare (AIHW) National Hospital Morbidity Database, and mortality data from the AIHW Mortality Database.

Notifications

The NNDSS database includes information about cases of VPDs reported by laboratories and health workers to state and territory authorities under public health legislation. State and territory notification criteria were based on the National Health and Medical Research Council (NHMRC) surveillance case definitions[4] in most jurisdictions. The case definitions presented for each disease in the results section are those of the NHMRC[4] with the exception of pneumococcal disease, which is taken from Roche, *et al*.[5] New standard national surveillance case definitions have recently been developed and were adopted in some jurisdictions during the period covered by this report. However, any changes in case definitions are unlikely to have a significant impact on the notification rates presented.

Notifications with an onset between 1 January 2000 and 31 December 2002 were analysed for this report. The variables extracted for analysis were: disease, date of disease onset, age at onset, Indigenous status and state or territory where the notification originated. Following an assessment of completeness of the Indigenous status field (see below), notifications were included from New South Wales, the Northern Territory, South Australia and Western Australia. Detailed data are presented for *Haemophilus influenzae* type b (Hib) disease, hepatitis A, acute hepatitis B, measles, meningococcal disease, pertussis, and pneumococcal disease. Notification data are not presented for VPDs with few or no notifications in the period (diphtheria, mumps, polio, rubella, tetanus), or for varicella, for which a vaccine was not widely used during this period. Summary data for these are provided in the Appendix. Data are not provided for influenza notifications due to the low level of completeness of the Indigenous status field.

Hospitalisations

The AIHW National Hospital Morbidity Database receives administrative, demographic and clinical information about patients admitted to public and private hospitals in Australia. Data are received by financial year of hospital separation. The three most recent years for which data were available (1999/00, 2000/01 and 2001/02) were examined. Data for 2001/02 are provisional because, at the time of publication, New South Wales data were under revision. The variables extracted for analysis were: date of separation, age at admission, state or territory of hospitalisation, Indigenous status and diagnoses. Diagnoses included principal and other with up to 31 diagnoses for each admission, coded using the International Statistical Classification of Diseases and Related Health Problems, 10th Revision, Australian Modification, 1st Edition (ICD–10–AM). Eligible separations were those with the code of interest listed in any diagnosis field except for hepatitis B, where only principal diagnoses were included. Detailed data are presented for hepatitis A, acute hepatitis B, influenza and pneumonia, measles, meningococcal disease, pertussis, and pneumococcal disease. Separation data are not presented in the results section for those VPDs with few or no separations during the period (diphtheria, mumps, polio, rubella, tetanus), with no specific ICD10 code (Hib disease), or for which a vaccine was not widely used during this period (varicella). Summary data for these diseases are provided in the Appendix.

Deaths

Death data were obtained from the AIHW Mortality Database. These data are supplied annually to the AIHW by the Australian Bureau of Statistics (ABS). Analysis was by year of registration for the three most recent years for which data were available (2000 to 2002). The variables extracted were: age, state or territory of registration, Indigenous status, and underlying cause of death, coded using the International Classification of Disease, 10th Revision (ICD–10). Deaths where the disease of interest was recorded as the underlying cause are used in this report. Following previous practice,[1] mortality data for those jurisdictions considered to have the most complete coverage of Aboriginal and Torres Strait Islander deaths (Queensland, South Australia, Western Australia and the Northern Territory) were combined. For diseases included in the results section, numbers of deaths are presented by age group where appropriate, and as summary data. For those VPDs not included in the results section (diphtheria, mumps, polio, rubella, tetanus and varicella), summary data are provided in the Appendix.

Calculations

Incidence rates in Aboriginal and Torres Strait Islander people were calculated using ABS estimates of Indigenous populations as at mid-2001.[6] Incidence rates for other (presumed non-Indigenous) persons were calculated using as the denominator the total ABS estimated resident population as at mid-2001, minus the relevant Indigenous population. Rates for all ages combined were age standardised to the ABS Australian population estimates for 2001,[6] for both those categorised as Indigenous and other. Rate ratios for Indigenous versus other persons were calculated for each disease, with age-specific rate ratios where appropriate. All rates are presented as average annual rates per 100,000 total population or population by age group, as appropriate.

Report structure for individual diseases

For each disease, data are presented in the following format:

- description of salient clinical and epidemiological features;
- case definitions (NHMRC 1994 unless otherwise specified);
- distribution by Indigenous status (and age where appropriate); and
- comment on the data presented.

Vaccination coverage data

Data on coverage for various vaccines at the national level were provided by the Health Insurance Commission (from the Australian Childhood Immunisation Register [ACIR]) and the Australian Bureau of Statistics (from the 2001 National Health Survey).

The ACIR is administered by the Health Insurance Commission (HIC) for the Australian Government Department of Health and Ageing, and records the vaccination service details of children aged less than seven years from data supplied by vaccine providers. Vaccination coverage estimates derived from ACIR data have been reported in *Communicable Diseases Intelligence* since early 1998. A complete description of the method for calculating coverage estimates by age cohorts is given elsewhere.[7] National data on coverage in children recorded as Aboriginal and Torres Strait Islander have not been published previously.

The 2001 National Health Survey included a supplementary survey of 3,198 Aboriginal and Torres Strait Islander adults and children in order to improve the precision of estimates of Indigenous people. The sample covered all areas of Australia, including sparsely settled areas. When combined with the main survey, the total Indigenous sample size was 3,681.[8] This survey provided national data on the immunisation status of Aboriginal and Torres Strait Islander people for the first time in Australia. This report includes more detailed tabulations not previously published. Immunisation status was collected by face-to-face interview for both adults and children. Respondents were asked to consult written records if available. Vaccinations were regarded as given if at least a date of vaccination was supplied. Children were regarded as vaccinated appropriately for age if the vaccine was administered up to one month after the recommended age. Thus, data from the National Health Survey are not directly comparable with the ACIR due to differences in both the methods of data collection and calculation of vaccination status.

Data quality and notes on interpreting data

As a consequence of differences in data quality and availability, the time periods and states and territories included in this report differ between data collections. Comparing data from the different collections is therefore problematic and should take account of the various factors outlined below.

Indigenous identification

The quality of Indigenous health statistics depends on both the accuracy of Indigenous population estimates and the level of completeness and accuracy of reporting achieved in the collection of Indigenous status for the condition of interest.[1] Considerable work has been done in recent years on assessing and improving the quality of Indigenous statistics in national and state and territory administrative data collections.[1] More work is needed to improve the quality of the data, as large variations in quality exist between data collections, between States, and over time. For this report, data and analyses chosen for inclusion are in most instances similar to previous publications on those data collections.[1] This is not the case where there have been changes in data quality since the last publication, or no previous assessment has been carried out, as specified below.

Notification data

Indigenous identification

The proportion of notifications lacking identification of Indigenous status were analysed by state, year and disease. Adequate levels of completeness of Indigenous status identification between 2000 and 2002 were defined as at least 60 per cent for a substantial majority of the diseases analysed. This level of completeness was achieved for New South Wales, South Australia, Western Australia and the Northern Territory. After first establishing that the notification incidence estimates were not dominated by any one of these four States for all diseases of interest (data not shown), estimates are presented for the four jurisdictions combined. Although a previous presentation of notifications for the period 1998–2000[3] excluded New South Wales, data completeness for this State between 2000 and 2002 was found to be comparable to that of South Australia and Western Australia. As a high proportion of influenza notifications lacked identification of Indigenous status in all states and territories, other than the Northern Territory, influenza was excluded from further analysis.

Overall, estimated Indigenous notification rates can be considered underestimates, due to the incomplete recording of Indigenous status.

Other issues

A major consideration in interpreting notification data is that they represent only a proportion of the total cases occurring in the community. This proportion is usually unknown and may vary between diseases, with infections diagnosed by a laboratory test more likely to be notified. Data accuracy may also vary between states and territories, due to the use of different case definitions for surveillance and varying reporting requirements by medical practitioners, laboratories and hospitals. In addition, data accuracy may change over time as new diagnostic tests are introduced or surveillance practices change.

Hospitalisation data

Indigenous identification

Although the overall proportion of hospitalisations lacking identification of Indigenous status was low (less than 5%) from 1999/00 to 2001/02, variability in completeness and in accuracy of identification have been previously documented.[3,9] The proportion of Aboriginal and Torres Strait Islander people correctly identified in hospital records has varied between 44 per cent and 100 per cent in studies conducted in various jurisdictions since 1997.[9] Following previous practice,[1] hospital separations are presented for all jurisdictions combined. Indigenous hospitalisation rates can also be considered underestimates due to the incomplete identification of Aboriginal and Torres Strait Islander people.

Other issues

There are limitations associated with the use of ICD–10–AM codes to identify cases. Hospital coding errors have been reported to occur more commonly for diseases that the coder was less familiar with and for admissions with multiple diagnoses.[10] Assignation of codes is based on information in medical records, as recorded by clinicians, without strict case definitions. This is in contrast with the more stringent case definitions used for notification data. Indigenous hospitalisations where the code of interest was in any diagnosis field were included, and the relative importance of these cannot be gauged. For acute hepatitis B, cases were only included where the code of interest was the principal diagnosis, as previous studies have found a substantially lower proportion of principal diagnoses compared to other diseases.[11,12]

It should also be noted that the hospitalisation database contains a record for each admission, which means that there are separate records for each readmission or inter-hospital transfer. This is unlikely to have a major impact on the numbers reported for most diseases reviewed, as they are acute illnesses, but may inflate the numbers of hospitalisations where inter-hospital transfer is more frequent, either because of remote residence or the severity of the illness.

Death data

Indigenous identification

The accuracy of reporting Indigenous status on deaths has been previously evaluated by comparing the reported number with an expected, or predicted, number of Indigenous deaths.[1] Reporting was found to be acceptable for deaths in 1997–1999 in Queensland, South Australia, Western Australia and the Northern Territory. Following previous practice,[1] reported deaths from these four States only have been presented in this report. These combined rates may still underestimate Indigenous death rates due to under-reporting.

Other issues

Mortality data were analysed by year of registration rather than by year of death. Approximately six per cent of deaths in a particular calendar year are registered in the subsequent year, mostly deaths in the previous December. Issues associated with the accuracy of the ICD codes used for hospital separations may also apply to mortality data.

Vaccination coverage data

Indigenous identification

The completeness of reporting of Indigenous status on the ACIR was analysed by comparing the number of children recorded as Aboriginal or Torres Strait Islander on the ACIR with ABS 2001 Census data. Indigenous status is currently either not routinely reported or not transferred to the ACIR from the Australian Capital Territory, Queensland and Tasmania, so these jurisdictions were not included in this report. For New South Wales, the Northern Territory, South Australia, Victoria and Western Australia, 61 per cent of the estimated cohort of Aboriginal or Torres Strait Islander children aged 12–18 months and 65 per cent of the estimated cohort aged 24–30 months were recorded as Aboriginal or Torres Strait Islander on the ACIR. The accuracy of the information recorded in the Indigenous status field was assessed by comparison of Hib vaccines received, adapting a method developed by Hull, *et al*.[13] Until May 2000, different Hib vaccines were recommended for Indigenous (Hib vaccine conjugated to outer membrane protein of *Neisseria meningitidis*, Pedvax) and non-Indigenous (Hib vaccine conjugated to a mutant diphtheria toxin, HibTiter) children. Children born between 1 January and 30 April 2000 and recorded on the ACIR as receiving a Hib vaccine and being Aboriginal or Torres Strait Islander were 27 times more likely to have received a dose of Pedvax. Children recorded as receiving a dose of Pedvax were nine times more likely to be recorded as Aboriginal or Torres Strait Islander than children who received other Hib vaccines. These data suggest an acceptable level of correlation between the recording of Indigenous status and the receipt of a vaccine recommended only for Aboriginal and Torres Strait Islander children.

Other issues

General limitations of data available from the ACIR must be considered when used to estimate vaccination coverage. A study conducted in 2001 found that the ACIR underestimated overall Australian immunisation coverage by 2.7 per cent at 12 months of age and five per cent at 24 months.[14] However, for Aboriginal and Torres Strait Islander children these estimates may not be valid, as there is the issue of both under-reporting of vaccinations given to the ACIR and incomplete identification of Indigenous children. This means that ACIR coverage estimates could overestimate or underestimate coverage, depending on whether those children not identified as Aboriginal and Torres Strait Islander have higher or lower than average vaccination coverage. The ACIR holds records only for children up to seven years of age. Coverage is calculated only for children registered on Medicare; however, current data show that by the age of 12 months practically all Australian children have been registered with Medicare (personal communication, Kathi Williams, Health Insurance Commission, April 2004).

Indigenous population estimates

Estimation of the size and age composition of the Aboriginal and Torres Strait Islander population is difficult. Increases in census counts of Aboriginal and Torres Strait Islander people between 1966 and 1996 are far greater than can be explained by simple demographic factors (births minus deaths).[15] Other factors thought to be important include changes in: the propensity to identify as Indigenous in the Census; the proportion of children with only one Aboriginal and Torres Strait Islander parent identified as Indigenous; and Census enumeration procedures.[3,15]

In this report, Australian Bureau of Statistics estimates of Aboriginal and Torres Strait Islander population figures, based on 2001 Census data,[6] are used.

Results

Table 1 summarises the main findings, showing the reported incidence per 100,000 total population in Indigenous and other (presumed non-Indigenous) people and the ratio of incidence in Indigenous to other (presumed non-Indigenous) people. The estimates of incidence in Indigenous people are based on incomplete ascertainment of Indigenous status and can be considered as minimum estimates. Table 1 illustrates the large burden (incidence) and relative burden (rate ratio) of almost all VPDs in Indigenous people. When measured across all age groups in the Indigenous population, pertussis, influenza and pneumococcal disease have the highest incidence, though the differential burden, as measured by the rate ratio, is highest for *Haemophilus influenzae* type b (Hib) disease and hepatitis A. It is important to note that the Indigenous population is significantly younger than the non-Indigenous population and that both the absolute and relative burdens are greatest in the youngest age groups for most VPDs.

Haemophilus influenzae type b disease

Haemophilus influenzae is a fastidious Gram-negative bacterium, which occurs in both encapsulated and unencapsulated forms. Before Hib vaccines became available, one encapsulated serotype, type b (Hib), caused at least 95 per cent of invasive infections due to *H. influenzae* (those associated with isolation of the organism from a normally sterile site) in children.[16,17] The most common manifestation of invasive Hib disease was meningitis, with children aged less than 18 months most at risk.[17–19] Aboriginal children had a particularly high risk of Hib meningitis, with a recorded incidence among children living in Central Australia among the highest in the world and an earlier age of onset than non-Indigenous children.[20] Survivors of Hib meningitis commonly had neurological sequelae such as deafness and intellectual impairment. Epiglottitis, a potentially fatal inflammation of the epiglottis obstructing breathing, was the other major category of infection, but was rare in Aboriginal and Torres Strait Islander children.[20] Less common manifestations of invasive Hib disease include a range of focal infections (cellulitis, septic arthritis, pneumonia, pericarditis, osteomyelitis) and septicaemia without focus.

Table 1. Summary of notification or hospitalisation rates of vaccine preventable diseases in Australia, 2000 to 2002*

Disease	Notification or hospitalisation rates — all ages†			Indigenous age group with peak incidence	
	Indigenous	Other‡	Incidence rate ratio	Age group	Notification or hospitalisation rate
Invasive Hib disease	1.2	0.1	9.7	0–4	10.0
Hepatitis A	9.1	3.1	2.9	0–4	37.1
Hepatitis B	7.2	1.6	4.4	15–24	14.1
Influenza§	49.3	17.1	2.9	0–4	127.3
Measles	0.2	0.4	0.6	–	–
Meningococcal disease	7.2	3.4	2.1	0–4	50.7
Pertussis	41.8	46.9	0.9	0–4	89.7
Pneumococcal disease	44.7	9.9	4.5	0–4	87.0

* Notifications (New South Wales, Northern Territory, South Australia and Western Australia only) where the date of onset was between 1 January 2000 and 31 December 2002, except for pneumococcal disease, which is from 1 January 2001 to 31 December 2002.

† Notifications per 100,000 population (unless otherwise specified), age standardised to the Australian Bureau of Statistics Australian estimated population 2001.[6]

‡ Includes records where Indigenous status was not stated.

§ Influenza data are hospitalisations (all states and territories) where the month of separation was between 1 July 1999 and 30 June 2002.

Haemophilus influenzae type b

Case definitions

Notifications

(a) A clinically compatible illness (meningitis, epiglottitis, cellulitis, septic arthritis, osteomyelitis, pneumonia, pericarditis or septicaemia) *and* either:

isolation of *Haemophilus influenzae* type b from blood *or*

detection of Hib antigen (in a clinically compatible case) *or*

detection of Gram-negative bacteria of characteristic appearance where the organism fails to grow in a clinical case

or

b) A confident diagnosis of epiglottitis by direct vision, laryngoscopy or X-ray.

Hospitalisations and deaths

Hospitalisations and deaths were not analysed as there are no ICD–10–AM/ICD–10 codes which specify Hib as a causative organism, as opposed to *Haemophilus influenzae* (type unspecified).

Distribution by age and Indigenous status

In 2000–2002, there were a total of 47 notifications of invasive Hib disease in the jurisdictions with adequate data, 24 in children 0–4 years, of whom 11 (46%) were identified as Aboriginal and Torres Strait Islander (Table 2). The notification rate of 10 per 100,000 in Aboriginal and Torres Strait Islander children under five years of age gave a rate ratio of 15 for this age group, compared to four in persons five years and over. Cases in Aboriginal and Torres Strait Islander children were not wholly or predominantly reported from any one jurisdiction.

A similar number of notifications were received for persons 0–4 years and five years of age and over. Among 0–4 year olds, there was a striking difference in the annual notification rates for Aboriginal and Torres Strait Islander (10 per 100,000) and other children (0.7 per 100,000).

Comment

The Hib immunisation program in Australia commenced in April 1993, with catch-up immunisation for children up to five years of age from July 1993. Until June 2000, Aboriginal and Torres Strait Islander children were scheduled to receive a different Hib vaccine (conjugated to the outer membrane of *Neisseria meningitidis* type C, PRP–OMP) than other children who received a vaccine conjugated to a mutant diphtheria toxin (CRM197). Since June 2000, all Australian children have received PRP–OMP vaccine.

Following the introduction of Hib vaccination in 1993, children aged up to 15 years and children born since the introduction of the program up to 10 years of age by July 2003 were eligible to receive it. Indigenous status was poorly reported in the National Notifiable Diseases Surveillance System until the late 1990s, but an enhanced surveillance scheme for invasive Hib disease, established in 1993, included Indigenous status.[21] Vaccination has had a striking impact on the incidence of Hib disease in the age groups targeted by immunisation programs, among both Indigenous and non-Indigenous children.[21] Compared to an incidence of 35–40 per 100,000 in the general population and up to 280 per 100,000 in Aboriginal and Torres Strait Islander children living in the Northern Territory,[22] notification rates presented in this report (0.7 and 10.0 respectively in 2000–2002) represent a reduction of almost 98 per cent since vaccination was introduced.

Table 2. *Haemophilus influenzae* type b notifications, selected Australian States,* 2000 to 2002, by Indigenous status

Age group (years)	Indigenous status	Notifications (2000–2002)		
		n	Rate†	Rate ratio
0–4	Indigenous	11	10.0	14.7
	Other	13	0.7	
5 and over	Indigenous	2	0.3	3.6
	Other	21	0.1	
All ages‡	Indigenous	13	1.2	9.7
	Other	34	0.1	

* New South Wales, Northern Territory, South Australia and Western Australia only, where the date of onset was between 1 January 2000 and 31 December 2002.

† Average annual age-specific rate per 100,000 population.

‡ Rates for all ages combined are age standardised to the Australian Bureau of Statistics Australian population estimates for 2001.[6]

Although the number of cases has decreased markedly, the proportion of total Hib disease cases occurring in Aboriginal and Torres Strait Islander people has increased from around seven per cent before 1993 to 15 per cent in 2000.[21] This report shows a further increase in the differential between Aboriginal and Torres Strait Islander people and others to a rate ratio of 10 overall and 15 among children under five years of age. However, the point estimate for incidence in Aboriginal and Torres Strait Islander children under five years of 10.0 per 100,000 compares favourably with that among American Indian children in 1998–2000.[21] A similar pattern of increasing disparity in Hib disease rates has also been observed between Maori-Pacific Islander children and other children in New Zealand.[23] The available data indicate that Hib immunisation rates are acceptable in Aboriginal and Torres Strait Islander children in some geographic areas.[22] However, the role of regional variations in immunisation coverage should be examined as a likely reason for this increasing disparity. Crowded living conditions may also be a factor, being associated with high levels of Hib colonisation in the nasopharynx. In the meantime, the experience of similar Indigenous populations in Alaska[24] suggests that continued use of a Hib vaccine which is immunogenic after the first dose is appropriate in Aboriginal and Torres Strait Islander children.

Hepatitis A

Infection with the hepatitis A virus (HAV), a picorna virus, produces symptoms with a wide range from subclinical hepatitis to acute hepatitis with jaundice and, in the most severe cases, fulminant hepatitis leading to death. The single most important factor in determining the outcome of HAV infection is age. In symptomatic adult cases, onset of clinical symptoms is usually abrupt with fever, anorexia, malaise, nausea and abdominal discomfort followed by jaundice. In contrast, over 90 per cent of infections acquired before the age of five years are clinically silent. In adults, the proportion of infected individuals showing symptoms is thought to be around 90 per cent.[25,26]

Distribution by Indigenous status and age

Of the total of 1,012 notifications of hepatitis A recorded in the relevant jurisdictions, 113 (11%) were identified as occurring in Aboriginal and Torres Strait Islander people, as were 82 (6%) of the 1,309 hospitalisations recorded nationally. The overall rate ratio of cases identified as Indigenous to those presumed to be non-Indigenous was three for notifications and two for hospitalisations. Both hospitalisation (16 per 100,000) and notification (37 per 100,000) rates were highest among children 0–4 years identified as Indigenous, with the absolute number of cases higher than in other children (Table 3) and rate ratios of 57 and 22 respectively. This excess morbidity falls sharply with age, with smaller but substantial Indigenous versus other rate ratios among children 5–14 years of age (8 for notifications, 7 for hospitalisations), decreasing to two or less from the age of 15 years. Figures 1 and 2 illustrate the striking difference between young children and others when comparing both notifications and hospitalisations for Indigenous and other people.

Hepatitis A

Case definitions

Notifications

a) Detection of anti-hepatitis A virus IgM antibody, in the absence of recent vaccination

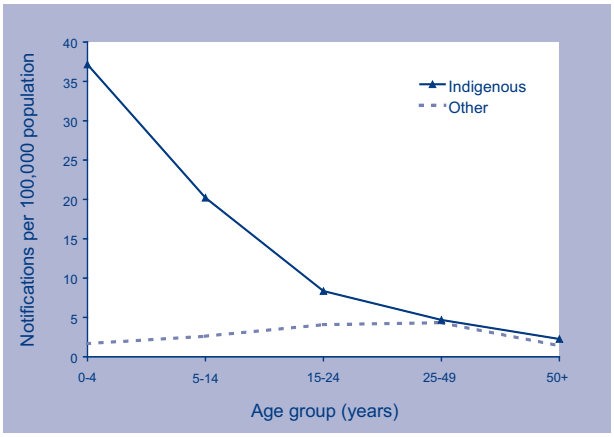
or

b) A clinical case of hepatitis (jaundice ± elevated aminotransferase levels, without a non-infectious cause) *and* an epidemiological link to a serologically confirmed case.

Hospitalisations and deaths

The ICD–10–AM/ICD–10 codes B15 (hepatitis A) were used to identify hospitalisations and deaths.

Figure 1. Hepatitis A notification rate, selected Australian States,* 2000 to 2002,† by age group and Indigenous status



* New South Wales, South Australia, Western Australia and the Northern Territory.

† Notifications where the date of onset was between 1 January 2000 and 31 December 2002.

Table 3. Hepatitis A notifications, hospitalisations and deaths, Australia, 1999 to 2002,* by age group and Indigenous status

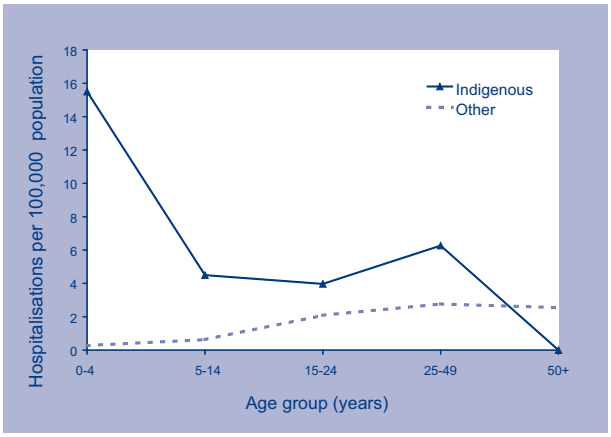
Age group (years)	Indigenous status	Notifications (2000–2002)			Hospitalisations (July 1999–June 2002)			Deaths (2000–2002)
0–4	Indigenous	41	37.1	22.2	28	15.5	56.8	1
	Non-Indigenous	32	1.7		10	0.3		0
5–14	Indigenous	44	20.2	7.7	16	4.5	7.1	0
	Other	105	2.6		49	0.6		0
15–24	Indigenous	13	8.4	2.0	10	4.0	1.9	0
	Other	163	4.1		161	2.1		0
25–49	Indigenous	13	4.7	1.1	28	6.3	2.3	0
	Other	479	4.3		585	2.8		0
50+	Indigenous	2	2.3	1.6	0	0.0	0.0	0
	Other	120	1.4		422	2.5		2
All ages‡	Indigenous	113	9.1	2.9	82	4.5	2.1	1
	Other	899	3.1		1,227	2.1		2

* Notifications (New South Wales, Northern Territory, South Australia and Western Australia only) where the date of onset was between 1 January 2000 and 31 December 2002; hospitalisations (all States) where the date of separation was between 1 July 1999 and 30 June 2002; deaths (Queensland, Northern Territory, South Australia, Western Australia) where the date of death was recorded between 1 January 2000 and 31 December 2002.

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with unknown ages. Rates for all ages combined are age standardised to the Australian Bureau of Statistics Australian population estimates for 2001.[6]

Figure 2. Hepatitis A hospitalisation rate, Australia, 1999 to 2002,* by age group and Indigenous status



* Hospitalisations where the date of separation was between 1 July 1999 and 30 June 2002.

Comment

The data in this report show a high burden of hepatitis A among Aboriginal and Torres Strait Islander children, with a progressive decrease in incidence with increasing age. The high rate ratio for hospitalisations due to hepatitis A in children under the age of five years is particularly noteworthy, indicating that higher rates of disease are reflected in significantly higher levels of morbidity in Aboriginal and Torres Strait Islander children. One death was coded as due to hepatitis A in an Aboriginal and Torres Strait Islander child in the reporting period, and other such deaths have been reported recently in North Queensland.[27]

The pattern of acquisition of hepatitis A is known to vary substantially according to living standards. More advantaged communities have delayed or no exposure to hepatitis A, with the majority seronegative even in middle age, while communities living in crowded and/or less hygienic circumstances acquire infection and immunity to hepatitis A at an early age. In the Northern Territory in 1994, a serosurvey in rural Aboriginal populations found hepatitis A to be hyperendemic, with acquisition of the virus predominantly in the first five years of life,[28] as is characteristic of disadvantaged living conditions. It was argued that, in such circumstances, immunisation is of little

benefit and may produce a cohort whose immunity could subsequently wane.[28] Although case-fatality and hospitalisation rates are low in children, with a high rate of infection some serious outcomes can be expected. In comparable Indigenous populations in the United States of America (USA), with similar high infection rates and age distribution, community-wide hepatitis A immunisation was recommended[29] and has resulted in dramatic reductions in the incidence of hepatitis A.[30] The recommendations have been expanded more recently to include the routine vaccination of children in areas where the rate of hepatitis A exceeds 20 per 100,000 population.[29]

In Australia, an immunisation program for hepatitis A was commenced among children from 18 months of age in North Queensland in 1999, in response to two deaths in Far North Queensland Aboriginal and Torres Strait Islander children from fulminant hepatitis A.[27] Early results from this program indicate that despite targeting only Aboriginal and Torres Strait Islander children, substantial reductions in hepatitis A across all sectors of the population groups have occurred.[31] Currently, no funded hepatitis A immunisation program exists in Australia outside of north Queensland. The favourable experience with hepatitis A immunisation programs in north Queensland, and in high incidence areas of the United States of America, is in accord with experience in other highly endemic areas.[32] The Australian Technical Advisory Group on Immunisation will examine the use of hepatitis A vaccination for children, particularly in relation to Indigenous children, in 2004.

Hepatitis B (acute)

Acute infection with hepatitis B virus (HBV), a hepadnavirus, may produce a range of conditions from subclinical hepatitis to acute hepatitis with jaundice and, rarely, fulminant hepatitis. Only a small proportion of HBV infections are clinically recognised, with less than 10 per cent of children and 30–50 per cent of adults experiencing clinical symptoms.[33] Onset of illness, when it occurs, is usually insidious with anorexia, vague abdominal discomfort, nausea and vomiting and sometimes arthralgia and rash, often progressing to jaundice. The principal cause of morbidity and mortality from hepatitis B is chronic infection, which may occur irrespective of symptoms. Chronic infection can lead to cirrhosis of the liver and hepatocellular carcinoma, usually over a prolonged period.[34] The risk of chronic infection is greatest in those infected as infants, particularly if infected in the perinatal period. Accordingly, preventive efforts have been focussed, in both the Indigenous and non-Indigenous community in Australia, on preventing maternal-infant transmission.

Hepatitis B (acute)

Case definitions

Notifications

People who have a positive hepatitis B surface antigen (HBsAg) *and* one of the following:

hepatitis B core antibody (Anti-HBc) IgM

or

demonstration of a clinical illness consistent with acute viral hepatitis (jaundice, elevated aminotransferase levels).

Hospitalisations

ICD–10–AM code B16 (acute hepatitis B) was used to identify hospitalisations.

As in previous reports,[12] hospitalisations were included only where the relevant ICD code was the principal diagnosis (which was in 30% of all hospitalisations which included acute hepatitis B). This is a much lower proportion than for the other diseases but similar to previous analyses of hepatitis B hospitalisations.[33]

Deaths

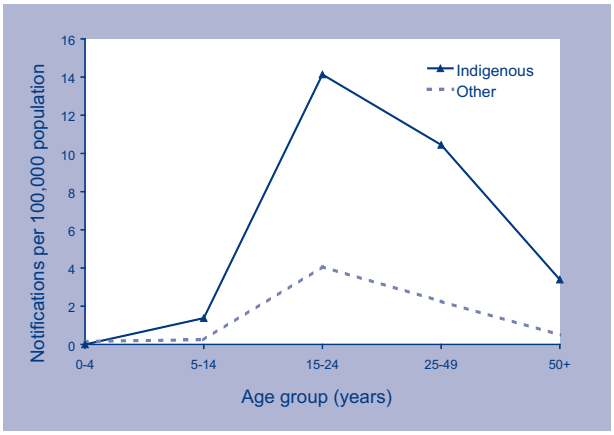
ICD–10 code B16 (acute hepatitis B) was used to select deaths from acute hepatitis B.

Distribution by Indigenous status and age

Of the total of 526 notifications of acute hepatitis B recorded in the relevant jurisdictions, 57 (11%) were identified as occurring in Aboriginal and Torres Strait Islander people, as were 30 (6%) of the 463 hospitalisations recorded nationally. The overall rate ratio for cases identified as Indigenous compared to those presumed to be non-Indigenous was four for both notifications and hospitalisations (Table 4). In contrast to hepatitis A, no notified or hospitalised cases of hepatitis B were recorded as Indigenous among children 0–4 years. Notification rates for acute hepatitis B then increased progressively among Aboriginal and Torres Strait Islander people, reaching a peak among 15–24 year olds and continuing to show a higher incidence in absolute and relative terms compared with other groups at all ages, maximal in those over 50 years (Figure 3). This was also reflected in hospitalisations (Figure 4). The highest notification rates per 100,000 were seen in both Indigenous and other populations in the 15–24 years (14 versus 4) and

25–49 years (10 versus 2) age groups. The highest hospitalisation rates per 100,000 were seen in cases aged 25–49 years cases identified as Indigenous (5 per 100,000) with a rate ratio of four. Of the 10 deaths recorded, two were in persons identified as Indigenous, both aged 25–49 years.

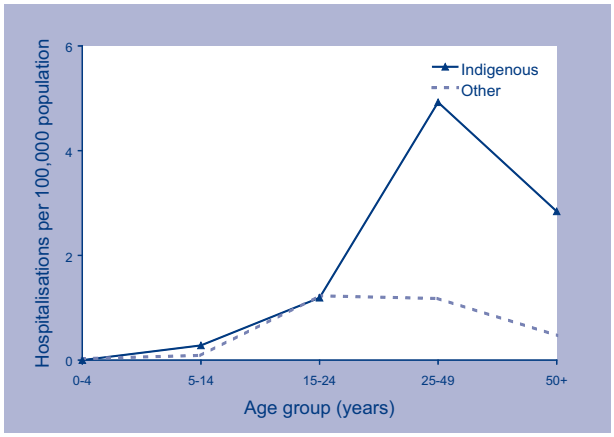
Figure 3. Acute hepatitis B notification rate, selected Australian States,* 2000 to 2002,† by age group and Indigenous status



* New South Wales, South Australia, Western Australia and the Northern Territory.

† Notifications where the date of onset was between 1 January 2000 and 31 December 2002.

Figure 4. Acute hepatitis B hospitalisation rate, Australia, 1999 to 2002,* by age group and Indigenous status



* Hospitalisations where the date of separation was between 1 July 1999 and 30 June 2002.

Table 4. Acute hepatitis B notifications, hospitalisations and deaths, Australia, 1999 to 2002,* by age group and Indigenous status

Age group (years)	Indigenous status	Notifications (2000–2002)			Hospitalisations (July 1999–June 2002)			Deaths (2000–2002)
0–4	Indigenous	0	0.0	0.0	0	0.0	0.0	0
	Other	3	0.2		1	0.0		0
5–14	Indigenous	3	1.4	5.5	1	0.3	3.1	0
	Other	10	0.2		7	0.1		0
15–24	Indigenous	22	14.1	3.5	3	1.2	1.0	0
	Other	162	4.1		98	1.3		2
25–49	Indigenous	29	10.4	4.6	22	4.9	4.2	2
	Other	249	2.3		250	1.2		1
50+	Indigenous	3	3.4	6.8	4	2.8	6.0	0
	Other	43	0.5		77	0.5		5
All ages‡	Indigenous	57	7.2	4.4	30	2.8	3.7	2
	Other	469	1.6		433	0.8		8

* Notifications (New South Wales, Northern Territory, South Australia and Western Australia only) where the date of onset was between 1 January 2000 and 31 December 2002; hospitalisations (all States) where the date of separation was between 1 July 1999 and 30 June 2002; deaths (Northern Territory, South Australia, Queensland, Western Australia) where the date of death was recorded between 1 January 2000 and 31 December 2002.

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with unknown ages. Rates for all ages combined are age standardised to the Australian Bureau of Statistics Australian population estimates for 2001.[6]

Comment

The mortality and morbidity from hepatitis B among Aboriginal and Torres Strait Islander people has been recognised for at least two decades and led to childhood hepatitis B vaccination programs being introduced in the Northern Territory in the 1980s.[33] Aboriginal and Torres Strait Islander newborns were also targeted for risk-based hepatitis B immunisation programs, along with children born to hepatitis B surface antigen positive mothers or parents born in countries with a high incidence of hepatitis B from this time. In contrast to hepatitis A, the greatest differential morbidity from hepatitis B is in the age groups over 15 and particularly over 25 years. This is consistent with some impact from immunisation programs, as the oldest cohorts of children targeted for immunisation would now be around 15 years of age.

The highest incidence of acute hepatitis B and the greatest differential between Indigenous and other hepatitis B incidence has always been in adolescents and young adults,[33] and remains so in these data. Hepatitis B immunisation could be considered for adolescents and young adults, including prison populations, until birth cohorts eligible for hepatitis B vaccine reach adulthood. While providing information on acute disease, these data do not reflect the significant chronic disease burden from hepatitis B for Aboriginal and Torres Strait Islander people, including liver cancer.

Influenza and pneumonia

Influenza is an acute respiratory illness caused by influenza type A or B viruses. Onset of clinical symptoms is typically abrupt with fever, cough, myalgia and prostration. Outbreaks of variable severity occur almost every winter in Australia and are associated with significant morbidity and mortality, particularly in the elderly and chronically ill. The most common complication of influenza is pneumonia. It is generally believed that hospitalisations and deaths coded as influenza significantly underestimate disease burden, with excess all-cause pneumonia and influenza combined, during the influenza season, being a better indicator of true disease burden.[35]

Distribution by Indigenous status and age

Rates of hospitalisation were higher in all Indigenous age groups for both influenza and influenza and pneumonia combined, with the highest rate ratio (7) for influenza and pneumonia combined in the 25-49 years age group (Table 5). While rates of influenza and pneumonia combined are substantially higher than for influenza alone, the pattern of distribution by age group is very similar (Figures 5 and 6, allowing for different scales). The rate ratio for deaths was highest in those aged 25–49 (28), 15–24 (15) and 0–4 years (17).

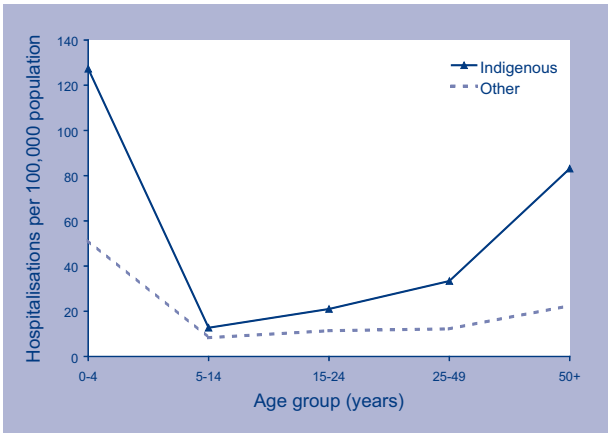
Influenza and pneumonia

Case definitions

Hospitalisations and deaths

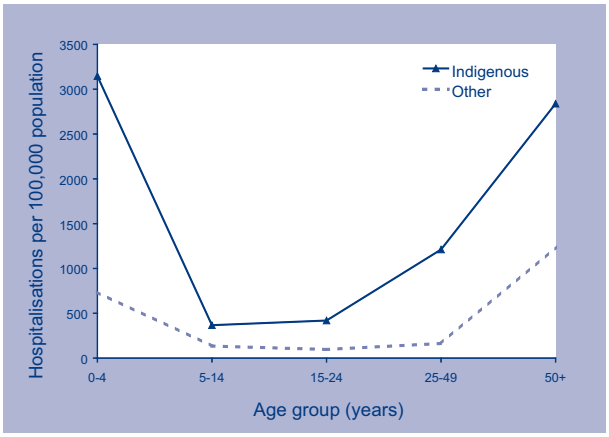
The ICD–10–AM/ICD–10 codes J10 and J11 (influenza) were used to identify hospitalisations and deaths from influenza. The ICD–10–AM/ICD10 codes J10 to J18 were used to identify hospitalisations and deaths from influenza and all-cause pneumonia combined.

Figure 5. Hospitalisation rate for influenza, Australia, 1999 to 2002,* by age group and Indigenous status



* Hospitalisations where the date of separation was between 1 July 1999 and 30 June 2002.

Figure 6. Hospitalisation rate for influenza and all pneumonia combined, Australia, 1999 to 2002,* by age group and Indigenous status



* Hospitalisations where the date of separation was between 1 July 1999 and 30 June 2002.

Table 5. Influenza and pneumonia hospitalisations and deaths, Australia 2000 to 2002,* by age group and Indigenous status

Age group (years)	Indigenous status	Hospitalisations (July 1999–June 2002)						Deaths (2000–2002)			
		Influenza			Influenza and pneumonia			Influenza	Influenza and pneumonia		
		n	Rate†	Rate ratio	n	Rate†	Rate ratio	n	n	Rate†	Rate ratio
0–4	Indigenous	230	127.3	2.5	5,682	3,146	4.3	0	18	10.0	17.4
	Other	1,877	51.2		26,885	733		3	21	0.6	
5–14	Indigenous	45	12.7	1.5	1,304	367	2.7	0	1	0.3	10.9
	Other	643	8.3		10,471	135		0	2	0.0	
15–24	Indigenous	53	21.0	1.8	1,056	419	4.4	0	1	0.4	15.3
	Other	883	11.4		7,383	96		1	2	0.0	
25–49	Indigenous	149	33.4	2.7	5,416	1,212	7.4	0	34	7.6	27.8
	Other	2,591	12.2		34,536	163		3	58	0.3	
50+	Indigenous	117	83.1	3.7	3,997	2,838	2.3	2	76	54.0	2.6
	Other	3,725	22.5		204,601	1,236		54	3,486	21.0	
All ages‡	Indigenous	594	49.3	2.9	17,455	1,580	3.2	2	130	19.1	3.1
	Other	9,719	17.1		283,876	495		61	3,569	6.2	

* Hospitalisations (all States) where the date of separation was between 1 July 1999 and 30 June 2002; deaths (Queensland, Northern Territory, South Australia and Western Australia) where the date of death was recorded between 1 January 2000 and 31 December 2002.

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with unknown ages. Rates for all ages combined are age standardised to the Australian Bureau of Statistics Australian population estimates for 2001.[6]

Comment

The relatively high morbidity from influenza and related conditions in older Aboriginal and Torres Strait Islander adults led to a specific program for influenza and pneumococcal vaccine being funded nationally for Indigenous adults over 50 years in 1999 and those aged 15–49 years with risk factors.[36] This is in contrast to the non-Indigenous population, for whom influenza vaccine is funded only from age 65 years and pneumococcal vaccine is funded only in Victoria for this age group. Young children also experience high morbidity from influenza, with minimal estimates, such as those in this report based on hospital coding data, still showing a high incidence of hospitalisation under five years of age. Although only five per cent of deaths coded as attributable to influenza nationally were in persons under five years and none in Aboriginal and Torres Strait Islander children, there was a twofold differential in the influenza hospitalisation rate between Indigenous and other children.

The importance of young children, both in terms of their own high morbidity from influenza and their role in transmission of influenza to adults, has been increasingly appreciated in recent years.[37] This has led to a recommendation in the United States of America that all children between six and 24 months of age receive influenza vaccine.[38] This recommendation is particularly pertinent to Aboriginal and Torres Strait Islander children in Australia, where influenza may contribute significantly to overall respiratory morbidity in children and adults they are in contact with, especially in crowded living conditions. Evaluation of the reach and impact of the current National Indigenous Pneumococcal and Influenza Immunisation (NIPII) program in adults is currently underway, and the potential role of influenza immunisation in children or universally in at least some communities of Aboriginal and Torres Strait Islander people is needed. Subsequently, consideration of the role of influenza immunisation in young Aboriginal and Torres Strait Islander children would be appropriate.

Measles

Measles is an acute and highly communicable disease caused by a morbillivirus. The clinical picture includes a prodromal fever, rash, conjunctivitis, coryza, cough and Koplik spots on the buccal mucosa. Complications include otitis media, pneumonia and encephalitis. Subacute sclerosing panencephalitis occurs very rarely as a late sequel.[35]

Distribution by Indigenous status

Only 113 notifications and 172 hospitalisations recorded as being associated with measles were seen in the relevant jurisdictions or Australia respectively during the reporting period examined. Of these, only 3 (2.6%) notifications and 2 (1.1%) hospitalisations were recorded as Indigenous, with equivalent incidence in Indigenous and other persons (Table 6). No deaths were recorded in the reporting period.

Comment

Two significant milestones in measles control occurred in Australia in 1998; the Measles Control Campaign (MCC), involving catch-up immunisation of 1.3 million children aged 5–12 years, and the moving of the second dose of measles-mumps-rubella (MMR) vaccine from 12 to four years.[39] This reporting period follows the MCC and is in a period when transmission of measles in Australia is believed to have been largely limited to introduction of virus from overseas, with limited local transmission among age-groups with high levels of immunity from immunisation (under 15 years) or past infection (over 35 years).[40] Measles outbreaks have been almost entirely confined to children who were unimmunised, either because of young age or refusal to vaccinate, and to young adults aged 18–30 years.[41]

Measles

Case definitions

Notifications

An illness characterised by all the following features:

a generalised maculopapular rash lasting three or more days, *and*

a fever (at least 38º C if measured), *and*

cough or coryza or conjunctivitis or Koplik spots

or

Demonstration of measles specific IgM antibody

or

A fourfold or greater rise in measles antibody titre between acute and convalescent phase sera obtained at least two weeks apart, with tests preferably conducted at the same laboratory

or

Isolation of measles virus from a clinical specimen

or

A clinically compatible case epidemiologically related to another case.

Hospitalisations and deaths

The ICD–10–AM/ICD–10 code B05 (measles) was used to identify hospitalisations and deaths. Subacute sclerosing panencephalitis was not included in these analyses.

Table 6. Measles notifications, hospitalisations and deaths, Australia, 1999 to 2002,* by age group and Indigenous status

Age group (years)	Indigenous status	Notifications (2000–2002)			Hospitalisations (July 1999–June 2002)			Deaths (2000–2002)
All ages‡	Indigenous	3	0.2	0.6	2	0.1	0.4	0
	Other	110	0.4		170	0.3		0

* Notifications (New South Wales, Northern Territory, South Australia and Western Australia only) where the date of onset was between 1 January 2000 and 31 December 2002; hospitalisations (all States) where the date of separation was between 1 July 1999 and 30 June 2002; deaths (Queensland, Northern Territory, South Australia and Western Australia) where the date of death was recorded between 1 January 2000 and 31 December 2002.

† Average annual rate per 100,000 population.

‡ Includes cases with unknown ages. Rates are age standardised to the Australian Bureau of Statistics Australian population estimates for 2001.[6]

Measles was associated with high levels of morbidity among Aboriginal and Torres Strait Islander children in the past, prompting the Northern Territory to introduce immunisation at nine months of age, with a subsequent booster, for Aboriginal children.[42] This program ended in the Northern Territory in 1998[43] and no excess morbidity from measles is evident in this or other Australian jurisdictions. The successful control of measles in Aboriginal and Torres Strait Islander people is a reflection of the almost total success of immunisation in preventing measles transmission, in contrast to other VPDs such as pertussis or Hib disease. It illustrates the importance of universal programs, across all relevant age groups in the population, in disease prevention in both Indigenous and non-Indigenous people.

Meningococcal disease

Clinical manifestations of meningococcal disease include meningitis, meningococcaemia without meningitis (which varies in presentation from fulminant to chronic) and septic arthritis.

Meningococcal disease

Case definitions

Notifications

Isolation of *Neisseria meningitidis* from a normally sterile site

or

Detection of meningococcal antigen in joints, blood or cerebrospinal fluid

or

Detection of Gram-negative intracellular diplococci in blood or cerebrospinal fluid.

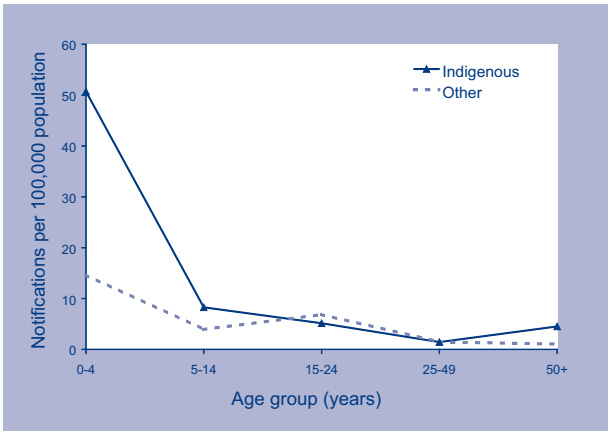
Hospitalisations and deaths

The ICD–10–AM/ICD–10 code A39 (meningococcal infection) was used to identify hospitalisations and deaths.

Distribution by Indigenous status and age

Notification and hospitalisation rates (per 100,000 population) showed a progressive decrease with increasing age in both Indigenous and other persons (Table 7). The notification rate fell from a peak of 51 in the 0–4 age group for Indigenous and 15 in other children, to eight and four respectively in 5–14 year olds, with the lowest rates in 25–49 year olds in both groups (Figure 7). Overall, the rate ratio for notifications comparing Indigenous versus others was two, but was close to unity among 15–49 year olds, differing only at the extremes of age. The pattern for hospitalisations was similar, with again the lowest hospitalisation rates among Aboriginal and Torres Strait Islander people aged 15–49 years, although one of the five Indigenous deaths recorded occurred in this age band (Figure 8).

Figure 7. Meningococcal disease notification rate, selected Australian States*, 2000 to 2002,† by age group and Indigenous status



* New South Wales, South Australia, Western Australia and the Northern Territory.

† Notifications where the date of onset was between 1 January 2000 and 31 December 2002.

Serogroup distribution by Indigenous status and age

Serogroup data were not available for hospitalisations and were missing for 63 per cent of Indigenous and 45 per cent of other notifications, so conclusions about serogroup distribution are subject to significant limitations. In notifications where serogroup was known, a higher proportion of cases identified as Indigenous were serogroup B (73% compared to 59% in other cases) with a lower proportion due to serogroup C (27% compared to 38%).

Table 7. Meningococcal disease notifications, hospitalisations and deaths, Australia, 1999 to 2002,* by age group and Indigenous status

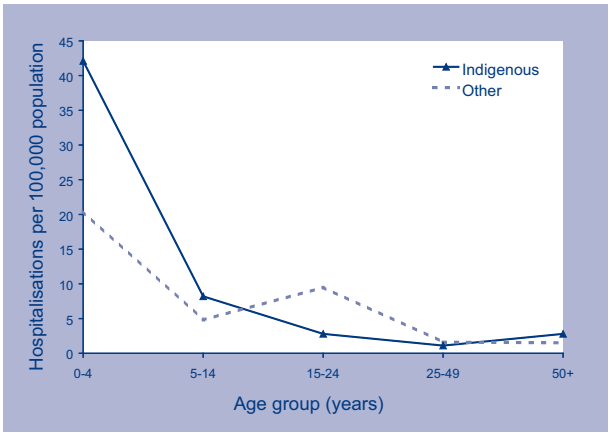
Age group (years)	Indigenous status	Notifications (2000–2002)			Hospitalisations (July 1999–June 2002)			Deaths (2000–2002)
		n	Rate†	Rate ratio	n	Rate†	Rate ratio	n
0–4	Indigenous	56	50.7	3.5	76	42.1	2.1	4
	Other	280	14.6		731	20.4		12
5–14	Indigenous	18	8.3	2.1	29	8.2	1.7	0
	Other	158	3.9		364	4.8		3
15–24	Indigenous	8	5.1	0.7	7	2.8	0.3	1
	Other	273	6.9		756	9.5		5
25–49	Indigenous	4	1.4	1.0	5	1.1	0.7	0
	Other	162	1.5		349	1.6		8
50+	Indigenous	4	4.5	4.3	4	2.8	1.9	0
	Other	91	1.1		240	1.5		5
All ages‡	Indigenous	92	7.2	2.1	121	5.5	1.3	5
	Other	975	3.4		2,440	4.3		33

* Notifications (New South Wales, Northern Territory, South Australia and Western Australia only) where the date of onset was between 1 January 2000 and 31 December 2002; hospitalisations (all States) where the date of separation was between 1 July 1999 and 30 June 2002; deaths (Queensland, Northern Territory, South Australia and Western Australia) where the date of death was recorded between 1 January 2000 and 31 December 2002.

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with unknown ages. Rates for all ages combined are age standardised to the Australian Bureau of Statistics Australian population estimates for 2001.[6]

Figure 8. Meningococcal disease hospitalisation rate, Australia, 1999 to 2002,* by age group and Indigenous status



* Hospitalisations where the date of separation was between 1 July 1999 and 30 June 2002.

Comment

The available data on meningococcal disease were limited by lack of serogroup information, which is important given the introduction of conjugate meningococcal C vaccine into the Australian Standard Vaccination Schedule (ASVS) for all children 1–19 years in 2002. The overall pattern of invasive meningococcal disease shown here indicates a secondary peak in notifications and hospitalisations among 15–24 year olds presumed non-Indigenous persons not evident in Indigenous people. In both groups, the age-specific incidence is highest among 0–4 year olds, being about tenfold higher in Indigenous 0–4 year olds (51) than in 15–24 year olds (5) and twofold higher in the comparable (presumed) non-Indigenous age groups. In keeping with this, the greatest differential disease burden was in 0–4 year olds, with a rate ratio of Indigenous to other notifications of 3.5. Indigenous deaths accounted for 13 per cent of the total, similar to the proportion of notifications (9%), but four of five these deaths (80%) occurred in 0–4 year olds compared with 12 (36%) of other deaths.

Historically, the incidence of meningococcal disease has been disproportionately higher among Aboriginal and Torres Strait Islander Australians, with well-recorded outbreaks in Central Australia[44] and north-west Queensland due to type A and type C disease.[45] The current pattern appears, from the limited data available, to be more one of serogroup B disease in the youngest children, similar to that seen in Maori and Pacific Islander children in New Zealand,[46] in whom living conditions have been shown to be an important disease risk factor.[47] Vaccines protecting against serogroup B disease will be an important consideration for Aboriginal and Torres Strait Islander children in the future, but will need to be effective against a wider range of serogroup B subtypes than the vaccine currently being evaluated in New Zealand.[48]

Pertussis

Pertussis (whooping cough) is an acute illness, caused by the *Bordetella pertussis* bacterium, involving the respiratory tract. The typical illness begins with an irritating cough that gradually becomes paroxysmal and lasts for 1–2 months or longer. Paroxysms are characterised by repeated violent coughs, followed by a characteristic crowing or high-pitched inspiratory whoop. Infants less than six months old, adolescents and adults often have fewer classical symptoms without paroxysms or whoop.[49]

Distribution by Indigenous status and age

The age-specific pattern of notifications among Indigenous and other people differed substantially, even though the overall notification rates were almost identical at 42 and 47 respectively per 100,000. For Aboriginal and Torres Strait Islander people, there was an almost twofold higher notification rate among 0–4 year olds and over 50 year olds, with relatively lower notification rates in those aged 5–49 years (Figure 9). In contrast, there was a relative excess of hospitalisations for Aboriginal and Torres Strait Islander people in all age groups, except 25–49 year olds, with an overall rate ratio of two (Figure 10). Hospitalisations were predominantly in those aged 0–4 years, (130/150, 87%) of those recorded as Indigenous and (957/1478, 62%) of other children.

Pertussis

Case definitions

Notifications

Isolation of *Bordetella pertussis* from a clinical specimen

or

Elevated *B. pertussis*-specific IgA in serum *or* the detection of *B. pertussis* antigen in a nasopharyngeal specimen using immunofluorescence with history of a clinically compatible illness

or

An illness lasting two weeks or more with one of the following:

paroxysms of coughing, *or*

inspiratory whoop without other apparent causes, *or*

post-tussive vomiting.

or

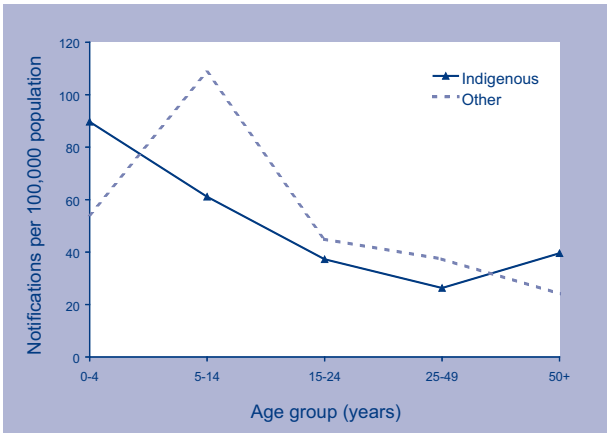
An illness characterised by a cough lasting at least two weeks in a patient who is epidemiologically linked to a laboratory confirmed case.

Hospitalisations and deaths

The ICD–10–AM/ICD–10 code A37 (whooping cough) was used to identify hospitalisations and deaths.

In view of the large differential in rates for 0–4 year olds and relatively large case numbers, incidence in the under one year age group was also examined. The notification rate (per 100,000 population) was substantially higher in Indigenous (308) compared to other infants (120), with a rate ratio twice that seen in the 0–4 years age group. However, while hospitalisation rates under 12 months were more than threefold higher than the overall 0–4 years age group at 196 per 100,000 Indigenous and 89 per 100,000 other, there was no difference between the rate ratios at 0–11 months and 12–59 months of age. Seven deaths were recorded as due to pertussis during the reporting period, five in presumed non-Indigenous children under 12 months of age and two in presumed non-Indigenous adults over 50 years.

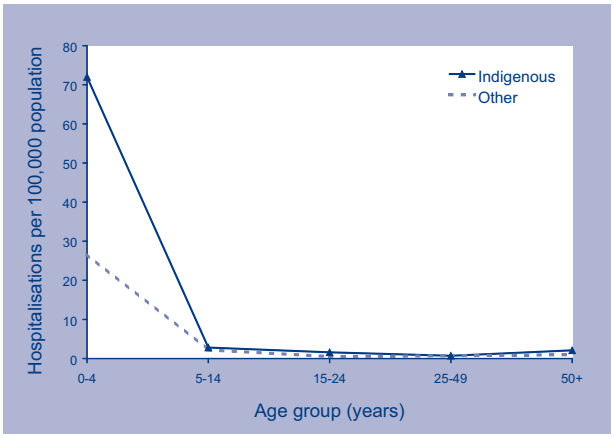
Figure 9. Pertussis notification rate, selected Australian States,* 2000 to 2002,† by age group and Indigenous status



* New South Wales, South Australia, Western Australia and the Northern Territory.

† Notifications where the date of onset was between 1 January 2000 and 31 December 2002.

Figure 10. Pertussis hospitalisation rate, Australia, 1999 to 2002,* by age group and Indigenous status



* Hospitalisations where the date of separation was between 1 July 1999 and 30 June 2002.

Table 8. Pertussis notifications, hospitalisations and deaths, Australia, 1999 to 2002,* by age group and Indigenous status

Age group (years)	Indigenous status	Notifications (2000–2002)			Hospitalisations (July 1999–June 2002)			Deaths (2000–2002)
		n	Rate†	Rate ratio	n	Rate†	Rate ratio	n
0–4	Indigenous	99	89.7	1.7	130	72.0	2.8	0
	Other	1,038	54.3		957	26.1		5
5–14	Indigenous	133	61.1	0.6	10	2.8	1.3	0
	Other	4,372	108.6		167	2.2		0
15–24	Indigenous	58	37.3	0.8	4	1.6	3.1	0
	Other	1,798	44.9		40	0.5		0
25–49	Indigenous	73	26.3	0.7	3	0.7	0.9	0
	Other	4,130	37.3		150	0.7		0
50+	Indigenous	35	39.6	1.6	3	2.1	2.2	0
	Other	2,083	24.1		164	1.0		2
All ages‡	Indigenous	408	41.8	0.9	150	6.2	2.4	0
	Other	13,528	46.9		1,478	2.6		7

* Notifications (New South Wales, Northern Territory, South Australia and Western Australia only) where the date of onset was between 1 January 2000 and 31 December 2002; hospitalisations (all States) where the date of separation was between 1 July 1999 and 30 June 2002; deaths (Queensland, Northern Territory, South Australia and Western Australia) where the date of death was recorded between 1 January 2000 and 31 December 2002.

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with unknown ages. Rates for all ages combined are age standardised to the Australian Bureau of Statistics Australian population estimates for 2001.[6]

Comment

Pertussis is the disease least well controlled of all the diseases with long-standing, well-established vaccination programs. It has the highest notification rate for all ages for the total Australian population[12] and, with the exception of influenza, meningococcal and pneumococcal diseases and varicella, the highest hospitalisation rates. The data in this report are the first to examine this in relation to Indigenous status nationally, although data have been reported previously from the Northern Territory.[50] Interpretation of the data is complex, because of the marked differences between age groups in immunisation history and disease severity as measured by hospitalisation and deaths. Despite this, the relatively large number of cases compared with other VPDs among Aboriginal and Torres Strait Islander people allows some interesting conclusions.

First, the greatest relative excess of pertussis is in 0–4 year olds, most marked in infants under 12 months of age and in hospitalisations. Second, the relatively lower notification rates and rate ratios seen between five and 49 years of age are not reflected in hospitalisation data. This could be artefactual, due to diagnostic practice or lack of access to laboratory facilities in more remote areas, or a real phenomenon, perhaps related to greater exposure to pertussis and more long-lasting immunity in Aboriginal and Torres Strait Islander adults and older children. Even in presumed non-Indigenous children, the age group 5–14 years is a diverse one in relation to vaccination history, as an additional dose of pertussis vaccine was included in the ASVS from 1994. More detailed analysis shows a progressive cohort effect among this age group, shown by high rates in the oldest members of the cohort and low rates among the younger ones, similar that of 1–4 year olds.[51] From the beginning of 2004, an additional dose of a pertussis-containing vaccine will replace diphtheria-tetanus vaccine for all Australian 15–17 year olds.[52] It will be important to monitor trends following this change according to Indigenous status to fully examine its impact.

Pneumococcal disease

Pneumococcal disease is caused by the bacterium *Streptococcus pneumoniae* (pneumococcus). Pneumococci are frequently isolated from the upper respiratory tract and can spread directly from the nasopharynx to cause infection in other parts of the respiratory tract (otitis media, sinusitis, pneumonia) or enter the bloodstream. Manifestations include meningitis, pneumonia and infection at a number of less common sites, as well as septicaemia without focal infection. Invasive pneumococcal disease (IPD) is defined as a sterile site isolate of *Streptococcus pneumoniae*, usually from blood. In the absence of a sterile site isolate, a presumptive diagnosis of pneumococcal pneumonia may be based on a sputum isolate of *Streptococcus pneumoniae* and/or clinical features such as the chest X-ray appearance and prompt response to antibiotic therapy. Invasive pneumococcal disease has been notifiable in Queensland and the Northern Territory since 1997. From January 2001, invasive pneumococcal disease became notifiable Australia-wide.

Pneumococcal disease

Case definitions

Notifications

Isolation from, or detection in, blood, cerebrospinal fluid or other sterile site, of *Streptococcus pneumoniae*

Hospitalisations

The ICD–10–AM codes used to identify hospitalisations were: G00.1, pneumococcal meningitis; A40.3, pneumococcal septicaemia (together considered to be a proxy for invasive pneumococcal disease); and J13, pneumococcal pneumonia.

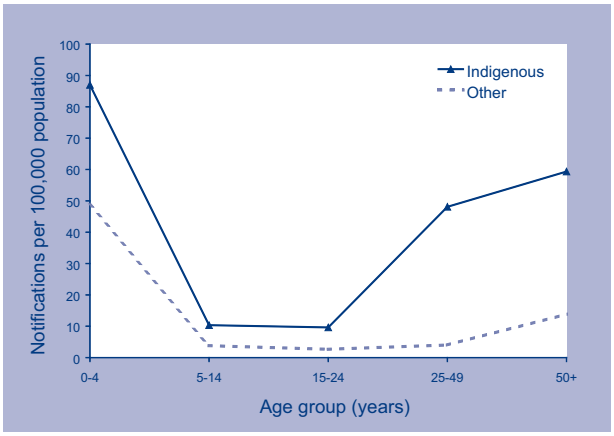
Deaths

ICD–10 codes G00.1 and A40.3 were used (together considered to be a proxy for invasive pneumococcal disease) to select deaths from IPD.

Distribution by Indigenous status and age

Notification and hospitalisation rates for invasive pneumococcal disease were higher for all Indigenous age groups with an overall Indigenous: other rate ratio of five for notifications and hospitalisations (Table 9). Rates per 100,000 population were highest in both Indigenous and other populations for the 0–4 years age group (notifications 87 versus 49; hospitalisations 45 versus 26) and 50+ years age group (notifications 59 versus 14; hospitalisations 29 versus 8). Importantly, the incidence of IPD began to increase significantly in the 25–49 years Indigenous age group to an incidence of 48 per 100,000 compared with four per 100,000 in other persons. The incidence rate ratios remained significantly higher at approximately three even for the lowest incidence age group of 5–24 years, with the notification rate among Aboriginal and Torres Strait Islander young people remaining 10 per 100,000, equivalent to that seen among other people over 50 years of age (Figure 11). The age distribution was similar for notifications of IPD and

Figure 11. Invasive pneumococcal disease notification rate, selected Australian States,* 2001 to 2002,† by age group and Indigenous status



* New South Wales, South Australia, Western Australia and the Northern Territory.

† Notifications where the date of onset was between 1 January 2001 and 31 December 2002.

Table 9. Invasive pneumococcal disease notifications, hospitalisations and deaths, Australia, 1999 to 2002*, by age group and Indigenous status

Age group (years)	Indigenous status	Notifications† (2001–2002)			Hospitalisations‡ (July 1999–June 2002)			Deaths (2000–2002)
		n	Rate§	Rate ratio	n	Rate§	Rate ratio	n
0–4	Indigenous	64	87.0	1.8	81	44.8	1.7	2
	Other	620	48.6		944	25.7		6
5–14	Indigenous	15	10.3	2.7	24	6.8	4.0	0
	Other	102	3.8		130	1.7		1
15–24	Indigenous	10	9.6	3.7	15	6.0	8.2	1
	Other	70	2.6		58	0.8		0
25–49	Indigenous	89	48.1	11.9	108	24.2	13.4	1
	Other	297	4.0		384	1.8		0
50+	Indigenous	35	59.4	4.3	41	29.1	3.7	1
	Other	800	13.9		1,290	7.8		9
All ages‖	Indigenous	214	44.7	4.5	269	22.0	4.5	5
	Other	1,926	9.9		2,806	4.9		16

* Notifications (New South Wales, Northern Territory, South Australia and Western Australia only) where the date of onset was between 1 January 2001 and 31 December 2002; hospitalisations (all States) where the month of separation was between 1 July 1999 and 30 June 2002; deaths (Queensland, Northern Territory, South Australia and Western Australia) where the date of death was recorded between 1 January 2000 and 31 December 2002.

† Notifications are presented for two years only as invasive pneumococcal disease became nationally notifiable in January 2001. Completeness of the Indigenous status data field was suboptimal for New South Wales data in 2001.

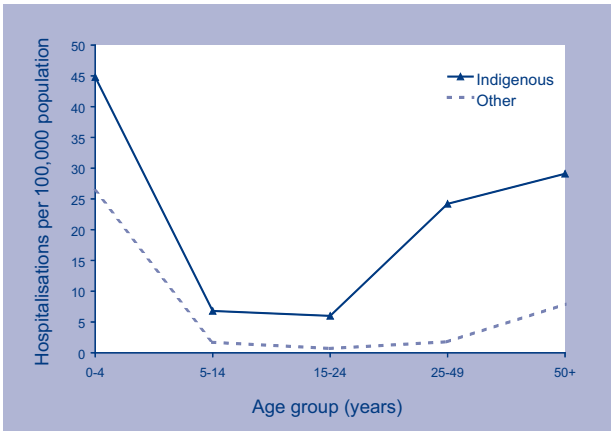
‡ Using hospitalisations for pneumococcal meningitis and pneumococcal septicaemia as a proxy for invasive pneumococcal disease

§ Average annual age-specific rate per 100,000 population.

‖ Includes cases with unknown ages. Rates for all ages combined are age standardised to the Australian Bureau of Statistics Australian population estimates for 2001.[6]

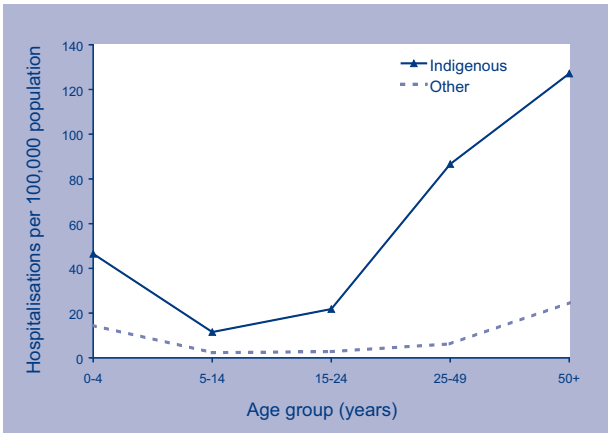
for hospitalisations. For hospitalisations coded as pneumococcal pneumonia, without septicaemia or meningitis, a similar pattern was also found, but was accentuated among those over 50 years and showed a much lower hospitalisation rate for 0–4 year olds (Figures 12 and 13). Death was recorded as due to IPD in 21 cases in the reporting period, 24 per cent of whom were Aboriginal and Torres Strait Islander. In 0–4 year olds, 2/8 deaths (25%) were in Aboriginal and Torres Strait Islander children, 2/3 (67%) among 5–49 year olds and 1/10 (10%) of deaths occurring in over 50 year olds.

Figure 12. Hospitalisation rate for pneumococcal meningitis and septicaemia, Australia, 1999 to 2002,* by age group and Indigenous status



* Hospitalisations where the date of separation was between 1 July 1999 and 30 June 2001.

Figure 13. Hospitalisation rate for pneumococcal pneumonia (not coded as meningitis or septicaemia), Australia, 1999 to 2002,* by age group and Indigenous status



* Hospitalisations where the date of separation was between 1 July 1999 and 30 June 2002.

Comment

Invasive pneumococcal disease is well recognised as causing a significant disease burden among Aboriginal and Torres Strait Islander people, especially in Northern and Central Australia.[53,54] The data presented in this report reinforce the importance of IPD as a priority for vaccine prevention in Aboriginal and Torres Strait Islander people of all ages. In particular, the data indicate that IPD has an incidence in younger Aboriginal and Torres Strait Islander adults (25–49 years) almost as high as in the age group currently targeted for pneumococcal vaccine, those over 50 years. It should be noted that the pattern of age-specific incidence seen for IPD is almost identical to that seen for influenza and pneumonia.

Currently, IPD is targeted for vaccine prevention among Aboriginal and Torres Strait Islander children 0–2 years of age, except in Central Australia and adjacent areas, where the conjugate pneumococcal vaccine program is extended to five years. From the age of 15 years, only adults with risk factors for IPD are included in recommendations for funding for polysaccharide vaccine, with the exception of the Northern Territory, where all Aboriginal and Torres Strait Islander adults aged 15 years or more are eligible. From the age of 50 years, funded vaccine is provided for all Aboriginal and Torres Strait Islander adults.

It is too early for definitive data to be available on the impact of the conjugate vaccine program among Aboriginal and Torres Strait Islander infants, but available data indicate that vaccine coverage is sub-optimal, particularly in the south-east of Australia. For adults, data from the program in North Queensland indicate that, in a setting where high coverage with pneumococcal polysaccharide vaccine has been achieved, substantial disease reductions have been observed.[55] However, recent data from the USA in Navajo populations suggest sub-optimal efficacy of pneumococcal polysaccharide vaccine, particularly in adults with underlying medical conditions such as alcoholism.[56] Based on the data presented here on disease rates, and assuming that vaccine effectiveness is adequate, a universal pneumococcal polysaccharide vaccination program for 25–49 year old Aboriginal and Torres Strait Islanders may be justified and should be further examined.

Vaccination coverage

Australian Standard Vaccination Schedule 1998 to 2003

The Australian Standard Vaccination Schedule for children aged 0–6 years changed in the second half of 1998, in mid 2000 and in January and September 2003. In 1998, the second dose of measles-mumps-rubella vaccine (previously given at 12–13 years) was moved to four years. For children born after May 2000, full vaccination at 12 months of age required immunisation against hepatitis B as well as three doses of diphtheria-tetanus-pertussis vaccine (DTP) and oral poliomyelitis (OPV) vaccines. Full immunisation against *Haemophilus influenzae* type b disease at 12 months required, from 2000, two doses of PRP–OMP (*Haemophilus influenzae* type b polysaccharide conjugated to the outer membrane protein of *Neisseria meningitidis*) vaccine for all children. The neonatal dose of hepatitis B vaccine (scheduled for all newborns since May 2000) is not accounted for in ACIR coverage estimates. In the second year of life, a dose of MMR vaccine is scheduled at 12 months of age as well as booster doses of DTP (at 18 months) and Hib vaccine (at 12 months); the 18 month dose of (DTP) was removed in September 2003. The 7-valent pneumococcal conjugate vaccine (7vPCV) was introduced for children at high risk of invasive pneumococcal disease (including all Aboriginal and Torres Strait Islander children) in June 2001. The meningococcal C conjugate vaccine (MenCCV) was introduced for all children in January 2003. The current ASVS is shown in Table 10.

Specific recommendations for Aboriginal or Torres Strait Islander people

There are several differences between the vaccines recommended for Indigenous and non-Indigenous Australians, mainly due to differences in disease incidence. The Australian Standard Vaccination Schedule contains recommendations specifically for Aboriginal and Torres Strait Islander people for influenza, pneumococcal disease and Hib disease. There are also additional recommendations for Aboriginal and Torres Strait Islander people for tuberculosis and hepatitis A vaccines, which are limited to some geographical regions.

The 23-valent pneumococcal polysaccharide vaccine is recommended for all Aboriginal and Torres Strait Islander people aged 50 years and over, and for those aged 15–49 years who have high-risk underlying conditions. In the Northern Territory, vaccination is recommended for all Aboriginal and Torres Strait Islanders aged 15 years or more. A single re-vaccination is generally recommended five years later (see the *Australian Immunisation Handbook* 8th edition for more details).[57] Annual influenza vaccination is also recommended for Aboriginal and Torres Strait Islander adults aged 50 years and over, and for those aged 15 to 49 years with high-risk underlying conditions. These vaccines are funded under the National Indigenous Pneumococcal and Influenza Immunisation Program. The recommendations for non-Indigenous adults differ in that vaccination for these two diseases is recommended from 65 years of age instead of 50. Influenza vaccine is provided for non-Indigenous adults aged 65 or more years under the National Immunisation Program. Pneumococcal vaccine is not publicly funded for non-Indigenous adults except in Victoria, although it is subsidised on the Pharmaceutical Benefits Scheme.

The 7-valent pneumococcal conjugate vaccine is recommended for all children at two, four and six months of age. It is provided free under the National Immunisation Program for all Aboriginal and Torres Strait Islander children and for non-Indigenous children with high risk conditions. Aboriginal and Torres Strait Islander children living in high incidence areas (Northern Territory, South Australia, Western Australia, Queensland) should also receive a dose of 23-valent vaccine at 18–24 months. Catch up vaccination is recommended for unvaccinated children up to five years of age in Central Australia and up to two years of age in other areas.

PRP-OMP (purified polysaccharide conjugated to an outer membrane protein carrier) Hib vaccine is immunogenic at an earlier age and therefore requires fewer doses than other Hib vaccines. For PRP-OMP vaccine, doses are recommended at two, four and 12 months of age, while other Hib vaccines require an extra dose at six months. Since Hib vaccine was first introduced onto the ASVS in 1993, PRP–OMP has been recommended for Aboriginal and Torres Strait Islander children, due to their higher risk of disease under six months of age. Other Hib vaccines were recommended for non-Indigenous children until May 2000. Since then, either PRP–OMP or PRP–T/HbOC vaccines are recommended for non-Indigenous children.

Since 1999, hepatitis A vaccine has been provided for all Aboriginal and Torres Strait Islander children in North Queensland at 18 and 24 months of age. Catch-up vaccination is provided to children up to six years of age.

Vaccination with BCG (Bacille Calmette-Guérin) vaccine for tuberculosis is recommended for all Aboriginal and Torres Strait Islander neonates in high incidence areas.

Table 10. The Australian Standard Vaccination Schedule[57]

Age	Vaccine								
Birth	Hepatitis B*								
2 months	Hepatitis B*	DTP_a	Hib‡	IPV‖			7vPCV**		
4 months	Hepatitis B*	DTP_a	Hib‡	IPV‖			7vPCV**		
6 months	Hepatitis B*	DTP_a	Hib§	IPV‖			7vPCV**		
12 months	Hepatitis B*		Hib‡		MMR			MenCCV	
18 months						VZV¶	23vPPV††		
2 years									
4 years		DTP_a		IPV‖	MMR				
10–13 years	Hepatitis B†					VZV¶			
15–17 years		dTp_a							
50 years and over		dT					23vPPV‡‡		Influenza (annual)‡‡
65 years and over							23vPPV		Influenza (annual)

Note: Vaccines currently funded under the National Immunisation Program are shaded in Table 10 above. Vaccines lightly shaded are funded for targeted, at-risk populations only.

* Four doses of hepatitis B vaccine recommended for each child, the timing of doses varies according to the vaccine formulation used.

† Two or three catch up doses recommended for children not previously immunised.

‡ Three doses of PRP–OMP recommended for all children.

§ A 4-dose schedule of PRP–T or HbOC may be given to non-Indigenous children as an alternative.

‖ IPV is recommended but not funded by the National Immunisation Program; OPV is funded as an alternative.

¶ Only for children with a negative history of disease or vaccination.

** Recommended for all children, provided free only to: all Aboriginal and Torres Strait Islander children aged up to two years, Indigenous children in Central Australia aged up to five years, non-Indigenous children in Central Australia aged up to two years, and all children under five years with medical risk factors that predispose them to high rates or high severity of pneumococcal infection.

†† Aboriginal and Torres Strait Islander children only.

‡‡ Aboriginal and Torres Strait Islander adults only, vaccines provided under the National Indigenous Pneumococcal and Influenza Immunisation Program.

Vaccine key

Hepatitis B	Hepatitis B vaccine	MMR	Measles-mumps-rubella vaccine
DTPa	Diphtheria-tetanus-acellular pertussis infant/child formulation	VZV	Varicella-zoster vaccine
dTpa	Adult diphtheria-tetanus-pertussis vaccine	7vPCV	7-valent pneumococcal conjugate vaccine
Hib	*Haemophilus influenzae* type b (Hib) vaccine (PRP–OMP, as monovalent or in combination)	23vPPV	23-valent pneumococcal polysaccharide vaccine
IPV	Inactivated poliomyelitis vaccine (in combination)	MenCCV	Meningococcal C conjugate vaccine
OPV	Oral poliomyelitis vaccine	Influenza	Influenza vaccine

Vaccination for Japanese encephalitis virus is recommended for all residents of the outer Islands of the Torres Strait aged over one year of age, and non-residents staying at least 30 days during the wet season.

Calculating vaccination coverage estimates from the ACIR

The methodology for calculating cohort-based vaccination coverage from the ACIR was described by O'Brien *et al*.[7] Using this method, a cohort of children is defined by date of birth in 3-month groups; the first cohort was born between 1 January 1996 and 31 March 1996. To minimise duplicate records, the cohort includes only children enrolled with Medicare. The vaccination status of each cohort is assessed at the two key milestones of 12 months and 24 months of age. Coverage is measured several months after the due date for completion of each milestone, to allow for delayed notification to the ACIR. It is assumed that notification of receipt of a later vaccine dose implies receipt of earlier doses, even if no earlier vaccination is recorded ('third dose assumption').[58]

A child is now defined as 'fully vaccinated' at 12 months of age if he or she has received a third dose of DTPa and poliomyelitis vaccine (oral or inactivated), a second dose of Hib vaccine (PRP–OMP), and either a second or a third dose of hepatitis B vaccine. At 24 months of age a child is defined as fully vaccinated if he or she has received the third dose of DTPa, OPV and Hib, either a third or fourth dose of hepatitis B and a first dose of MMR. ACIR coverage estimates have been reported in *Communicable Diseases Intelligence* since 1998.[7] Coverage estimates for Aboriginal and Torres Strait Islander children are not published separately except in those jurisdictions where agreement has been reached with the relevant jurisdictional organisation(s).

The completeness of data on Indigenous status was assessed by State and Territory. As a result, data were included only for New South Wales, Northern Territory, South Australia, Victoria and Western Australia. See Methods for more information.

Vaccination coverage estimates from the ACIR for Indigenous versus other children

Coverage estimates from the ACIR for Aboriginal and Torres Strait Islander children and children not identified as Indigenous aged one and two years are shown in Table 11. For Aboriginal and Torres Strait Islander children, 82.2 per cent of one year olds and 90.1 per cent of two year olds were fully immunised. The proportion fully immunised at 12 months of age was considerably higher for children not identified as Indigenous (9% higher than Indigenous children) and less so at two years of age (1% higher).

Table 11. Coverage estimates from the Australian Childhood Immunisation Register for Indigenous and other children 'fully vaccinated' at age 1 and 2 years*

Vaccine	Indigenous status	1 year	2 years
Hepatitis B	Indigenous	94.0	97.9
	Other	94.8	95.5
DTP	Indigenous	84.8	96.7
	Other	92.7	95.7
OPV	Indigenous	84.1	95.2
	Other	92.6	94.5
Hib	Indigenous	93.0	92.9
	Other	94.4	92.9
MMR	Indigenous	N/A†	94.2
	Other	N/A†	93.1
All vaccines	Indigenous	82.2	90.9
	Other	91.2	91.3

Source: Australian Childhood Immunisation Register, Health Insurance Commission.

* 3-month cohorts, age at 30 September 2003, calculated at 31 December 2003. Coverage assessment date was 12 or 24 months after the last birth date of each cohort. Includes data from New South Wales, Northern Territory, South Australia, Victoria, Western Australia only.

† Not included in coverage estimates for that age group.

Coverage at 12 months of age was lower for Aboriginal and Torres Strait Islander children for each single vaccine. However, at two years of age, the differences in coverage for individual vaccines between Indigenous and other children were less marked. Coverage was higher in Aboriginal and Torres Strait Islander two year olds for hepatitis B (Indigenous 97.9%, other 95.5%), DTP (Indigenous 96.7%, other 95.7%), OPV (Indigenous 95.3%, other 94.5%) and MMR (Indigenous 94.2%, other 93.1%).

It should be noted, however, that coverage estimates for Aboriginal and Torres Strait Islander children include only those who are registered on the ACIR and identified on it as Indigenous. Children so identified may not be representative of all Aboriginal and Torres Strait Islander children, and so estimates based on these could either overestimate or underestimate coverage among young Indigenous children.

Calculating vaccination coverage estimates from the National Health Survey

The methodology for calculating vaccination coverage estimates in the National Health Survey (NHS) has been published.[8] In contrast to ACIR estimates, NHS estimates for children are combined for different ages, in this case 2–6 years of age. The NHS was conducted in 2001, so for this age group, three different schedules have applied—those of 1996, 1998 and 2000. Some of the schedule changes applied only to children born after that date, while others applied to all children below a certain age, depending on the vaccine and dose concerned. The vaccination status for each child was calculated according to the number of doses received, compared to the number recommended under the schedule applying to a child in that age group. An allowance of one month was made for late vaccinations, so that at the time of interview, if a child was due for a vaccination in the previous month and had not received it, that dose was not included in the calculation of vaccination status. Adult respondents were asked whether they had been vaccinated for influenza in the last 12 months, and for pneumococcal disease in the last five years.

Vaccination coverage estimates from the National Health Survey for Indigenous versus non-Indigenous children

Coverage estimates from the National Health Survey for Indigenous and non-Indigenous children were provided by the ABS and are presented here for children aged 2–6 years in Table 12. Estimated coverage in this group was considerably lower for both Indigenous and non-Indigenous children than recorded on the ACIR for one and two year olds.

For Aboriginal and Torres Strait Islander children in non-remote areas, estimated coverage ranged from 85 per cent for one or more doses of MMR to 50 per cent for three or more doses of Hib. Coverage for non-Indigenous children was statistically significantly higher for DTP, OPV and Hib, and almost so for MMR. Coverage for non-Indigenous children ranged from 94 per cent for one or more doses of MMR to 67 per cent for four or more doses of Hib. The vaccine with the largest difference in coverage between Indigenous and non-Indigenous children was Hib (17%), while the smallest difference was for MMR (9%). Parents of Indigenous children were less likely to have referred to immunisation cards or records (56%, 95% CI 43–69%) than parents of non-Indigenous children (80%, CI 77–83%). The proportion of parents of Aboriginal and Torres Strait Islander children who reported they either did not know whether their child was vaccinated, or did not know how many doses they had received, ranged from six per cent for MMR to 23 per cent for Hib. For parents of non-Indigenous children, the proportions ranged from six per cent for OPV to nine per cent for Hib.

Table 12. Coverage estimates (with 95% confidence intervals) from the National Health Survey for Indigenous and non-Indigenous children 'fully immunised', aged 2–6 years*

Vaccine	Indigenous status	Coverage (%)
DTP†	Indigenous	70 (62–78)
	Non-Indigenous	82 (79–85)
OPV‡	Indigenous	76 (70–82)
	Non-Indigenous	87 (84–90)
Hib§	Indigenous	50 (41–59)
	Non-Indigenous	67 (64–70)
MMR‖	Indigenous	85 (78–92)
	Non-Indigenous	94 (92–96)

Source: Unpublished data from the National Health Survey, provided by the Australian Bureau of Statistics.

* Children living in non-remote areas only.

† Four or more doses.

‡ Three or more doses.

§ Indigenous children three or more doses, non-Indigenous children four or more doses.

‖ One or more doses.

Vaccination coverage estimates from the National Health Survey for Indigenous versus non-Indigenous adults

Coverage estimates for influenza and pneumococcal vaccines in age groups relevant for the fully funded vaccine programs among Indigenous and non-Indigenous adults are shown in Table 13. Pneumococcal vaccine coverage was significantly higher in Aboriginal and Torres Strait Islander adults at 50–64 years (Indigenous 20%, non-Indigenous 3%) and 65 years and over (Indigenous 47%, non-Indigenous 28%). Influenza vaccine coverage was significantly higher in Aboriginal and Torres Strait Islander adults at 50–64 years (Indigenous 47%, non-Indigenous 26%) but not when those aged 65 years and over were added (50+ years; Indigenous 51%, non-Indigenous 47%).

Data on remoteness were available for Aboriginal and Torres Strait Islander people only. For those aged 50 years and over, coverage was substantially higher in remote areas than in non-remote areas for both influenza vaccine (75% vs 45%) and pneumococcal vaccine (48% vs 19%).[59]

Other data

Previously published national data on immunisation coverage in Aboriginal and Torres Strait Islander people are largely limited to the 1996 'Wronski Report'.[60] This report estimated levels of immunisation coverage in Aboriginal and Torres Strait Islander children by surveying community controlled Aboriginal and Torres Strait Islander Health Services and regional health bodies. Of 25 services providing data, estimated coverage in two years old and five years old children varied from 14 per cent to 100 per cent, with generally higher coverage in non-urban areas compared to urban areas.[60] A more recent study[13] has estimated national immunisation coverage using Australian Childhood Immunisation Register records with receipt of PedvaxHIB immunisation as a proxy for Aboriginal and Torres Strait Islander status. Using this method, coverage in Aboriginal and Torres Strait Islanders at 12 months (72%–76%) and 24 months (64%–73%) was considerably lower than in others (90%–94% and 81%–88% respectively). As in the National Health Survey, coverage was significantly lower in children residing in 'accessible' compared to 'remote' areas.[13]

Other data on immunisation coverage in Aboriginal and Torres Strait Islander children have come largely from specific areas in particular States. Surveys of immunisation providers' records have reported rates from 36 per cent fully vaccinated for children up to 11 years of age on the North Coast of New South Wales in 1992[61] to 73 per cent by two years of age in Central Australia in 1985.[62] In Far North Queensland in 1996, coverage was estimated to be less than 42 per cent by two years of age.[63] Studies examining computerised immunisation registers in the Northern Territory have reported uptake rates of above 95 per cent for most vaccines at two years of age in remote communities in 1993.[64] Overall coverage, measured as fully immunised at six years of age, was estimated as 77 per cent for the whole Northern Territory in 1996.[65] Studies have consistently reported significantly higher coverage rates in remote communities compared to urban areas in Northern Australia[63,65,66] but the reverse was found in Northern New South Wales.[61]

Table 13. Vaccination coverage estimates from the National Health Survey for Indigenous and non-Indigenous adults, by age

Vaccine	Indigenous status	50–64 years	65+ years	Total 50+ years
Influenza*	Indigenous	47 (38–56)	71 (50–92)	51 (43–59)
	Non-Indigenous	26 (24–28)	75 (74–76)	47 (46–48)
Pneumococcal†	Indigenous	20 (15–25)	47 (29–65)	25 (19–31)
	Non-Indigenous	3 (2–4)	28 (26–30)	14 (13–15)

Source: Unpublished data from the National Health Survey, provided by Australian Bureau of Statistics.

* Vaccinated in the last 12 months.

† Vaccinated in the last five years.

Comment

Immunisation coverage from the National Health Survey is generally lower than that from the ACIR and the NHS figures show a larger discrepancy between rates in Indigenous and non-Indigenous children. There are several possible reasons for this. First, the NHS coverage estimates only include children whose vaccination status could be determined. The data above also show that parents of Aboriginal and Torres Strait Islander children were less likely to have written records to refer to. They were therefore more likely to report that they did not know the status of their child and therefore not be recorded as vaccinated. Under-reporting of vaccination to the ACIR also occurs, but it has been estimated at 2.5 per cent for the general population,[14] considerably lower than the 6–9 per cent of non-Indigenous children with unknown vaccination status in the NHS, and 8–23 per cent for Indigenous children. NHS estimates did not include children from remote areas. Given that coverage has frequently been shown to be higher in Indigenous populations in remote compared to urban areas,[59,66] this may also have contributed to underestimating Indigenous coverage.

For those children who are recorded as Indigenous on the ACIR, coverage is similar to that in the general population by two years of age. Coverage for DTP and OPV were lower than in the general population at 12 months of age, but there was little difference by two years. As there are no doses of DTP or OPV recommended between one and two years of age, this indicates that the main reason for lower coverage at 12 months for those recorded as Indigenous on the ACIR is delayed receipt of the vaccines due at 0–6 months.

Approximately 65 per cent of the estimated number of Aboriginal and Torres Strait Islander children born between June 2001 and December 2002 are recorded as Indigenous on the ACIR. To the extent that data completeness is an indicator of access to health care then these children may be more likely to be vaccinated than those whose status has not been recorded. It is likely, therefore, that the ACIR and NHS respectively provide an upper and lower limit of the coverage in Aboriginal and Torres Strait Islander children and the difference between Indigenous and non-Indigenous children. Improved reporting of Indigenous status on the ACIR in future may enable the monitoring of trends over time and geographical differences. Ideally, any future NHS examining immunisation status in an enhanced sample of Aboriginal and Torres Strait Islander children should seek to map this to the ACIR, for identification of both Indigenous and immunisation status.

In adults, coverage was higher for Aboriginal and Torres Strait Islanders for pneumococcal vaccine in those aged 50 years and over and for influenza vaccine at 50–64 years. This higher coverage is probably attributable to the provision of free vaccine through the National Indigenous Pneumococcal and Influenza Immunisation program. There was no significant difference in influenza vaccine coverage at 65 years and over, where vaccine is recommended and provided free to the general population.

Discussion

This report gives, for the first time, detailed data on VPDs and vaccine coverage in Aboriginal and Torres Strait Islander people and compares these data with those for the population not recorded as Indigenous. These data add to the more general data on communicable disease and health in the report(s) of the Australian Institute of Health and Welfare and the Australian Bureau of Statistics.[1] They have direct implications for prevention, both in considering new or expanded vaccine programs and in improving the population coverage of existing programs.

With the exception of hepatitis A, all VPDs considered in detail in the report are currently targeted by population-wide programs. Each VPD highlights different issues for consideration. For childhood programs, Hib disease, pertussis and measles provide interesting contrasts. In the case of Hib disease, great progress has been made in preventing this life-threatening infection across the population, but the residual disease burden, though much diminished, is falling disproportionately on Aboriginal and Torres Strait Islander people. More information is needed on the reasons for this, as vaccination coverage data from the NHS and ACIR do not show a consistent picture. In contrast, ACIR data show little difference in younger children aged one and two years. For pertussis, although there is little difference in incidence rates across all ages, this is not the case in the youngest children. Both Hib disease and pertussis can affect very young infants below six months of age and the data suggesting delayed immunisation in Aboriginal and Torres Strait Islander children may be particularly relevant here. In the case of measles, where early receipt of vaccine is not so critical and a recent nationwide campaign was delivered in schools, good control of equivalent degree is evident for Indigenous and non-Indigenous people.

Different issues are highlighted by examination of the data on disease incidence and vaccine coverage for hepatitis B, meningococcal disease, influenza and pneumococcal disease, all with established programs targeting a wider age group. In the case of hepatitis B, Aboriginal and Torres Strait Islander infants have been included in targeted programs since the late 1980s and the oldest will now be more than 15 years of age. In the Northern Territory, where the program was universal rather than targeted, high coverage may have been more successful than in regions with targeted programs. Certainly the data showing high incidence and significant differential incidence in the 5–14 years old age group suggest that there may have been sub-optimal coverage. Indeed, relatively high rates of hepatitis B notification for Aboriginal and Torres Strait Islander young people suggest that a program targeting this age group should be considered. A national program of meningococcal C conjugate vaccination has been in place since early 2003. Available notification data indicate that the significantly higher rates of invasive meningococcal disease in Aboriginal and Torres Strait Islander people are primarily in children under two years and are mostly type B, for which no licensed vaccine is available. It will be important to have more complete serogroup-specific data available for Aboriginal and Torres Strait Islander cases in the future.

High morbidity from influenza and pneumococcal disease in Aboriginal and Torres Strait Islander people has been targeted by a funded vaccine program for adults over 50 years since 1999. Children under two years of age have been targeted for a funded program of conjugate pneumococcal vaccine since 2001. The data presented here show that the incidence of these diseases remains high and, in the adult program, there is significant scope for increasing vaccine coverage. This is particularly the case in adults aged 50–64 years where coverage, although higher than in non-Indigenous adults for whom there is no funded program, remains low. There is also a high disease burden from influenza and pneumococcal disease in 25–49 years old Aboriginal and Torres Strait Islander adults, potentially justifying expansion of the program to all Aboriginal and Torres Strait Islander adults in this age group. However, it will be important to first identify means of achieving higher coverage in the young adult population.

In the last category is hepatitis A, where no national vaccine program exists for any population group. In north Queensland, hepatitis A has been targeted by a vaccination program for Aboriginal and Torres Strait Islander children under five years of age with dramatic falls in disease incidence extending to other age groups and the non-Indigenous population.[31] This experience, and similar experiences with hepatitis A programs in Indigenous communities in the USA, suggest that hepatitis A should also be strongly considered for a national program, at least in high incidence areas, as is done in the USA.

In summary, the data in this report reveal some important areas where increased attention is justified and consideration of expanded programs and mechanisms of enhancing existing programs should be expedited. Limitations of the available data relate both to VPD and vaccine coverage data, though the issues in each differ. For VPD data, incomplete identification of Aboriginal and Torres Strait Islander status is a problem, tending to underestimate disease burden. However, even the available data indicate a substantially higher burden from VPDs in almost all instances, with the limitation that information about Aboriginal and Torres Strait Islander people living in the southern parts of the country and in urban areas is less complete. For vaccine coverage, there is the possibility of overestimating completeness of coverage through the ACIR, depending on the characteristics of children identified as Indigenous in it. On the other hand, the range of years and small sample sizes for individual years significantly limit the interpretation of data from the National Health Survey, as does lack of availability of records. Expansion of the ACIR to include other age groups, should this occur in the future, may be able to address this problem.

Acknowledgements

We thank the members of the National Indigenous Immunisation Reference Group for their assistance in reviewing drafts and consulting with the organisations they represent.

Australian Bureau of Statistics:
Mr Dan Black, Ms Katrina Poyser

Australian Government Department of Health and Ageing, Public Health Division:
Ms Jeanette Baird, Dr Paul Roche, Dr Jenean Spencer, Ms Sharon Tuffin

Office for Aboriginal and Torres Strait Islander Health:
Mr Gerard De Ruyter, Ms Sue Green, Dr Ana Herceg, Mr Bernard Pearce, Ms Joy Savage

Australian Divisions of General Practice:
Dr Diana Terry

Australian Institute of Health and Welfare:
Dr Fadwa Al-Yaman

Cooperative Research Centre on Aboriginal Health:
Dr Sue Vlack

National Aboriginal Community Controlled Health Organisation:
Ms Rachel Atkinson, Dr Sophie Couzos

National Advisory Group on Aboriginal and Torres Strait Islander Health Information and Data:
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National Immunisation Committee:
Dr Christine Selvey, Ms Dallas Young

We wish to acknowledge the following organisations for provision of data for this report.

The Surveillance and Epidemiology Section, Population Health Division, Australian Government Department of Health and Ageing, for data from the National Notifiable Diseases Surveillance System.

The Australian Institute of Health and Welfare, Hospitals and Mental Health Services Unit and Population Health Unit, for data from the National Hospital Morbidity and Mortality databases.

The Australian Bureau of Statistics, for vaccination coverage data from the National Health Survey (special thanks to Mr Danny Cook).

The Health Insurance Commission, for vaccination coverage data from the Australian Childhood Immunisation Register.

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases is a collaborating unit of the Australian Institute of Health and Welfare and receives funding from the Australian Government Department of Health and Ageing.

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Appendix

Summary of notifications, hospitalisations and deaths for vaccine preventable diseases, Australia, 1999 to 2002,* by Indigenous status

Disease†	Indigenous status	Notifications (2000–2002)			Hospitalisations (July 1999–June 2002)			Deaths (2000–2002)		
		n	Rate‡	Rate ratio	n	Rate‡	Rate ratio	n	Rate‡	Rate ratio
Diphtheria§	Indigenous	0	–	–	0	–	–	0	–	–
	Other	1	–		2	–		0	–	
Hib disease (invasive)‖	Indigenous	13	1.2	9.7	–	–	–	–	–	–
	Other	34	0.1		–	–		–	–	
Hepatitis A	Indigenous	113	9.1	2.9	82	4.5	2.1	1	–	–
	Other	899	3.1		1,227	2.1		2	–	
Hepatitis B (acute)	Indigenous	57	7.2	4.4	30	2.8	3.7	2	–	–
	Other	469	1.6		433	0.8		8	–	
Influenza¶	Indigenous	–	–	–	594	49.3	2.9	2	–	–
	Other	–	–		9,719	17.1		61	–	
Measles	Indigenous	3	0.2	0.6	2	0.1	0.4	0	–	–
	Other	110	0.4		170	0.3		0	–	
Meningococcal disease	Indigenous	92	7.2	2.1	121	5.5	1.3	5	–	–
	Other	975	3.4		2,440	4.3		33	–	
Mumps**	Indigenous	5	0.3	0.4	9	0.4	1.8	0	–	–
	Other	270	0.9		129	0.2		1	–	
Pertussis	Indigenous	408	41.8	0.9	150	6.2	2.4	0	–	–
	Other	13,528	46.9		1,478	2.6		7	–	
Pneumonia	Indigenous	NN	NN	–	17,455	1,580	3.2	130	19.1	3.1
	Other	NN	NN		283,876	495		3,569	6.2	
Pneumococcal disease	Indigenous	214	44.7	4.5	269	22.0	4.5	5	–	–
	Other	1,926	9.9		2,806	4.9		16	–	
Poliomyelitis††	Indigenous	0	–	–	2	0.3	2.8	0	–	–
	Other	0	–		54	0.1		0	–	
Rubella‡‡	Indigenous	5	0.5	0.4	1	0.1	0.6	0	–	–
	Other	310	1.1		81	0.1		0	–	
Tetanus§§	Indigenous	1	0.3	16.2	0	–	–	0	–	–
	Other	6	0.0		80	0.1		1	–	
Varicella‖‖	Indigenous	NN	NN	–	197	8.9	1.2	0	–	–
	Other	NN	NN		4,285	7.6		10	–	

* Notifications (New South Wales, Northern Territory, South Australia and Western Australia only) where the date of onset was between 1 January 2000 and 31 December 2002; hospitalisations (all states) where the month of separation was between 1 July 1999 and 30 June 2002; deaths (Queensland, Northern Territory, South Australia and Western Australia) where the date of death was recorded between 1 January 2000 and 31 December 2002.

† See results section for case definitions. For diseases not included in Section 3, case definitions are listed below.

‡ Average annual rate per 100,000 population, age standardised to the Australian Bureau of Statistics Australian population estimates for 2001.[6]

§ Notifications: isolation of toxigenic *Corynebacterium diphtheriae*, plus either pharyngitis or laryngitis, and toxic symptoms. Hospitalisations: ICD–10 codes A36.0, A36.1, A36.2 or (A36.8 + I41.0). Deaths: A36. One notification of a cutaneous infection acquired overseas.

‖ Hospitalisations and deaths not included because there is no ICD–10 code specific to *Haemophilus influenzae* type b.

¶ Notifications not included due to low completeness of Indigenous status field.

** Notifications: Isolation of mumps virus, rise in mumps antibody, or clinically compatible illness. Hospitalisations and deaths: ICD–10 code B26.

†† Notifications: Acute flaccid paralysis without apparent cause. Hospitalisations and deaths: ICD–10 code A80. Hospitalisations include vaccine-associated polio and imported cases.

‡‡ Notifications: generalised macropapular rash, fever, epidemiological link to a confirmed case, plus arthralgia/arthritis, lymphadenopathy or conjunctivitis. Hospitalisations and deaths: ICD–10 code B06.

§§ Notifications: a clinically compatible illness without other apparent cause. Hospitalisations and deaths: ISD–10 code A35.

‖‖ Hospitalisations and deaths: ICD–10 code B01.

NN Not notifiable.

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

The January 2004 edition of the *Infection Control Guidelines for the Prevention of Transmission of Infectious Diseases in the Health Care Setting* (ICG), was recently endorsed by the Australian Health Minister's Advisory Council.

The electronic version of this document can be downloaded from http://www.icg.health.gov.au. A limited number of hard copy folders of the document will be distributed nationally to public and private hospitals, aged care facilities, peak health organisations, key staff in state and territory health departments, health libraries and universities, in mid to late June 2004.

The Australian Government Department of Health and Ageing has prepared these guidelines under the auspices of the Communicable Diseases Network Australia, which is a subcommittee of the National Public Health Partnership. The National Health and Medical Research Council's Special Expert Committee on Transmissible Spongiform Encephalopathies has also endorsed the Creutzfeldt-Jakob disease (CJD) component of the ICG.

This document replaces the *Infection control in the health care setting—guidelines for the prevention of transmission of infectious diseases*,[1] and incorporates guidelines on patient management and infection control relating to CJD and other human transmissible spongiform encephalopathies (thus replacing *Creutzfeldt-Jakob Disease and Other Human Transmissible Spongiform Encephalopathies: Guidelines on Patient Management and Infection Control*.[2]

These guidelines are a reference document providing current 'best practice' national guidelines for infection control procedures in Australian health care settings, and should be considered in association with state and territory legislative requirements that affect work practices of the health care establishment and/or health care worker. (Note: if the recommendations in this document conflict with state or territory guidelines, the statutory requirements of the state or territory take precedence.

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Annual report of the National Influenza Surveillance Scheme, 2003

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Abstract

Surveillance of influenza in Australia is based on laboratory isolation of influenza viruses, sentinel general-practitioner practices for influenza-like illness, and absenteeism data from a major national employer. In 2003, the peak in influenza activity was in August which was later than in 2002. In 2003, 3,604 laboratory-confirmed cases of influenza were notified to the National Notifiable Diseases Surveillance System, which was marginally lower than for the previous year. Ninety-four per cent of the circulating viruses were influenza A. This was the highest proportion in the last five years. Nine hundred and thirty-five isolates were antigenically analysed: 928 were A(H3), two were A(H1) strains and five were influenza B viruses. The majority (98%) of the A(H3) subtypes were A/Fujian/411/2002(H3N2)-like and have shown a significant antigenic drift. The 2003 Australian influenza vaccine contained A/Panama/2007/99, which induced 2–4-fold lower antibody response against the drifted strain. An A/Fujian/411/2002(H3N2)-like virus has been incorporated in the Australian influenza vaccine for 2004. In 2003, the influenza vaccine was given to 77 per cent of Australians aged over 65 years; the same up take as in 2002. *Commun Dis Intell* 2004;28:160–168.

Keywords: influenza, surveillance, vaccine, general practice, influenza-like illness

Introduction

Influenza is an acute self-limiting viral disease of the upper respiratory tract. The health and economic impact of influenza largely arises from related complications such as lower respiratory tract infections and exacerbation of cardiopulmonary and other chronic diseases. These complications result in excess hospitalisation and mortality.

Influenza infections are seasonal in temperate climates (June to September in the Southern Hemisphere and December to April in the Northern Hemisphere), but may occur throughout the year in tropical regions. The seasonal activity of influenza virus varies from year to year with some years marked by larger epidemics with higher morbidity and mortality. In Australia in 2002, influenza and pneumonia were the underlying causes of 3,084 deaths.[1] Although the infection affects all age groups, those aged 0–4 years or 65 years and over, or those with chronic medical problems have higher rates of morbidity and mortality.

The potential for an epidemic of influenza is dependent on the susceptibility of the population and the ability of the viruses to evolve. There are three types of influenza viruses, A, B and C. Influenza virus types are further subtyped by the antigenic properties of two surface glycoproteins: haemagglutinin (H) and neuraminidase (N). Fifteen H and nine N subtypes have been identified for influenza A, and of these, three H (H1, H2, H3) and two N (N1 and N2) have been found in humans, while all subtypes have been found in aquatic birds. Only one H and one N have been identified for influenza B viruses. Influenza virus A and B are the cause of widespread annual epidemics as these viruses evolve by mutation of genes encoding the H and N. This gradual change or 'antigenic drift', is responsible for the emergence of variant strains of viruses able to evade the immunity conferred by previous infection or vaccination.

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Influenza virus A is also known to cause pandemics (worldwide epidemics) at irregular intervals, either by direct introduction of new haemagglutinin subtypes into the human population from animals or birds, or by genetic reassortment between an avian or animal influenza virus and a human influenza virus. This latter process is called 'genetic shift' and results in a new influenza virus. Unlike the seasonal epidemics of influenza, where attack rates depend on age, reflecting immunity conferred from previous infection, in pandemic influenza all age groups are equally susceptible. Last century the emergence of new strains of influenza A caused pandemic influenza with a global impact on morbidity and mortality rates: A(H1N1) in 1918; A(H2N2) in 1957; and A(H3N2) in 1968.

An effective national surveillance system is an essential component for the control of seasonal epidemics and the preparedness for possible pandemics. Virological and epidemiological monitoring are important components of influenza surveillance. The main objectives of virological and epidemiological surveillance of influenza are:

(i) early detection of epidemics to enable the implementation of public health measures such as the vaccination of high risk groups, outbreak 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.

In 2003, the Communicable Diseases Australia website (http://www.cda.gov.au/index.htm) published influenza surveillance data fortnightly during the influenza season. This annual influenza report is a summary of the surveillance information gathered by various systems in 2003.

Surveillance methods

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

1. notifications of laboratory-confirmed influenza required by legislation in most state and territories, and nationally notifiable 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 and strain data of circulating influenza viruses 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

In all jurisdictions with the exception of the Australian Capital Territory and South Australia, laboratory-confirmed influenza is a notifiable disease under state and territory legislature. In the Australian Capital Territory and South Australia, laboratory reports are also collected and sent to NNDSS although influenza is not a notifiable condition. In this report, data are analysed by the date of onset in order to present disease occurrence during the reporting period, but when this was not available the earliest date from specimen collection date or notification date was used.

Laboratory surveillance

LabVISE is a national scheme of sentinel laboratories that report influenza diagnosis all year round. In 2003, 16 laboratories from all jurisdictions except the Northern Territory contributed to the scheme. Data were reported to LabVISE monthly and were analysed by the specimen collection date.

WHO Collaborating Centre for Reference and Research on Influenza

The WHO Collaborating Centre for Reference and Research on Influenza is part of an international network for the surveillance of influenza viruses. It reports on the subtypes, performs antigenic analysis of influenza viruses isolated throughout the year and classifies them in accordance to the standard nomenclature for influenza viruses. The standard nomenclature is based on type, the place where they were first identified, sequential number, and year of isolation. For example, A/Sydney/5/97 denotes influenza A virus that was first isolated in Sydney and was isolate number 5 for the year in 1997. The main application of strain characterisation is 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.

The Centre 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 assist in vaccine formulation decisions. The Centre's data together with that from the WHO Collaborating Centres in Japan, the United Kingdom and the United States of America are reviewed at the World Health Organization (WHO) consultations, which take place twice yearly, to provide recommendations to national and regional authorities regarding vaccine formulation.

Sentinel general practitioner surveillance

Sentinel general practitioner surveillance schemes for influenza monitor the consultation rates for influenza-like illness (ILI). In Australia, there are five such schemes: the Australian Sentinel Practice Research Network (ASPREN) which collects data at a national level, 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. ASPREN and the Northern Territory Tropical Influenza Surveillance Scheme report ILI rates throughout the year, while the other sentinel surveillance schemes report from May to October each year.

Sentinel general practices contributing to the ASPREN scheme are mostly located in capital cities and larger regional centres on the east coast of Australia (Map). In 2003, an average of 47 (range 32–62) general practices reported ILI cases on an average of 4,962 (range 2,138–6,587) consultation per week.

The Northern Territory Tropical Influenza Surveillance reported cases of ILI as a rate per 1,000 consultations per week. Throughout the year, eight to 14 centres reported to the surveillance system with an average of 756 (range, 230–1,074) consultations per week.

In 2003, the New South Wales Influenza Surveillance program collected reports from New South Wales practitioners who are part of ASPREN and from five out of 17 Public Health Units (Southern New South Wales, New England, Illawarra, Central Coast, Northern Sydney, Western Sydney and South Eastern Sydney). Thirty-seven (range 17–50) general practitioners reported ILI cases weekly from May to October on an average of 3,933 (range 1,723–5,335) consultations per week.

The Victorian Infectious Diseases Reference Laboratory, the WHO Collaborating Centre for Influenza and the Department of Human Services contributed to the Victorian Influenza Surveillance Scheme. In 2003, the Victorian Influenza Surveillance Scheme also enlisted the Melbourne Locum Service. Overall, 79 general practitioners from metropolitan (20 sites) and rural (21 sites) regions were recruited to report ILI rates per 100 consultations per week, between May and September. These practices reported on average 7,542 (range 5,315–14,376) consultations per fortnight. ILI was reported as a rate per 100 and has been converted to rate per 1,000 consultations in this report to allow comparisons with other sentinel schemes.

In Western Australia, 16 general practices, 12 in the metropolitan area (Perth) and four in rural regions (one each in Kalgoorlie, Busselton, Tom Price and Geraldton) participated in ILI surveillance from May to October. Data were reported as the number of cases of ILI per practice per week. The number of consultations was not recorded and hence the number of practices was used as the denominator.

Map. Geographic distribution of ASPREN sentinel general practice sites, Australia, 2003

Absenteeism surveillance

Australia Post, a major nation-wide employer, provided sick leave absenteeism data collected weekly between March and September 2003. Absenteeism, defined as an absence due to illness for at least three consecutive days, was presented as a rate per 100 employees per week, on an average of 33,246 employees per week.

Hospitalisation data

The Australian Institute of Health and Welfare provide data on hospital separations in public and private hospitals. The number of separations with a primary diagnosis of influenza due to identified influenza viruses (ICD–10AM = J10) and influenza where the virus was not identified (ICD–10AM = J11) are reported. Data for the 2002/03 financial year were not 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 varied between sentinel surveillance schemes (Table 1) which makes comparison of ILI among the different schemes difficult.

National Notifiable Diseases Surveillance System

In 2003, 3,604 laboratory-confirmed cases of influenza were reported to NNDSS, which represents a two per cent decrease from the number of notifications in 2002. All jurisdictions reported laboratory-confirmed influenza to NNDSS, although Tasmania reported very few cases due to limited access to laboratory testing for influenza.

Notifications of laboratory-confirmed influenza started to increase in early August and peaked in the last week of August (Figure 1). Compared to 2002, the influenza season started late and was characterised by a rapid rise in cases.

A comparison of notification rates in each jurisdiction (with the exception of the Australian Capital Territory and Tasmania) is shown in Figure 2. The highest notification rates occurred in August in New South Wales (85 cases per 100,000 population), Victoria (89 cases per 100,000 population) and Queensland (131 cases per 100,000 population); and in September in the Northern Territory (629 cases per 100,000 population), South Australia (134 cases per 100,000 population), and Western Australia (185 cases per 100,000 population).

Figure 1. Notifications of laboratory-confirmed influenza to the National Notifiable Diseases Surveillance System, Australia, 2002 and 2003, by week of onset

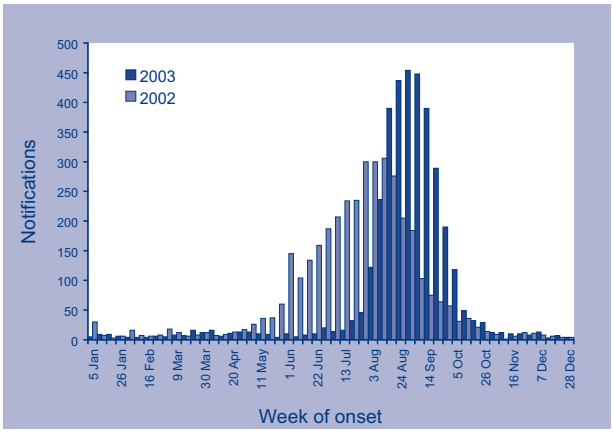


Figure 2. Notification rates of laboratory-confirmed influenza, Australia, 2003, by jurisdiction and month of onset

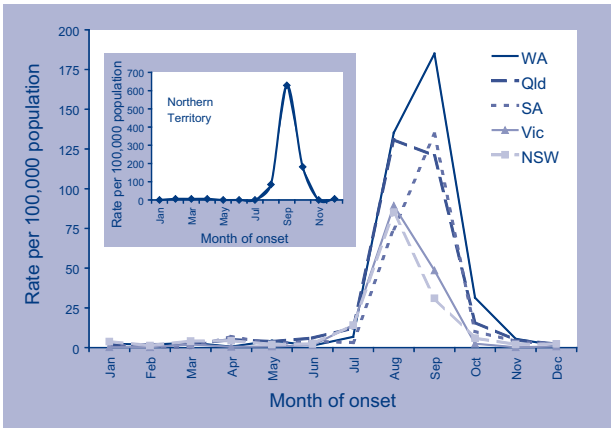


Table 1. Case definitions of influenza-like-illness used in Australian sentinel practice schemes, 2003[2]

Program	Case definition
Victorian Influenza Surveillance Scheme	Fever, cough, fatigue
Western Australian sentinel general practices	Fever, cough, fatigue
New South Wales state program, Northern Territory and ASPREN	Six of the following nine symptoms with sudden onset (<12 hours previously): cough, rigours or chills, fever, prostration and weakness, myalgia, redness of mucous membranes, influenza in close contacts

National age-specific notification rates are shown in Figure 3. The overall female to male ratio was 0.9:1. The 0–4 year age group had the highest notification rate (135 cases per 100,000 population), representing 48 per cent of all notifications.

Infants under the age of one accounted for 38 per cent of notifications in the 0–4 years age group and had the highest notification rate at 257 cases per 100,000 population (Figure 3 insert). Notification rates among the over 65 years age group were marginally lower in 2003 (16 cases per 100,000 population) compared to 2002 (18 cases per 100,000 population).

Laboratory surveillance

A total of 2,071 laboratory diagnoses of influenza were reported to LabVISE participating laboratories, of which 94 per cent of isolates were influenza A (Figure 4). The overall A to B ratio in 2003 was 16:1. The peak of influenza reports from LabVISE occurred in September, which was a fortnight later than the peak influenza activity observed in the NNDSS surveillance data. This is because LabVISE data were analysed by the date of specimen collection while NNDSS data were analysed by date of onset.

WHO Collaborating Centre for Reference and Research on Influenza

The WHO Collaborating Centre for Reference and Research on Influenza received 935 isolates or clinical specimens that yielded viable influenza viruses (477 less than for 2002) and they were all analysed antigenically. Of these viruses 928 (99.3%) were A(H3) strains with only two A(H1) viruses and five influenza B isolates. Sequence analysis of the variable (HA1) region of the haemagglutinin was undertaken for 61 strains (1 H1; 56 H3; and 4 B) and of the neuraminidase for 41 strains (39 A and 2 B). The majority (98%) of the A(H3) viruses were genetically and antigenically distinguishable from the reference strain A/Moscow/10/99 and the 2003 vaccine strain A/Panama/2007/99 (Table 2, Figure 5), and were similar to the new reference strain A/Fujian/411/2002. The majority of the 19 A/Moscow/10/99–like viruses identified were isolated early in the year (between January and May) whilst the first of the A/Fujian/411/2002-like viruses were isolated in Queensland and Victoria during late June. New Zealand also experienced predominantly A(H3) influenza and a similar change from A/Moscow-like to A/Fujian-like strains was also observed, although a few A/Fujian-like strains were seen there early in the season.

Genetic analysis of the Australian A(H3) isolates demonstrated that the neuraminidase antigen of these viruses remained more closely related to that of strains circulating in the 2003 season than to that of the new reference virus A/Fujian/411/2002 (Figure 6). Of the two A(H1) isolates one was A(H1N1), the other A(H1N2), however both remained antigenically close to the reference and vaccine strain A/New Caledonia/20/99. Of the five influenza B viruses analysed four were antigenically and genetically B/Sichuan/379/99 lineage viruses with one B/Hong Kong/330/2001-like strain (Figure 7).

Figure 3. Notification rates of laboratory-confirmed influenza, Australia, 2003, by age group and sex, and infants aged under four years, by age and sex

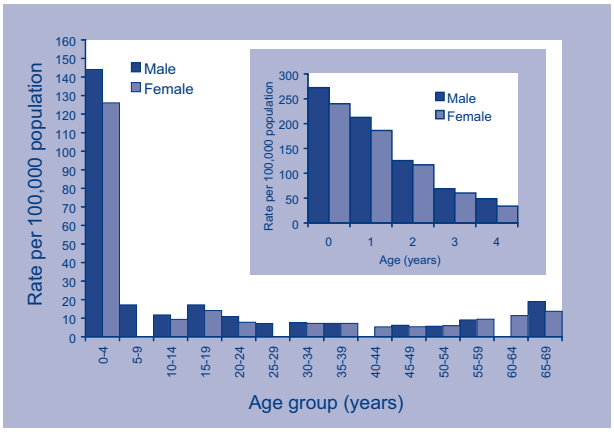


Figure 4. Laboratory reports of influenza diagnoses reported to LabVISE, Australia, 2003, by type and month of specimen collection

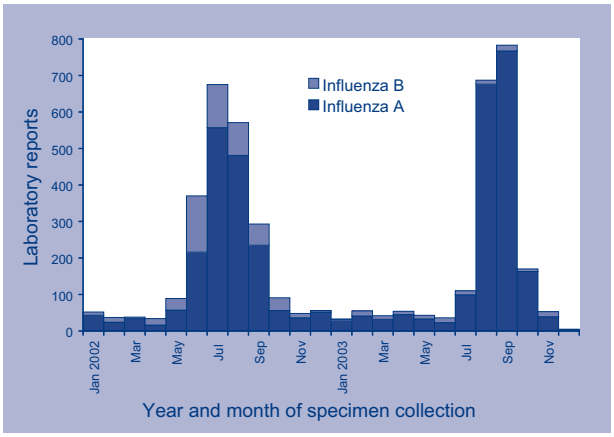


Figure 5. Evolutionary relationships between influenza A(H3) haemagglutinins (HA1 region)

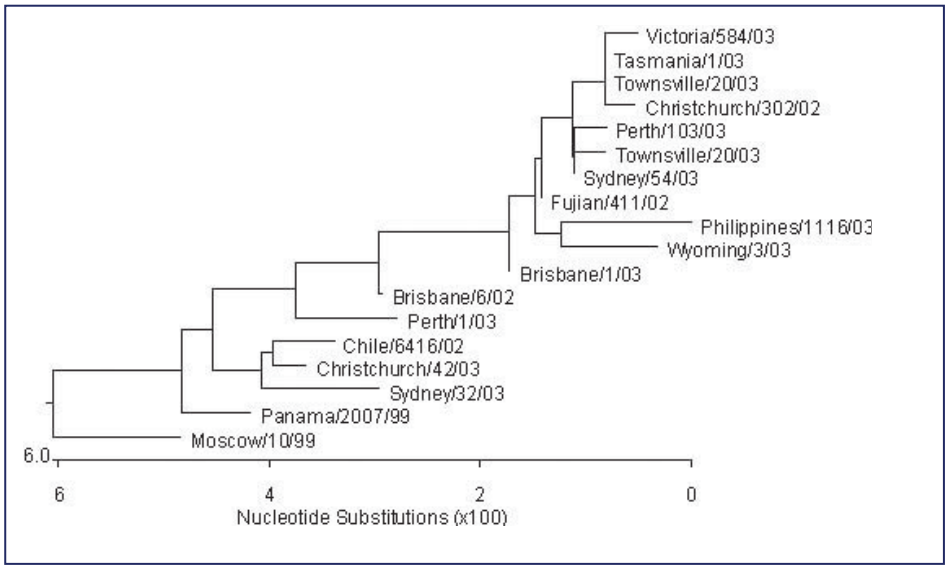


Figure 6. Evolutionary relationships between influenza N2 neuraminidases

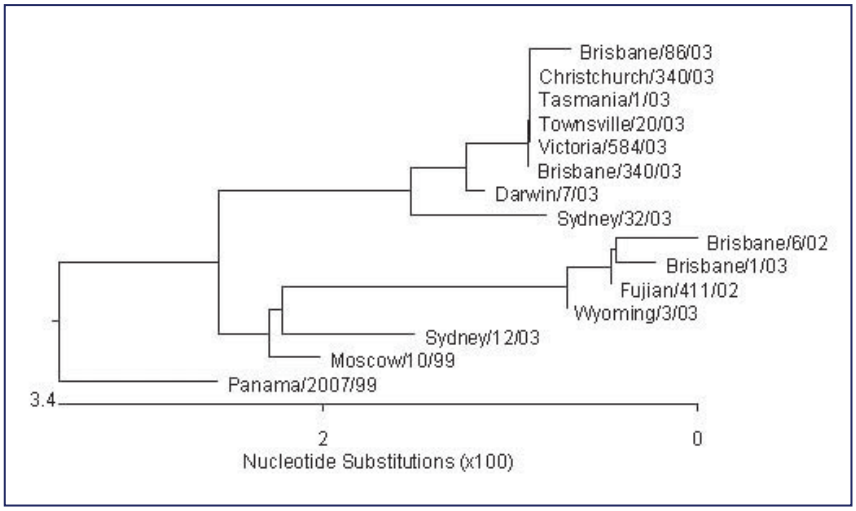
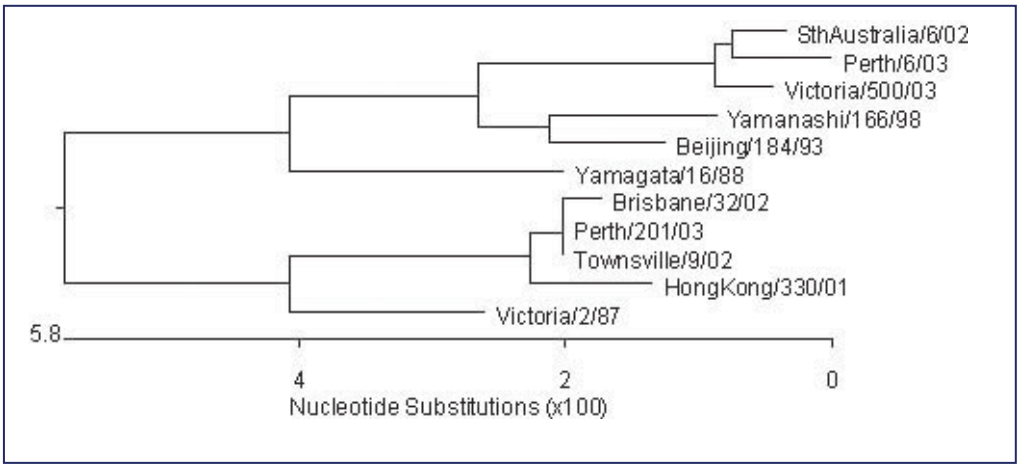


Figure 7. Evolutionary relationships between influenza B haemagglutinins (HA1 region)



Consistent with the antigenic drift in the A(H3) isolates demonstrated by ferret antisera (Table 2), serological studies conducted with pre– and post-vaccination human sera from recipients of vaccine containing the A/Panama/2007/99 strain, showed 2–4-fold lower antibody titres to the recent A/Fujian/411/2002-like strains and a reduction of about 20 per cent in the number of recipients achieving antibody levels in the protective range. While the 2003 vaccine contained a B/Hong Kong/330/2001-like strain, and the majority of the small number of influenza B isolates were B/Sichuan/379/99 lineage strains, similar numbers of younger adults and only slightly reduced numbers of older adults achieved antibody levels in the protective range to the B/Sichuan/379/99-like isolates.

Evidence of the emergence of A/Fujian/411/2002 (H3N2)-like strains was first recognised in the latter part of the 2002/03 Northern Hemisphere winter. As a consequence, WHO deferred its choice of an A(H3N2) vaccine strain at the February 2003 vaccine consultation. However, in the absence of a suitable A/Fujian/411/2002-like vaccine strain by March 2003, Northern Hemisphere vaccines for the 2003/04 winter contained the A/Panama/2007/99 virus as in the preceding winter. During the subsequent six months A/Fujian/411/2002-like strains became the predominant circulating influenza viruses and were responsible for extensive and severe outbreaks in some Northern Hemisphere countries during the 2003/04 winter. An A/Fujian-like virus has now been incorporated into the formulation for the Australian 2004 and the 2004/05 Northern Hemisphere vaccines.

Sentinel general practice surveillance

In 2003, reports of influenza-like illness from ASPREN sites started to increase earlier than in the 2002 season. ILI reports increased first in early March then in late May and peaked in mid-August (Figure 8). The peak ILI rate was 24 cases per 1,000 consultations in 2003 compared to 18 cases per 1,000 consultations in 2002. In contrast to previous years, ILI rates did not return to the base line but remained at seven cases per 1,000 consultations to the end of the year.

The Northern Territory Tropical Influenza Surveillance Scheme data showed two peak ILI rates (Figure 9); one in the first week of April, (21 ILI per 1,000 consultations) and the other in mid-September (36 ILI per 1,000 consultations). In 2002, the highest ILI rate was reported at the end of July with a rate of 39 ILI per 1,000 consultations.

Figure 8. ASPREN consultation rates for influenza-like illness, Australia, 2002 and 2003, by week of report

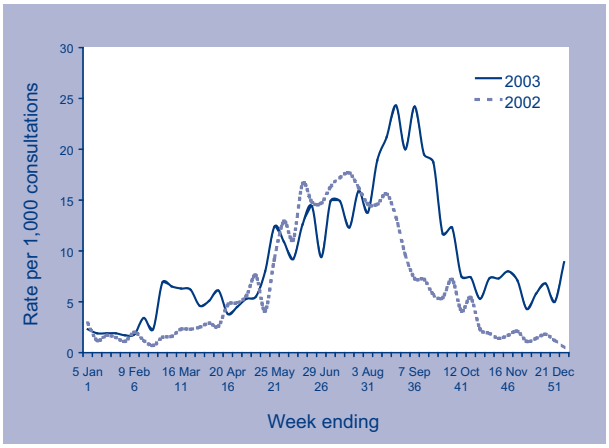


Figure 9. Consultation rates for influenza-like illness, Northern Territory, 2002 and 2003, by week of report

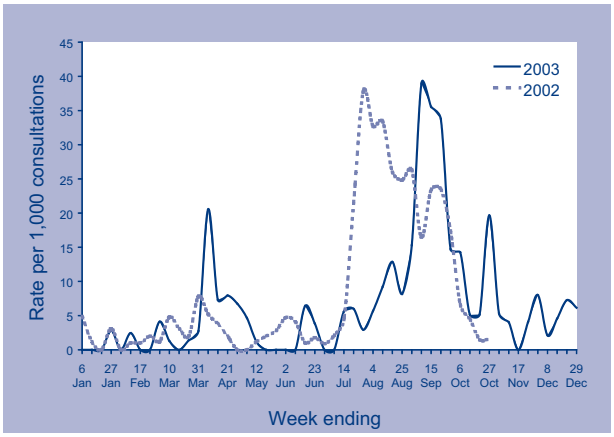


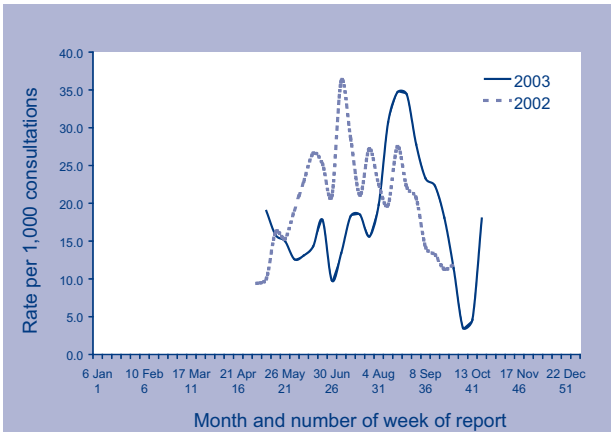
Table 2. Antigenic comparisons of influenza A(H3) viruses isolated by the haemagglutination-inhibition test

Virus antigen	Ferret serum Reciprocal HI titre			
	A/Panama	A/New York	A/Fujian	A/Wyoming
A/Panama/2007/99	1,280	160	40	40
A/New York/55/2001	1,280	1,280	320	640
A/Fujian/411/2002	80	80	1,280	2,560
A/Wyoming/3/2003*	320	80	2,560	2,560

* A/Wyoming is an A/Fujian-like strain that has been approved for vaccine manufacture.

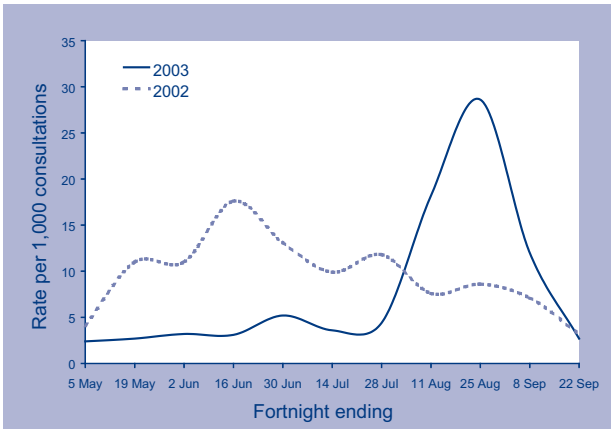
In New South Wales, ILI surveillance ran from May to October 2003. The peak ILI rate (35 ILI per 1,000 consultations) was reported for the weeks ending on 18 and 25 August (Figure 10). The peak ILI rate was similar in magnitude to that of 2002 but occurred one month later.

Figure 10. Consultation rates for influenza-like illness, New South Wales, 2002 and 2003, by week of report



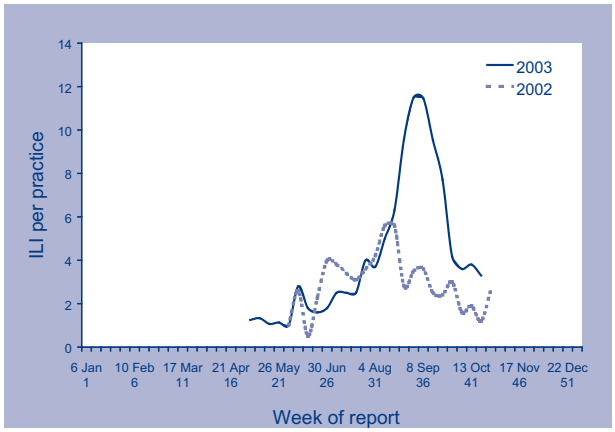
In Victoria, in 2003, ILI rates started to rise in early August and peaked at the end of that month (28 cases per 1,000 consultations). Compared to 2002, the peak ILI rate in 2003 was higher in magnitude (by 63%) and was reached two months later (Figure 11). A separate report on influenza in Victoria is included in this issue of *Communicable Diseases Intelligence* (pp. 175–180).

Figure 11. Consultation rates for influenza-like illness, Victoria, 2002 and 2003, by week of report



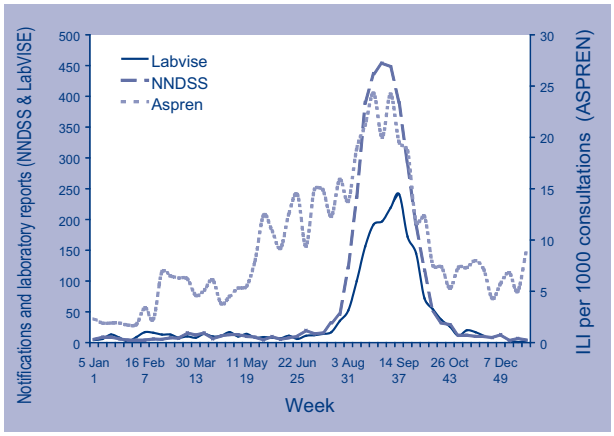
In Western Australia, ILI rates are calculated by number of cases per sentinel general practitioner practice. In 2003, the ILI rate per practice peaked in September at 12 cases per practice per week, more than twice the rate of 3.5 cases per practice reported during the peak period in 2002 (Figure 12). A separate report on influenza in Western Australia in 2003 is included in this issue of Communicable Diseases Intelligence (pp. 169–174).

Figure 12. Consultation rates for influenza-like illness, Western Australia, 2002 and 2003, by week of report



All indices of national influenza activity in Australia in 2003 indicated that the influenza season peaked between 17 August and 7 September (Figure 13).

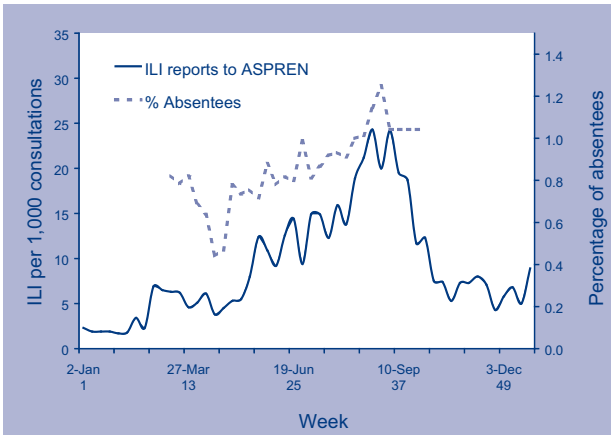
Figure 13. Laboratory reports to LabVISE, notifications to NNDSS and consultation rates in ASPREN of influenza, Australia 2003, by week of report



Absenteeism surveillance

Absenteeism surveillance is a non-specific index of influenza activity. The peak in absenteeism coincided with the peak influenza activity as measured by NNDSS and ASPREN (Figure 14). National absenteeism rates in 2003 peaked during the week ending on 28 August, at 1.3 per cent (n=415): an increase of 46 per cent from the average of 0.9 per cent absentees per week (n=284) during the reporting period.

Figure 14. Rates of absenteeism and consultation rates of influenza-like illness, Australia, 2003, by week of report



Discussion

According to most indices of influenza activity, the 2003 influenza season in Australia started later than the 2002 season. Influenza A was the predominant virus type diagnosed throughout the season with an A to B ratio of 16:1, the highest recorded over the last five years. The ratio of influenza A isolates submitted to the WHO Centre for antigenic analysis was even considerably higher (190:1). In the past, roughly equal levels of influenza A and B have been seen in every second year, interspersed with predominantly influenza A years.

In 2003, ILI rates started to increase five months before an increase in laboratory-confirmed influenza notifications. ILI reports may have been influenced by heightened attention to respiratory symptoms following the emergence of the severe acute respiratory syndrome or may be due to other respiratory pathogens, which cause ILI such as respiratory syncytial virus. ILI reporting is important timely information, but a non-specific index of influenza activity, which has to be interpreted in conjunction with the laboratory-based surveillance.

The National Health and Medical Research Council recommends annual influenza vaccination for all Australians aged over 65 years. In 2003, the vaccination coverage of Australians aged over 65 years was 77 per cent which was the same as 2002.[3] Compared to 2002, notification rates of influenza declined in the over 65 age group but increased among the 0–4 year age group and remained unchanged in the rest of the age groups. Influenza vaccination can mitigate the impact of morbidity and mortality from annual influenza epidemics on the most susceptible populations.[4]

The Australian influenza vaccine for 2003 contained A/New Caledonia/20/99(H1N1)-like, A/Moscow/10/99 (H3N2)-like and B/Hong Kong/ 330/2001-like antigens. The WHO reference centre identified that there had been significant antigenic drift in the A(H3) subtype and the majority of influenza isolates in 2003 were A/Fujian/411/2002(H3N2)-like. Consequently, vaccine-induced responses against the drifted strains were lower than those for A/Moscow-like viruses and some reduction in vaccine effectiveness may have been expected.

The majority of outbreaks in the Northern Hemisphere 2003/04 winter were also due to A(H3N2) A/Fujian-like strains. The United States of America recorded severe outbreaks with associated excess mortality characteristic of an A(H3N2) season and there were some reports of serious infections in children.[5]

For 2004, the recommended Australian influenza vaccine retained B/Hong Kong/330/2001-like, and A/New Caledonia/20/99(H1N1)-like virus components, and replaced A/Moscow/10/99 (H3N2)-like virus with an A/Fujian/411/2002-like virus.

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The Influenza Surveillance Program in Western Australia, 2003

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Abstract

In the winter of 2003 Western Australia experienced its largest epidemic of influenza for at least five years, with activity peaking in August and September. The season was short resulting in very high numbers of cases during the peak weeks. Activity in country areas followed the peak of Metropolitan activity. Influenza A virus was detected in 28.3 per cent of the sentinel samples, and influenza B in less than one per cent. Both routine and sentinel detections and the overall estimates of influenza-like illnesses (ILI) seen by general practitioners at sentinel practices peaked in August and September 2003. The combination of influenza detections and an increase in ILI seemed to be the most accurate predictor of the beginning of winter influenza activity. There was a shift in age distribution for influenza A compared with 2003. Both the sentinel surveillance and routine samples demonstrated an increase of influenza in children and young adults. The majority of influenza A isolates were identified as A/Fujian/411/2002-like, a variant of the A/Moscow strain included in the vaccine. Despite this mismatch there did not seem to have been any noticeable increase in the risk of influenza infection in the vaccinated populations from the sentinel practices, nor was there a relative increase in disease among the highly vaccinated elderly population. A number of other respiratory viruses were identified as causes of influenza-like illness in the sentinel samples. Rhinoviruses and human metapneumovirus were the most common, the latter occurring mainly in adults. *Commun Dis Intell* 2004;28:169–174.

Keywords: Influenza, surveillance, Western Australia, Sentinel General Practice

Introduction

Influenza viruses are major respiratory pathogens, causing significant illness and mortality in Australia each winter. In 1999 a national influenza surveillance program was established in Australia. This included community-based surveillance, using sentinel general practices[1] and surveillance of routinely collected respiratory samples from paediatric and adult patients. This surveillance program provided both a system for detecting the entry and spread of new influenza strains and generated valuable information to medical and public health practitioners about influenza activity each winter.

The Division of Microbiology and Infectious Diseases at PathCentre in Western Australia is one of the Australian National Influenza Centres. A general practitioner based surveillance program has operated since 1999 with the support of the Health Department of Western Australia. In 2003 Western Australia experienced the worst outbreak of influenza for five years, almost exclusively due to an influenza A strain that was poorly matched to the vaccine strain. This report presents the results collected by the Western Australia Influenza Surveillance Program during the 2003 season, as well as routine influenza virus infections diagnosed at PathCentre.

Methods

Influenza surveillance was conducted in Western Australia for 25 weeks from the week beginning Monday 5 May (Week 19) to the week beginning 20 October (Week 43).

During the 2003 winter season, 16 medical practices were recruited to the Western Australia Influenza Surveillance Program. Figure 1 shows the locations of the sentinel practices within Western Australia. The majority (12) of these were based in the Perth metropolitan area. Country practices included Kalgoorlie (Goldfields), Busselton (Southwest), Tom Price (Pilbara) and Geraldton (Midwest). Participating general practitioners (GPs) recorded the number of patients seen with an influenza-like illness (ILI) each week. An ILI was defined as an acute upper respiratory tract infection characterised by fever (or feverishness), cough and fatigue.[2] Nose and throat swabs were collected from the first patient seen on

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Monday, Tuesday and Wednesday each week with an ILI of less than 96 hours duration. Swabs were placed in viral transport medium and were stored and transported at 4° C. For each sample the GP was asked to record the symptoms, date of onset of symptoms, vaccination history and to estimate the likelihood of the illness being due to infection with an influenza virus.

Specimens were tested at PathCentre using in-house polymerase chain reaction (PCR) assays which identified a number of common respiratory viruses including influenza A, B and C, parainfluenza types 1, 2 and 3, respiratory syncytial virus (RSV) and human metapneumovirus (hMPV). In addition to PCR testing, samples were inoculated onto two tissue culture cell lines [MDCK and human fibroblast (HF) cells] for virus culture. Rapid culture of samples for influenza was carried out by inoculating samples onto coverslips seeded with MDCK cells. Tubes were spun for 60 minutes at 4° C and then incubated at 37° C for two days. Cells were then fixed in PBS/acetone and any influenza virus isolates were identified by fluorescence using FITC-labelled specific monoclonal antibodies (DakoCytomation).

All influenza isolates were sent on dry ice to the WHO Collaborating Centre for Reference and Research on Influenza in Melbourne for full strain analysis.

Other virus isolates (including rhinoviruses, adenoviruses, enteroviruses) were identified initially by the type of cytopathic effect (CPE) they caused in HF cells and this result was confirmed by PCR.

Routine specimens

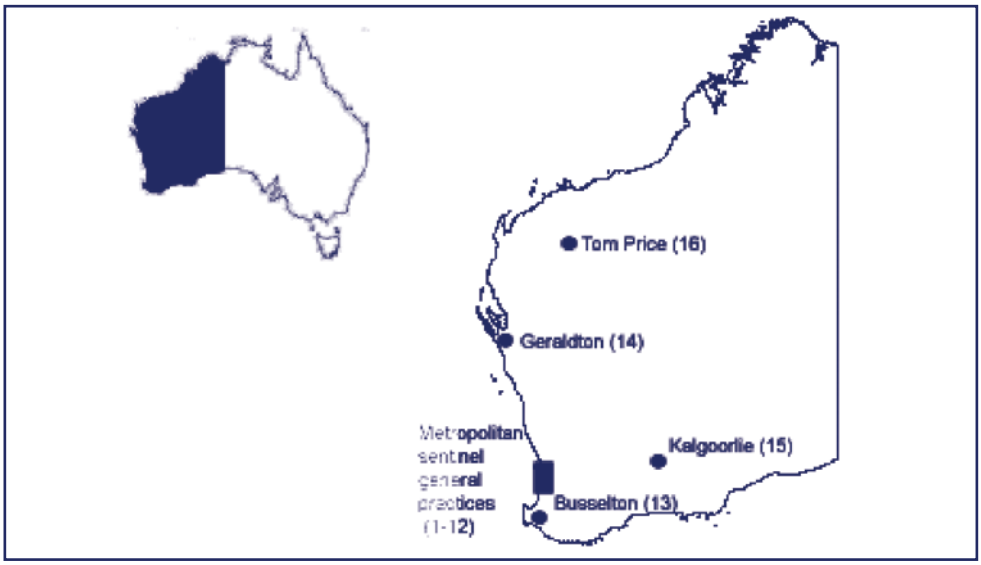
Routine samples are received at PathCentre throughout the year, from patients with respiratory infections from both metropolitan and country regions throughout Western Australia. If requested, these samples were tested for influenza and other respiratory viruses by the same methods as those used for the sentinel GP samples. We collated the total number of patients positive, either by PCR or virus culture, for influenza viruses and included these figures in the weekly reports. In addition, the number of blood samples sent for influenza serology was also recorded and reported weekly. Respiratory serology requests are usually undertaken for adults with proven or suspected lower respiratory tract infection.

Serologically diagnosed cases (influenza complement fixation tire of 1:160 or greater) were also recorded in order to improve the monitoring of influenza infections in adults.

Data collection and reporting

Results obtained from the general practitioner surveillance program as well as routine influenza detections from PathCentre and the Princess Margaret Hospital for Children were presented in the weekly Influenza News report that was sent out to the Health Department of Western Australia, all participating GPs and a number of other interested groups. As the number of practices reporting varied from week to week, the weekly information on ILIs was presented as the number of ILIs per reporting practices. Results of influenza detections (sentinel and routine) from PathCentre were also reported weekly to FluNet (available from: http://www.who.int/GlobalAtlas/home.asp).

Figure 1. Map of Western Australia showing the location of the 2003 sentinel general practices



Results

Sentinel general practices

A total of 276 samples were sent to PathCentre from 15 sentinel practices. One practice did not submit any samples this season. The ratio of males to females was approximately equal (135:141). Although 19 samples were sent with no date of onset recorded, over 97 per cent of the remaining samples were taken within the recommended 96 hours of onset of symptoms. According to the forms filled out by the doctors 203/276 (74%) of samples met the proposed case definition of fever, cough and fatigue. All patient samples were included in the analysis.

Influenza A virus was detected by PCR in 28.3 per cent of the sentinel samples. There was a single detection of influenza B virus. The positive rate was 36 per cent among patients who had request forms that showed symptoms meeting the case definition, but only 5.4 per cent for those who did not.

GPs were also asked to comment on their clinical impression of the patient and to estimate the likelihood of the patient's illness being caused by infection with an influenza virus (Table 1). The strength of the clinical impression of influenza showed a clear relationship to the actual influenza rates.

Influenza activity was substantially higher in 2003 than in the two previous years (Figure 2). Activity was even lower in 1999 and 2000 with reduced numbers of samples submitted by sentinel practices and only six and 15 influenza cases respectively, reported from sentinel samples. Influenza A activity in 2003 was first detected in early July and peaked in September and October (Weeks 35 to 39). Influenza B activity was detected only once. This was in the week beginning on 20 October 2003. The serology requests showed the first obvious rise but this preceded the beginning of the influenza season. Rises in ILI/practice and influenza detections from sentinel practices occurred 2–3 weeks later (Figure 3).

Figure 2. Comparison of influenza detections obtained from sentinel samples in 2001, 2002 and 2003

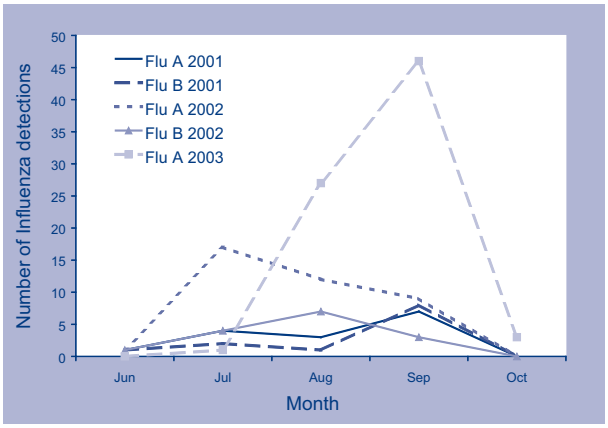


Figure 3. Incidence of sentinel GP influenza-like illnesses reporting and influenza detections, routine influenza detections and respiratory serology requests during the period of influenza surveillance, 2003

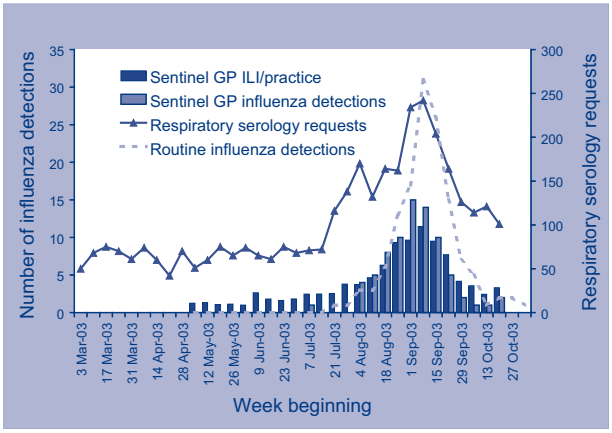
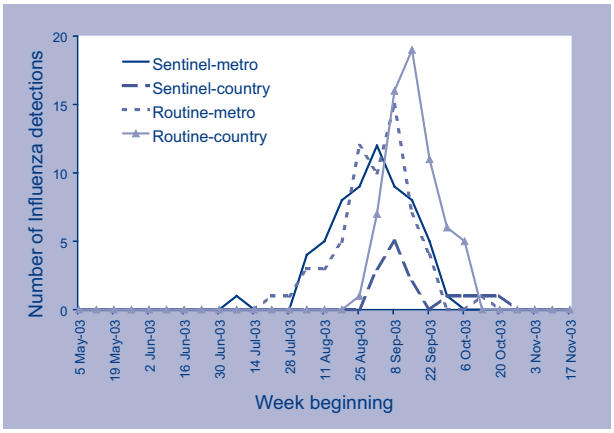


Table 1. Analysis of GP estimates of influenza virus infections in patients from sentinel practices

Respiratory virus detected	Almost certain influenza n=67		Probable influenza n=146		Likely influenza n=57		Not stated n=6	
	n	%	n	%	n	%	n	%
Influenza	38	56.7	35	24.0	6	10.5	0	0.0
Other virus	3	4.5	23	15.8	12	21.0	1	16.7
Not detected	26	38.8	88	60.3	39	68.4	5	83.3

When metropolitan and country areas were analysed separately (Figure 4) activity peaked in the Perth metropolitan area in September (week 35 to 37) and in late September and October in country areas (week 37 to 39).

Figure 4. Timing of detections of influenza A viruses from both routine and sentinel samples, 2003



Influenza A virus was detected in patients from all age groups but the largest percentage of positive samples were found in the 11–30 year age groups (43/89, 48%, Table 2).

Influenza infection was responsible for the illness in 32.3 per cent of the unvaccinated (68/211) compared with only 12.5 per cent (7/56) of the vaccinated individuals, i.e. unvaccinated patients with an ILI were 2.5 times more likely to have influenza than were vaccinated patients.

Routine samples received at PathCentre

Between 5 May 2003 and 26 October 2003 (the period of the surveillance program) there were 129 detections of influenza A and 2 detections of influenza B in the routine samples received by the PathCentre. These showed a time distribution similar to that shown by the sentinel general practices (Figure 4). Outside that period there was minimal influenza activity, with only five influenza A detections and two influenza B detections.

Typing of influenza isolates

In 2003 the laboratory referred a total of 111 influenza virus isolates to the WHO Collaborating Centre in Melbourne, of which 106 were type A, three type B and two could not be recovered. The latter were two influenza A isolates that were found by us to be positive for influenza virus, by PCR and fluorescence. Forty-five influenza isolates were obtained from the sentinel samples and 66 from routine specimens sent to the PathCentre. Forty-three of the sentinel positives were typed as influenza A, one as influenza B and one could not be recovered. Of the 66 isolations from routine samples, 63 were identified as influenza A, two as influenza B and one could not be recovered. The majority of influenza A isolates (104/106) isolated in Western Australia in 2003 were identified as the H3N2 subtype and were typed as A/Fujian/411/2002-like, a new variant of the A/Moscow strain that circulated in 2002. The remaining two isolates typed as A/Moscow/10/99-like. In 2003 relatively few cases of influenza B infection were detected in Western Australia and all were typed as B/Sichuan.

Table 2. Influenza detections in sentinel samples, 2003, by age group and vaccination status

Age group	Total samples	Influenza A				Influenza B			
		Positive samples	Male	Female	Vaccination history (Y/N/U)	Positive samples	Male	Female	Vaccination history (Y/N/U)
0–10	28	6	3	3	0/6/0				
11–20	46	26	17	9	2/23/1				
21–30	43	17	9	8	1/16/0				
31–40	53	10	4	6	1/7/2				
41–50	28	9	6	3	2/6/1				
51–60	32	9	5	4	0/9/0	1	1	0	0/1/0
>60	45	1	1	0	1/0/0				
Totals	275*	78	45	33	7/67/4	1	1	0	0/1/0

Vaccination history: N = not vaccinated, Y = vaccinated, U = unknown vaccination status of positive cases.

* Includes one patient of unknown age.

Other respiratory viruses

In addition to influenza A and B, 39 (14%) specimens of sentinel samples were positive for a number of other respiratory viruses: 5 parainfluenza, eight RSV, 11 human metapneumovirus, 17 rhinoviruses and one adenovirus. This included three dual infections, two with human metapneumovirus and influenza A, and one with parainfluenza 3 and influenza A.

Overview of the age distribution of influenza in 2003

The age distribution of the cases diagnosed as part of the sentinel general practitioner surveillance (Figure 5) showed a peak in adolescents and young adults that was not evident in 2002. Patients seen in the sentinel general practices are usually those with mild illness who seek attention from general practitioners and who may not necessarily have been sampled unless they were part of the surveillance. It does not necessarily reflect influenza distribution in more severely ill patients who warrant routine testing by their general practitioner or who present to hospital. Therefore the age distribution for influenza cases diagnosed from routine testing by culture, PCR or serology was also determined (Figure 6). This showed the expected peak in the elderly but, like the sentinel practice data, there was a relative increase in cases in the younger age groups compared with 2002.

The number of Influenza B cases was insufficient to comment on the age distribution.

Figure 5. Age distribution of patients with influenza confirmed by virus detection in surveillance samples from sentinel general practitioners

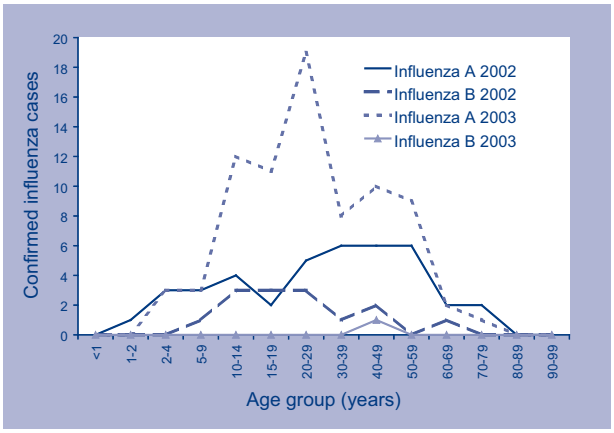
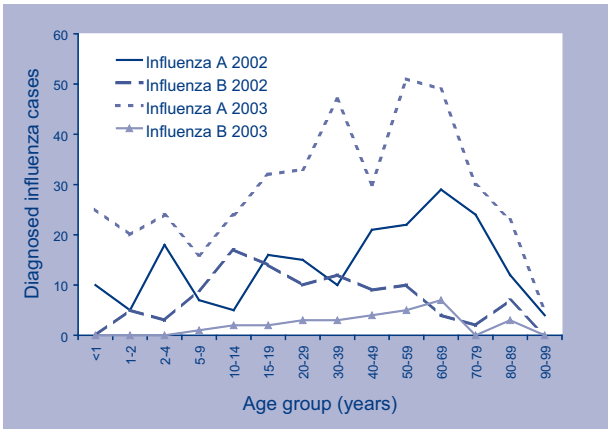


Figure 6. Age distribution of influenza cases diagnosed at PathCentre in 2002 and 2003, either by virus detection or serology



Discussion

In 2003 Western Australia experienced the largest influenza epidemic for at least five years. Additionally, the season was short resulting in very high numbers of cases during the peak weeks. The epidemic was predominantly caused by infection with influenza A H3 Fujian virus. Influenza A activity was first detected in the Perth metropolitan regions and then appeared to spread to country regions of the State. This increase in activity in country areas coincided with a decline in activity in Perth metropolitan areas.

The first indicator of the winter increase was a rise in the number of requests for respiratory serology, but this did not correlate with influenza activity in either sentinel or routine samples. However, there was significant RSV and rhinovirus activity detected in the routine PathCentre samples at that time (data not shown), which may have contributed to a rise in adult respiratory illness prior to the influenza season. The appearance of influenza in the sentinel samples accompanied by a rise in ILI seemed to give the earliest clear indication of the beginning of the influenza season. All indicators then rose together and peaked around the same time, including a secondary peak in the respiratory serology requests. The peak in routine influenza detections was slightly later than the peak in sentinel influenza activity. The higher proportion of country samples (which peaked later) among the routine positives compared with the sentinel positives would explain that difference (Figure 4).

The influenza A activity in 2003 showed an increase in disease in children and young adults compared with 2002, and this was seen in both the sentinel and routine samples. This may reflect an altered pathogenicity for this strain. Information from the following Northern Hemisphere winter indicated a possible increase in severe illness in children due to influenza A/H3 Fujian,[3] though this is not yet verified. Also our data is an underestimate of routinely diagnosed influenza in children, as it does not include children diagnosed at our major children's hospital. Therefore it is possible that the magnitude of the shift towards disease in children may also be underestimated.

The influenza A strain that emerged in 2003 (A/Fujian/411/2002-like) is an H3N2 drift variant of the A/Moscow/10/99-like strain that was circulating in 2002. The Australian vaccine for 2003 contained the A/Moscow strain and not the new variant and there was concern that vaccine-induced protection would be reduced for this strain. There was no obvious evidence of this in the GP sentinel data, where influenza was no more common as a cause of ILI in vaccinated individuals than it had been in previous years. Also a decline in vaccine effectiveness would be expected to result in a greater increase in illness among highly vaccinated population such as the elderly. The age distribution determined from the routine diagnostic samples did not show any relative increase in influenza among the highly vaccinated population aged 65 years or over and, in fact a lower proportion of cases occurred in those aged 60 years or older than in 2002 (Figure 6). However, we do not know whether the severity of illness was different. While this data is not conclusive, it does not indicate any substantial impact from the mismatch between the vaccine and the circulating strains.

In 2002 the dominant subtype of influenza B had been B/Hong Kong, which was included in the Australian vaccine for 2003. Relatively few cases of influenza B infection were detected in Western Australia and all were typed as B/Sichuan. In the absence of any significant activity it is not possible to speculate on the impact of the mismatch between the vaccine and the circulating strains.

A number of other respiratory viruses were also detected in sentinel samples. This was more likely to occur if the clinical impression reported by the GP was 'likely' or 'probable', rather than in the 'highly probable' group. It indicated that a strong clinical impression of influenza did largely separate influenza from other viruses. This may not necessarily apply to the same extent in seasons when influenza activity is lower or if the circulating strain causes milder illness. As in previous years, rhinoviruses were the most common respiratory pathogens besides influenza. It was interesting to see that hMPV was again detected in our surveillance population. The majority (8/11) occurred in patients aged 30 years or more, and activity occurred throughout the surveillance period. Relatively little is known about this virus as it was only identified in the past few years.[4] It is known to cause severe lower respiratory tract infections in young children, the elderly and immunosuppressed patients[5] and these data suggest that, like RSV, it circulates in the community as a mild respiratory illness of adults.

Acknowledgements

This surveillance program is funded by the Health Department of Western Australia.

We would like to thank all the participating general practitioners and their staff.

In addition, we thank all specimen reception staff and laboratory staff at PathCentre, Perth and country laboratories for arranging transportation and processing specimens. Thanks also to Robyn Wylie for her help preparing reports. We acknowledge the collaboration of the WHO Collaborating Centre for Reference and Research on Influenza.

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Higher than normal seasonal influenza activity in Victoria, 2003

Joy Turner,[1] Thomas Tran,[2] Chris Birch,[3] Heath Kelly[4]

Abstract

Influenza surveillance in Victoria comprises surveillance of patients with influenza-like illness (ILI) from sentinel general practices and laboratory based reporting of influenza detections, predominantly from hospital inpatients. Surveillance of patients with ILI seen by the Melbourne Medical Locum Service (MMLS) was conducted for the first time in 2003, when the influenza season was characterised by a late onset with higher than normal seasonal activity. Influenza A (H3N2) was the predominant circulating influenza virus type, with 99 per cent of sub-typed viruses identified as a drifted strain, A/Fujian/411/2002-like. Sentinel and hospital laboratory surveillance both indicated low levels of circulating influenza A (H1N1) and influenza B. Although the proportion of patients with ILI detected through MMLS surveillance was greater than the proportion from sentinel general practices, the ILI pattern was comparable between the two surveillance systems. *Commun Dis Intell* 2004;28:175–180.

Keywords: influenza, surveillance

Introduction

Influenza surveillance in Victoria comprises ascertainment of patients with influenza-like illness (ILI) through sentinel general practices and laboratory based reporting of influenza detections, predominantly from hospital inpatients.[1] Laboratory confirmed influenza is also notified to Victoria's Department of Human Services. ILI surveillance from the Melbourne Medical Locum Service (MMLS) was conducted for the first time in 2003.[2] The aim of surveillance is to facilitate the early detection of influenza, define its distribution and provide information on circulating influenza virus strains. The sentinel surveillance program is based at the Victorian Infectious Diseases Reference Laboratory (VIDRL).

In this report we aim to review the 2003 influenza season in Victoria and to compare surveillance data from sentinel general practices with that from the MMLS.

Methods

Influenza surveillance was conducted in Victoria for 22 weeks between 28 April and 28 September 2003 (weeks 18–39 inclusive).

Sentinel general practice surveillance

Seventy-nine general practitioners (GPs) from 19 metropolitan and 21 rural practices were recruited for sentinel ILI surveillance. Practices in specific locations were targeted to ensure an even distribution throughout metropolitan and regional Victoria. GPs were required to report weekly on the total number of consultations and the number of patients with ILI. The ILI case definition of fever, cough and fatigue was developed from a review of Victorian and Western Australian surveillance data.[3] A standard data collection form was used to record age, sex and vaccine status. Data were submitted without patient identifiers.

In addition to surveillance of patients with ILI, GPs were asked to collect a nose and throat swab from those patients who fulfilled the case definition for influenza and had presented within three days of onset of symptoms. Specimen collection was at the discretion of the GP. Information on each patient's vaccination status, symptoms and date of onset and the GP's clinical impression of the likelihood of influenza (almost certain, probable or less likely) was forwarded with each sample.

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Laboratory testing

Specimens were tested at VIDRL using an in-house respiratory multiplex polymerase chain reaction (PCR) adapted from published assays.[4,5,6] This assay identifies influenza A (H3N2 and H1N1), influenza B, adenovirus, picornavirus (enterovirus and rhinovirus), respiratory syncytial virus (RSV) and parainfluenza viruses. A small amount of bovine viral diarrhoea virus (BVDV) was spiked into each specimen prior to RNA extraction to act as a control for inhibitors of the PCR. When influenza virus was detected, aliquots of the original specimens were forwarded to the WHO Collaborating Centre for Reference and Research on Influenza for virus strain sub-typing. Specimens from outbreaks of influenza that had been tested at VIDRL were also forwarded for sub-typing.

Laboratory surveillance of hospital in-patients

In addition to sentinel practice surveillance, laboratory based surveillance of influenza was carried out by four metropolitan laboratories at The Royal Children's Hospital, Monash Medical Centre, The Alfred Hospital and VIDRL (diagnostic specimens other than specimens tested as part of surveillance). These laboratories reported all influenza that was identified by virus isolation, direct detection of viral antigen or nucleic acid assays.

Data collation and reporting

Rates for ILI were calculated per 100 consultations using the total number of weekly consultations as the denominator. Consultation and laboratory surveillance data were collected on a weekly basis and reports were generated fortnightly. These were disseminated to participating general practitioners, other interested health professionals and State and Commonwealth Departments of Health. Reports were also posted on the VIDRL web site (http://www.vidrl.org.au).

The ILI activity was described using a set of threshold values.[7] These were defined as; baseline <0.25 ILI cases per 100 patients/week, which reflects the influenza activity for the majority of the year; normal seasonal activity 0.25 – <1.5 ILI cases per 100 patients/week; higher than expected seasonal activity 1.5 – 3.5 ILI cases/100 patients/week; and epidemic activity above 3.5 ILI cases per 100 patients/week.

Melbourne Medical Locum Service

In addition to the sentinel general practice surveillance, data were collected for the first time on a weekly basis from the Melbourne Medical Locum Service, which provides a 24-hour, seven days a week medical service to patients within metropolitan Melbourne, although there is no information on the representativeness of the patients seen. Most locum consultations occur after normal working hours and usually involve general practice patients with an acute medical problem. Patients are seen in their homes. All information, including a list of the patient's presenting symptoms and a final working diagnosis from the attending doctor, is routinely entered into a database maintained by the MMLS. Data were extracted using the working diagnosis of 'flu' or 'influenza'. MMLS transmitted data electronically to VIDRL each week.[2]

Results

Sentinel surveillance

Of the 40 sentinel practices recruited throughout metropolitan Melbourne and rural Victoria, 39 provided data (Figures 1a and 1b), with an average of 34 practices (85% of those participating) contributing ILI data per week. There were 143,168 consultations recorded of which 1,277 (0.9%) were for ILI.

The weekly GP consultation rate for ILI peaked at higher than normal seasonal activity. ILI activity started to rise from week 31, reaching a peak of 3.4 per 100 patients by week 34 and declining to baseline activity by week 39 (Figure 2). Figure 3 compares the ILI consultation rate in 2003 with the rates from 1997 to 2002. This was the first reported influenza season since 1998 where peak levels were higher than the expected normal seasonal activity.

Laboratory testing of specimens from sentinel practices

Over the 2003 surveillance period, specimens were collected from 540 patients with ILI. Thirty-six samples were inhibited and were not included in further analysis. Of the 504 non-inhibited specimens, 184 (37%) were confirmed as influenza. Influenza A (H3N2) was the predominant virus detected (98% of all influenza PCR positive specimens) with only three (2%) influenza B PCR positive specimens.

Figure 1a. Distribution of sentinel surveillance sites in rural Victoria

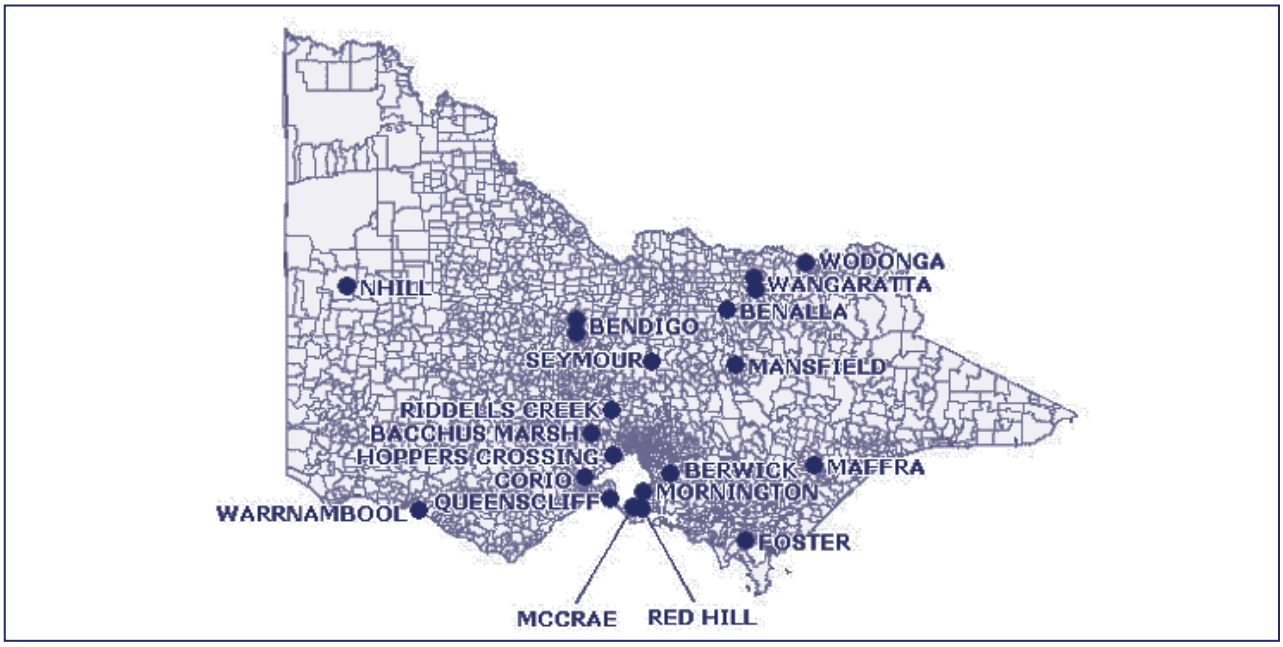


Figure 1b. Distribution of sentinel surveillance sites in metropolitan Victoria

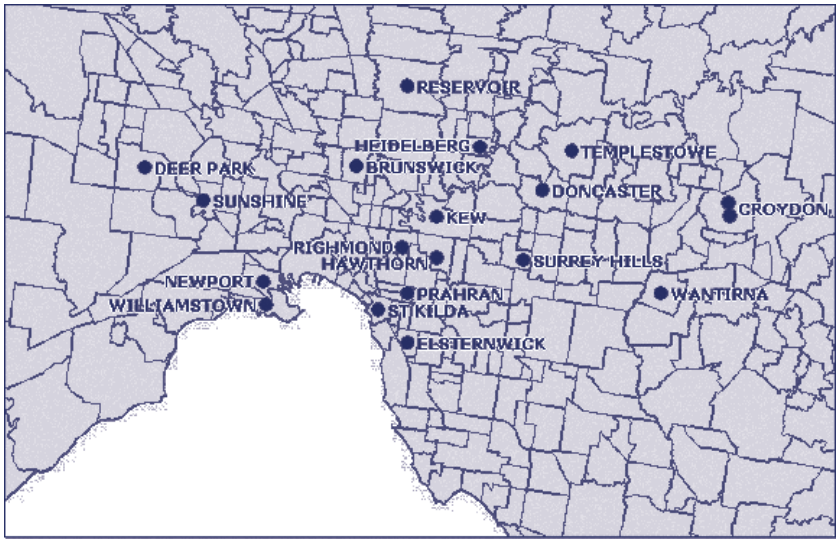


Figure 2. General practice surveillance for influenza-like illness from metropolitan and regional sentinel sites, 2003

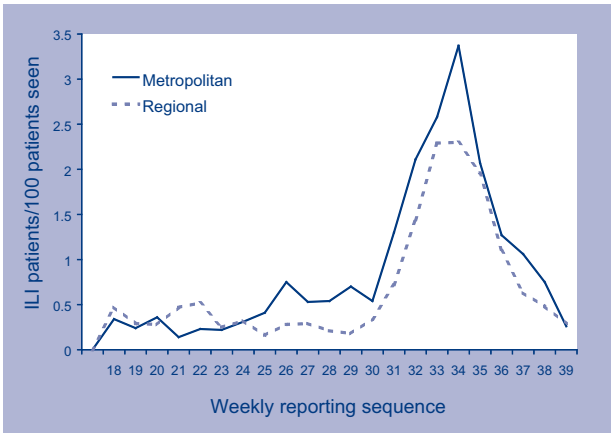
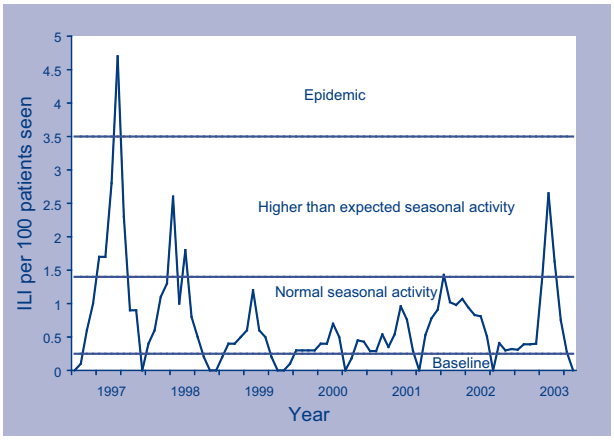


Figure 3. Fortnightly consultation rates for influenza-like illness, Victoria, 1997 to 2003



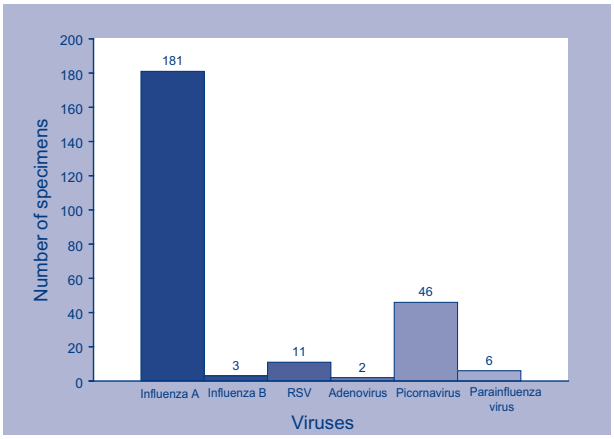
Aliquots of all PCR positive influenza A and influenza B specimens were sent to the WHO Collaborating Centre for Influenza for virus strain sub-typing. Of the 115 isolates recovered, 114 (99%) were typed as A/Fujian/411/2002-like and one (1%) was A/Moscow/10/99-like.

In addition to influenza A and B viruses, 65 specimens (13% of all patients with ILI) were PCR positive for other respiratory viruses, of which picornaviruses and respiratory syncytial virus were the most common non-influenza viruses (Figure 4).

Clinical diagnosis of influenza

Sentinel surveillance GPs were asked to forward their clinical impression (almost certain, probable or less likely) of the likelihood of influenza with each sample. Perception of certainty of influenza diagnosis is shown in the Table for the entire influenza season and for the peak season in the metropolitan area only. Over the entire season, it appeared that the GPs' perception of the likelihood of influenza was not associated with an increase in the laboratory confirmation of influenza. However, when the analysis was restricted to metropolitan GPs and the weeks when ILI activity was highest, the proportion of laboratory confirmed influenza increased with the GPs' perception of the likelihood of influenza.

Figure 4. Sentinel surveillance-PCR results for 2003



During the entire season, for any level of diagnostic certainty, the positive predictive value for the diagnosis of influenza was 37 per cent, but this rose to 47 per cent when considering only metropolitan GPs during the weeks when ILI activity was high.

Patient vaccination status was not analysed as status was unknown for 172 (34%) patients and not validated for those in whom it was reported.

Table. General practitioner perception of the likelihood of influenza by weeks of the surveillance season

All general practitioners for the entire influenza season				
Respiratory virus detected	**GP certainty of diagnosis**			
	Almost certain n=191 number (%) detected	**Probable n=228** number (%) detected	**Less likely n=35** number (%) detected	**Not stated n=50** number (%) detected
Influenza	47 (24.6%)	99 (43.4%)	26 (74.3%)	12 (24.0%)
Other virus	35 (18.3%)	19 (8.3%)	6 (17.1%)	5 (10.0%)
Not detected	109 (57%)	110 (48.2%)	3 (8.6%)	33 (66.0%)
Metropolitan general practitioners only - weeks 31 to 38 inclusive				
Respiratory virus detected	**GP certainty of diagnosis**			
	Almost certain n=111 number (%) detected	**Probable n=136** number (%) detected	**Less likely n=15** number (%) detected	**Not stated n=23** number (%) detected
Influenza	64 (57.7%)	57 (41.9%)	5 (33.3%)	9 (39.1%)
Other virus	6 (5.4%)	13 (9.6%)	1 (6.7%)	2 (8.7%)
Not detected	41 (36.9%)	66 (48.5%)	9 (60%)	12 (52.2%)

Outbreaks

Specimens from five outbreaks of respiratory disease were tested by VIDRL during 2003, of which four were confirmed to be due to influenza A (H3N2). A/Fujian/411/2002-like influenza was detected from all outbreak specimens that were forwarded for virus strain sub-typing to the WHO Collaborating Centre for Influenza.

Comparison of sentinel and laboratory surveillance

Similar proportions of influenza A and influenza B were detected from sentinel and hospital laboratory surveillance. Of the 445 influenza detections from the four laboratories, 444 (99%) were influenza A and one (1%) was influenza B, compared with 181 (98%) influenza A and 3 (2%) influenza B from sentinel surveillance. One influenza A (H1N1) subtype was identified from laboratory surveillance but none were identified from sentinel surveillance.

Comparison of sentinel surveillance and laboratory surveillance influenza positive specimens by age shows a higher percentage of surveillance specimens to be in the 5–35 year age range, whereas the hospital laboratory surveillance was more likely to identify the younger group from 0–4 years (Figure 5).

Melbourne Medical Locum Service

During weeks 18 to 39 of the surveillance period the MMLS recorded a total of 25,630 consultations, of which 661 (2.5%) were ILI patients. By comparison, the sentinel general practitioner surveillance recorded 143,168 consultations, of which 1,277 (0.9%) were ILI patients. Although the proportion of patients with ILI seen by MMLS was greater than sentinel surveillance the ILI pattern was comparable between the two surveillance systems (Figure 6).

Figure 5. Comparison between surveillance and laboratory results, by age group

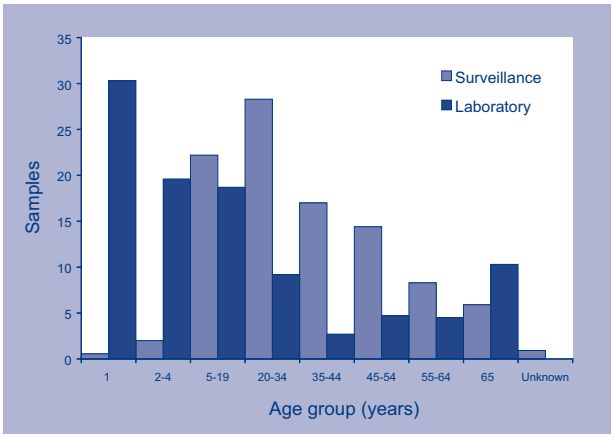
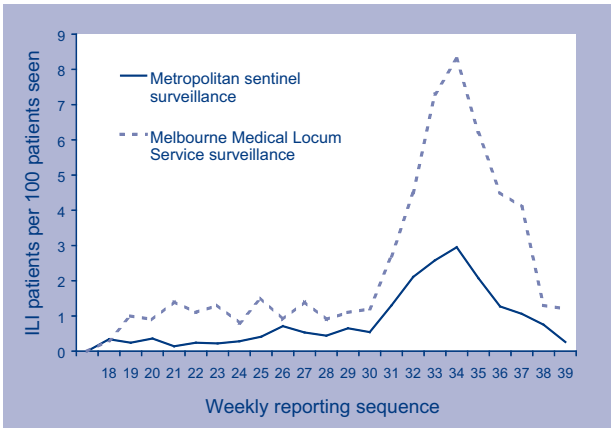


Figure 6. Comparison of metropolitan sentinel surveillance and Melbourne Metropolitan Locum Service surveillance, 2003



Discussion

Using threshold levels to describe the ILI activity reported from GP sentinel surveillance, the 2003 influenza season was described as higher than normal seasonal activity. Like 2000, the influenza season of 2003 was characterised by initial low levels of circulating influenza with a late peak. It was however, the first reported influenza season where peak levels reached higher than normal seasonal activity since 1998.

The season was characterised by the identification of a new strain of A (H3N2) influenza. The WHO Collaborating Centre for Reference and Research on Influenza introduced the reference strain of A/Fujian/411/2002-like for reporting of antigenic analysis. According to the Centre this is a drift variant of A/Moscow/10/99-like strain.[8]

The 2003 influenza season vaccine contained the following:[9]

- A/New Caledonia/20/99(H1N1)–like virus
- A/Moscow/10/99(H3N2)–like virus
- B/Hong Kong/330/2001–virus

Although A/Fujian was not included in the vaccine formulation for 2003, it was thought that the A/Moscow vaccine strain would provide some protection from A/Fujian infection.[8] The WHO Collaborating Centre for Reference and Research on Influenza reported that 'the A/Fujian-like viruses are still related to the A/Moscow-like strain included in the current vaccine and the vaccine has been demonstrated to induce antibodies to the A/Fujian-like strains, but generally at a reduced level. This suggests the possibility that there may be some reduction in the protective value of the vaccine, however this cannot be quantified.'[8]

The GPs' positive predictive value for influenza appeared to be lower in 2003 than in most previous years.[7] Of the 504 patients tested for influenza and other respiratory viruses, GPs indicated that they were almost certain that 191 patients had influenza. Influenza was detected in only 25 per cent of these specimens compared to 2002 when the comparable rate was 54 per cent. The GPs were rewarded with continuing medical education points from the Royal Australian College of General Practitioners for participation in the sentinel influenza surveillance program. In 2003 the College introduced a requirement that a minimum of five swabs needed to be obtained to qualify for these points. This, coupled with a very late seasonal peak, may have accounted for the decreased rate of detection of influenza when GPs were confident of the diagnosis. We performed a restricted analysis of metropolitan GPs in the weeks when ILI activity was high because transport to the laboratory is faster in the metropolitan area and testing may therefore have been more likely to detect influenza viral RNA when it was present. In this restricted analysis, the proportion of patients with an ILI confirmed to have influenza increased with the GP's certainty of the diagnosis. However the one GP whose positive predictive rate was 100 per cent from six patients was a regional practitioner who sent the first nose/throat swab from a patient with ILI in week 34, near the peak of ILI activity.

Victorian ILI surveillance has traditionally focused on consultations with metropolitan and regional GPs. The introduction of the MMLS surveillance for the first time this year provided an additional indicator of influenza activity. Overall the pattern of ILI was consistent between the two systems. This had also been demonstrated in a retrospective comparison of MMLS and sentinel surveillance data.[2] As anticipated, there was a higher proportion of patients with ILI from MMLS. This could be attributed to the different nature of the practices and possibly to differing case definitions for ILI. Although sentinel general practitioners use a specific case definition, data extracted from the MMLS database were based on reference to 'flu' or 'influenza'. Collection of surveillance data from MMLS will continue all year, whereas the sentinel surveillance is only conducted during the influenza season from May to September. In general, the use of MMLS surveillance needs to be tested using the same case definition as the sentinel surveillance but, for determining relative seasonal ILI rates, has the potential to simplify what is currently a labour intensive scheme. However collecting specimens from patients seen by MMLS GPs proved to be logistically difficult and it appears that sentinel practitioners will continue to be needed for the collection of specimens from patients with an ILI.

Acknowledgements

We would like to thank the general practitioners and their staff, the hospital laboratories and the generous support of the pathology providers who have all assisted us in the 2003 program. We are grateful to all specimen reception and laboratory staff at VIDRL. We extend a special thanks to the MMLS for generously providing their locum service data. We acknowledge the collaboration of the WHO Collaborating Centre for Reference and Research on Influenza. The sentinel surveillance program is supported in part by the Department of Human Services, Victoria.

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Severe acute respiratory syndrome surveillance in Australia

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Abstract

In March 2003, the World Health Organization (WHO) issued a global alert recommending active worldwide surveillance for severe acute respiratory syndrome (SARS). This paper describes the epidemiological features of cases reported by Australian states and territories to the Australian Government Department of Health and Ageing between 17 March and 31 July 2003. There were 138 people investigated for SARS: 111 as suspect and 27 as probable. Five probable cases were reported to WHO after review of other possible diagnoses and Australia-specific exclusion criteria had been applied. An additional probable case identified by laboratory testing overseas, but who was not under investigation when in Australia, was also reported to WHO. The method by which surveillance for SARS was rapidly established provided an opportunity to examine Australia's planning and preparedness for future respiratory disease epidemics such as influenza. *Commun Dis Intell* 2004;28:181–186.

Keywords: Severe acute respiratory syndrome, surveillance, Australia, influenza

Introduction

On 12 March 2003, the World Health Organization (WHO) issued a global alert about cases of a new, highly infectious severe atypical pneumonia referred to as severe acute respiratory syndrome (SARS).[1] Thought to have originated in Guangdong province, China in November 2002, SARS spread to 27 countries worldwide and two administrative regions of China. Mainland China, Hong Kong Special Administrative Region of China, Taiwan, Singapore, Vietnam and Canada were particularly affected.[2]

The global alert recommended countries undertake appropriate surveillance to detect cases of SARS. Symptoms of SARS included high fever (greater than 38° C), cough, shortness of breath or breathing difficulties.[3] On 16 March 2003 the Joint Executive Group, comprised of state and territory members from the Communicable Diseases Network Australia (CDNA), called by the acting Chief Medical Officer, commenced daily teleconferences to respond to the threat of importation of SARS. An Australian government inter-departmental taskforce for SARS was established on 28 March 2003. The Australian Government Department of Health and Ageing (DoHA) authorised the activation of an Incident Room on 4 April 2003 which coordinated the national public health response to SARS. This report summarises the people under investigation as suspected and probable cases of SARS notified to DoHA from 17 March to 31 July 2003.

Methods

Case definition

During the reporting period of March to July 2003 the WHO SARS case definition was used to determine those under investigation.[4] A person was classified as under investigation as a suspect case if they had a history of high fever (greater than 38° C); *and* cough, shortness of breath or breathing difficulty; *and* had resided or travelled in a SARS affected area or had contact with a SARS case in the 10 days prior to onset of symptoms. A person was classified as under investigation as a probable case if they met the criteria to be under investigation as a suspect case *and* had evidence of pneumonia by chest x-ray or acute respiratory distress syndrome or was positive for SARS coronavirus by one or more assays.

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Persons investigated as probable or suspect cases were excluded if an alternative diagnosis (including if there was a clinical response to antibiotic treatment) was made. Cases were also excluded if they met Australia-specific criteria, which included no convincing possibility of exposure (in transit for less than 8 hours in an area designated by WHO as SARS-affected, and remaining within the airport) or—for cases investigated as suspect—the illness was mild and self-limiting. The Australia-specific exclusion criteria—although subjective—were used to increase the specificity of the case definition in a non-SARS affected country where prevalence would be expected to be extremely low, to avoid an undue burden on health-care facilities created by the excess patient management and respiratory isolation required for suspect or probable SARS cases.

Notifications

State and territory health authorities raised clinicians' awareness of SARS by contacting hospitals and health alerts to general practitioners; however the mechanisms used varied between jurisdictions. Cases were initially notified by clinicians to local and jurisdictional public health authorities voluntarily, although during the surveillance period most jurisdictions listed SARS as a notifiable disease to make reporting mandatory. SARS notifications from each jurisdiction were reported to DoHA from 17 March to 25 July 2003. Data were collected initially by verbal report at daily teleconferences and through a nationally developed questionnaire which could be submitted by email or facsimile.

Following investigation, persons that met the case definition for a probable case were reported to WHO. Initially, WHO requested that both suspect and probable cases be reported. After 22 March only probable cases were reported via the WHO website.

Results

There were 138 persons under investigation for SARS notified to DoHA between 17 March and 31 July 2003. Of these, 111 were investigated as suspect cases and 27 were investigated as probable cases (Figure 1).

Six probable cases were reported to WHO; three from Victoria, two from New South Wales and one from Queensland. One of the probable cases was identified by SARS coronavirus serological testing overseas. This case was not hospitalised, and was not detected or investigated by routine surveillance methods for SARS when in Australia.

Eighty-one per cent of the remaining 21 cases initially under investigation as probable cases were excluded on the basis of an alternate diagnosis (including 9 cases with a clinical response to antibiotic treatment) and 19 per cent were excluded on the basis of no convincing possibility of exposure. One case was excluded for no clearly identified reason (Table 1).

Six cases met the case definition for suspected SARS. The alternate diagnosis of mild self-limiting illness accounted for the exclusion of 27 per cent of persons investigated as suspect, 44 per cent were excluded by other alternate diagnoses. Twenty per cent were excluded on the basis of no convincing exposure and five people excluded without a specific diagnosis provided.

Figure 1. Persons under investigation for severe acute respiratory syndrome and probable cases, Australia, 1 November 2002 to 10 July 2003, by week of onset

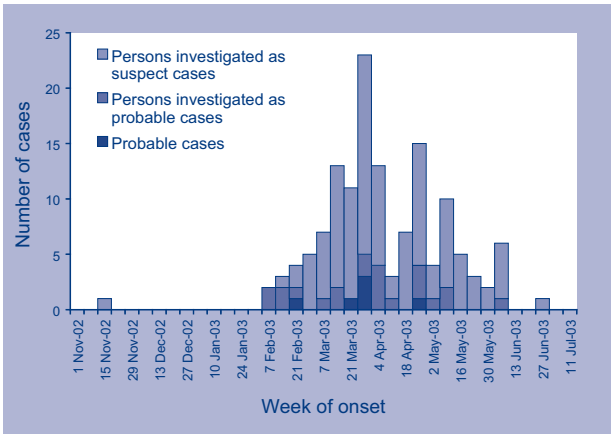

Table 1. Status of persons investigated for severe acute respiratory syndrome

	Suspect	Probable	Total
Final SARS classification	6	6*	12
No convincing exposure	21	4	25
Alternate diagnosis			
Cancer	1	1	2
Cellulitis	2	0	2
Chlamydia pneumoniae infection	1	0	1
Chlamydia (unspecified) infection	4	0	4
Chronic obstructive airway disease	1	0	1
Coronavirus infection†	1	0	1
Epstein-Barr virus infection	1	0	1
Exacerbation of asthma	2	0	2
Haemophilus (unspecified) infection	1	0	1
Influenza virus (Type A) infection	6	1	7
Influenza virus (Type B) infection	2	1	3
Maxillary sinusitis	1	0	1
Measles	1	0	1
Mycoplasma (unspecified) infection	3	1	4
Parainfluenza virus infection	2	1	3
Pharyngitis	1	0	1
Picornavirus/rhinovirus infection	3	1	4
Respiratory syncytial virus infection	1	1	2
Streptococcus (Group A) infection	1	0	1
Streptococcus pneumoniae infection	3	1	4
Typhoid fever	1	0	1
Unspecified bacterial infection	2	0	2
Unspecified viral infection	1	0	1
Upper respiratory tract infection	4	0	4
Responded to antibiotics	5	9	14
Mild self-limiting illness	28	0	28
Not provided	5	1	6
Total	111	28	139

* Includes probable case identified by serology overseas

† Does not include SARS coronavirus.

There were persons investigated for SARS in each jurisdiction. Notification rates of persons investigated as suspect and probable cases were highest in the Australian Capital Territory and the Northern Territory respectively (Table 2).

The male to female ratio among persons investigated for SARS was 1.4:1. This ratio was the same when stratified into those investigated as suspect and probable cases, although the ratio was 0.5:1 in those who met the case definition for a probable case. The median age of people investigated for SARS was 41 years (range 7 months to 89 years). The highest number of people under investigation as suspect and probable cases occurred in the 50–59 year and 0–9 year age groups respectively. The 10–19 year age group had the lowest rate of people under investigation, both as suspect and probable cases (Figure 2). The median age of those meeting the case definition for a probable case was 20 years (range 1–45 years).

Of the 138 persons investigated for SARS, 129 (93%) had cough, 60 (43%) had shortness of breath and 44 (32%) reported breathing difficulty. All persons under investigation as probable cases reported cough, and there was nearly a twofold greater reporting of the other symptoms compared to persons under investigation as suspect cases. Eighty-one per cent (n=22) of persons investigated as probable were hospitalised compared to 62 per cent (n=69) of persons investigated as suspect. Three of the six probable cases were hospitalised.

The most common SARS-affected areas where persons investigated as a probable case had travelled or resided were Singapore (14, 52%) and Hong Kong (10, 37%). Four (14%) reported travel history to China (1 to Beijing and 3 to Guangdong province) and Toronto, Canada. One person investigated as a probable case (4%) each had travelled to Hanoi, Vietnam and Taiwan. Contact with a suspect or probable SARS case in the 10 days prior to onset of symptoms was reported by four persons investigated as suspect cases.

Figure 2. Rate of persons under investigation for severe acute respiratory syndrome and probable cases, Australia, by age

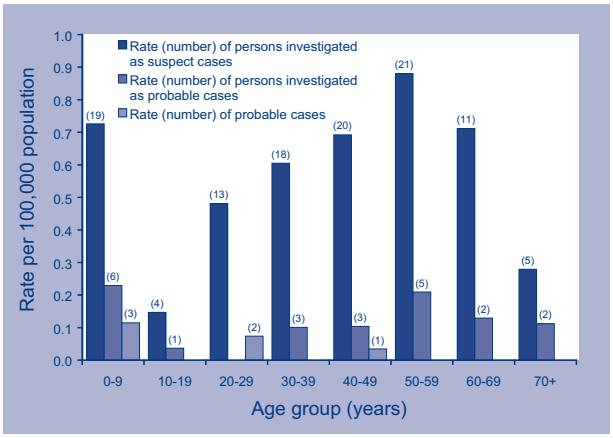

Table 2. Number and rate of persons under investigation for severe acute respiratory syndrome per 100,000 population, by jurisdiction

	n		Rate	
Jurisdiction	Suspect	Probable	Suspect	Probable
ACT	5	0	1.6	0
NSW	48	10*	0.7	0.2
NT	1	1	0.5	0.5
Qld	14	5	0.4	0.1
SA	7	1	0.5	0.1
Tas	0	1	0	0.2
Vic	26	8	0.5	0.2
WA	10	2	0.5	0.1
Total	111	28		

* Includes probable case identified by serology overseas

Discussion

Surveillance was a key component of Australia's response to SARS. Through the CDNA, a nationally coordinated approach to surveillance and public health management (including airport screening and contact tracing) was achieved. However, there were several limitations of the surveillance methods used including the poor positive predictive value of the case definition, inconsistencies with reporting and lack of data completeness, possible under-reporting and subjective exclusion criteria. The global SARS outbreak has highlighted changes and enhancements that could be made to current surveillance mechanisms for future respiratory disease epidemics such as influenza.

The case definition for SARS was very broad, which, while essential for the identification of cases in SARS-affected areas, was probably not specific enough in non-SARS-affected countries. This was recognised by WHO which rescinded its earlier directive for the reporting of all suspect cases in addition to probable cases. In an analysis of various cohorts, SARS was serologically or virologically identified in 26 per cent of cases meeting the WHO case definition for probable SARS in non-SARS affected areas compared to 79 per cent in SARS affected areas.[5] The age distribution of people under investigation as suspect and probable cases in Australia (Figure 2) is very different to that observed in SARS-affected countries where there were few cases in children and modal age groups were between 20 and 40 years.[6,7] This distribution most likely represents a generalised pattern of respiratory disease and travel habits, and reflects the poor positive predictive value of the case definition in a non-SARS-affected country.

Early clinical signs of SARS mimic many other respiratory diseases, and therefore a laboratory-confirmed diagnosis of an alternative cause was the most definitive method of exclusion. Less than half of the people under investigation as suspect or probable cases were excluded because of an alternative diagnosis involving a known aetiology. This highlights ongoing difficulties in determining aetiology for community acquired pneumonia which remains unknown in up to 70 per cent of cases.[8] It is highly unlikely whether five of the six probable cases in Australia reported to WHO actually had SARS: they did not test positive by polymerase chain reaction or serology, and were not very ill. Conversely, some of the alternate diagnoses were not entirely convincing when little was known about SARS. Although the validity of some of these may be questioned, many were difficult to follow up and this highlights the ongoing need for better communication between clinicians and public health authorities.

Reporting of surveillance data from jurisdictions and communication with DoHA through teleconferences was timely and open. Completion of questionnaires by most jurisdictions was also timely, although national data collation was difficult due to consistently slow return of questionnaires by one jurisdiction, use of a locally developed data collection form instead of the nationally developed form in one jurisdiction, and clinical data not always being timely and complete. The number of people meeting the criteria to be investigated as SARS cases is likely to have been under-reported and inconsistent as initial notifications were given voluntarily and listing of SARS as a notifiable disease was not uniform across jurisdictions. Furthermore, the drop in number of cases that were reported after March (Figure 1) could be indicative of reporting fatigue, but might also be attributed to fewer people travelling to affected countries.

In future similar efforts for influenza or other respiratory disease epidemics, improvements to national collation of data could be made by uniform reporting methods, in particular by electronic reporting rather than by facsimile, and standardised use of the data collection form. Linkage of the questionnaire to the database to create a one-step data entry process could streamline the collection and collation of data. Alternatively, reporting could be further streamlined by direct online reporting to DoHA from jurisdictions or public health units which would also ensure a uniform transition to usage of updated data collection forms.

Lack of completeness of clinical data was an important issue that highlighted the need to strengthen the links between public health and clinical data collection, particularly through clear communication in relation to the division of labour, data that is required, and how it can be reported in an easy and user-friendly way. It also highlighted the difficulties in obtaining complete clinical and laboratory workup for mild respiratory illnesses, especially those that are transient and self limiting.

The rapid and extensive allocation of resources required for the SARS response has also highlighted a need to examine surge capacity at primary care,[9] jurisdictional and national levels. Most stakeholders needed to make a substantial and prolonged response to SARS at the expense of other investigations, programs and routine activities. An assessment to estimate the impact of the response may assist in a more efficient future response to an influenza pandemic which would most likely be far greater in magnitude.

Australia may have been fortunate to avoid a SARS outbreak in the early stages of the global epidemic when very little was known about it. Careful evaluation of the implementation and method of surveillance both here and overseas are required for Australia to be well prepared for the possible re-emergence of SARS and future global outbreaks of influenza and other infectious respiratory diseases.

Acknowledgements

The authors thank members of the Joint Executive Group of the Communicable Diseases Network Australia, the Public Health Laboratory Network, state and territory health departments and local public health units, laboratories and hospitals for their efforts in the collection, reporting and verification of data for people under investigation as suspect and probable cases of SARS. We thank Leslee Roberts, Gina Samaan, Sally Munnoch, David Hogan and Megge Miller of the Department of Health and Ageing SARS Team for their assistance in data collection and collation, and Scott Cameron, Rod Givney and Gina Samaan for critical review of the manuscript. We also thank people who were under investigation for SARS and their families for their cooperation in the data collection.

The Master of Applied Epidemiology program is funded by the Australian Government Department of Health and Ageing.

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Annual report of the Australian Gonococcal Surveillance Programme, 2003

The Australian Gonococcal Surveillance Programme

Abstract

The Australian Gonococcal Surveillance Programme monitors the antibiotic susceptibility of *Neisseria gonorrhoeae* isolated in all Australian states and territories. In 2003 the *in vitro* susceptibility of 3,772 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 17 per cent but ranged up to 27 per cent in larger urban centres. Quinolone resistance in gonococci (QRNG) remained widespread and increased in most states but most markedly in Victoria. Nationally, 14.4 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 showed some decreased susceptibility to ceftriaxone (MIC 0.06 mg/L or more) and were concentrated in New South Wales. 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. In New South Wales, the number of available cultures decreased and represented 30 per cent of the national total. In Victoria, the number of isolates increased and was 25 per cent of all gonococci examined. *Commun Dis Intell* 2004;28:187–193.

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

Introduction

Antimicrobial resistance (AMR) in *Neisseria gonorrhoeae* affects not only the individual patient who may fail to be cured by ineffective dosage regimens, but also compromises public health efforts aimed at control of gonococcal disease through use of programmatic treatments. Standardised treatment protocols for gonorrhoea utilise single dose treatments designed to cure 95 per cent or more of cases.[5] The continuing increase in AMR in gonococci has led to the progressive loss of utility of different antibiotic classes in recent years. The effectiveness of the penicillins has long been compromised in much of Australia and quinolone-based treatment schedules have also been discontinued in some jurisdictions because of high levels of resistance to these agents. The net effect of increasing AMR in gonococci has been the decreased use of cheaper oral antibiotics and increased use of expensive injectable agents, most notably third generation cephalosporin antibiotics, as the agents of choice. Reports of treatment failure from Japan with cephems and monobactams were accompanied by evidence of raised MICs to cefixime (an oral agent not available in Australia) and ceftriaxone in gonococci already resistant to quinolones and penicillins.[1,2,3] Gonococcal strains with these resistances have been detected in Australia.[4]

Laboratory analyses assist the control and treatment of gonorrhoea by confirmation of the diagnosis, application of typing for gonococcal strain differentiation and provision of antibiotic susceptibility data. 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. 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 2003 calendar year.

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Methods

The AGSP is a component of the National Neisseria Network 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. The increasing use of non-culture based methods of diagnosis has the potential to reduce the size of the sample 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 and distribution, although some inferences on the latter may be also drawn from these data.

Gonococci isolated in, or 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.[6] The AGSP also conducted a program-specific quality assurance program.[7] Antibiotic sensitivity data were submitted quarterly to a coordinating laboratory which collated the results and also conducted the quality assurance program. Additionally, the AGSP received data on the sex of the patient and site of isolation of gonococcal strains. Where available, data on the geographic source of acquisition of antibiotic-resistant isolates were included in analyses.

Results

Number of isolates

There were 3,772 gonococcal isolates referred to, or isolated in, AGSP laboratories in 2003. The source of and site of infection with these isolates are shown in the Table. One thousand one hundred and sixteen gonococci (30% of the Australian total) were isolated in New South Wales, 920 (24.5%) in Victoria, 637 (16.9%) in Queensland, 517 (13.8%) in the Northern Territory, 343 (9.1%) in Western Australia, and 227 (6%) in South Australia with small numbers in Tasmania (9) and the Australian Capital Territory (3). Of the total, 3,679 remained viable for susceptibility testing.

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

Nationally, 179 (5%) fewer isolates were received in 2003 than in 2002. The number of isolates fell by 509 in New South Wales and 48 in the Northern Territory but rose by 226 in Victoria, 95 in South Australia and 49 in Queensland. In Western Australia numbers were stable in 2003. Numbers in other centres remained low.

Table. Source and number of gonococcal isolates, Australia, 2003, by sex, anatomical site and state or territory

		State or territory						
	Site	NSW	NT	Qld	SA	Vic	WA	Aust*
Male	Urethra	720	294	437	131	614	246	2,449
	Rectal	181	2	35	53	152	15	440
	Pharynx	101	0	12	28	94	5	240
	Other/NS	44	13	13	3	8	4	85
	Total	1,046	309	497	215	868	270	3,214
Female	Cervix	53	188	128	7	47	69	495
	Other/NS	14	17	12	5	5	4	57
	Total	67	205	140	12	52	73	552
Unknown	Total	3	3	0	0	0	0	6
Total*		1,116	517	637	227	920	343	3,772

* Includes isolates from Tasmania (9) and the Australian Capital Territory (3).

NS Not stated.

Source of isolates

There were 3,214 strains from men and 552 from women, with a male to female (M:F) ratio of 5.8:1, only slightly higher than the 5.6:1 ratio for 2002. The number of strains from men decreased by 121 and from women by 42. The M:F ratio was again high in New South Wales (15.6:1) and Victoria (16.7:1) where strains were more often obtained from urban populations. The lower ratios in Queensland (3.5:1) Western Australia (3.7:1), and the Northern Territory (1.5:1) reflected the large non-urban component of gonococcal disease in those regions. Male rectal and pharyngeal isolates were most frequent in South Australia (38% of isolates from men), New South Wales (27%) and Victoria (28%). These percentages are higher than in 2001 and 2002 but also may reflect clinical sampling practices in those States. About 2.5 per cent of isolates are shown as being isolated from 'other' or unknown sites. These included 14 cases of disseminated gonococcal infection in men (0.4%) and 10 (1.8%) in women. 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. There were a small number of isolates from the eyes of both new-born and older infants and also adults, and from pelvic and Bartholin's abscesses in women.

Antibiotic susceptibility patterns

In 2003 the AGSP reference laboratories examined 3,677 gonococcal isolates for sensitivity to penicillin (representing this group of antibiotics), ceftriaxone (representing later generation cephalosporins), ciprofloxacin (representing quinolone antibiotics) and spectinomycin and for high level resistance to tetracycline (TRNG). As in past years the patterns of gonococcal antibiotic susceptibility differed between the various states and territories. For this reason data are presented by region as well as aggregated for Australia as a whole.

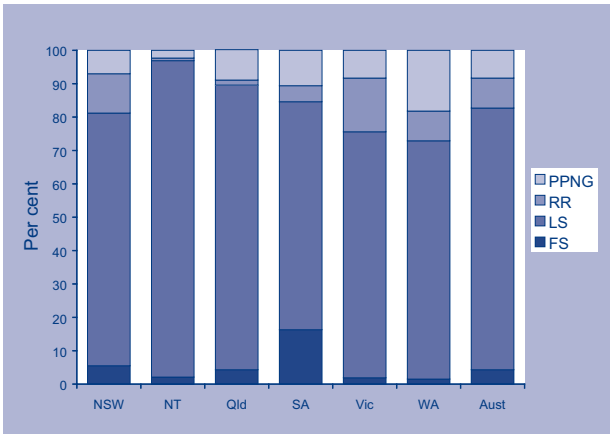
Penicillins

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 (MIC) in mg/L which is the least amount of antibiotic which inhibits *in vitro* growth under defined conditions. The categorisation of strains in Australia in 2003 by penicillin MIC is shown in Figure 1. The MIC reflects the expression of multiple and different chromosomal changes present in an organism.[8] 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.

The number (333) and proportion (9%) of isolates resistant to penicillin by chromosomal mechanisms, CMRNG, in 2003 was lower than in 2002 (421, 10.9%) and 2001 (558, 15.3%). Strains of this type were concentrated in Victoria where 147 were detected (16.1% of all isolates) compared with 76 CMRNG, (11%) in 2002. A small increase in the proportion of CMRNG to 4.8 per cent was seen in South Australia in 2003. In Western Australia the number and proportion of CMRNG (29, 8.9%) was little changed from 2002. The number and proportion of CMRNG in Queensland (9, 1.5%) continued to decrease compared to 2002, (26 CMRNG, 4.6%) and 2001 (101, 17.3%). A large decrease in CMRNG was also seen in New South Wales in 2003 when 130 (11.8%) CMRNG were seen compared with 2002, (275 CMRNG, 17%). In the Northern Territory, four CMRNG strains represented 0.8 per cent of all isolates in 2003.

Figure 1. Penicillin resistance of gonococcal isolates, Australia, 2003, 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 *Neisseria gonorrhoeae.*

The number of PPNG isolated in 2003 (306) was 32 more than in 2002 and the proportion of all isolates (9%), was also more than the previous year (7.1%). Again the distribution of PPNG differed significantly by region. Western Australia once again had the highest proportion of PPNG, (59 PPNG representing 18.2% of all gonococci). South Australia had 24 PPNG (10.6%), Queensland 56 (9.1%) and Victoria 76 (8.3%). New South Wales had the highest number of PPNG (78, 7%) The 11 PPNG in the Northern Territory represented 2.3 per cent of all isolates there. Two PPNG were found in Tasmania but none in the Australian Capital Territory. Information on the geographic location of the acquisition of PPNG was available for only 80 of the 306 infections and most data were from New South Wales and Western Australia. In Western Australia, local acquisition was prominent (30 of 39 cases) and in New South Wales overseas acquisition was recorded in 26 of 40 cases. Cambodia, China, Fiji, Hong Kong, Indonesia, Papua New Guinea, the Philippines, Thailand, Viet Nam, the United States of America and the United Kingdom were identified as countries of PPNG acquisition.

Ceftriaxone

No instance of treatment failure following a 250 mg dose of ceftriaxone and attributable to altered MICs has been described in Australia to date. However in 2001, a small number of strains in a few states showed a small increase in ceftriaxone MICs. In 2002, there were 21 gonococci with ceftriaxone MICs > 0.03 mg/L isolated in Australia. In 2003, this number declined to a total of 10 with seven of these in New South Wales, two in South Australia and one in Queensland. Isolates were usually also resistant to quinolones and penicillins, but spectinomycin sensitive.

Spectinomycin

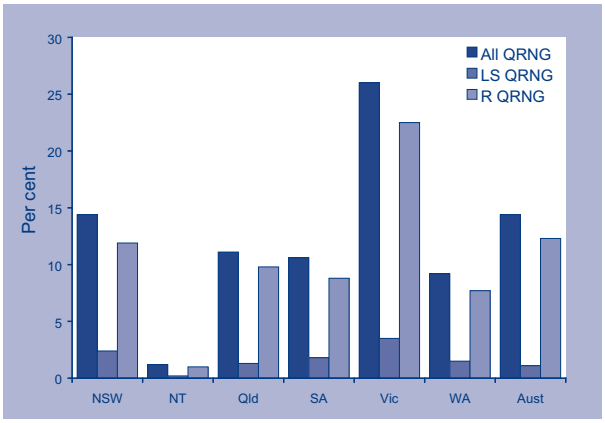
All isolates were susceptible. Resistance most often occurs as a result a single step ribosomal change.

Quinolone antibiotics

Gonococcal 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 a lower level of resistance, (0.06 – 0.5 mg/L) 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 (1 mg/L or more), 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 2003, a total of 529 (14.4%) gonococci had some level of resistance to quinolones (QRNG) (Figure 2) an increase over the 389 (10%) recorded in 2002 but less than the 638 (17.5%) seen in 2001. Most QRNG (452, 85%) had resistance at a high level (MICs ≥ 1 mg/L).

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, by region, Australia, 2003



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

The highest proportion of QRNG was seen in Victoria where the 237 QRNG were 26 per cent of the total number examined. This is a substantial increase in both the number (98) and proportion (14%) of QRNG seen in Victoria in 2002. In New South Wales there were 159 QRNG (14.4%), in South Australia 24 (10.6%), Western Australia 30 (9.2%) and in Queensland 69 (11.1%). In other jurisdictions the numbers of QRNG were low (Northern Territory, 6; Tasmania, 3; Australian Capital Territory, 1).

Information on acquisition of QRNG was available in 110 of the 529 cases. In New South Wales, 41 infections were acquired locally and 41 overseas, but in Western Australia only seven of 28 cases were acquired locally. Overseas acquisition was from many sources. In addition to the countries listed above for PPNG acquisition, QRNG were acquired from contacts in Malaysia 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 upsurge in TRNG isolation in 2002 when 442 (11.4%) strains of this type were detected nationally. In 2003, the number (411) and proportion (11.2 %) of TRNG detected throughout Australia was little changed. Most TRNG were found in New South Wales (125, 11.3% of isolates). Western Australia (79, 24%) again had the highest proportion of TRNG. The proportion of TRNG in Queensland (69 isolates, 11.1%) and Victoria (106 isolates, 11.6%) was similar to that in New South Wales. Lower numbers were found in South Australia (17) and the Northern Territory (11) and single isolates of TRNG were found in Tasmania and the Australian Capital Territory.

Discussion

The examination of the susceptibility of gonococci is undertaken principally to determine the patterns of susceptibility of prevalent gonococci to those antibiotics currently recommended for single-dose treatment. The WHO recommends that once resistance to an antibiotic has reached a level of five per cent, then use of that agent should be discontinued. Before surveillance can be reliably performed, a number of technical obstacles that arise from the fastidious nature of the gonococcus need to be overcome. These include the need for specialised isolation and testing methods and that a sufficient number of isolates are examined. The AGSP adopted standardised methods from its inception and conducts a program-specific quality assurance survey. The number of isolates available in Australia in 2003 remained sufficient for the purpose of detecting resistance at the five per cent level. The limitations of culture-based diagnosis, especially in remote settings, decreases the number of gonococci available for testing so that the AGSP currently examines all isolates available to its members, rather than a sample of these isolates. The increasing use of non-culture based methods for the diagnosis of gonorrhoea also decreases the number of gonococcal isolates available for testing. A continuing commitment to maintenance of culture-based systems is required for the purposes of AMR surveillance, while molecular methods for gonococcal susceptibility testing remain problematic.[9]

The wider introduction of nucleic acid amplification testing for diagnostic purposes 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 2003, although the overall number of isolates was little altered from previous years, a change in the number of isolates in New South Wales and Victoria was notable. While the number of isolates from New South Wales, which had progressively increased over a number of years, declined in 2003 to 1,100 from a peak of 1,600 in 2002, in Victoria the number of gonococci examined increased to 900 from the 700 seen in both 2002 and 2001. As a consequence, the New South Wales gonococci decreased as a proportion of all Australian isolates from 40 per cent in 2002 to 30 per cent in 2003 while the Victorian percentage increased from 18 per cent to 25 per cent. The reasons for these alterations in numbers in New South Wales and Victoria remain uncertain. In both States, gonorrhoea continued to be concentrated in homosexually active males. In New South Wales the ratio of isolates from men and women was unaltered and rectal and pharyngeal isolates from men were again prominent—27 per cent of all male isolates in both 2003 and 2002. In Victoria, the ratio of isolates from men and women increased to 16.7:1 from 10.5:1 in 2002 and the proportion of rectal and pharyngeal isolates in men also increased from 23 per cent to 28 per cent.

Considerable regional variation in susceptibility of gonococci to antibiotics was again observed in Australia in 2003. Previous AGSP data has demonstrated that antibiotic resistant gonococci are more prevalent in larger urban centres than in rural centres.[4] Because of these pronounced regional differences in patterns of AMR in gonorrhoea in Australia, standard treatment regimens are best derived from a consideration of local patterns of susceptibility rather than aggregated national data.

Oral penicillins remain the mainstay of treatment in rural settings in a number of jurisdictions and patterns of resistance to the penicillins altered little in 2003. In the Northern Territory, both CMRNG and PPNG rates were low. In contrast, penicillin resistance continues at a high rate in urban centres in 2003 and penicillin resistance in New South Wales, Victoria, South Australia, Queensland and Western Australia ranged between 10 and 27 per cent. In Victoria and New South Wales, most of this resistance was chromosomally mediated, but in Western Australia, South Australia and Queensland PPNG were prominent.

Patterns of quinolone resistance have shown considerable volatility over a number of years. Until 1999, QRNG were particularly concentrated in homosexually active males in New South Wales and Victoria and the QRNG were predominantly in the lower MIC range (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 trend to higher levels of MICs in QRNG 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). In 2003, a further upward shift in quinolone resistance in gonococci was observed. More QRNG were detected (14.5% of all gonococci examined) and one in eight of all gonococci in Australia were resistant at an MIC of 1 mg/L or more of ciprofloxacin. Most of this increase in QRNG occurred in Victoria. The number of QRNG there increased from 98 to 237 and QRNG as a proportion of all gonococci doubled in 2003. Smaller increases in the numbers of QRNG detected occurred in South Australia, New South Wales and Queensland, with a slight decline in Western Australia. Acquisition data on QRNG were difficult to obtain, but sustained domestic transmission remained important in New South Wales. There was a significant rate of acquisition of QRNG from countries close to Australia where rates of QRNG remain very high.[10] Use of quinolones, including more recently available members of this group, are unsuitable for the treatment of individuals who acquire gonorrhoea overseas.

Repeated attention has been drawn to the appearance of gonococci with decreased susceptibility to third generation cephalosporin antibiotics in recent AGSP reports. While it is emphasised that no treatment failures have been documented in Australia with gonorrhoea treated with ceftriaxone when a 250 mg dose was used, reports from Japan have confirmed treatment failures with other oral third generation agents including cefixime.[11] Laboratory data indicated that these treatment failures in Japan were accompanied by increased MICs with mosaic *penA* genes in the gonococci.[3] These gonococci were also resistant to penicillins and quinolones and their presence has been reported in regional surveys and elsewhere.[10,12] The AGSP uses the Japanese isolates for comparative purposes. Gonococci encountered in Australia have characteristics similar to those reported in Japan. In 2003 these isolates were 10 in number from apparently sporadic cases and MICs did not increase over the values seen in 2001 and 2002. 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 a National Neisseria Network laboratory.

All gonococci tested in Australia in 2003, including those with altered cephalosporin susceptibility, were susceptible to spectinomycin.

There is little cause for complacency in the current AGSP data. The incidence of antibiotic resistance in *N. gonorrhoeae* shows no evidence of a decline, and resistance to commonly used agents is at levels that require continuing surveillance to ensure that optimal antibiotic treatment is available for both individual case management and disease control.

Acknowledgements

Participating laboratories in the AGSP (to whom isolates should be referred):

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The AGSP thanks the Australian Government Department of Health and Ageing for continued financial support and the many laboratories, private and public, throughout Australia for submission of isolates for testing.

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Annual report of the Australian Meningococcal Surveillance Programme, 2003

The Australian Meningococcal Surveillance Programme

Abstract

This tenth report by the National Neisseria Network, a nation-wide collaborative laboratory program, describes 494 laboratory-confirmed cases of meningococcal disease in Australia, diagnosed in 2003. The phenotypes (serogroup, serotype and serosubtype) and antibiotic susceptibility of 303 isolates of *Neisseria meningitidis* from invasive cases of meningococcal disease were determined, and an additional 191 cases were confirmed by non-culture-based methods. The age distribution of invasive meningococcal disease showed a typical primary peak in those aged four years or less which was predominantly serogroup B meningococci. A secondary peak in adolescents and young adults contained a larger proportion of serogroup C infections. Nationally, the majority of isolates were serogroup B (183 isolates, 60.4%) or serogroup C (102 isolates, 33.6%) meningococci. The number of serogroup C isolates in Victoria decreased from 72 in 2002 to 33 in 2003 and in Tasmania the number of serogroup C isolates decreased from 14 to five. Smaller decreases in serogroup C isolate numbers were recorded in most other jurisdictions but the number increased in the Australian Capital Territory from four to seven isolates. Serogroup B isolate numbers also decreased nationally but by a smaller amount. However in South Australia serogroup B infections more than doubled, and there were also increases in the Northern Territory and Australian Capital Territory. The serogroup C phenotype C:2a:P1.4 remained prominent in Victoria but elsewhere in Australia it was detected only in low numbers. About two thirds of all isolates showed decreased susceptibility to the penicillin group of antibiotics (MIC 0.06 to 0.5 mg/L). A single isolate from the Australian Capital Territory was penicillin resistant at 1 mg/L and two, one each from South and Western Australia were rifampicin resistant. *Commun Dis Intell* 2004;28:194–206.

Keywords: antibiotics, antimicrobial resistance, cephalosporin, ceftriaxone, ciprofloxacin, penicillin, Neisseria meningitidis*, meningococcal, surveillance*

Introduction

The National Neisseria Network (NNN) is a collaborative national program of reference laboratories in each state and territory of Australia. NNN provides surveillance data relevant to the public health control of invasive meningococcal disease (IMD), namely diagnosis, antimicrobial resistance surveillance, and organism typing, including both isolate-based and non-culture derived methodologies. The first reports from the meningococcal surveillance program, which began in 1994, relied on data derived from examination of isolates from culture-positive cases of IMD, in particular, their phenotype and antibiotic susceptibility. Increasingly, data derived from non-culture-based methods, notably the genotype and diagnoses based on nucleic acid amplifications assays (NAA), have been included. The information is provided to supplement that from clinical notification schemes.

During 2003, a publicly funded program to vaccinate children and adolescents with serogroup C conjugate vaccine, commenced in Australia, but at different times in different States and Territories. This report analyses information gathered by the NNN on laboratory-confirmed cases of IMD in the calendar year 2003. The format of previous annual reports published in *Communicable Diseases Intelligence*[1–9] has been followed for comparative purposes, but this report includes additional data on IMD diagnosed by non-culture methods.

Methods

The NNN is a long term collaborative program for the laboratory surveillance of the pathogenic *Neisseria*, *Neisseria meningitidis* and *N. gonorrhoeae*.[1–10] A network of reference laboratories in each state and territory (see acknowledgements) performs and gathers laboratory data on cases of IMD 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 (porin) 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 has 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.[10]

sensitive	MIC $\leq$ 0.03 mg/L;
less sensitive	MIC 0.06 – 0.5 mg/L;
relatively resistant	MIC $\geq$ 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

Increasingly, additional laboratory confirmation of suspected cases of IMD is made available by means of non-culture-based methods including NAA and serological techniques. NAA testing is essentially by polymerase chain reaction techniques[11] 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 Public Health Laboratory Service reference laboratory, United Kingdom, as assessed for Australian conditions.[12–14] Where age, sex and outcome data for patients with non-culture-based diagnoses are available these were also recorded. The site of a sample of a positive NAA is also used to define the clinical syndrome. This separation is not possible for cases diagnosed serologically.

Results

Number of isolates from culture-confirmed cases

A total of 303 invasive isolates of meningococci were examined in 2003, 90 less than the 393 examined in 2002, and 35 less than the 338 seen in 2001. The decrease in the number of isolates in 2003 was principally due to the lower number of culture positive cases in Victoria where there has been considerable volatility in the number of isolates in recent years. In 2001 in Victoria, there were 77 culture positive isolates, and 129 in 2002, decreasing again to 69 (22.6% of all isolates in Australia) in 2003. The number of isolates in Tasmania (10, 3%) was half that recorded in 2002. Smaller decreases in numbers were also seen in New South Wales (93, 30%, from 110 in 2002), Western Australia (25, 9%, from 35) and Queensland (69, 20%, from 76). The number of culture-positive cases in the Australian Capital Territory (11, 3%) and the Northern Territory (7, 2%) both increased from the five seen in each jurisdiction in 2002 and in South Australia the number almost doubled from 13 to 25, (9%) (Table 1).

Seasonality

Fifty-six (19%) of cases occurred between 1 January and 31 March, 57 (19%) between 1 April and 30 June, 111 (36%) between 1 July and 30 September and 79 (26%) between 1 October and 31 December. A winter peak of meningococcal disease is usual.

Table 1. *Neisseria meningitidis* isolates, Australia, 2003, by serogroup and state or territory

State or territory	Serogroup										Total	
	B		C		A	Y		W135		NG*		
	n	%	n	%	n	n	%	n	%	n	n	%
ACT	3	27	7	63	0	0	0	1	10	0	11	3.4
NSW	61	66	25	27	0	5	5	1	1	1	93	30.4
NT	6	86	0		0	0		1	14	0	7	2.3
Qld	33	52	28	44	1	1	4	0		0	63	20.6
SA	20	80	2	8	0	1	4	1	4	1	25	9.2
Tas	5	50	5	50	0	0		0		0	10	3.3
Vic	33	48	33	48	0	2	3	1	1	0	69	22.6
WA	22	88	2	8	0	1	4	0		0	25	8.2
Australia	183	60	102	34	1	10	3	5	1.5	2	303	100

* Not viable for serogrouping or not serogroupable.

Age group

The age distribution of patients infected with invasive isolates in each state and territory is shown in Table 2. Nationally, the peak incidence of meningococcal disease was again in those aged four years and under. Those aged less than one year or in the 1–4 year age group accounted for 40 (13.2%) and 53 (17.5%) cases respectively. The combined total of culture positive cases in these two groups (93) is less than that in 2002 (108). However, these two age groups comprised a slightly higher proportion of all cases in 2003. A secondary disease peak is also usual in the 15–19 year age group. The total of 52 cases (17.1%) in this age group in 2003 was substantially less than the 95 (24.2%) seen in 2002, but almost the same as the 54 cases (16%) recorded in 2001. The total of 35 cases (11.5%) in those aged 20–24 years was the same proportion as the 45 cases seen in 2002. Those aged 15–24 years together accounted for 87 (28.6%) cases, that is, almost the same proportion as the four years and under age group.

Serogroup, serotype and serosubtype (phenotype) distribution

The distribution of the isolates by serogroup is shown in Tables 1 and 2. Nationally, 183 serogroup B isolates represented 60.4 per cent of all strains, lower in number, but an increase in the proportion of all cases when compared with 2002 data (210 cases, 53.5%). The 102 serogroup C strains isolated (33.6% of all cases) was a substantial decrease in both the number and proportion seen in recent years. In 2002, there were 162 serogroup C cases (41.2% of all culture positive IMD), while in 2001 there were 122 serogroup C isolates (36%) detected and in 2000, 128 (33%) isolates. The other culture positive cases were serogroup W135 (5, 1.6%), serogroup Y (10, 3.3%) and a single case of serogroup A IMD from Queensland.

When data were disaggregated by state and territory, some differences in disease distribution were again noted and changes in patterns observed in recent years were recorded. Serogroup B meningococci predominated in Western Australia (22, 88%), the Northern Territory (6, 86%), and New South Wales (61, 66%). While these proportions differ little from data obtained in 2002, the number of strains isolated was less in 2003. In South Australia, serogroup B isolates also predominated with an increased number of 20 strains representing 80 per cent of all meningococcal isolates. In Victoria (33 isolates, 48%) and Tasmania (5, 50%) the number of serogroup B strains isolated decreased in 2003 but was higher as a proportion of all meningococci grown. In Queensland, both the number (33) and proportion (52%) of serogroup B strains detected decreased.

The number of serogroup C isolated nationally decreased from 162 in 2002 to 102 in 2003 and as a proportion of all culture positive cases of IMD from 41 per cent to 34 per cent. The largest reduction in the number of serogroup C isolates was in Victoria where 33 strains were isolated in 2003 and 72 in 2002. Serogroup C strains were 48 per cent of all Victorian isolates in 2003 and 55 per cent in 2002. The number of serogroup C strains also decreased in all other jurisdictions except the Australian Capital Territory. In the Australian Capital Territory the number of strains rose from four in 2002 to seven in 2003. The other notable reduction in the number of serogroup C isolates was in Tasmania, from 14 isolates in 2002 to five in 2003. In other states, the reduction in serogroup C isolates from 2002 figures was more modest. In New South Wales the 25 isolates (27%) were nine fewer than in 2002 and in Queensland, the Northern Territory, South Australia and Western Australia the reduction in the number of serogroup C isolates from 2002 was one or two only.

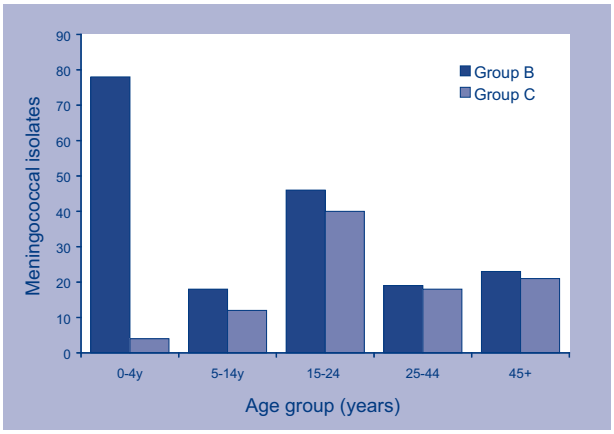
Table 2. Serogroup B and C meningococcal isolates, Australia, 2003, by state or territory

State or territory		Age group (years)										Total
		<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	NS	
ACT	Total	0	1	1	2	4	0	0	2	1	0	11
	B	0	0	1	1	0	0	0	1	0	0	3
	C	0	1	0	1	4	0	0	1	0	0	7
NSW	Total	18	20	5	3	14	5	11	8	9	0	93
	B	15	19	2	2	7	1	6	6	3	0	61
	C	3	1	3	1	5	4	5	1	2	0	25
NT	Total	2	1	1	0	0	0	2	1	0	0	7
	B	2	1	1	0	0	0	1	1	0	0	6
	C	0	0	0	0	0	0	0	0	0	0	0
Qld	Total	8	3	4	4	14	10	8	8	4	0	63
	B	8	1	2	1	8	4	5	3	1	0	33
	C	0	2	1	2	6	6	3	5	3	0	28
SA	Total	3	7	0	2	5	3	2	0	3	0	25
	B	2	7	0	2	4	1	2	0	2	0	20
	C	0	0	0	0	0	2	0	0	0	0	2
Tas	Total	0	3	1	0	1	1	3	0	0	1	10
	B	0	1	0	0	1	1	1	0	0	1	5
	C	0	2	1	0	0	0	2	0	0	0	5
Vic	Total	6	8	3	4	10	14	10	10	4	0	69
	B	4	6	2	2	6	7	2	2	2	0	33
	C	1	2	1	2	4	6	8	7	2	0	33
WA	Total	3	10	2	0	4	2	2	1	1	0	25
	B	3	9	2	0	2	2	2	1	1	0	22
	C	0	0	0	0	2	0	0	0	0	0	2
Australia	n	40	53	17	15	52	35	38	30	22	1	303
	%	13.2	17.49	5.6	4.9	17.1	11.55	12.54	9.9	7.26	0.33	
Serogroup B Australia	n	34	44	10	8	28	16	19	14	9	1	183
	%	18.5	24.04	5.56	4.37	15.3	8.79	10.38	7.65	4.91	0.54	60.39
Serogroup C Australia	n	4	8	6	6	21	18	18	14	7	0	102
	%	3.92	7.84	5.88	5.88	20.58	1.64	17.64	13.72	6.86	0	33.66
Other Australia	n	2	1	1	1	3	1	1	2	6	0	18
												5.94

NS Not stated.

Serogroup distribution was typically age-associated, with serogroup B disease concentrated in younger age groups and serogroup C infections increasing as a proportion of all isolates in adolescents and young adults (Table 2). In 2003, 78 of 93 (84%) isolates in those aged less than four years were serogroup B and the 12 serogroup C isolates comprised 13 per cent of cultures nationally in this age group (Figure 1). In those aged 5–14 years, 18 serogroup B meningococcal cultures represented 56 per cent of the 32 isolates and the 12 serogroup C strains 38 per cent. There were 87 isolates in those aged 15–24 years in 2003 of which 44 (50.5%) were serogroup B and 39 (44.8%) were serogroup C. In 2002, there were 140 isolates in this age group of which 60 (43%) were serogroup B and 73 (52%) were serogroup C. In older age groups serogroup B (42) and serogroup C (39) isolates were in about equal numbers nationally.

Figure 1. Number of serogroup B and C isolates, Australia, 2003, by age



Jurisdictional differences in the distribution of serogroup B and C meningococcal isolates were also evident in 2003 with notable changes in the 15–24 year age groups when compared with 2002 data (Table 2). In all centres except Tasmania and the Australian Capital Territory serogroup B markedly predominated in those aged four years or less. In Western Australia, South Australia and the Northern Territory, serogroup B isolates predominated in most age groups. In New South Wales, serogroup B was especially prominent in these younger age groups in that only four of 38 cases were of serogroup C. A distinct shift in the number and proportion of serogroup C isolates was observed in the adolescent and young adult (15–24 year) age group in Victoria. In 2002, 35 of 56 (62%) isolates in this age group were serogroup C whereas in 2003, 10 of 24 (42%) isolates were serogroup C. In Queensland however, the number and proportion of serogroup B and C isolates in the 15–24 year age group was identical in both years.

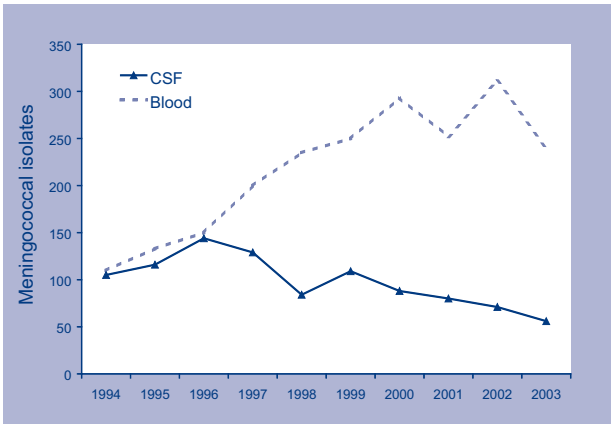
Considerable phenotypic heterogeneity amongst invasive isolates was again present. The predominant serotypes/serosubtypes in each state and territory are shown in Table 3. Serogroup B meningococci are in general more heterogeneous, but also more difficult to characterise by serological methods and a number could not be phenotyped. Twenty-five isolates of the B:4:P1.4 phenotype were identified in Victoria, New South Wales, Queensland and the Australian Capital Territory. Historically, the other common phenotype circulating has been B:15:P1.7 but only nine strains of this type were seen, seven of them in New South Wales. Of interest were four B serotypes of 2a or 2b, all with a separate serosubtype. These serotypes are more often seen in serogroup C organisms. If these subtypes become more common, extensive genotypic investigation would be warranted.

All except one of the typeable serogroup C isolates was of serotype 2a. Phenotype C:2a:P1.4, prominent in Victoria in recent years, was again commonly seen and 29 of the 33 serogroup C strains were of this phenotype. There were 10 such isolates in Victoria in 1999, 24 in 2000, 19 in 2001 and 55 in 2002. The same phenotype was also seen in most other jurisdictions. In the Australian Capital Territory and Tasmania this subtype was seen exclusively in those strains fully phenotyped, but was only found infrequently in Queensland and New South Wales. Serotype 2b strains were rare.

Site of isolation

There were 55 isolates from CSF either alone or with a blood culture isolate and 238 from blood cultures alone. There were seven isolates from synovial fluid and one each from skin, eye and peritoneum. 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, the same as in 2002 but lower than that recorded in preceding years.

Figure 2. Numbers of meningococcal isolates from cerebrospinal fluid and blood culture, Australia, 1994 to 2003



Outcome data for cases with sterile site isolates

Outcome data (survived or died) were available for 214 of the 303 patients from whom isolates were obtained (71%). Sixteen deaths were recorded in this group (7.5%) (Table 4). Outcomes were available for 123 (67%) serogroup B infections and 76 (75%) serogroup C infections. There were 5 (4%) deaths in serogroup B infections and 11 (14.4%) in serogroup C infections.

Table 3. Commonly isolated serotypes and serosubtypes and phenotypes of *Neisseria meningitidis* of interest, 2003, by state or territory

State or territory	Serogroup B				Serogroup C			
	Serotype	n	Serosubtype	n	Serotype	n	Serosubtype	n
ACT	4	1	1.4	1	2a	7	1.4	6
NSW	4	17	1.4	8	2a	20	1.5	8
			1.6,3	3			1.5,2	3
			1.14	2			1.2	2
			nst**	4			1.4	3
	2a	3	1.2	1			nst	4
			1.5	1	2b	1	1.5,2	1
			1.5,2	1	1	2	1.15	1
	15	9	1.7,(16)	7	NT	3	1.15	2
	1	7	1.14	2			1.5,2	1
			1.7	1				
	nt*	24	4	4				
			1.14	4				
			1.15	2				
			1.7	1				
			nst	12				
NT	14	4	nst	4				
Qld	4	7	1.4,(7)	6	2a	23	1.5	13
			nst	1			1.2	1
	1	5	1.4,(7)	2			1.4	3
	14	2	1.5	1			nst	4
			nst	1	nt	5	1.5	3
	15	2	1.7	1			nst	2
			nst	1				
	nt	16	1.4,(7)	6				
			1.15	2				
			nst	5				
SA	1	5	1.14	3	2a	2	nst	1
			1.7	2				
	15	3	1.4	1				
			1.7	1				
	nt	9	1.4	1				
			1.14	3				
			1.15	2				
Tas	ND	5			2a	4	1.4	4
Vic	4	12	1.4	10	2a	33	1.4	29
			1.5,2	1			1.5,10	3
			1.16	1			1.14	1
	14	2	1.14	2				
	15	2	1.4	1				
	2b	1	1.16	1				
	nt	16	1.15	8				
			1.14	3				
			nst	4				

Table 3. Commonly isolated serotypes and serosubtypes and phenotypes of *Neisseria meningitidis* of interest, 2003, by state or territory, continued

State or territory	Serogroup B				Serogroup C			
	Serotype	n	Serosubtype	n	Serotype	n	Serosubtype	n
WA	1	2	1.1,7	1	2a	2	1.4	1
			1.5	1			nst	1
	14	1	nst	1				
	15	1	1.7	1				
	nt	16	1.4	6				
			1.5	1				
			1.15	1				
			nst	8				

nt Not serotypeable.

nst Not serosubtypeable.

Table 4. Outcome data (survived, died) for culture positive cases of invasive meningococcal disease, 2003, by syndrome and serogroup

Disease type	Outcome	Serogroup					Total
		B	C	Y	W135	NG	
Meningitis	Survived	23	12	2	0	0	37
	Died	2	0	0	0	0	2
	Total	25	12	2	0	0	39
Septicaemia	Survived	93	50	5	5	2	155
	Died	3	11	0	0	0	14
	Total	96	61	5	5	2	169
All cases	Survived	118	65	8	5	2	198
	Died	5	11	0	0	0	16
	Total	123	76	8	5	2	214

NG Not groupable.

There were two deaths in 39 patients (5%) with meningitis where outcome was recorded. Both of these patients were infected with a serogroup B strain. Fourteen deaths were recorded in 169 bacteraemic patients (8.2%). There were 96 cases of serogroup B meningococcal bacteraemia with three deaths (3.1%) and 61 cases were caused by serogroup C strains among whom 11 fatalities were recorded (18%). No fatalities were recorded with serogroup Y (5 cases) or W135 (5 cases) bacteraemia.

Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillin

Three hundred isolates were available for determination of their susceptibility to penicillin. Using defined criteria, 99 strains (33%) were fully sensitive to penicillin and 200 (67%) less sensitive (MIC 0.06 to 0.5 mg/L). These proportions are similar to those observed in recent years. One isolate from a blood culture had an MIC of 1 mg/L and six isolates had MICs of 0.5 mg/L.

Other antibiotics

All isolates were susceptible to ceftriaxone (and by extrapolation to other third generation cephalosporins) and to ciprofloxacin. A single isolate from South Australia was rifampicin resistant at an MIC of 32 mg/L and another from Western Australia displayed high level resistance.

Number and sources of non-culture diagnoses of invasive meningococcal disease in 2003

One hundred and ninety-one additional cases of IMD were diagnosed by non-culture methods in 2003. This is a similar number to that diagnosed by these means in 2002. One hundred and sixty-nine cases were diagnosed by NAA (Table 5) and another 22 solely by serology using enzyme immunoassay (EIA).

With NAA, it was also possible to categorise the disease type by source of specimen in a manner similar to that used for culture positive cases. Of the 169 cases positive by NAA, 75 were from CSF or CSF and blood, 91 from blood only and three from joint fluid. This is a different IMD syndrome 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 NAA based diagnosis, the ratio of positive diagnoses from blood to CSF was 1.2:1. The site of infection could not be determined by laboratory testing in serologically diagnosed cases.

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

In addition to diagnostic NAA, molecular techniques can also be used to ascertain the serogroup involved in the disease process. In most centres this is still limited to serogroup B and C determinations. Of the 169 cases where a NAA-based diagnosis was made, a serogroup was also determined in 145 instances (Table 5); 101 cases diagnosed by NAA were with serogroup B, 43 with serogroup C and one with W135. An additional EIA can also be performed to identify serogroup C infection serologically. Eight of the 22 serologically identified cases were confirmed as serogroup C.

The serogroup and age distribution of NAA diagnosed cases is shown in Table 6. Nationally, the number of cases in children aged less than four years was 44 (26%) a lower proportion than that diagnosed by culture-based procedures (30.3%). In 40 of these cases, the serogroup was determined and 38 were serogroup B infections. The proportion of cases diagnosed by NAA testing in the 15–24 year age group (31.3%) was slightly higher than the proportion diagnosed by culture-based methods (29.4%) in the same group. Using NAA, 56 cases were detected in this age group. However, the proportion of serogroup C (n=24) cases found equalled the number of serogroup B (n=25) cases detected. Eight of the 22 serologically-confirmed cases were in the 15–24 year age group and two were in those aged four years or less.

Table 5. Nucleic acid amplifications test-based diagnosis of invasive meningococcal disease, Australia 2003, by serogroup and state or territory

State or territory	Serogroup				Total
	B	C	W135	ND	
ACT	0	3	0	0	3
NSW	42	13	0	5	60
NT	5	0	0	0	5
Qld	15	7	0	7	29
SA	6	0	0	0	6
Tas	3	3	0	3	9
Vic	20	14	1	9	44
WA	10	3	0	0	13
Total	101	43	1	24	169

ND Not determined.

Table 6. Nucleic acid amplifications test-based diagnoses of invasive meningococcal disease, Australia, 2003, by age, serogroup and jurisdiction

State or territory	Serogroup	Age group										Total
		<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	NS	
ACT	C					1			2			3
	Total					1			2			3
NSW	B	4	12	2		8	5	1	5	5		42
	C			2	2	4	2	3				13
	Total	4	12	6	2	12	7	6	6	5		60
NT	B	2	2				1					5
	Total	2	2				1					5
Qld	B	2	2	1	3	3	2	1	1			15
	C			1		2	4					7
	Total	3	3	2	4	7	8	1	1			29
SA	B		1		3		1		1			6
	Total		1		3		1		1			6
Tas	B		1			1			1			3
	C					1		1	1			3
	Total		1	2	1	2		1	2			9
Vic	B	4	1	3	2	3		5	2			20
	C		2		2	3	4	2		1		14
	Total	4	5	4	4	7	6	11	2	1		44
WA	B		7	1		1		1				10
	C					1	2					3
	Total		7	1		2	2	1				13
Australia	B	12	26	7	8	16	9	8	10	5		101
	C		2	3	4	12	12	6	3	1		43
	Total	13	31	15	14	31	25	20	14	6		169

NS Not stated (totals include other serogroups).

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

For IMD diagnosed by NAA based tests, the outcome was known in 86 instances, with eight deaths recorded (9.3%). There were five deaths (4 of serogroup C and one of serogroup B) (14%) in the 35 cases where the CSF examined was positive by NAA. Three deaths where NAA was positive only on a blood sample were recorded out of a total of 48 cases where outcome data was available. Serogroup C was identified in two cases and serogroup B in one. Of the 78 cases where survival was recorded, the diagnosis was made by NAA using CSF samples in 30 instances. Twenty-four of these were due to serogroup B infections and five to serogroup C organisms. Forty-five cases who survived were diagnosed as having IMD using NAA with a blood sample. Twenty-four infections were with serogroup B, nine with serogroup C meningococci with a single case of W135 disease. The other cases where the serogroup was determined and survival was recorded were three cases of septic arthritis, two with serogroup C and one with serogroup B. The serogroup was not determined in 12 cases where survival was recorded.

Combined data for all laboratory-confirmed IMD by age, jurisdiction and serogroups B and C are shown in Table 7.

Table 7. All laboratory-confirmed cases of invasive meningococcal disease, Australia 2003, by age, jurisdiction and serogroup

State or territory	Serogroup	Age group										Total
ACT	B	0	0	1	1	0	0	0	1	0		3
	C	0	1	0	1	5	0	0	3	0		10
	Total	0	1	1	2	5	0	0	4	1		14
NSW	B	19	31	4	2	15	6	7	11	8		103
	C	3	1	7	3	11	7	8	2	2		44
	Total	23	33	13	6	31	13	21	15	15		170
NT	B	4	3	1			1	1	1			11
	C											0
	Total	4	3	1			1	2	1			12
Qld	B	10	3	3	4	11	6	6	4	1		48
	C		2	2	2	9	10	3	6	3		37
	Total	11	6	6	8	22	18	11	10	4		96
SA	B	2	8		5	4	2	2	1	2		26
	C						2					2
	Total	3	8		5	5	4	2	1	3		31
Tas	B		2			2	1	1	1		1	8
	C		2	1		1		3	1			8
	Total		4	3	1	3	2	4	2		1	20
Vic	B	8	7	5	4	9	7	7	4	2		53
	C	1	4	1	4	7	10	10	7	3		47
	Total	10	14	7	8	17	20	21	12	5		114
WA	B	3	16	3		3	2	3	1	1		32
	C					3	2					5
	Total	3	17	3		6	4	3	1	1		38
Australia	B	46	70	17	16	44	25	27	24	14	1	284
	C	4	10	11	10	36	31	24	19	8		153
	Other	4	6	6	3	9	6	13	3	7		57
	Total	54	86	34	29	89	62	64	46	29	1	494

NS Not stated (totals include other serogroups).

Discussion

There were 494 laboratory-confirmed cases of IMD in 2003, 303 (61.3%) by culture and 191 (38.7%) by non-culture-based methods. The 303 isolates examined by NNN laboratories in the Australian Meningococcal Surveillance Programme in 2003 was the lowest number recorded since 1996. The 393 isolates recovered in 2002 was the highest number examined in any year by the NNN. The annual numbers of isolates examined from 1997 to 2002 have ranged between 323 and 388. In the 2002 report, specific mention was made of the changes in the number of isolates from Victoria in recent years. The 41 isolates in 1998 increased to 94 in 1999 and to 108 in 2000, declined to 77 in 2001 only to increase to 129 in 2002. In 2003, a substantial reduction to 69 isolates was recorded and this accounted for most of the national reduction in numbers from the 2002 figure. Smaller decreases in numbers were noted in New South Wales (17), Western Australia (12) and Queensland (7). Isolate numbers more than doubled in the Australian Capital Territory and in South Australia and also increased in the Northern Territory.

The considerable fluctuation in isolate numbers both nationally and by jurisdiction means that data on isolation rates must be examined with caution if they are applied to determine trends in disease rates. The NNN has consistently pointed to reasons for differences in the number of isolates available for examination and the number of clinically notified cases. Clinical surveillance case definitions

allow for inclusion of culture negative cases under certain criteria and this number is also influenced by the 'early treatment' practices advocated for management of IMD. Since 1999, the NNN has included data on non-culture-based testing for IMD. The use of these tests has increased progressively, however the introduction and uptake of non-culture-based diagnostic methods has varied in different jurisdictions over time and new forms of NAA with increased sensitivity have been introduced. As a result, the diagnostic basis for IMD confirmation has progressively altered. The total of 191 instances of non-culture-based diagnoses in 2003 was similar to the 187 cases confirmed by these means in 2002. However in 2003, the proportion of all cases confirmed by culture declined to 61 per cent from the 68 per cent in 2002. A further potential bias in the data arises in the different sensitivity of various test methods and the influence of clinical practice. The ratio of cases of meningitis to those of bacteraemia in culture-confirmed cases is one example. Figure 2 shows trends in this ratio over a number of years. Currently, culture-confirmed cases of meningitic IMD are one quarter of bacteraemic cases. In contrast, when NAA were first introduced, cases were confirmed as positive from CSF samples at 2.5 times the rate of diagnoses from blood. In 2002 and now in 2003, this ratio has been reversed with NAAT on blood yielding 1.2 times the rate of diagnoses from CSF. Contributing to these trends have been the increasing use of appropriate blood samples for NAA and the reluctance of clinicians to perform lumbar puncture early in the disease process.

In earlier NNN reports, analyses have concentrated on data derived from culture-confirmed cases. The increasing use of NAA has been recognised in this report by inclusion of Table 6 which shows a breakdown of NAA diagnoses by jurisdiction, age and serogroup. Table 7 includes the same information, but shows all laboratory-confirmed cases. The data on culture-based cases alone (Table 2) continue to be included for longer-term comparative purposes. Some differences are noted when parameters derived from culture-based and non-culture-based cases are compared. A primary disease peak is usual in those aged less than four years with a secondary peak in those aged 15–24 years. In 2003, 31 per cent of cases confirmed by culture were in the younger age group and 29 per cent in the older age group. In those cases confirmed by NAA, 20 per cent were aged four years or less and 33 per cent aged 15–24 years. For all laboratory-confirmed cases, 28 per cent were in the 0–4 year age group and 30.5 per cent occurred in the older age group. Some differences were also noted in the relative proportions of serogroup B and C disease diagnosed by different methods. Serogroup B cases were 60 per cent of all cases diagnosed by both culture and NAA, but serogroup C cases diagnosed by culture were 33 per cent of all diagnoses by this method but 25 per cent of all NAA diagnoses. The final example of differences in data due to the diagnostic test method used occurs in the proportion of serogroup B and C disease recorded by age. In those aged 15–24 years, the ratio of serogroup B to serogroup C disease differs little with infection due to both serogroups in similar proportions irrespective of test method. However in the younger age group, serogroup B isolates were cultured 6.5 times more often than serogroup C meningococci whereas with NAA, serogroup B disease was diagnosed 19 times as often as serogroup C infection.

Irrespective of these differences, the predominant disease pattern throughout the country was with serogroup B meningococci (284 cases, 57.4%) and 153 (31%) serogroup C cases (Table 7). There has also been a substantial reduction in the number of serogroup C infections, most notably in Victoria. It is tempting to attribute these changes to the effect of vaccination programs with serogroup C conjugate vaccines. This type of analysis is beyond the scope of this report. As well as the limitations of the data presented here, it should be remembered that fluctuations in the rates of IMD can occur naturally. In Victoria there was also a smaller decrease in the number of serogroup B meningococci cultured and in New South Wales, Western Australia and Queensland the reduction in numbers of serogroup B isolated was greater than the decrease in numbers of serogroup C isolates. Any assessment of the impact of the vaccination program on IMD rates will thus require a continuing and detailed analysis. Other important considerations in these analyses include the possibility of replacement of serogroup C strains by other serogroups by 'vacuum filling'. In South Australia in 2003, the number of serogroup B infections more than doubled. A further concern is the ability of meningococci to undergo genetic recombination. A limited number of clonal complexes are responsible for most IMD globally. The ET–37 clonal complex or its genetic variant ET–15 for example, comprise related strains that may express a serogroup B, C, Y or W135 capsule. The possibility that a switch from serogroup C to other sialic acid containing capsular types may occur is well documented and it would seem that a small number of such isolates were again present in Australia in 2003. While a multivalent polysaccharide conjugate vaccine containing A, C, Y and W135 antigens is close to release, there is no realistic prospect of a serogroup B capsule vaccine. Rather, vaccination strategies for serogroup B meningococci are currently concentrated on porin subtype vaccines such as those currently under trial in New Zealand. For this reason close attention must be paid to analysis of serogroup B subtypes and any evidence of their clonal expansion. In addition, meningococcal heterogeneity may arise from variation within the *porA* and *porB* genes or, separately, from their recombination. The typing data contained

in this and earlier reports suggests that these phenomena are also occurring and warrant continued monitoring.

Recent NNN reports noted that the age distribution of IMD showed a primary peak in those aged four years or less and was predominantly with serogroup B meningococci while the secondary peak in adolescents and young adults contained a larger proportion of serogroup C infections. This pattern was again observed in 2003, irrespective of the diagnostic method by which the data were generated.

Mortality data were assessable in only a proportion of cases and must be interpreted with caution. Excess mortality has been linked to serogroup C infections in more complete studies and has been consistently recorded in NNN data. The NNN does not attempt collection of morbidity data associated with IMD.

A penicillin MIC of 1 mg/L was detected in a single strain in 2003. Outcome/MIC correlates are difficult to establish in IMD because of the fulminant nature of fatal cases of meningococcal disease. Additionally, methodological differences may significantly alter the MIC value reported so that little guidance for Australian conditions is available from overseas studies. Meningococci are however generally slower to develop antimicrobial resistance and NNN trend data show no recent shifts in penicillin MICs of invasive strains. All isolates were susceptible to the third generation cephalosporins and ciprofloxacin. Occasional isolates demonstrate rifampicin resistance and one isolate with an MIC of 32 mg/L and another with high level resistance were detected in 2003.

This report completes a decade of NNN analysis of laboratory-confirmed cases of IMD in Australia. Significant changes to diagnostic and analytical capacity have occurred in this period placing some limitations on the value of comparative analyses. However the data generated is an essential adjunct to clinically based surveillance systems for IMD. For further details the relevant NNN member in each jurisdiction should be contacted.

Acknowledgements

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 Australian Government Department of Health and Ageing provided a grant for the National Neisseria Network.

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OzFoodNet: enhancing foodborne disease surveillance across Australia: quarterly report, January to March 2004

The OzFoodNet Working Group

Introduction

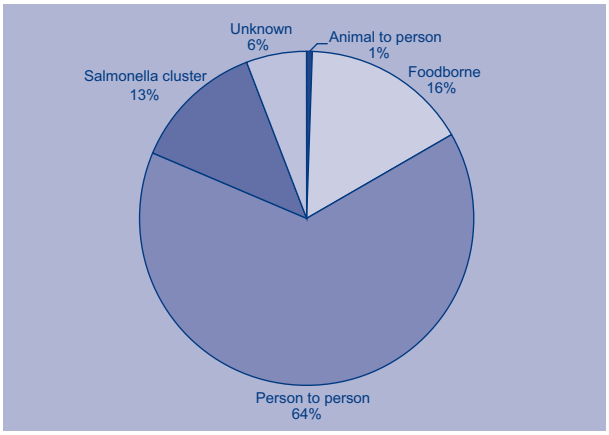
The Australian Government Department of Health and Ageing established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease. This quarterly report documents investigations of gastroenteritis outbreaks and clusters of disease potentially related to food occurring around Australia. The first quarter of the year is the peak season for many foodborne infections, such as *Salmonella* and *Campylobacter*. For information on sporadic cases of foodborne illness, see Communicable Disease Surveillance, Highlights for 1st quarter 2004 in this issue.

This report summarises the occurrence of foodborne disease outbreaks and cluster investigations between January and March 2004. Data were reported from all Australian State and Territory jurisdictions and a sentinel site in the Hunter region of New South Wales. The data in this report are provisional and subject to change as results of outbreak investigations can take months to finalise.

Foodborne disease outbreaks

During the first quarter of 2004, OzFoodNet sites reported 152 outbreaks of gastrointestinal infections (Figure 1). Seventy-one per cent (107) of these outbreaks were spread from person-to-person or were of unknown transmission affecting 2,629 people, hospitalising 61 and causing six fatalities. The majority of these outbreaks occurred in aged care facilities (57%), hospitals (12%) and child-care centres (9%). Recent reports from the United Kingdom have reported the emergence of a new norovirus strain since 2001 that may have increased virulence.[1] In recent years, there has also been a marked increase in the use of molecular diagnosis of these infections leading to increased recognition of this massive problem for healthcare agencies and the community. Outbreaks of gastroenteritis not transmitted by food are often not reported to health agencies or the reports are delayed, meaning that these figures significantly under-represent the true burden of these infections.

Figure 1. Mode of transmission for gastrointestinal outbreaks reported by OzFoodNet sites, January to March 2004



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

Twenty-four outbreaks were due to foodborne transmission compared to 27 in the fourth quarter of 2003 (Table). The outbreaks affected 280 people and 23 people were hospitalised. There were no fatalities in these outbreaks. Eleven outbreaks were due to *Salmonella* infection, three outbreaks of norovirus infection, three outbreaks of ciguatera poisoning and one outbreak of *Bacillus cereus* poisoning. The remaining six outbreaks were of unknown aetiology, affecting a total of 67 people. Nine of the outbreaks occurred in association with meals at restaurants and four in association with meals prepared by commercial caterers. Ten outbreaks occurred in January, eight in February and six occurred in March 2004.

OzFoodNet sites conducted four retrospective cohort studies and two case control studies to investigate these foodborne outbreaks. Fifty-eight per cent of outbreak investigations relied on descriptive epidemiology alone. One outbreak investigation obtained both epidemiological evidence of an association with a food vehicle and microbiological evidence of the agent in the food. In three outbreaks, investigators obtained analytical epidemiological evidence only, and in a further three microbiological evidence only, was found.

Table. Outbreaks of foodborne disease, January to March 2004 by OzFoodNet sites*

State	Month	Setting category	Agent responsible	Number exposed	Number affected	Evidence	Responsible vehicles
NSW	January	Restaurant	*S.* Typhimurium 170	Unknown	2	D	Suspected tartare sauce, fish and chips
	January	Institution	*S.* Typhimurium 135	Unknown	6	D	Suspected chicken or eggs
	January	Hospital	Unknown	6	5	D	Beef curry
	February	Caterer	Unknown	72	20	A	Mushroom soup
	February	Community	*S.* Typhimurium 9	Unknown	4	D	Duck eggs
	February	Caterer	*S.* unknown	14	12	N	BBQ chicken and rice
	February	Restaurant	Unknown	Unknown	7	D	Fried rice
NT	January	Restaurant	*S.* Typhimurium 108	Unknown	9	D	Unknown
Qld	January	Restaurant	Norovirus	Unknown	4	D	Frozen oysters
	January	Home	Ciguatoxin	2	2	D	Golden spotted trevally
	January	Community	*S.* Typhimurium 44	Unknown	12	D	Sushi
	February	Restaurant	Ciguatoxin	Unknown	4	D	Coral trout
	February	National take-away	*B. cereus*	Unknown	6	M	Potato and gravy
	March	Caterer	Norovirus	Unknown	8	D	Unknown
	March	Restaurant	Unknown	6	5	D	Sandwiches
	March	Home	Ciguatoxin	Unknown	2	D	Fish species unknown
	March	Restaurant	*S.* Zanzibar Var 15+	Unknown	5	D	Unknown
SA	March	Community	*S.* Typhimurium 108	Unknown	13	AM	Cream cakes
	January	Community	*S.* Saint Paul	Unknown	4	A	Eggs
Vic	January	Restaurant	*S.* Typhimurium 9	Unknown	90	M	Pizza and pasta
	February	Hospital	Unknown	Unknown	14	D	Unknown
	March	Restaurant	Unknown	Unknown	16	A	Suspect spaghetti bolognaise
	February	Community	*S.* Typhimurium 126	Unknown	11	D	Suspected eggs
WA	January	Caterer	Norovirus	37	19	M	Prawns and cold meats

* No foodborne outbreaks were reported from the Australian Capital Territory, Tasmania or the Hunter sites.

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

A Analytical epidemiological association between illness and one or more foods.

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

During the quarter, Queensland reported a small outbreak of norovirus associated with imported oysters from Japan. This followed similar outbreak investigations in Western Australia and the Northern Territory in November 2003.[1] The brand of oyster was different in each of the three outbreaks. Norovirus was not isolated from the oysters, but traceback investigations found that all implicated products were harvested from the same oyster system in Japan. This 'outbreak of outbreaks' was investigated by several Australian health departments, the National Centre for Epidemiology and Population Health, public health laboratories, Food Standards Australia New Zealand, and the Australian Quarantine and Inspection Service. OzFoodNet coordinated the investigation, which relied on good cooperation with Japanese authorities.

There were nine outbreaks of foodborne illness in Queensland. Three outbreaks were due to ciguatera following consumption of golden spotted trevally, coral trout and an unknown species of fish. Two of these outbreaks occurred at home, while one occurred at a restaurant. Queensland reported a small outbreak of *S.* Zanzibar 15+var affecting five people of Pacific Island origin. Common food outlets were investigated, but no food vehicle or source was identified. In an outbreak of *S.* Typhimurium 44 four of six cases interviewed had eaten sushi in the 48 hours prior to onset of their illness. Three cases had purchased their sushi from the same premises. There was one small outbreak of *Bacillus cereus* associated with potato and gravy from a take-away food premises. One outbreak of unknown aetiology occurred following a meal of sandwiches. There were two outbreaks of norovirus; one of which was associated with the frozen oyster meat mentioned previously, and one where the food vehicle was not identified.

The Victorian Department of Human Services reported two outbreaks of *Salmonella* infection and two of unknown aetiology. One outbreak of *Salmonella* Typhimurium 9 occurred at a pizza restaurant and affected 90 people. Multiple foods were positive for *S.* Typhimurium 9, including leftover cooked chicken, ham, salami and marinara mix. Pizza is a food commonly reported to be associated with foodborne outbreaks as short cooking times may not kill microorganisms.[3] An outbreak of *S.* Typhimurium 126 was a community-wide outbreak affecting 11 people. There were nine cases in an initial cluster. Six out of nine cases ate the same brand of organic eggs. One case had leftover uncooked vegetable patties bound with raw egg, which were positive for *S*. Typhimurium 126. The particular brand of eggs were sampled but no salmonellae were isolated. The Department of Agriculture collected drag swabs from the farm and sampled eggs, all of which were negative. Two further cases were investigated: both cases shared a smoothie containing raw eggs. The brand of eggs was not the same as that identified in the earlier cases. The two outbreaks of unknown aetiology were consistent with *Clostridium perfringens.* Spaghetti bolognese was the food vehicle in one of these outbreaks, while the other was unknown.

New South Wales reported seven outbreaks during the quarter, four of which were due to *Salmonella.* Three of these outbreaks were due to *S.* Typhimurium. One outbreak of *S.* Typhimurium 135 in a correctional facility was suspected to be caused by chicken or eggs, while another outbreak of *S.* Typhimurium 9 was attributed to duck eggs. The food vehicles in the remaining outbreaks included beef curry, seafood, mushroom soup, barbecue chicken and rice, and fried rice. Three outbreaks were of unknown aetiology.

The Northern Territory reported a single outbreak of *S.* Typhimurium 108 associated with a café. No food vehicle was identified despite sampling mayonnaise, chicken loaf and tartare sauce. *S.* Typhimurium 108 has an identical phage type pattern to *S.* Typhimurium 170, which is currently one of Australia's most common phage types of *S.* Typhimurium. During the quarter there were three foodborne outbreaks due to phage types 108 or 170.

South Australia reported an outbreak of *S.* Typhimurium 108 associated with cream cakes from a single bakery. Thirteen of 22 people interviewed were affected and five people were hospitalised. A case control study demonstrated an association between illness and consumption of cream cake, continental cake, sponge cake and cream Black Forest cake. No source of *Salmonella* was identified within the bakery. There is an urgent need for food safety agencies to determine the exact critical control points in bakeries, as these foods are consistently identified as the cause of *Salmonella* outbreaks.[3,4] South Australia also reported a small outbreak of four cases of *S.* Saintpaul where a cohort study identified an association with consumption of boiled eggs at a workplace function.

Western Australia reported an outbreak where 19 of 37 people on a cruise became ill with norovirus infection. Illness was associated with consumption of prawns and cold meats. The caterer for the cruise was unregistered.

There were no outbreaks of foodborne illness reported from the Australian Capital Territory, Tasmania or the Hunter OzFoodNet sites during the quarter.

Cluster investigations

During the third quarter of 2003, Australian states and territories conducted 18 investigations into clusters of various *Salmonella* serovar infections, including *S.* Typhimurium 126 in Western Australia; *S.* Typhimurium 9, *S.* Typhimurium 6 var 1, *S.* Typhimurium 141, *S.* Typhimurium 12, and *S.* Enteritidis 26 in Queensland; *S.* Typhimurium 197, *S.* Typhimurium 12, *S.* Typhimurium 9, *S.* Typhimurium 170, *S.* Virchow 8, *S.* Anatum, *S.* subsp I 16 :1,v:–, *S.* Cerro, *S.* Infantis and *S.* Oranienberg in Victoria; *S.* Zanzibar in South Australia; and *S.* Typhimurium 4 and *S.* Typhimurium 12 in New South Wales.

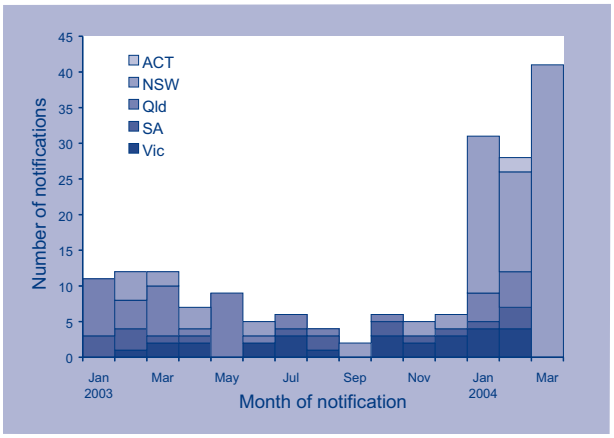
Queensland reported that seven of 19 cases of *S.* Typhimurium 9 interviewed had consumed kebabs from take-away shops in the 48 hours prior to their illness. Five of these cases purchased chicken kebabs in Brisbane from several locations, while two other cases purchased kebabs from a shop in northern New South Wales. Food samples including chicken meat, beef, lamb, sauces and salad ingredients collected from five shops in Brisbane and the Gold Coast were negative for all pathogens tested. Queensland also reported a cluster of four locally-acquired cases of *S.* Enteritidis 26 in a northern Queensland town. Interviews of three cases did not identify any common exposures. One patient owned egg-laying hens. Egg and environmental samples taken from the chicken coop were negative for *Salmonella*.

Queensland also reported a small cluster investigation into two cases of *S.* Paratyphi biovar Java 3b var 10, who had purchased aquarium fish from a common pet shop. These cases were included in the national case series investigation into these infections. In 2003, there were 72 cases of *S.* Paratyphi biovar Java notified to the National Enteric Pathogen Surveillance Scheme (NEPSS) and phage type 3b var 10 accounted for 21 cases (personal communication, J Powling, NEPSS, May 2004).

The Australian Capital Territory reported an investigation into two cases of locally-acquired *S.* Typhimurium 104L. The two cases were a 51-year-old female and a 1-year-old male who lived in the same suburb. Case households shopped at the same supermarkets, but no common foods were identified. Investigations into this cluster are continuing.

Multiple jurisdictions reported increases in *Salmonella* Typhimurium 12 during the quarter (Figure 2). Links could not be identified between cases. Since the majority of these *S.* Typhimurium 12 cases occurred in New South Wales, the Hunter OzFoodNet site coordinated the multi-state investigation into this increase in *S.* Typhimurium 12 and initiated a case control study of *S.* Typhimurium 12 in New South Wales to explore hypotheses for the increase, which included consumption of salad vegetables and fruit. The results from this study are not yet available.

Figure 2. Notifications of *Salmonella* Typhimurium 12, January 2003 to 23 March 2004, by jurisdiction and date of notification



Summary

Salmonella incidence increased during the quarter and was responsible for 46 per cent of foodborne outbreaks. Large numbers of norovirus outbreaks continued to be reported in aged care facilities and hospitals. Several outbreaks were related to eggs, although traceback efforts were unsuccessful. A third outbreak of norovirus implicating a different brand of oysters imported from Japan was significant, resulting in action to remove the oysters from the marketplace. These outbreaks have resulted in reconsideration of the safety of oysters harvested from contaminated waters in other countries.

Acknowledgements

We would like to thank state, territory and public health unit investigators, public health laboratories, and local government environmental health officers who contributed data to this report.

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Foodborne disease outbreaks in Australia, 1995 to 2000

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Abstract

Health agencies are increasingly conducting systematic reviews of foodborne disease outbreak investigations to develop strategies to prevent future outbreaks. We surveyed state and territory health departments to summarise the epidemiology of foodborne disease outbreaks in Australia from 1995 to 2000. From 1995 through 2000, 293 outbreaks were identified, with 214 being of foodborne origin. One hundred and seventy-four (81%) had a known aetiology, and accounted for 80 per cent (6,472/8,124) of illnesses. There were 20 deaths attributed to foodborne illness. Of the 214 outbreaks, bacterial disease was responsible for 61 per cent of outbreaks, 64 per cent of cases and 95 per cent of deaths. The most frequent aetiology of outbreaks was *Salmonella* in 75 (35%) outbreaks, *Clostridium perfringens* in 30 (14%), ciguatera toxin in 23 (11%), scombrotoxin in 7 (3%) and norovirus in 6 (3%). Salmonellosis was responsible for eight of the 20 (40%) deaths, as was *Listeria monocytogenes*. Restaurants and commercial caterers were associated with the highest number of outbreak reports and cases. Outbreaks in hospitals and aged care facilities were responsible for 35 per cent of deaths. The most frequently implicated vehicles in the 173 outbreaks with known vehicles were meats 64 (30%), fish 34 (16%), seafood 13 (6%), salad 12 (6%), sandwiches 11 (5%) and eggs 9 (4%). Chicken, the most frequently implicated meat, was associated with 27 (13%) outbreaks. This summary demonstrates the serious nature of foodborne disease and supports the move to risk-based food safety interventions focusing on mass catering and hospital and aged care facilities. *Commun Dis Intell* 2004;28:211–224.

Keywords: foodborne disease, disease outbreaks, surveillance, Salmonella, Campylobacter, hepatitis A, *Clostridium perfringens*, ciguatera toxin, *Staphylococcus aureus, Listeria monocytogenes, Bacillus cereus, Escherichia coli, Shigella,* norovirus, toxoplasma, scombroid

Background

Foodborne disease is a significant cause of morbidity and mortality throughout the world.[1] Contaminated food causes serious outbreaks that can result in significant societal costs. In addition, outbreaks have major implications for the food industry through lost earnings, lawsuits and damaged consumer confidence.[2,3]

Many countries systematically review outbreaks to develop strategies to prevent foodborne illness.[3,4] These reviews allow health agencies to identify high-risk foods, hazardous food processing procedures, and pathogens commonly associated with foodborne disease outbreaks. Summary findings can then be translated into policy to reduce foodborne disease. In the United States of America, reviews of outbreak data led to changes in the recommendations about eating undercooked eggs and hamburgers and the development of processing standards for ready to eat foods.[5,6,7]

In Australia, doctors notify foodborne disease outbreaks to state and territory health departments under state public health laws.[8] Members of the public, local government authorities and other agencies may voluntarily report foodborne outbreaks. Health agencies use standard techniques to investigate these outbreaks, and summarise the results for historical and legal purposes.[9] Health agencies only publish the results of a very small proportion of these investigations, making summary reviews more important.

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There have been several reviews of foodborne disease outbreaks in Australia, although their accuracy and completeness has varied considerably.[10–12] Until the establishment of the OzFoodNet program in 2000, there had been no systematic collection of national foodborne disease data.[13,14] The objective of this study was to summarise the epidemiology of foodborne disease outbreaks in Australia from 1995 to 2000, as background to the OzFoodNet initiative.

Methods

Identification of outbreaks

We surveyed state and territory health departments in 1998, 2000 and 2002 to elicit summary information on confirmed and suspected outbreaks in their jurisdiction from January 1995 to December 2000. Medline searches were conducted using the keywords Australia, foodborne disease, food poisoning, outbreak, and the specific pathogens (*Salmonella, Campylobacter*, hepatitis A, *Clostridium perfringens*, ciguatera toxin, *Staphylococcus aureus, Listeria monocytogenes, Bacillus cereus,* enterohaemorrhagic *Escherichia coli, Salmonella* Typhi, *Shigella, Vibrio,* Norwalk virus, toxoplasma, scombroid); state and commonwealth health agency bulletins, conference proceedings of the Australian Society of Microbiology, the Public Health Association, and the Communicable Diseases Network Australia were reviewed to identify any other foodborne outbreaks.

For each outbreak, health department staff completed a survey form that identified the pathogen, date and setting of outbreak, number of cases, number of fatalities, the attack rate among those consuming the implicated food, the mode of transmission, the type of study and whether food microbiology was positive. We provided a dummy outbreak report as an example to guide respondents in the use of the form.

Definition of foodborne outbreak

An outbreak was defined as foodborne if two or more people experienced a similar illness after sharing a common food or meals. However, for some settings and pathogens further analytical epidemiological and/or microbiological evidence was required. For example, outbreaks occurring in an institution required epidemiological or microbiological evidence of foodborne transmission to be included as a foodborne outbreak. For outbreaks due to hepatitis A, *Shigella* or *Giardia*, which are more commonly transmitted person-to-person in Australia, epidemiological and/or microbiological evidence was required for inclusion regardless of the setting. For outbreaks due to pathogens that are more commonly associated with foodborne transmission such as *Salmonella, Campylobacter, Staphylococcus aureus, Bacillus cereus* and *Clostridium perfringens*, and fish toxins such as scombrotoxin and ciguatoxin, that occurred after common foods or meals were shared, only epidemiological (or microbiological) evidence was required to identify that the outbreak was foodborne. However, the specific food vehicle responsible for the outbreak did not have to be identified.

Clusters of seemingly unrelated cases of *Salmonella* of a common subtype clustered in time and place were not included unless a particular food vehicle or a particular meal was implicated through investigation. If the pathogen was unknown, epidemiological evidence implicating a specific food vehicle was required. Waterborne outbreaks were not included in this summary. Where health department summary outbreak forms provided inadequate information, we contacted outbreak investigators or reviewed departmental reports to determine the evidence of foodborne disease transmission.

Classification of food vehicle and outbreak settings

Vehicles were grouped into a hierarchical classification system for food vehicle types. We adapted the food classification system used in the Centers for Disease Control and Prevention Electronic Foodborne Outbreak Reporting System (EFORS) (personal communication, Alana C Sulka, March 2002). Vehicles were coded into four levels of classification. For example, level one foods include broad food groups such as dairy, meat, fish, seafood and eggs. Level two breaks the foods into a second level such as cheese (dairy), beef (meat), herring (fish), crab (seafood) and egg sauces (eggs). Levels three and four became increasingly more specific.

Common Australian foods that were not in this system were added where relevant, such as pork rolls, which featured a number of times as vehicles for transmission in outbreaks. These were defined as level 1—sandwiches; level 2—sandwich, red meat based, and level 3—sandwich pork. The ‘soups and sandwiches’ descriptor used in EFORS was separated to reflect separate meal types, as there is no reason to combine these food types in an Australian setting.

Food vehicle groupings were presented as level 1 except where meats were broken down to level 2, oysters were shown as a level 2 subset of seafood, and fruit juice was shown as a level 3 subset of ‘non-dairy beverages’.

A coding system was used for classifying the settings where outbreaks occurred. We looked separately at where the food was prepared and where the food was eaten as the former more accurately reflects, where possible, food handling errors may have occurred. Most of the settings definitions are self evident. Restaurants included cafes and meals served in hotels where patrons sat down to eat. Take-away included milk bars and fast food outlets. Commercial caterer was defined as a setting in which food was produced for a special function or group (e.g. wedding and airlines) either in a private function room or at a location distant from the commercial caterer's kitchen. Contaminated primary produce was defined as food that routinely underwent no further processing before consumption. Commercial manufactured food was defined as foods that were prepared by large commercial processing groups and widely distributed.

Data management and analysis

Summary information was entered into Epi Info 6.04b and analysed in Epi Info 2002 and Excel 2000. The number of foodborne disease outbreaks per capita by state or territory were calculated using 1998 Australian Bureau of Statistics population projections.[15]

Results

Participation and response

There were 293 outbreak reports for the six-year period. Of these, 214 outbreaks were due to foodborne transmission and were included in the analysis. Seventy-nine outbreaks were excluded from analysis. For 78 outbreaks there was no epidemiological association with a food source, including:

- 37 outbreaks of unknown aetiology;
- 15 outbreaks of viral aetiology (mostly noroviruses);
- 5 bacterial outbreaks (4 of *Salmonella* and 1 of *Campylobacter*) in institutional settings;
- 6 outbreaks that were suspected person-to-person transmission including 3 *Shigella* outbreaks, two parasitic outbreaks and one hepatitis A outbreak;
- 10 waterborne outbreaks; and
- 5 *Salmonella* clusters.

One foodborne outbreak was excluded because the cases were infected while travelling overseas.

Time and state or territory of outbreak

From 1995 through 2000, there were 214 outbreaks of gastroenteritis of foodborne origin resulting in 8,124 cases. More outbreaks were reported annually during the data collection period from 1998–2000 compared to the retrospective collection of the 1995–1997 data (Table 1). Victoria and New South Wales recorded the highest number of outbreaks during the six-year period, the average number of outbreaks per million population by jurisdiction for the six-year period was highest in the Northern Territory, South Australia and Victoria. There were six multistate outbreaks resulting in 945 cases. Outbreaks were more frequently reported in the warmer months of October through March (Figure) predominantly due to the higher incidence of *Salmonella* outbreaks in these months. One hundred and seventy-four (81%) outbreaks had a known aetiology and these outbreaks accounted for 79 per cent (6,472/8,124) of illnesses (Table 2). The median number of cases for foodborne outbreaks was 17 (range 2 to 862). There were 20 deaths associated with the outbreaks, equating to a fatality rate of 0.3 per cent.

Figure. Seasonality of all foodborne disease and *Salmonella* outbreaks, 1985 to 2000

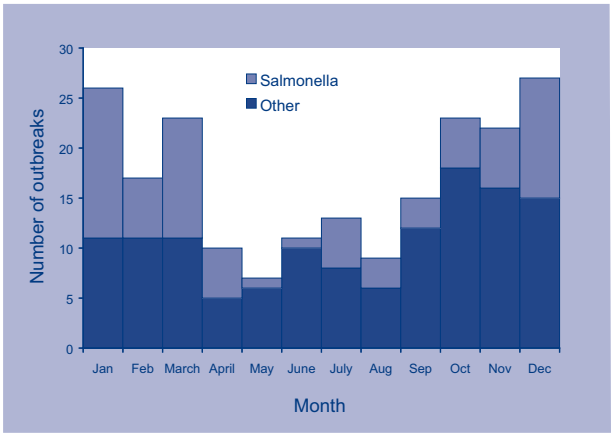

Table 1. Foodborne disease outbreaks and number of people affected, 1995 to 2000, by state or territory and year

		1995	1996	1997	1998	1999	2000	Total 1995–2000	Average annual reports per million population 1995–2000
New South Wales	Cases	71	144	475	226	422	216	1,554	41
	Outbreaks	5	6	10	11	8	8	48	1
Victoria	Cases	–	69	1,213	358	669	325	2,634	95
	Outbreaks	–	2	11	19	24	17	73	3
Queensland	Cases	–	564	–	119	189	127	999	48
	Outbreaks	–	4	–	3	14	12	33	2
South Australia	Cases	97	173	89	378	543	60	1,340	150
	Outbreaks	2	3	4	7	4	5	25	3
Western Australia	Cases	4	–	59	146	72	151	432	39
	Outbreaks	1	–	3	8	2	6	20	2
Tasmania	Cases	–	32	–	15	–	–	47	17
	Outbreaks	–	1	–	1	–	–	2	1
Australian Capital Territory	Cases	26	–	–	–	–	23	49	26
	Outbreaks	1	–	–	–	–	1	2	1
Northern Territory	Cases	–	–	20	43	5	56	124	108
	Outbreaks	–	–	1	2	1	1	5	4
Australia*	Cases	362	1,243	2,322	1,339	1,900	958	8,124	72
	Outbreaks	11	18	30	52	53	50	214	2

* Six multi-state outbreaks and 945 associated cases were only included in the total for Australia hence the annual columns for each state or territory do not sum to the total for Australia.

Aetiological agent

Of the 214 outbreaks, bacterial disease was responsible for 61 per cent of outbreaks, 64 per cent of cases and 95 per cent of deaths (Table 2). Most frequent were non-typhoidal salmonellae in 75 (35%) outbreaks, *Clostridium perfringens* in 30 (14%), ciguatera toxin in 23 (11%), scombrotoxin in 7 (3%), and norovirus in 6 (3%). *Campylobacter* spp. and *Listeria monocytogenes* were responsible for only 6 (3%) and 5 (2%) outbreaks respectively. Salmonellosis was responsible for eight of the 20 (40%) deaths, as was *Listeria monocytogenes*. Enterohaemorrhagic *E. coli* was responsible for two deaths, and *C. perfringens* and hepatitis A, one death each.

The largest outbreaks were 862 cases of illness associated with Asian-style pork rolls in Victoria in 1997 in which *Salmonella* Typhimurium phage type 1 was confirmed as the aetiological agent,[16] 502 cases of illness in South Australia in 1999, associated with unpasteurised orange juice in which *Salmonella* Typhimurium phage type 135A was confirmed as the aetiological agent (personal communication, Ingrid Tribe, SA Department of Human Services, May 2004) and 466 cases of hepatitis A in New South Wales in 1998, associated with oysters.[17]

Fourteen different serotypes of *Salmonella* were responsible for foodborne outbreaks, with serotype Typhimurium responsible for 49 (65%) of all *Salmonella* outbreaks and 2,716 (66%) of all *Salmonella* cases. The most common phage types of *S.* Typhimurium were phage type 135 (16 outbreaks, 1,014 cases); phage type 9, (13 outbreaks, 454 cases); and phage type 64 (4 outbreaks, 81 cases). *S.* Virchow was the second most common serotype responsible for eight outbreaks, with phage type 34 accounting for seven outbreaks (78 cases). The following serotypes were also implicated: Chester (4 outbreaks, 62 cases); Bredeney (2 outbreaks, 183 cases); Heidelberg (2 outbreaks, 507 cases); Mbandaka (2 outbreaks, 175 cases). Serotypes Anatum, Bareilly, Brandenburg, Hessarek, Mississippi, Muenchen, Oranienburg, and Saintpaul each caused one outbreak with a total of 248 cases ranging from two to 102 cases per outbreak.

Table 2. Foodborne disease outbreaks, cases, and deaths, Australia, 1995 to 2000, by aetiology (% of total)

Aetiology	Outbreaks		Cases		Deaths		Median number of cases per outbreak	Range
	n	%	n	%	n	%		
Bacterial	131	61	5,356	64	19	95	17	2–862
Salmonella	75	35	4,123	51	8	40	18	2–862
Clostridium perfringens	30	14	787	10	1	5	25	2–171
Campylobacter spp.	6	3	136	2	–	–	14	4–74
Listeria monocytogenes	5	2	41	<1	8	40	5	4–23
Staphylococcus aureus	5	2	78	<1	–	–	13	2–33
Bacillus cereus	2	1	28	<1	–	–	14	4–24
Enterohaemorrhagic *E. coli*	3	1	35	<1	2	10	6	6–23
Salmonella Typhi	1	0.5	4	<1	–	–	–	–
Shigella	2	1	42	<1	–	–	–	13–29
Streptococcus pyogenes	1	0.5	72	<1	–	–	–	–
Vibrio cholerae non O1, non O139	1	0.5	10	<1	–	–	–	–
Viral	8	4	780	10	1	5	40	10–466
Norovirus	6	3	297	4	–	–	40	10–97
Hepatitis A	2	<1	483	6	1	5	–	17–466
Protozoal (toxoplasma)	1	0.5	12	<1	0		–	–
Chemical	34	16	324	4			6	2–56
Ciguatera	23	11	179	2	–	–	5	2–33
Scombroid	7	3	34	<1	–	–	4	3–9
Dinophysis species	2	1	78	<1	–	–	–	22–56
Wax ester (escolar)	2	1	33	<1	–	–	–	14–19
Unknown	40	19	1,652	20	0		28	5–200
Total	214	100	8,124	100	20	100	17	2–862

Setting of outbreaks

Setting of food preparation

Ninety-four per cent (202/214) of outbreaks were associated with food prepared commercially or in settings other than private residences. Restaurants were associated with the highest number of outbreak reports and commercial caterers with the highest number of cases (Table 3).

The median number of cases per outbreak was much higher for commercial caterers (30 cases), take-away (23 cases) and commercially manufactured food (17 cases) than for restaurants (13 cases) reflecting the relative scale of production in these industry sectors. Aged care and hospital settings were responsible for 35 per cent of deaths despite being associated with only five per cent of outbreaks and less than three per cent of cases. There were seven deaths among 231 (3%) cases in aged care and hospital settings compared to 13 deaths among 5,297 cases (0.3%) in other settings in which deaths occurred.

Table 3. Foodborne disease outbreaks, cases, and deaths, Australia, 1995 to 2000, by setting prepared (% of total)

Setting prepared	Outbreaks		Cases		Deaths		Median number of cases per outbreak	Range
	n	%	n	%	n	%		
Restaurants	60	28	1,084	13	4	20	13	3–96
Commercial caterer	43	20	2,264	28	1	5	30	5–500
Contaminated primary produce	34	16	996	13	2	10	8	2–466
Take-away non franchised	17	8	1,397	17	–	–	23	2–862
Commercial manufactured food	14	7	967	12	5	25	17	2–502
Private residence	12	6	237	3	–	–	16	4–55
Aged care institution	8	4	167	2	4	20	22	4–37
Institution not elsewhere specified	5	2	304	4	–	–	72	17–85
Camp	4	2	252	3	–	–	22	9–200
Fair, festival, other temporary/mobile service	4	2	125	2	1	5	20	12–74
Hospital	3	1	64	<1	3	15	13	5–46
School	3	1	188	2	–	–	74	24–90
Grocery store/deli/ supermarket	3	1	24	<1	–	–	8	6–10
Military institution	1	<1	8	<1	–	–	–	–
Contaminated imported food	1	<1	17	<1	–	–	–	–
Other	1	<1	3	<1	–	–	–	–
Unknown	1	<1	27	<1	–	–	–	–
Total	214	100	8,124	100	20	100	17	2–862

Setting of food consumption

The setting where people consumed the implicated food was usually the same as where the food was prepared. The exception was where foods were purchased from a take-away store, a grocery store, delicatessen, or a supermarket. Foods contaminated in a commercial manufacturing setting or where the food was grown, were also consumed elsewhere. Foods prepared in settings where consumption occurred elsewhere resulted in 39 outbreaks and 3,288 cases in the community, and 37 outbreaks and 398 cases clustered in private residences.

Implicated food vehicles

A food vehicle was implicated in 173 (81%) of the 214 outbreaks. The most frequent vehicles were meats 64 (30%), fish 34 (16%), seafood 13 (6%), salad 12 (6%), sandwiches 11 (5%), desserts 9 (4%) and eggs 9 (4%) (Table 4). Chicken was the most frequently implicated meat and was associated with 27 (13%) outbreaks. Fish was the next most common cause of outbreaks but it was responsible for a relatively smaller number of cases as most were small outbreaks of ciguatera poisoning. When all outbreaks containing foods in which egg was the main high-risk ingredient were collated, a total of 16 potentially egg-associated outbreaks were identified. *Salmonella* was the aetiological agent for 14 of the 16 potentially egg-associated outbreaks. Salad, egg, dessert, and sandwich (pork roll) associated outbreaks tended to result in higher numbers of cases per outbreak. Seventy per cent (565/803) of cases attributed to seafood outbreaks were due to oyster consumption in the Wallis Lake oyster outbreak responsible for 446 cases of hepatitis A.[17]

Selected food vehicles, aetiological agents and settings combinations

Meats, particularly chicken were associated with 33 per cent of *Salmonella* and 60 per cent of *C. perfringens* outbreaks (Table 5). Restaurants, commercial caterers, and take-away settings were associated with 61 per cent of *Salmonella* outbreaks (Table 6). Meats were associated with a significant number of outbreaks across a range of settings (Table 7).

Table 4. Foodborne disease outbreaks, cases, and deaths, Australia, 1995 to 2000, by vehicle of transmission (% of total)

Vehicle	Outbreaks		Cases		Deaths		Median number of cases per outbreak	Range
	n	%	n	%	n	%		
Meats	64	30	1,846	23	7	35	17	2–200
Chicken	27		899		3		21	2–171
Beef	9		313		1		15	2–200
Pork	4		126		–	–	26	15–60
Lamb	2		16		–	–	–	4–12
Processed meats-consumed cold	6		97		2		16	8–24
Other meats*	16		395		1		20	4–85
Fish	34	16	281	4	–	–	6	2–33
Seafood	13	6	803	10	1	5	22	2–466
Oysters	3		565		1		97	2–466
Salads	12	6	587	7	3	15	29	4–176
Sandwiches	11	5	1,321	16	1	5	42	11–862
Eggs	9	4	773	10	–	–	36	7–500
Desserts	9	4	439	6	–	–	45	8–102
Grains	5	2	178	2	–	–	37	24–48
Dairy	5	2	81	1	–	–	12	9–27
Specialty/ethnic dishes	5	2	46	<1	1	5	6	3–24
Soup	2	<1	80	<1	–	–	–	13–67
Fruit	2	<1	60	<1	1	5	–	6–54
Vegetables	1	<1	54	<1	–	–	–	–
Fruit juice	1	<1	502	6	–	–	–	–
Miscellaneous	37	17	1,004	13	6	30	10	3–164
Unknown	4	2	69	<1	–	–	17	10–26
Total	214	100	8,124	100	20	100	17	2–862

* Includes meats in above categories that may be mixed together and meats not in above categories, or where type of meat was not known.

Discussion

This is the most comprehensive summary of foodborne outbreaks in Australia since the publication of a summary from 1980 to 1995 by Crerar, *et al.*[11] Crerar's 16 year summary identified 128 outbreaks—an average of eight outbreaks per year and six deaths in 15 years. Our collection of outbreaks identified 214 outbreaks over six years and 20 deaths—an average of 36 outbreaks per year and an average of 52 outbreaks during the contemporaneous collection from 1998 to 2000. We believe this reflects an improvement in the documentation of outbreaks due to improved availability of records and better recollection of more recent outbreaks. It is unlikely to be due to increased diagnostic capability as primary identification methods for the most common aetiological agents in this review have not changed substantially during this period. This highlights the importance of contemporaneous reporting of outbreaks in a standardised format. The new OzFoodNet outbreak surveillance system facilitates such reporting but this must be well supported at the local and state level.[14]

This summary of outbreaks contains some important information for policy makers involved in preventing foodborne illnesses. These data suggest a need for initiatives in commercial catering and aged care and hospital catering settings and for special attention to be given to particular high risk foods.

Table 5. Foodborne disease outbreaks and number of people affected, Australia, 1995 to 2000, by selected aetiological agents and food vehicle implicated

	Aetiological agent																			
	Salmonella				*Clostridium perfringens*				Norovirus				*Campylobacter*				Unknown			
	Outbreaks		Cases		Outbreaks		Cases		Outbreaks		Cases		Outbreaks		Cases		Outbreaks		Cases	
Vehicle	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Meats	25	33	658	17	18	60	572	73	1	17	96	32	1*	17	4	3	11	28	401	24
Chicken	10		335		6		312		1		96		–	–	–	–	6		112	
Beef	4		67		2		32		–	–	–	–	–	–	–	–	3		214	
Pork	2		37		1		29		–	–	–	–	–	–	–	–	1		60	
Lamb	–	–	–	–	2		16		–	–	–	–	–	–	–	–	–	–	–	–
Processed meats–consumed cold	4		61		–		–	–	–	–	–	–	–	–	–	–	–	–	–	–
Other meats†	5		158		7		183		–	–	–	–	–	–	–	–	1		15	
Eggs	8	11	701	17	–		–	–	–	–	–	–	–	–	–	–	–	–	–	–
Sandwiches	7	9	1,205	29	–		–	–	–	–	–	–	–	–	–	–	3	8	103	6
Desserts	6	8	254	6	–		–	–	1	17	51	17	–	–	–	–	2	5	134	8
Fruit	2	3	60	1	–		–	–	–	–	–	–	–	–	–	–	–	–	–	–
Seafood	2	3	14	< 1	2	7	76	10	2	33	111	37	–	–	–	–	1	3	27	2
Dairy	1	1	12	< 1	1	3	27	3	–	–	–	–	3	50	42	31			–	–
Fish	1	1	26	< 1	–	–	–	–	–	–	–	–	–	–	–	–	1	3	9	< 1
Fruit juice	1	1	502	12	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Salads	1	1	21	< 1	–	–	–	–	–	–	–	–	1	17	74	54	8	20	459	28
Vegetables	1	1	54	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Specialty/ethnic dishes	–	–	–	–	2	7	32	4	–	–	–	–	–	–	–	–	1	3	5	< 1
Grains	–	–	–	–	–	–	–	–	1	17	29	10	–	–	–	–	3	8	125	8
Soups	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2	5	80	5
Miscellaneous	18	24	573	14	6	20	70	9	1	17	10	3	–	–	–	–	8	20	309	19
Unknown	2	3	43	1	1	3	10	1	–	–	–	–	1	17	16	12	–	–	–	–
Total	75	100	4,123	100	30	100	787	100	6	100	297	100	6	100	136	100	40	100	1,652	100

* Poultry.

† Includes meats in above categories that may be mixed together and meats not in above categories, or where type of meat was not known.

Table 6. Foodborne disease outbreaks and number of people affected, Australia, 1995 to 2000, by selected aetiological agents and setting where the food was prepared

Setting Prepared	Aetiological agent																			
	Salmonella				*C. perfringens*				Norovirus				*Campylobacter*				Unknown			
	Outbreaks		Cases		Outbreaks		Cases		Outbreaks		Cases		Outbreaks		Cases		Outbreaks		Cases	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Restaurants	19	25	395	10	8	27	89	11	3	50	120	40	2	33	20	15	15	38	336	20
Commercial caterer	16	21	992	24	9	30	388	49	2	33	80	27		–	–	–	14	35	754	46
Contaminated primary produce	6	8	164	4		–	–	–	1	17	97	33	1	17	12	9	–	–	–	–
Take-away non-franchised	11	15	1,316	32		–	–	–	–	–	–	–	–	–	–	–	2	5	59	4
Commercial manufactured food	10	13	933	23	2	3	2	< 1	–	–	–	–	–	–	–	–	–	–	–	–
Private residence	4	5	26	< 1	2	7	63	8	–	–	–	–	–	–	–	–	2	5	74	5
Aged care	2	3	25	< 1	5	17	138	18	–	–	–	–		–	–	–	–	–	–	–
Institution not elsewhere specified	1	1	85	2	1	3	56	7	–	–	–	–	1	17	74	54	1	3	17	1
Camp	1	1	22	< 1	–	–	–	–	–	–	–	–	2	33	30	22	1	3	200	12
Fair, festival, other temporary/ mobile service	2	3	101	3	2	7	24	3	–	–	–	–	–	–	–	–	–	–	–	–
Hospital	1	1	46	1	–	–	–	–	–	–	–	–	–	–	–	–	1	3	13	< 1
School	–	–	–	–	1	3	24	3	–	–	–	–	–	–	–	–	2	5	164	11
Grocery store/deli/supermarket	2	3	18	< 1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Military institution	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	3	8	< 1
Contaminated imported food	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Other	–	–	–	–	1	3	3	< 1	–	–	–	–	–	–	–	–	–	–	–	–
Unknown	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	3	27	2
Total	75	100	4,123	100	30	100	787	100	6	100	297	100	6	100	136	100	40	100	1,652	100

Table 7. Foodborne disease outbreaks and number of people affected, Australia, 1995 to 2000, by food vehicle and selected settings where the food was prepared

Vehicle	Setting																					
	Restaurant				Take-away				Commercial caterer				Commercial manufactured food				Private residence				Hospital/aged care			
	Outbreaks		Cases		Outbreaks		Cases		Outbreaks		Cases		Outbreaks		Cases		Outbreaks		Cases		Outbreaks		Cases	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Meats	20	33	372	34	6	35	203	15	11	26	499	22	8	57	105	11	6	50	94	40	3	27	105	46
Chicken	10		242		6		203		5		314		2	14	14	2	2		38		1		32	
Beef	2		23		–	–	–	–	2		89		2	14	17	2	2		38		1		37	
Pork	1		15		–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1		36	
Lamb	1		4		–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Processed meats–consumed cold	–	–	–	–	–	–	–	–	1		23		3	21	51	5	1		13		–	–	–	–
Other meats*	6		88		–	–	–	–	3		73		1	7	23	2	1		5		–	–	–	–
Eggs	2	3	96	9	–	–	–	–	4	9	580	26	–	–	–	–	–	–	–	–	2	18	25	11
Sandwiches	–	–	–	–	5	29	1,096	79	3	7	84	4	1	7	78	8	–	–	–	–	1	9	46	20
Desserts	2	3	84	8	3	18	68	5	2	5	111	5	1	7	102	11	–	–	–	–	–	–	–	–
Fruit	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Seafood	3	5	51	5	–	–	–	–	1	2	43	2	2	14	16	2	1	8	33	14	–	–	–	–
Dairy	–	–	–	–	–	–	–	–	1	2	27	1	–	–	–	–	–	–	–	–	–	–	–	–
Fish	9	15	72	7	–	–	–	–	1	2	26	1	–	–	–	–	1	8	4	2	–	–	–	–
Fruit juice	–	–	–	–	–	–	–	–	–	–	–	–	1	7	502	52	–	–	–	–	–	–	–	–
Salads	3	5	61	6	–	–	–	–	4	9	347	15	–	–	–	–	2	17	74	31	1	9	4	2
Vegetables					–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Grains	1	2	48	4	–	–	–	–	3	7	106	5	–	–	–	–	1	8	24	10	–	–	–	–
Soup	–	–	–	–	–	–	–	–	1	2	67	3	–	–	–	–	–	–	–	–	1	9	13	6
Specialty/ethnic dishes	2	3	11	1	1	6	3	< 1	–	–	–	–	–	–	–	–	–	–	–	–	1	9	8	4
Miscellaneous	16	27	256	24	2	12	27	2	10	23	338	15	1	7	164	17	1	8	8	3	2	18	30	13
Unknown	2	3	33	3	–	–	–	–	2	5	36	2	–	–	–	–	–	–	–	–				
Total	60	100	1,084	100	17	100	1,343	100	43	100	2,264	100	14	100	967	100	12	100	237	100	11	100	231	100

* Includes meats in above categories that may be mixed together and meats not in above categories, or where type of meat was not known.

High-risk settings

Outbreaks in aged-care and hospital facilities were associated with 35 per cent of all deaths. These data lend support to the recent draft National Risk Framework that prioritises catering for immune compromised people and commercial catering as high-risk settings.[12] Ensuring high-risk patients do not receive high-risk foods could prevent many of these deaths. In this review the case fatality rate was three per cent for aged care and hospital settings compared to 0.3 per cent for all foodborne outbreaks. This raises the need for urgent implementation of food safety plans in these settings. While *Listeria monocytogenes* outbreaks occur rarely, the high case-fatality rate of approximately 20 per cent highlights the need for attention, particularly in institutions where residents are immune-compromised.[18] The NSW Health Department issued a circular on listeriosis control in such settings which emphasises implementation of Hazard Analysis Critical Control Points (HACCP); separating high risk foods from high risk patients, food temperature control, and cross contamination control.[19] Policy makers and regulators should ensure that HACCP is implemented in these settings.

Commercial caterers were responsible for the largest number of cases of any setting or 27 per cent of all cases. Commercial caterers are a special risk because they prepare large volumes of food and may be required to cater in venues with inadequate cooking, storage, transport, cooling, and reheating facilities. Several aspects of commercial catering make it difficult for regulators to identify and inspect these operations. Commercial caterers may be highly mobile and prepare or serve food in different settings from week to week including private residences, leased function rooms, and public venues and their operations are often conducted on weekends. Identifying and inspecting these operations for compliance with the Food Standards Code will help ensure that appropriate facilities are used and reduce the potential for outbreaks.

High-risk foods

Food vehicles that need special attention for interventions include chicken, eggs, certain fish species, salads, seafood (particularly prawns and oysters) and sandwiches.

Chicken was the most common meat associated with outbreaks. Chicken is a commonly consumed food and needs to be produced in as safe a manner as possible. The high proportion of chicken associated outbreaks due to *Salmonella* and *Clostridium perfringens* suggest the need for efforts at the point of primary production, processing, and food preparation. Reduction of contamination at the processing level and appropriate food handling in restaurants and homes and effective cooking can reduce the risk of foodborne infection. Interventions to reduce bacterial contamination at farm and processing levels can significantly reduce human infection as has been demonstrated in Iceland and Denmark.[20,21]

Egg-based foods were commonly reported in this series of foodborne outbreaks. We cannot be certain how many of these outbreaks were due to direct contamination from the surface of eggs, from internally contaminated eggs, or contamination of an egg-based food. Nevertheless, the high proportion of egg associated outbreaks that were due to *Salmonella* suggests the need for vigilance. *Salmonella* Typhimurium phage types 9 and 135 were the predominant pathogens identified in egg-associated outbreaks. The link between eggs and outbreaks of salmonellosis has been well established in Europe and the United States of America due to transovarian transmission of infection.[22,23] There is no evidence of transovarian transmission of disease in Australia, nevertheless these data suggest that there is a need for the egg industry to monitor egg production hygiene issues closely. Additionally, control of temperature and prevention of cross contamination of eggs in processing, storage, and food preparation is extremely important. Since the compilation of these data there have been further egg-associated outbreaks, some of which were traced back to layer farms that were positive for the outbreak strain of *Salmonella.*[24] Even quite low contamination rates (e.g. < 0.01%) of eggs can pose a significant public health problem.[25]

Reef fish are a significant cause of ciguatera poisoning which causes a severe illness occasionally with a fatal outcome.[26] Better education of restaurateurs, fish wholesalers, and recreational fisherman is required to prevent the consumption of high-risk fish. Public health traceback investigations may identify specific reefs that are high risk for ciguatera. There are no suitable routine tests to detect ciguatera toxin in fish, nor can the toxin be detected in the fish by its appearance, odour, texture or taste. In general terms, the risk of poisoning is increased by consuming larger and presumably older fish, but poisoning can sometimes occur following the consumption of relatively small fish. These risks are increased for people who regularly consume fish soups using the head or viscera of smaller fish, where the toxin concentrations may be higher.

In this review seafood associated outbreaks included three oyster outbreaks (*Salmonella*, norovirus, and hepatitis A) and four prawn-associated outbreaks (hepatitis A, *C. perfringens* (2), and *Salmonella* Typhi). Hepatitis A, while rare, causes serious illness with an average of 12.5 days of work missed.[27] In this summary, the hepatitis A outbreak associated

with Wallis Lake oysters shows the potential of this disease to cause large widespread outbreaks. These outbreaks are difficult to investigate due to the long incubation period of hepatitis A.[17]

Salads were responsible for 12 outbreaks. While salads are not often considered a high-risk food the minimal post-harvest processing of salads requires good on-farm HACCP as consumers and retailers have minimal opportunity to prevent illness.[28,29]

Four outbreaks were associated with Asian-style pork rolls, a high risk food by virtue of the ingredients and method of preparation. One of these outbreaks was the largest single documented outbreak in our review with 862 cases.[16] Pork rolls include a range of high-risk foods including eggs, chicken liver pate, and pork.

Sandwiches are not usually considered a high-risk food. This perception and the use of sandwiches in mass catering—particularly where sandwiches may be stored for many hours at room temperature for convenience—may contribute to temperature abuse and subsequent food poisoning. Temperature control of sandwiches should be a high priority with a 'use by time' label to manage sandwich safety in mass catering settings.

Barriers to surveillance and limitations of outbreaks summaries

The number of outbreaks per million residents differs greatly across states and territories. This reflects differences in detection, and investigation of outbreaks, and formal systems of documentation rather than real differences in outbreak activity. This is exemplified by the disparity in outbreaks reported to the US Centers for Disease Control and Prevention from 1993 to 1995 with interstate incidence ranging from 73 outbreaks per million population to 0.65 in some years.[4] While much research is needed into the usefulness of this population-based ratio, it may be useful as a tool for evaluation of surveillance systems.[30,31] The lack of any reports from Victoria in 1995 is explained by difficulties in recall or identification of records rather than an absence of outbreaks or outbreak investigation. Victoria began to systematically record outbreaks in August 1995 (personal communication, Joy Gregory, December 2003). The prospective reporting of outbreaks to OzFoodNet from 2001 should redress these reporting disparities.

This summary is biased by patterns of reporting, analysis, investigation and laboratory testing for foodborne disease outbreaks. The number of outbreaks documented by setting is very dependent on reporting biases.[3] Patient and doctor behaviour is affected by the patient's symptoms. Patients are more likely to present to a doctor and be tested if their diarrhoeal illness lasts more than two days or involves bloody diarrhoea (personal communication, Gill Hall, October 2003). Outbreaks are more likely to be recognised and investigated where an established social, work, or familial group share a common meal—leading to greater recognition of restaurant and catered function outbreaks. The relatively smaller number of outbreaks associated with commercial manufactured food may reflect quality assurance processes in this sector. Alternatively, outbreaks due to high volume commercial products with wide distribution may have such low attack rates that a common source of illness is difficult to identify.

Patterns of laboratory testing may bias outbreak summaries leading to greater recognition of pathogens such as *Salmonella*, *Campylobacter*, and *Shigella* which are identified by routine stool cultures ordered by general practitioners. Norovirus, *C. perfringens*, *Staphylococcus aureus*, *Bacillus cereus* outbreaks are less likely to be detected because tests for these agents are not routinely ordered by general practitioners. Tests for these pathogens are usually requested by public health officials only after an outbreak with typical symptom or incubation period profile is recognised. Additionally many of these agents are only excreted in stool for a short period making detection less likely if there is a delay in stool testing.

The real time collection of outbreak summaries and enhancement of foodborne disease investigation and surveillance under OzFoodNet and advances in laboratory methods will ensure better information for food safety initiatives in the future. The number of cases and deaths are very likely an underestimate of those occurring even within the reported outbreaks as complete case ascertainment could not be confirmed and cases were not followed up to confirm recovery. Immune-compromised persons may die many weeks or months after suffering a foodborne disease, however, attributing the cause of death may be difficult.[32]

Data collected on potential factors contributing to outbreaks were not included here because of the lack of standard systems for inspection and criteria for linking inspection findings with outbreak causation. Because environmental inspections are always conducted after an outbreak, it is difficult to know if the conditions found during the inspection were associated with the outbreak.

Conclusion

The outbreaks reported here are biased by multiple layers of surveillance barriers and constitute only a small proportion of the outbreaks, cases and deaths that actually occurred. Nevertheless, food safety regulators rely on these summaries as they provide evidence of repeated failures in the system. This review highlights the major areas of concern during 1995 to 2000. During the period there were several serious and large outbreaks, causing at least 20 deaths. Clearly, this burden of illness justifies the attention given to foodborne disease and food safety in Australia.[33] There is an urgent need for Australia to prioritise initiatives for high-risk foods, high risk settings, and production methods to prevent outbreaks and sporadic disease.

Acknowledgements

We would like to thank the many health department staff that investigated foodborne outbreaks that contributed to this review. Thanks also to thank Mohinda Sarna, Rod Givney, Ingrid Tribe, Jane Raupach, David Peacock, Eddie O'Brien, Tony Watson, Brian MacKenzie and Steve Munyard for providing extra information about outbreaks, Katerina Ludenowski for research assistance, and Rosemarie Schmidt for editing. We also thank the many members of the public who report outbreaks and provide information to investigators. We thank the food manufacturers and handlers involved in outbreaks for their cooperation that is so essential to the prevention of future outbreaks. Joy Gregory, Russell Stafford and Martyn Kirk are funded by the Australian Government Department of Health and Ageing's OzFoodNet program of work.

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An outbreak of shigellosis in a child care centre

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Abstract

Outbreaks of shigellosis in child care are not commonly reported in Australia, however *Shigella* bacteria can easily spread in these settings. We report an outbreak of shigellosis in a child care centre and discuss the control measures implemented. This investigation identified 20 confirmed cases of *Shigella sonnei* biotype g and a further 47 probable cases in children and staff who attended a child care centre, and their household contacts. The investigation highlighted the importance of stringent control measures and protocols for dealing with outbreaks of *Shigella* and other enteric infections in the child care setting, and the importance of prompt notification by both doctors and child care centres, of suspected outbreaks. *Commun Dis Intell* 2004;28:225–229.

Keywords: disease outbreak, child care, shigellosis, Shigella sonnei

Introduction

Shigellosis is an acute enteric bacterial infection generally characterised by a mild and self-limiting gastroenteritis, however illness may be severe. Symptoms include diarrhoea accompanied by fever, nausea and sometimes toxaemia, vomiting, cramps, and tenesmus. In typical cases, the stools contain blood and mucus. Many cases present with watery diarrhoea. Convulsions may be an important complication in young children. Illness may last from several days to weeks with an average of four to seven days. Asymptomatic infections also occur.[1]

At the time of this outbreak there were no publications of outbreaks of shigellosis in child care centres in Australia, however they are considered high-risk settings due to close contact of children with each other and the low numbers of bacteria (10–100) required to cause illness.[1] Transmission of infection can occur through inadequate hand washing after defaecation or nappy changing, faecal contamination of nappy changing surfaces and fomites, or from person-to-person directly via the faecal-oral route. We report an outbreak of shigellosis in a child care centre and discuss the control measures implemented.

On 4 December 2000, during a routine investigation by the Communicable Diseases Section (CDS), Victorian Department of Human Services, of a shigellosis notification, the notifying medical practitioner advised that other children who attended the same child care centre as his patient were ill. The Director of the centre subsequently reported that approximately 15 children, some of their family contacts and a number of staff had been ill with gastroenteritis over a two and a half week period. At the time of the outbreak, there were approximately 70 children attending the centre and 11 staff employed. The centre has three rooms; nursery/baby room (0–1 years), toddler (2–3 years) and kindergarten (4–5 years), and operates from 7 am to 6 pm. Mixing of these groups in the centre occurs in the early morning and late afternoon.

Methods

A case series investigation and active case finding was conducted. A probable case was defined as any child or staff member of the child care centre, or their household contacts, who had been ill with a gastrointestinal illness consisting of diarrhoea and self-reported fever between 12 November and 22 December 2000. Cases were confirmed if S*higella sonnei* biotype g was isolated from a faecal specimen.

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Ethical approval was not needed as the investigation was carried out as part the Communicable Diseases Section core duties under the *Health Act 1958*.

The Director provided details on the number of staff and children attending the centre and consent to contact people. All ill persons or their parents were interviewed by phone using a standard questionnaire. This included demographics, clinical symptoms, whether hospitalised, environmental exposures and which room children attended within the child care centre if the case was a child, or the staff duties at the centre if the case was a staff member. If the case was a household contact of an attendee at the centre, then their occupation was obtained.

Local Government Environmental Health Officers conducted environmental inspections according to Department of Human Services' guidelines,[2] and organised collection of faecal specimens from cases. These were collected even if symptoms had subsided as *Shigella* can be excreted for up to four weeks after symptoms cease.[3] CDS staff also visited the centre to provide advice and assistance with implementation of control measures.

Control measures were based on reports from a similar outbreak in the United States of America[4] due to an inability to identify relevant protocols in Australia.

Specimens collected were submitted to the Microbiological Diagnostic Unit and the Victorian Infectious Diseases Reference Laboratory for microbiological analysis. Confirmation of species, sub-typing and antibiotic sensitivity analyses were conducted at the Microbiological Diagnostic Unit. Testing for viral pathogens was conducted at the Victorian Infectious Diseases Reference Laboratory.

Results

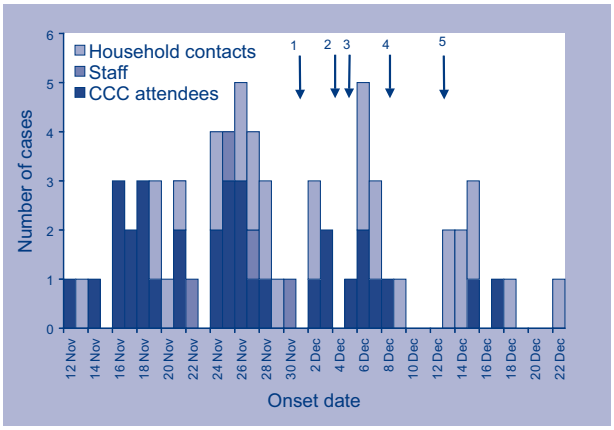
Epidemiological investigation

Sixty-seven probable cases were identified; 33 children (47% of children attending), four staff (37% of staff employed) and 30 household contacts (total denominator unknown). The age of cases ranged from 1–59 years, with household cases amongst children aged 1–17 years and adults aged 24–52 years. Twenty-seven cases were male. Two cases were hospitalised and there were no deaths. Onset of illness was between 12 November and 22 December 2000, with median duration of five days and range 0.5 to 14 days.

Characteristics of clinical details are shown in Table 1. Clinical illness was consistent with *Shigella*.

The first case was in a child from the toddler room (Figure). Staff reported illness near the middle of the outbreak, around the same time as the majority of the family contacts. Within four days after the onset of illness in the first case, cases were detected in all three rooms at the centre. Onset of illness in the first family member so early in the outbreak could not be explained. A source of illness was not identified for the case with the earliest onset.

Figure. Epidemic curve, child care centre outbreak, Victoria, 2000, by exposure category



1. Notification of first case received.
2. Outbreak identified.
3. Initial control measures implemented.
4. Case positive for both norovirus and *Shigella*.
5. Cohorting and antimicrobial treatment commenced.

Table 1. Clinical description for 63 cases with reported symptoms of *Shigella* associated with a child care centre, Victoria, 12 November to 22 December 2000

Symptom	Percentage
Diarrhoea	97
Watery diarrhoea	69
Fever	55
Abdominal pain	52
Vomiting	32
Blood in faeces	8

There were 12 pairs of siblings attending the centre (four pairs in the toddler and kinder rooms; three pairs in the kinder and baby room; three pairs all in the toddler room; one pair in the toddler and baby room; one pair in the baby room) and potentially contracted their illness from each other either at home spreading the illness to other rooms at the centre, or contracted their illness at the centre from other children in their room, or in the mixed morning and afternoon groups.

Environmental investigation

Clean-up procedures were implemented according to CDS guidelines, although a chlorine base sanitiser was not used for cleaning until 15 December 2000. Detailed information on the hygiene practices at the child care centre was incomplete, however the premises were in a good condition and it had an approved Food Safety Plan. Methods for cleaning toilets and nappy change tables were found to be inadequate. The levels of hygiene and cleanliness were evaluated according to the Department of Human Services' *Guidelines for the Investigation of Gastrointestinal Illness* [2] and *Staying Healthy in Child Care*,[5] and advice given according to these guidelines.

It was determined that children at the centre mix freely with each other at the beginning and end of each day, and were not confined to separate rooms. Staff worked in multiple rooms as child care workers; three of the staff positive for *Shigella,* worked in the toddler room, two of these also worked in the nursery and the fourth staff member occasionally assisted in all rooms as required and occasionally worked as a food handler.

Laboratory investigation

Faecal specimens were collected at a median of nine days after onset (range 0–29 days). Forty-nine stool specimens were submitted; 20 (41%) were positive for *Shigella sonnei* biotype g. Confirmed and probable cases by type of exposure are outlined in Table 2. One specimen in a child attending the centre was also positive for both *Shigella* and norovirus. Onset of illness in this child was 8 December, near the end of the outbreak.

One culture positive case was tested 29 days after their onset date. More adults (family contacts and staff) than children were culture positive (Table 2). This may be due to specimens in adults having been taken closer to their onset date making it more likely that they were still excreting *Shigella*.

Table 2. Outbreak cases by confirmation status and exposure type

Exposure type	Confirmed	Probable	Total
Toddler room	4	11	15
Kinder room	1	7	8
Nursery/baby room	2	8	10
Staff	4	0	4
Adult family contact	7	11	18
Sibling contact	2	10	12
Total	20	47	67

All isolates were resistant to streptomycin, tetracycline, sulphathiozole and trimethoprim; three were also resistant to ampicillin and the remaining were sensitive. All were sensitive to chloramphenicol, kanamycin, nalidixic acid, spectinomycin, gentamicin and ciprofloxacin.

Instances of isolates, with and without an antibiotic resistance marker, isolated from the same patient have been noted. Consequently, the result of both ampicillin-resistant and ampicillin-sensitive strains being isolated in this outbreak was not a surprise. The protocol for any testing requests a pure culture, i.e. taken from a single colony, thus laboratory tests may not have detected both sensitive and resistant isolates from the same patient. The original source of the infection may have been a mixture of both ampicillin-sensitive and ampicillin-resistant strains. Another explanation may be that the ampicillin resistance in some of the isolates may have been transferred from other enteric organisms within the patients' intestines. The isolates by all other parameters were indistinguishable other than the ampicillin resistance.

Control measures

The majority of cases had already occurred before CDS was notified of the first case (Figure), delaying control of the outbreak. Initial control measures such as clean-up procedures, enhanced hygiene and hand washing, were implemented on 5 December 2000. More stringent control measures, such as cohorting and antimicrobial therapy, were implemented on 13 December 2000. Onset of illness in the last case at the centre was 13 days after control measures were first implemented but only five days after more stringent measures were initiated (Figure).

Further transmission occurred in family contacts with the last case reporting an onset of illness on 22 December 2000. In order to minimise the impact on parents and staff, the child care centre remained open throughout the outbreak.

Case management

Those who still had diarrhoea were excluded from the child care centre until they had received an antibiotic sensitive to the *Shigella* strain for at least 72 hours[6] and diarrhoea had ceased. If a person could not or refused to take antibiotics, he or she could not return to the centre until asymptomatic and had two consecutive negative faecal specimens taken at least 24 hours apart.

Any child or staff member with a history of diarrhoea in the past month who were asymptomatic at the time of the investigation (irrespective of whether their faecal specimen was positive for *Shigella*) were presumed to have had shigellosis and to still be infectious. They were cohorted in a 'get well room' until they had taken appropriate antibiotics for at least 72 hours,[6] after which they could return to their usual room or duties whilst completing the full course of antibiotics. The use of a 'get well room' was used as an effective strategy in a similar outbreak overseas.[7]

Children in the 'get well room' were allocated a specific toilet and hand basin throughout the duration of the outbreak so as to prevent the spread of *Shigella* to others. As the centre had only one staff toilet, ill staff (who lived near work) agreed to use their home toilet rather than being excluded until antibiotics had been received for at least 72 hours. Similarly, ill staff could not conduct any cleaning or cooking at the centre for the same time period. One food handler was required to provide two consecutive negative faecal specimens taken at least 24 hours apart before returning to normal duties.

A letter of advice about the outbreak and *Shigella* fact sheet was given to all families of attendees. Parents were advised to take the letter with them to their doctor if their child or any household member had been or became ill. Family contacts in high-risk occupations (a registered nurse and supermarket worker) were identified during the interview process and provided with appropriate advice (such as recommendations for work exclusion) in order to prevent the outbreak from spreading further.

Treatment of cases with an appropriate antibiotic was recommended in this outbreak as this usually reduces duration of carriage to a few days.[1] Recommendations were based on the therapeutic guidelines[8] and the antibiotic sensitivity pattern of the first notified *Shigella* case. Ampicillin was initially recommended however parents complained that ampicillin was not available in syrup or powder form and amoxicillin was subsequently recommended for children too young to take tablets.[3]

Three cases (one child and two adults) had a strain resistant to ampicillin. An alternative antibiotic was not available for the child and norfloxacin was recommended for the adults.

Environmental measures

At least twice daily clean-up procedures were carried out throughout the centre. Diluted household bleach (one cup of bleach to nine cups of water) was used as a sanitiser; spray bottles with the bleach were used to clean door handles, knobs and surfaces and the solution was made up twice daily. Hand washing was re-emphasised and posters were displayed in bathrooms. Staff supervised the hand washing of children and assisted those children too young to wash their own hands. Loose soap in the toilets was replaced with liquid soap dispensers, which were washed and refilled at various times throughout the day; paper towels were provided for hand drying.

Food on common plates was not permitted and children in the cohort room were not allowed contact with children from other rooms. Separate batches of toys were used in the morning and the afternoon and were required to be cleaned and sanitised prior to use at each session. The nappy changing area was cleaned and sanitised twice daily.

New admissions to the centre were not permitted during the outbreak and the transfer of children to other centres was monitored.

Discussion

This investigation identified 20 confirmed cases of *Shigella sonnei* biotype g and a further 47 probable cases in children and staff who attended a child care centre, and in their household contacts. The extent of this outbreak is likely to be due to person-to-person transmission. The implementation of stringent clean-up procedures, enhanced hand washing and hygiene and the support of staff and families at the child care centre was an effective strategy in controlling the outbreak. While we did not investigate asymptomatic infection, once these procedures were fully implemented, transmission at the centre appeared to have ceased after five days and no further cases were reported.

The finding of one co-infection with norovirus is interesting and it is possible that other cases of norovirus were not detected, however we do not believe other cases of gastroenteritis in the centre were viral infections. Although a broad case definition was used, the clinical illness (Table 1) was consistent with *Shigella* and did not resemble the explosive nature of viral gastroenteritis outbreaks. In addition, over 30 per cent of the faecal specimens collected were tested within 0–10 days after onset of illness and if norovirus was present more positive specimens would have been expected. It is however plausible to assume that the stringent control measures implemented in this setting potentially prevented a concurrent viral gastroenteritis outbreak.

The interventions adopted in this outbreak of *Shigella* appeared to be effective. Excluding persons with diarrhoea from attending the child care centre until symptoms have ceased is critical. Active follow-up of illness in household members gave the opportunity to educate on gastrointestinal illnesses and ways of mini-

mising the spread to other family contacts. Persons in high-risk occupations were educated on exclusion requirements and personal hygiene at work.

Providing support to staff and parents was important. An earlier visit to the centre would have been beneficial in ensuring staff obtained clear and uniform information on the outbreak and the importance of adhering to control measures. Keeping the child care centre open but with extensive restrictions appears to have been justified. It prevented parents from taking their children to another centre and spreading the outbreak further. Allowing staff to work whilst excreting *Shigella* also eliminated the stress of insufficient staff.

Antibiotic treatment appeared effective with only two cases in attendees and no cases in staff occurring after this intervention (Figure). However, as faecal specimens were not collected after the completion of antimicrobial treatment, the true effectiveness of antimicrobial treatment in this setting could not be determined. Further evaluation of antibiotic effectiveness in outbreak settings would be beneficial. The lack of appropriately formulated antibiotic therapy for small children was problematic and we suggest that therapeutic guidelines are reviewed to take into account the lack of availability of ampicillin for small children and the use of amoxicillin in shigellosis.

This investigation also highlighted the importance of prompt notification by both doctors and child care centres of suspected outbreaks, so that investigations and control measures can be implemented in a timely fashion. Similarly, specific protocols for the management of outbreaks in childcare settings are essential and the Department of Human Services is incorporating those used in this outbreak into gastrointestinal illness management guidelines.

To further minimise such outbreaks, child care centres are required under the Health Infectious Diseases Regulations, to exclude children with diarrhoea until ceased or a medical certificate is provided (and a policy around this should be in place at child care centres). Child care centres should seek advice from their local health department where an outbreak is suspected or where advice is required regarding infectious diseases.

Acknowledgements

Thanks to the staff of the participating council for their assistance and support in this investigation, and to the staff, parents and children at the centre.

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Passive surveillance of antimicrobial resistance in Queensland public hospitals: the basis for a national system?

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Abstract

Australia currently has no system of passive surveillance of antimicrobial resistance in spite of the importance of surveillance in identifying and defining emergent resistance being generally accepted. Queensland Health Pathology and Scientific Services have developed flexible software for passive surveillance with the capacity to handle national data. The system imports raw data strings in delimited ASCII text format into a relational database and screens to exclude duplicates before the processing of the cumulative susceptibility data. It allows considerable flexibility in inquiry parameters and has the ability to 'drill down' to individual laboratory results. Examples of analytical output are given for 49,169 unique isolate results obtained in all Queensland Health Pathology Service laboratories from 1 January to 30 June 2003. The system could form the basis of a national system for passive antimicrobial resistance surveillance. *Commun Dis Intell* 2004;28:230–235.

Keywords: Passive surveillance, antimicrobial resistance, antibiogram

Introduction

The emergence of increasing levels of resistance in a growing number of major pathogens has led to the recognition of antimicrobial resistance as an important public health issue. Government sponsored reports in a number of countries including the United Kingdom, the United States of America and Australia have identified the need for action to deal with this emergent problem.[1,2,3] Furthermore, the World Health Organization has acknowledged the global nature of this problem and has recommended a concerted international approach to controlling the emergence and spread of antimicrobial resistance.[4] The importance of surveillance in identifying and defining emergent resistance is generally accepted. The Australian report (JETACAR)[3] recommended that a comprehensive surveillance system be established in Australia incorporating both active and passive components.[3]

Prior to the publication of JETACAR passive surveillance of resistance in a broad range of pathogens was conducted for a number of years by the National Antimicrobial Resistance Surveillance Program (NARSP) by collation of results obtained in 29 pathology laboratories both public and private.[5] However, due to the laborious nature of data collection and collation, NARSP publications lagged behind testing by several years at least. This tended to limit their utility in identifying emergent problems. The introduction of The Surveillance Network™ (TSN®), an American commercial computerised surveillance system, into Australia in 1998 promised for some time to fill the gap vacated by NARSP and to provide national passive surveillance data in close to 'real-time'.[6] However, the decision by TSN® to withdraw from Australia in 2003 leaves us without a national passive surveillance system.

Queensland Health Pathology and Scientific Services (QHPSS) have made the provision of passive antimicrobial resistance surveillance data to clinicians within Queensland public hospitals a high priority. The purpose of passive surveillance is to provide estimates of the prevalence of resistance phenotypes based on specimens submitted to clinical laboratories. The aim of the current project is to provide timely cumulative susceptibility data to interested health care professionals at a local, regional and state level.

This paper describes the creation of comprehensive state-wide network of passive surveillance incorporating all susceptibility data generated in our laboratories and gives some examples of its reporting output.

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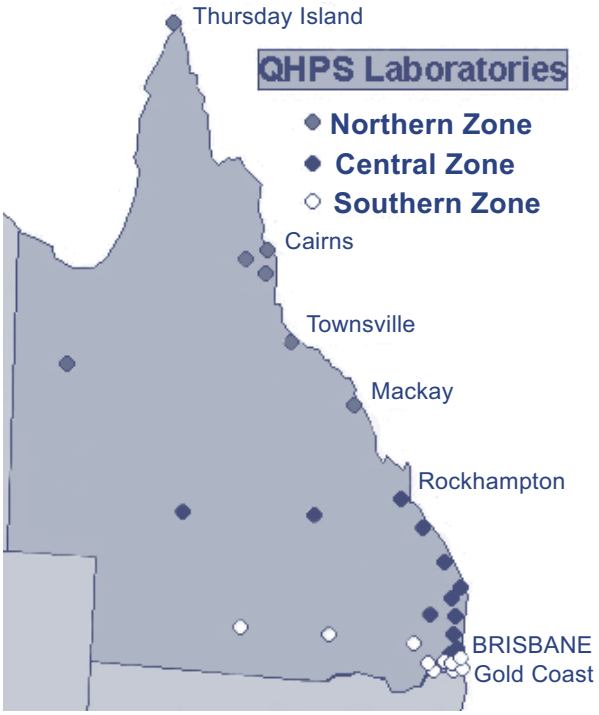
Methods

Queensland Health Pathology Service (QHPS) consists of a hierarchical, networked system of 32 laboratories (Figure) which vary in size from small remote laboratories serving rural communities to large multidivisional laboratories serving metropolitan tertiary referral hospitals. It provides laboratory services to all public hospitals but one in Queensland. Public hospitals in Queensland form part of health districts that in turn belong to three zones. The Northern Zone serves a population of 596,725, the Central Zone 1,365,076 and the Southern Zone 1,624,094 according to 2001 census data.

Figure. Location of Queensland Health Pathology Service laboratories



A single laboratory information system (LIS) (AUSLAB™, PJACC, Melbourne) is networked to all laboratories from a central computer. Twenty-four of the laboratories perform antimicrobial susceptibility testing. Since July 2002, all susceptibility testing is performed according to National Committee for Clinical Laboratory Standards (NCCLS)[7] or using automated methods based on NCCLS methods (Vitek®, bioMerieux, Missouri or MicroScan®, Dade Behring, Illinois). Specimen, isolate and susceptibility data ('S', 'I' or 'R' calls) are downloaded from the LIS using an 'autodump' function. A data field is included to specify the test method thus allowing data derived from any method to be captured and analysed separately if required. The raw data (strings in delimited ASCII text format) are imported into a relational database (Microsoft™ SQL Server 2000™) and processed through a screening algorithm to remove duplicates. Results are stored in separate tables in the relational database. Data are presented with a 3-tiered-architecture web application developed using Microsoft™ Dot Net™ technologies. A web browser is used as the user interface allowing access through the Queensland Health intranet or through remote access secure internet connection. The 'unique' isolate definition used for duplicate removal uses the following parameters: identical patient identifiers (name, patient record number, date of birth), identical organism name, identical susceptibility pattern and isolation within five days of a previous 'identical' isolate. Specimen type is also included in the algorithm from specimen category inquiries. Data from patients with multiple isolates were audited to ensure that the algorithm selected the first isolates of a particular profile and excluded all duplicates.

The database can be queried for all specimens or for particular categories of specimens. Specimens are categorised as blood, cerebrospinal fluid, ear/nose/throat, enteric, genital, respiratory, tissue/fluid/pus/prostheses, urine, infection control screening and other. Ad hoc inquiries are processed according to the following parameters: testing laboratory, health care facility, ward address, inpatient/outpatient status, zone, district, year/month, organism, antimicrobial, and specimen type. Data for clinical isolates and infection control screening isolates can be analysed separately. Reports state the number of isolates tested and the percentage susceptible.[8] For demonstration purposes the results reported here reflect results as recorded in the LIS. These would not necessarily have appeared in the pathology report.

Results

Susceptibility results were available for 52,563 isolates between 1 January and 30 June 2003. The screening algorithm excluded 6.5 per cent of isolates leaving 49,169 in the active database including 48,096 clinical and 1,073 infection control screening isolates. Some of the flexible data analysis capabilities of the system are demonstrated in the tables.

Tables 1 and 2 show the susceptibilities of common gram-negative and gram-positive blood culture isolates from all laboratories respectively. The variation of the proportion of blood culture isolates of *Staphylococcus aureus* that were methicillin-resistant (MRSA) in the three zones is shown in Table 3. Differences in the susceptibility of inpatient and outpatient MRSA isolates at one Southern Zone hospital are seen in Table 4. The ability to differentiate between clinical and infection control screening isolates is demonstrated by data from the same hospital (Table 5). Summary data for extended spectrum beta-lactamase (ESBL) producing *Klebsiella pneumoniae* and vancomycin resistant enterococci

Table 1. Antimicrobial susceptibilities of the 10 most common aerobic gram-negative isolates in blood cultures in all laboratories, January to June 2003

Organism	Amikacin		Amoxycillin		Cefotaxime		Ceftazidime		Cephalothin		Ciprofloxacin		Gentamicin		Meropenem		Timentin	
	%S	n	%S	n	%S	n	%S	n	%S	n	%S	n	%S	n	%S	n	%S	n
Acinetobacter baumannii	90.2	41	3.1	32	42.9	42	50.0	38	0.0	32	70.5	44	70.5	44	62.5	33	72.7	44
Enterobacter aerogenes	100.0	13									92.3	13	100.0	13	100.0	10		
Enterobacter cloacae	98.1	53									100.0	55	84.2	57	100.0	44		
Escherichia coli	100.0	484	54.7	525	99.8	492	100.0	428	69.4	520	99.0	514	99.4	526	100.0	371	89.2	454
Klebsiella oxytoca	100.0	27	0.0	28	96.4	28	100.0	21	63.0	27	100.0	28	96.4	28	100.0	17	84.6	26
Klebsiella pneumoniae	100.0	142	0.0	150	97.9	143	97.5	122	91.0	145	97.2	145	96.7	150	100.0	112	94.0	133
Proteus mirabilis	100.0	30	90.6	32	100.0	31	100.0	25	75.9	29	93.6	31	96.9	32	100.0	21	100.0	30
Pseudomonas aeruginosa	97.4	114					94.0	117			95.0	119	94.2	120	95.0	99	89.2	120
Serratia marcescens	97.4	38									100.0	37	92.1	38	100.0	29		
Stenotrophomonas maltophilia							60.0	15			43.8	16	33.3	3	0.0	1	37.5	16

Agent not recommended or not tested for this species.

Table 2. Antimicrobial susceptibilities of the 10 most common aerobic gram-positive isolates in blood cultures in all laboratories, January-June 2003 (excluding common skin flora)

Organism	Amoxycillin		Ciprofloxacin		Clindamycin		Erythromycin		Fusidic acid		Gentamicin		Penicillin G		Rifampicin		Vancomycin	
	%S	n	%S	n	%S	n	%S	n	%S	n	%S	n	%S	n	%S	n	%S	n
Enterococcus faecalis	100.0	59					21.2	33									100.0	59
Staphylococcus aureus			98.3	342	100.0	242	89.1	367	95.6	296	99.1	349	16.3	368	100.0	342	100.0	367
Staphylococcus aureus (MRSA)			44.9	69	61.5	52	22.2	72	95.8	72	36.6	71	0.0	72	95.8	72	100.0	72
Streptococcus agalactiae (Group B)	100.0	24					88.9	36					100.0	44			100.0	33
Streptococcus milleri group	100.0	14					95.8	24					100.0	36			100.0	26
Streptococcus mitis	100.0	5					80.0	10					73.3	15			100.0	10
Streptococcus oralis	100.0	7					66.7	9					73.3	15			100.0	9
Streptococcus pneumoniae							84.2	101					90.5	105			100.0	93
Streptococcus pyogenes (Group A)	100.0	13					96.9	32					100.0	46			100.0	28
Streptococcus sp. Group G	100.0	10					92.3	26					100.0	32			100.0	27

Agent not recommended or not tested for this species.

Table 3. Proportion of methicillin-resistant *Staphylococcus aureus* (MRSA) among blood culture isolates of *Staphylococcus aureus* and proportion of gentamicin susceptible MRSA, January to June 2003

Zone	Total *Staphylococcus aureus*	Methicillin-resistant *Staphylococcus aureus*		MRSA gentamicin susceptible
		n	%	%
Northern	94	11	11.7	72.7
Central	143	24	16.8	17.4
Southern	130	22	16.9	31.8

Table 4. Antimicrobial susceptibility of inpatient and outpatient isolates of methicillin-resistant *Staphylococcus aureus* at a metropolitan teaching hospital, January to June 2003

Antibiotic	Inpatients		Outpatients	
	%S	n	%S	n
Vancomycin	100.0	267	100.0	25
Fusidic acid	89.9	267	80.0	25
Clindamycin	74.9	267	84.0	25
Ciprofloxacin	43.1	267	76.0	25
Gentamicin	38.2	267	72.0	25
Tetracycline	33.7	267	72.0	25
Erythromycin	22.1	267	44.0	25
Rifampicin	78.2	266	92.0	25
Mupirocin	99.2	127	100.0	3

Table 5. Comparison of susceptibility of all methicillin-resistant *Staphylococcus aureus* isolates with those from clinical specimens and those from infection control screening specimens at a metropolitan teaching hospital, January to June 2003

Antimicrobial	All isolates		Clinical isolates		Screening isolates	
	%S	n	%S	n	%S	n
Ciprofloxacin	45.9	292	57.6	165	30.7	127
Clindamycin	75.7	292	80.0	165	70.1	127
Erythromycin	24.0	292	31.5	165	14.2	127
Fusidic acid	89.0	292	90.9	165	86.6	127
Gentamicin	41.1	292	48.5	165	31.5	127
Mupirocin	99.2	130	100.0	17	99.1	113
Rifampicin	79.4	291	81.8	165	76.2	126
Tetracycline	37.0	292	39.4	165	33.9	127
Vancomycin	100.0	292	100.0	165	100.0	127

(VRE) for the three zones are shown in Tables 6 and 7 respectively. Cumulative susceptibility to a variety of antimicrobials and reduced susceptibility to penicillin of *Streptococcus pneumoniae* isolated from sterile and non-sterile sites are shown in Table 8.

Discussion

We have endeavoured to display the versatility of the antibiogram software by presenting data derived from the entire state, the three Queensland Health zones and from an individual institution. The data are presented as recorded as the system does not at present include software to automatically identify improbable results. The current version of software does however provide a 'drill down' feature which allows individual anomalous results to be identified by laboratory number and testing laboratory for follow up.

We presented data concerning some key endemic and emerging resistant organisms. MRSA is of particular interest in Queensland due to the emergence of non-multiresistant strains causing severe community acquired infections.[9,10] Gentamicin susceptibility has been used as a surrogate marker for these strains and Table 3 shows marked differences in the gentamicin susceptibility of MRSA in the three zones. This suggests that community strains are probably most common in the Northern Zone and least common in the Central Zone. Examination of MRSA susceptibilities at one metropolitan teaching hospital in Table 4 demonstrates that outpatient isolates are more susceptible to non-beta-lactam antimicrobials, which is also in keeping with community acquisition of non-multiresistant strains. The varying cumulative susceptibility results displayed in Table 5 demonstrate the importance of separating results of infection control screening isolates from clinical isolates when reporting cumulative susceptibilities, in this case for MRSA.

Table 6. Antimicrobial susceptibility of extended spectrum beta-lactamase producing *Klebsiella pneumoniae* isolated in Queensland Health Zones, January to June 2003

Antibiotic	Northern zone		Central zone		Southern zone	
	%S	n	%S	n	%S	n
Amikacin	100.0	36	98.0	51	100.0	12
Amoxycillin	0.0	40	0.0	50	0.0	12
Cefotaxime	20.5	39	7.5	40	81.8	11
Ceftazidime	2.6	38	5.7	35	27.3	11
Cephalothin	2.6	38	0.0	50	0.0	12
Ciprofloxacin	43.6	39	89.8	49	16.7	12
Co-trimoxazole	13.2	38	15.7	51	16.7	12
Gentamicin	5.0	40	2.0	51	0.0	12
Imipenem	100.0	37	97.9	48	100.0	12
Meropenem	100.0	31	97.6	41	100.0	11
Netilmicin	nt	nt	66.7	3	100.0	11
Trimethoprim	9.1	33	9.8	41	27.3	11

nt Not tested

Table 7. Vancomycin resistant enterococci isolated from screening specimens in Queensland Health zones, January to June 2003

Zone	*Enterococcus faecium* (van B phenotype)	*Enterococcus faecalis* (van B phenotype)
Northern	0	0
Central	28	2
Southern	1	2

Table 8. Antimicrobial susceptibilities of *Streptococcus pneumoniae* in all laboratories, January to June 2003

Antimicrobial	Sterile sites			Non-sterile sites		
	%S	%I	n	%S	%I	n
Chloramphenicol	97.3		74	98.5		401
Co-trimoxazole	64.3		56	74.1		325
Erythromycin	82.6		109	85.1		578
Penicillin G	89.3	4.5	112	81.1	9.0	586
Tetracycline	90.9		77	85.0		454
Vancomycin	100.0		101	100.0		501

Comparison of susceptibilities of ESBL producing *K. pneumoniae* to cephalosporins in the three zones in Table 6 illustrates another potential pitfall in interpreting cumulative susceptibility results from different laboratories. The marked differences in susceptibility recorded here is due to differing reporting practices between laboratories: some record results as tested and suppress them while others record all as resistant and report them. Clearly, knowledge of reporting practices for organism/antibiotic combinations where susceptibility phenotypes are not reliable indicators of clinical utility, is an important element in reporting and interpreting cumulative data.

Analysis of infection control screening for VRE showed a large number of isolates of van B *E. faecium* in the Central Zone (Table 7). Drilling down revealed that this was due to an outbreak in one institution only. The emergence of resistance to penicillin and other antimicrobials in *S. pneumoniae* has been evident in Australia for over a decade.[11] Table 8 shows that, while isolates from blood and CSF are, as expected, more susceptible than isolates from non-sterile sites, the proportion of resistant isolates (penicillin MIC $\geq$ 2 mg/L) from sterile sites is 6.2 per cent, which is cause for concern.

The method of data transfer employed makes this passive surveillance system adaptable to output from any modern LIS. The system also allows for internet access with appropriate security. These features suggest it could be relatively easily adapted to provide the basis for a national system of passive surveillance. National input into specification of such a system would be required and a mechanism for providing this through the Australian Group for Antimicrobial Resistance has been proposed. While the Commonwealth has stated its commitment to the surveillance approach suggested by JETACAR,[12] a practical and cost effective solution to the requirement for passive surveillance is yet to be implemented. We suggest that the system described would, with appropriate modification, satisfy the requirement for national passive surveillance of antimicrobial susceptibility.

Acknowledgement

We acknowledge the contribution of the Microbiology Discipline Working Party and the Auslab Support Unit of QHPSS.

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Letter to the Editor

Was the egg a plausible source for the *Salmonella* Potsdam outbreak?

The paper, *Outbreak of* S. *Potsdam associated with salad dressing at a restaurant* (*Commun Dis Intell* 2003;27:508–512), implicated the egg as the 'most plausible source of *S.* Potsdam'. No tangible evidence to support this statement was provided and broader possibilities were not considered. The reasons for implicating the eggs were that another *Salmonella* serovar (*Salmonella* Infantis) was found on cloth used to clean the eggs and the egg was an ingredient common to the *S.* Potsdam positive dressings.

Is the presence of one serovar in the environment an indication of the presence of another *Salmonella* serovar in the same environment? Unless a scientific reference can support *S.* Infantis presence in environmental samples as an indicator for the presence of *S.* Potsdam, the scientific validity of the argument implicating the eggs is unsustainable.

Was the egg the only ingredient common to all *S.* Potsdam positive dressings?

Salad dressings were prepared by dividing a single batch of mayonnaise to make the five salad dressings (p. 510). *S.* Potsdam was only found in two. This could indicate that the mayonnaise base was not the primary source but that the two salad dressings once they had acquired *S.* Potsdam through cross contamination from another source merely provided good media for multiplication.

If the mayonnaise was the primary source, the main ingredient in the mayonnaise base was vegetable oil that in a kitchen with hygienic standards as described in the paper, could have been subject to cross contamination. Mayonnaise is prepared in restaurant kitchens by manual separation of the egg yolk and the white. Therefore, human hands were another possible common denominator. *S.* Potsdam has been isolated in Australia from potable water (J Powling, National Enteric Pathogens Surveillance System, personal communication). It does not appear that the water, a common denominator to all aspects of food preparation, was tested.

The human cases 'ate a variety of food items' and 73 per cent of the human cases did not consume salad dressings or egg-containing food items. The paper, while acknowledging this discrepancy, argued that the kitchen practices were conducive to cross contamination and this could explain the occurrence of further cases in people that had not eaten food that contained eggs. Is cross contamination possible only from eggs to other food items? A timely and broader range of food sampling could have cast light on this aspect. However, in view of the lack of tangible evidence to implicate the eggs and the opportunity for cross contamination 'between raw and prepared food' (p. 511), it is puzzling why cross contamination from other food items to food containing eggs was not considered as a plausible explanation.

A lag of 15 days was reported between the time the first case had eaten at the restaurant and the collection of samples that yielded *S.* Potsdam. During this time the bottles described as 'stained and containing food residues and odour, standing at high ambient temperatures', were continually handled by patrons and staff and topped up with 'partly used dressing stocks' (p. 511). The finding of *S.* Potsdam under these circumstances adds considerable weight to the hypothesis that the dressings were not the primary food vehicle.

Investigations of human food poisoning are likely to benefit from a broad epidemiological approach and from knowledge of farming practices.

The paper states that 'meat meal was the major component of laying hen feed at egg producer A'. This is a distorted view of practices in the poultry industry and places distorted epidemiological significance on the meat meal. Poultry rations are grain based and meat meal is not the major component in any poultry ration.

In April/May 2002, shortly after this outbreak, *S.* Potsdam, by the paper's own admission, 'a relatively uncommon serovar in Australia' (page 508), was reported in 10 adults in the Richmond-Tweed region of New South Wales. In late November until the end of December 2002 a multi-state cluster of *S.* Potsdam was reported from New South Wales, the Australian Capital Territory, Victoria and Tasmania. Fresh produce from Queensland was suspected as a source (July 2003 NEPSS annual report). Because of the significant chances for cross contamination in the restaurant and the lack of tangible evidence implicating the eggs, it is plausible that fresh produce in the restaurant was the source of *S.* Potsdam as early as February 2002.

The only *S.* Potsdam from egg source during 1988 to 2002, was from raw egg pulp (and not eggs, p. 508) in Western Australia in July 1990 (J Powling, personal communication). Egg pulp is harvested by manually or mechanically removing the egg contents from the shell. This process provides ample opportunities for cross contamination with bacteria that may not be present in the layer shed, or if present would not normally present a significant hazard.

Between 1996 and 2003, as part of the New South Wales Salmonella Enteritidis-Free Accreditation Scheme, 3,470 layer farm tests have been done on a monthly basis. *S.* Potsdam has never been detected (nor has *S. Enteritidis*). In a survey in Queensland. (J Cox, 9th Australian Poultry and Feed Convention, Gold Coast 1993), a variety of *Salmonella* spp. were reported in the Queensland layer environment but *S.* Potsdam was not detected on any of the 60 farms. These are significant, consistent epidemiological findings that provide additional evidence to question the plausibility of the eggs as a source in this case.

Although the majority of affected patrons in the restaurant did not consume egg-containing food, the egg farm was sampled on the 18 February 2002, two days before the *Salmonella* culture results from the various restaurant food items were available (personal communication, the Egg Producer). This perhaps demonstrates that eggs were considered the source before the facts were known and despite very strong hints suggesting other possibilities, none were elucidated. Indeed, the evidence in the paper and the broader epidemiological picture tends to suggest that eggs were unlikely to be the source of *S.* Potsdam and investigations of food poisoning require, from the onset, objective assessment of all possibilities.

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NSW Department of Primary Industries

Response to Letter to the Editor:

Salmonella Potsdam and eggs

In response to George Arzey's letter regarding the paper *Outbreak of* Salmonella *Potsdam associated with salad dressing at a restaurant, Commun Dis Intell* 2003;27:508–812.

As the title of our paper states we reported an outbreak of *Salmonella* Potsdam associated with salad dressing at a restaurant. We were able to culture *S.* Potsdam from two bottles of salad dressing at the restaurant. Given the difficulty in recalling food consumption many weeks past, it is not surprising that many of the cases could not recall consuming the contaminated salad dressings.

There are potential explanations for this outbreak, other than shell eggs. Alternative sources of *S.* Potsdam were investigated and described. *Salmonella* testing was undertaken on ingredients of the dressings (fresh dill, horseradish relish, anchovies, vinegar, parmesan cheese, vegetable oil, salt and pepper, sour cream, mustard) and other vehicles at the restaurant considered plausible, based on previous data on the distribution of *S.* Potsdam (including swabs of drinking water bottles) and all were found negative.

However, we made extensive commentary on shell eggs for three reasons:

1. Shell eggs found at the restaurant were heavily faecally contaminated and, given the restaurant's handling methods, represented a major food safety hazard at the time of our inspection.

2. The environmental findings at the egg producer identified multiple hazards in an industry that is currently unregulated and subject only to voluntary codes of practice under which only some part of the egg industry operate. Some of the most concerning hazards included *Salmonella* contamination of environmental surfaces including bulk feed and feed troughs, and numerous opportunities for cross-contamination between bird droppings and eggs.

3. Sixteen of 18 cultures collected from the egg producer, including meat meal and drinking water for the chickens, identified *Salmonella.*

Leanne Unicomb
Phillip Bird
Craig Dalton

What do we know about 7vPCV coverage in Aboriginal and Torres Strait Islander children?

Brynley P Hull,[1,2] Peter B McIntyre[3,4]

Abstract

In 2001, a publicly funded pneumococcal conjugate vaccine (7vPCV) program commenced for Aboriginal and Torres Strait Islander children aged under two years. At present, there is very little knowledge about the uptake of 7vPCV vaccine amongst Aboriginal and Torres Strait Islander children. This study examined the rollout and use of 7vPCV vaccine in Australia and estimated immunisation coverage for Indigenous children at the age of 12 months for 7vPCV vaccine. To calculate 7vPCV coverage we chose four consecutive 3-month birth cohorts born between 1 October 2001 and 30 September 2002. The immunisation status of children in each birth cohort was assessed at 12 months for the third dose of 7vPCV vaccine. The largest absolute number of 7vPCV doses was given in Queensland, the Northern Territory and New South Wales. As the 7vPCV program matured, a progressively higher proportion of total doses was administered to children under the age of 12 months consistent with the introduction of the program. For all jurisdictions except the Northern Territory and Western Australia, where it has remained reasonably constant, estimated coverage increased over the most recent birth cohorts but was still less than 50 per cent for all states except the Northern Territory, Queensland, and Western Australia. This study provides the first national measure of 7vPCV immunisation coverage among Indigenous children in Australia. With the likely improvement over time in the recording of 7vPCV vaccinations and Indigenous status on the Australian Childhood Immunisation Register, the validity of coverage estimates is likely to increase. *Commun Dis Intell* 2004;28:238–243.

Keywords: pneumococcal, vaccine, vaccination, indigenous children, Aboriginal, Torres Strait Islander

Introduction

Invasive pneumococcal disease became largely preventable in Australian children aged less than two years, for the first time, with the approval in December 2000, of the use of a pneumococcal conjugate vaccine to protect against the seven most common serotypes. Indigenous children in Central Australia have the highest documented incidence of invasive pneumococcal disease in the world, with high rates also seen in Indigenous children in other parts of northern Australia.[1,2,3] In 2001, a publicly funded pneumococcal conjugate vaccine (7vPCV) program commenced for children at high risk (Indigenous children under two years, Indigenous children in Central Australia aged up to five years, non-Indigenous children living in Central Australia aged up to two years, and all children with predisposing medical conditions aged under five years). The 7vPCV vaccine was first used in the Northern Territory, from June 2001, with a progressive rollout in other jurisdictions. At present, there are few data on the uptake of 7vPCV vaccine amongst Aboriginal and Torres Strait Islander children, especially in regions outside the Northern Territory.

Since May 2001, 7vPCV vaccination encounters have been recorded on the Australian Childhood Immunisation Register (ACIR). From the immunisation data finally entered onto the ACIR, the Health Insurance Commission (HIC) provides regular quarterly coverage reports at the national and state and

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territory level, calculated using the cohort method.[4] A cohort of children is defined by date of birth in three-month groups, assessed at the three key ages of 12 months, 24 months, and six years of age.

Initially, the field for recording Aboriginal and Torres Strait Islander status on the ACIR was incomplete and not used for official reports. However, since mid-2001, as completeness has improved, calculation of immunisation coverage estimates for Indigenous children using the ACIR has been made available to state and territory health departments.

The aims of the study were to:

1. Describe the rollout and use of 7vPCV vaccine in Australia since May 2001 by state, remoteness index, date of administration, and number of doses.

2. Calculate the number of children in a 12-month birth cohort assessed as Aboriginal or Torres Strait Islander on the ACIR, by state, and to compare these figures with the Australian Bureau of Statistics (ABS) estimates of the numbers of Aboriginal and Torres Strait Islander children by state.

3. Estimate immunisation coverage for four cohorts of Aboriginal and Torres Strait Islander children at the age of 12 months for 7vPCV vaccine and compare it with 'fully immunised' coverage at 12 months of age.

Methods

Immunisation status assessment

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases receives downloads of ACIR immunisation data from the HIC each quarter. This analysis was undertaken using ACIR data as at 31 December 2003. As pneumococcal conjugate vaccines were first recorded on the ACIR from May 2001, we analysed all vaccine encounters from this date onwards. We chose four 3-month birth cohorts, the first born between 1 October 2001 and 31 December 2001, the second born on 1 January 2002 to 31 March 2002, the third born on 1 April 2002 to 30 June 2002, and the fourth born on 1 July 2002 to 30 September 2002. The immunisation status of children, identified on the ACIR as Aboriginal and Torres Strait Islander, in the four birth cohorts was assessed at 12 months of age for pneumococcal conjugate vaccine. The third dose assumption was applied in this analysis.[5] The analysis was undertaken using the SAS software system.[6]

Aboriginal and Torres Strait Islander population denominators

In addition to ACIR Indigenous denominator data, data on the number of Aboriginal and Torres Strait Islander births in all Australian states and territories for the year 2002 was also obtained from the Australian Bureau of Statistics.[7] There are four estimates of the number of Aboriginal and Torres Strait Islander births in Australia calculated each year by the ABS. Each is based on a different collection, with a different propensity to identify Aboriginal or Torres Strait Islander status.[7,8] In this study, we used the expected Aboriginal and Torres Strait Islander births from 1996 Census-based experimental estimates and projections. These use the number of Aboriginal and Torres Strait Islander children in the 1996 Census to estimate fertility rates for 1996. Assuming this fertility rate to continue, and making other assumptions about mortality and interstate migration, the number of births in subsequent years was projected.

Accessibility/remoteness status

Based on the residential address postcode recorded on the ACIR, the residential status of children was defined as accessible or remote using the Accessibility/Remoteness Index of Australia (ARIA) developed by the then Department of Health and Aged Care.[9] There are five ARIA categories:

highly accessible – relatively unrestricted accessibility to a wide range of goods and services;

accessible – some restrictions to accessibility of some goods and services;

moderately accessible – significantly restricted accessibility of goods and services;

remote – very restricted accessibility of goods and services; and

very remote – very little accessibility of goods and services.

The ABS views the ARIA methodology as a suitable means to determine the remoteness of any part of Australia. The ARIA is being used for a variety of policy, administrative and statistical purposes and is used by a number of government agencies as a definition of remoteness. It is becoming a 'de-facto standard' on remoteness and was proposed as the national standard measure of remoteness for inclusion in the ABS 2001 census.[10]

Sensitivity analysis

A sensitivity analysis of the 7vPCV coverage estimates was also undertaken to assess the effects on coverage of using ABS population estimates as the denominator and using all doses of 7vPCV given to children on the ACIR as the numerator, with the assumption that only Indigenous children receive 7vPCV vaccine.

Results

The total number of 7vPCV doses recorded in Australia since mid-2001 are shown in Table 1. The largest absolute number of doses was given in Queensland, the Northern Territory, New South Wales and Western Australia. Three of these jurisdictions have the largest rural and remote Aboriginal and Torres Strait Islander populations and had the largest proportion of doses given to children residing in remote areas (Table 1). More than 80 per cent of 7vPCV doses were given to children residing in remote or very remote areas in the Northern Territory, although only 53 per cent of the total 0–4 year ACIR child population in the Northern Territory resides in these areas (not shown). Indeed, all jurisdictions where there is a substantial population residing in remote or very remote areas had a disproportionate amount of 7vPCV administered to children residing in these areas, in keeping with it being given predominantly to Indigenous children (Table 1).

An indication of the rollout of 7vPCV vaccination in each jurisdiction of Australia from May 2001 to December 2003 is provided in Table 2. For Australia as a whole there has been a steady increase in the number of 7vPCV doses administered over time with a large increase in the most recent 6-month period to December 2003 (Table 2). The Northern Territory, Western Australia and Queensland were the first jurisdictions to implement their 7vPCV programs and vaccinate significant numbers of children with 7vPCV. New South Wales, Victoria, South Australia and Tasmania were much later in rolling out their programs and have only begun to vaccinate significant numbers of children in the past year.

Approximately 40 per cent of all 7vPCV vaccine administered was given only as a single dose, suggesting that it was part of a catch-up campaign (not shown). This is consistent with 90 per cent of children who were aged between 12 and 32 months as at 31 December 2003 and aged 18 months or more at their first dose of 7vPCV having received just the one dose, and 43 per cent of children who were aged between 12 and 32 months as at 31 December 2003 and aged 7–17 months at their first dose of 7VPCV having received two doses of 7VPCV vaccine (Table 3). As the program has matured, a progressively higher proportion of total doses was administered to children under the age of 12 months, consistent with the tailing off of catch-up programs, the introduction of the program and the birth of new babies (not shown).

Table 1. Receipt of 7vPCV vaccine doses administered from May 2001 to December 2003 and recorded on the Australian Childhood Immunisation Register as at December 2003, by state or territory and accessibility/remoteness status

State	Highly accessible		Accessible		Moderately accessible		Remote		Very remote		Total	
	n	%	n	%	n	%	n	%	n	%	n	%
ACT	983	73.5	111	8.3	53	4.0	53	4.0	137	10.2	1,337	1.7
NSW	9,200	56.9	4,374	27.0	1,430	8.8	934	5.8	242	1.5	16,180	20.3
NT	450	2.7	4,781	10.7	752	4.5	3,445	20.7	10,185	61.3	16,613	20.9
Qld	7,166	28.6	6,690	26.9	3,852	15.5	1,456	5.9	5,752	23.1	24,866	31.2
SA	2,209	65.0	591	17.4	131	3.9	154	4.5	312	9.2	3,397	4.3
Tas	278	65.6	122	28.8	23	5.4	0	0.0	1	0.2	424	0.5
Vic	6,162	92.2	446	6.7	67	1.0	6	0.09	4	0.06	6,685	8.4
WA	2,436	24.1	1,069	10.6	1,216	12.0	893	8.8	4483	44.4	10,097	12.7
Aust	28,834	36.2	15,184	19.1	7,524	9.5	6,941	8.7	21,116	26.5	79,599	

Table 2. The roll-out of 7vPCV vaccination doses administered from May 2001 to December 2003 and recorded on the Australian Childhood Immunisation Register as at December 2003, by state or territory

State	May to December 2001		January to June 2002		July to December 2002		January to June 2003		July to December 2003		Total
	n	%	n	%	n	%	n	%	n	%	n
ACT	160	11.4	232	16.5	289	20.5	253	18.0	474	33.7	1,408
NT	5,043	28.2	3,840	21.5	3,290	18.4	2,960	16.6	2,742	15.3	17,875
NSW	195	1.2	1,987	12.0	3,700	22.3	4,038	24.4	6,638	40.1	16,558
Qld	3,834	15.1	4,544	17.9	5,080	20.0	5,383	21.1	6,612	26.0	25,453
SA	9	0.3	688	20.0	623	18.1	688	20.0	1,434	41.7	3,442
Tas	20	4.6	27	6.2	44	10.1	78	17.9	266	61.1	435
Vic	85	1.3	480	7.1	1,243	18.4	1,415	20.9	3,536	52.3	6,759
WA	2,576	24.2	2,310	21.7	2,020	19.0	1,842	17.3	1,906	17.9	10,654
Aust	11,922	14.4	14,108	17.1	16,289	19.7	16,657	20.2	23,608	28.6	82,584

Table 3. Age at the first dose of 7vPCV vaccination administered to children aged 12 to 32 months and recorded on the Australian Childhood Immunisation Register as at December 2003, by the total number of 7vPCV doses received

Age at first dose of 7vPCV	Number of total 7vPCV doses received		
	1 dose (%)	2 doses (%)	3 doses (%)
0–3 months	3.8	7.9	88.3
3–5 months	6.2	16.5	77.3
5–7 months	16.2	24.8	58.9
7–17 months	47.6	42.7	9.7
18+ months	90.0	10.2	0.2

Table 4 shows the number of Indigenous children recorded in Australia using two measures. The first was the number of children as measured by the Indigenous indicator on the ACIR in 2002, and the second, the best available estimates from the ABS. In most jurisdictions the numbers of children identified as Indigenous by the ACIR compared quite favourably with the ABS estimates. The numbers of children in the Northern Territory identified as Indigenous by the ACIR almost mirrored that of the ABS estimates, and the ACIR estimated greater than 68 per cent of the ABS numbers of Indigenous children in five other jurisdictions: Western Australia (85%), New South Wales (76%), South Australia (75%), Victoria (73%) and the Australian Capital Territory (69%).

Table 4. Comparison of the number of Aboriginal and Torres Strait Islander children, Australian Childhood Immunisation Register (ACIR) data versus Australian Bureau of Statistics (ABS)

State	Indigenous population		Accuracy of ACIR Indigenous data (%)‡
	ACIR data 2002*	ABS 2002 – (births)†	
ACT	72	105	68.6
NSW	2,714	3,568	76.1
NT	1,422	1,445	98.4
Qld	489	3,493	14.0
SA	498	665	74.9
Tas	109	482	22.6
Vic	495	680	72.8
WA	1411	1,653	85.4
Aust	7,210	12,094	59.6

* Numbers for the 12-month birth cohort born 1 January to 31 December 2002).

† Australian Bureau of Statistics. Births Australia 2002. Canberra: Australian Bureau of Statistics. Projected indigenous births from the 1996 Census.

‡ (The number of children estimated by the ACIR/the number of Census projection births)*100.

Table 5 shows a comparison of 7vPCV vaccine coverage estimates for Indigenous children for the four study birth cohorts. Data from Queensland on the Indigenous indicator had not been transmitted to the ACIR but coverage estimates were available from a Vaccine Information and Vaccine Administration System (VIVAS) analysis in 2002. For many jurisdictions, estimated coverage increased from the earliest cohort presented but was still less than 50 per cent in five jurisdictions. The Northern Territory was the best performer with greater than 74 per cent coverage for all four cohorts, well above other jurisdictions. With the exception of the Northern Territory, 7vPCV vaccine coverage estimates in all jurisdictions were considerably lower than 'fully immunised' coverage estimates for all scheduled vaccines at 12 months (Table 5). In a sensitivity analysis, estimated 7vPCV coverage was lower if the denominator was the ABS estimates and the numerator was Indigenous children receiving 7vPCV (not shown). Coverage increased considerably if *all* doses of 7vPCV on the ACIR were included (assuming only Indigenous children receive 7vPCV). Overall, it is difficult to estimate the relative contribution of under-immunisation of Indigenous children and under-enumeration of Indigenous status, but coverage appears unlikely to be greater than 60 per cent in any jurisdiction other than the Northern Territory.

Discussion

This analysis of recent ACIR data provides the first national measure of pneumococcal conjugate vaccine immunisation coverage among Indigenous children in Australia and, also provides the first examination of the rollout and pattern of use of 7vPCV vaccine in Australia by state, remoteness index, and number of doses. The main finding is that 7vPCV coverage amongst Indigenous children in Australia increased over the most recent birth cohorts but, although differing substantially, was sub-optimal in all states and territories except the Northern Territory. Using the more accurate Indigenous child denominator from ABS data rather than the ACIR denominator, coverage was even lower. Of particular note was that the proportion of Indigenous children in the four cohorts 'fully immunised' for age was considerably greater than 7vPCV coverage in all jurisdictions except the Northern Territory, and only slightly lower than for all children at 12 months of age.[11] This suggests that either 7vPCV is being administered to Indigenous children, but not being reported by providers in most jurisdictions, or 7vPCV is not being administered despite children being identified as Indigenous on the ACIR (although not necessarily at the point of immunisation service). Differential reporting is quite likely as the ACIR encounter form does not have a specific box for

Table 5. A comparison of 7vPCV and 'fully immunised' coverage estimates calculated from the Australian Childhood Immunisation Register for Aboriginal and Torres Strait Islander children born in four 3-month birth cohorts

State	Number*	% coverage for cohort born 1/10/01 – 31/12/01		% coverage for cohort born 1/1/02 – 31/3/02		% coverage for cohort born 1/4/02 – 30/6/02		% coverage for cohort born 1/7/02 – 30/9/02	
		7vPCV (median age at 3rd dose – months)	Fully immunised†	7vPCV (median age at 3rd dose – months)	Fully immunised	7vPCV (median age at 3rd dose – months)	Fully immunised	7vPCV (median age at 3rd dose – months)	Fully immunised
ACT	73	0.0 (na)	100.0	8.7 (8.1)	82.6	11.1 (9.6)	92.6	6.7 (11.5)	80.0
NSW	2,692	16.5 (8.8)	86.2	27.8 (6.8)	84.5	31.8 (7.1)	87.1	36.9 (6.8)	84.7
NT	1,424	79.8 (6.8)	88.9	75.0 (7.0)	85.9	80.2 (6.9)	85.3	74.1 (7.3)	79.2
Qld‡		58.0		58.0		58.0		58.0	
SA	513	39.1 (8.5)	80.9	46.5 (7.1)	84.5	37.9 (7.2)	81.9	44.9 (7.0)	81.1
Tas	122	9.1 (7.0)	87.9	9.7 (7.6)	90.3	3.2 (6.4)	87.1	7.4 (7.7)	88.9
Vic	519	12.0 (7.2)	86.5	15.2 (7.6)	86.2	14.8 (8.6)	88.3	16.7 (7.6)	88.3
WA	1,421	53.2 (7.2)	83.8	52.8 (7.1)	79.5	58.6 (7.1)	82.6	50.7 (6.9)	77.5
Aust	7,212	39.1	85.8	43.3	83.9	46.9	85.8	46.1	81.9

* Total number of Indigenous children in the four cohorts born 1 October 2001 to 30 September 2002.

† All of the third doses of diphtheria-tetanus-pertussis (DTP), poliomyelitis, and *Haemophilus influenzae* type b (Hibtiter) (or second dose Pedvax Hib) vaccines, and a 2nd or 3rd dose of hepatitis B vaccine used to assess whether a child is completely immunised at 12 months of age.

‡ Figure for Queensland calculated from VIVAS data. The proportion of Aboriginal and Torres Strait Islander children fully vaccinated for age for 7vPCV, for the period 1 July 2001 to 30 June 2002.

7vPCV vaccine reporting unlike other vaccines on the schedule, and general practitioners do not receive Service Incentive Payments for reporting administration of 7vPCV vaccine. However, non-administration of 7vPCV to children identified as Indigenous is also likely.

Interpretation of the data is made more difficult by being likely to be incomplete for many jurisdictions both for 7vPCV doses and Indigenous status. Firstly, it is known that the ACIR underestimates coverage for scheduled vaccines by 3–5 per cent because of failure of providers to report to it.[12] Failure to notify the ACIR is even more likely for 7vPCV vaccine, as it is not one of the vaccines assessed for the General Practice Immunisation Incentives scheme. Secondly, the reporting of Indigenous status to the ACIR is incomplete, varies by jurisdiction, and in some jurisdictions is not transferred to the ACIR as yet.

Despite these limitations, the data reported in this study are important given the lack of other data sources and, although incomplete, provide a basis for monitoring trends. The recording of 7vPCV vaccinations on the ACIR is likely to improve further over time as seen in the increase in coverage across the four cohorts examined. The completeness of Indigenous reporting on the ACIR is also likely to improve over time. The HIC is currently undertaking various Indigenous Access initiatives, which include an amended Medicare enrolment form that enables the voluntary recording of Indigenous status. As of July 2003, more than 800 notifications of Indigenous status had been lodged with the HIC through this amended Medicare form (K. Williams, personal communication, July 2003). Further, it is hoped that existing data transmission problems with the Indigenous field on VIVAS in Queensland will be resolved in the near future. This will have a significant impact on the numbers of Indigenous children being reported on the ACIR for Australia and will result in more accurate estimates of coverage of pneumococcal conjugate vaccine amongst Indigenous children in the future.

Additional data are also likely to made available from other sources against which these findings can be measured, in particular the recent 2001 ABS survey including Aboriginal and Torres Strait Islander children 0–6 years, released this year ,and improved data from the ACIR in many jurisdictions.

Acknowledgements

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases is supported by the Australian Government Department of Health and Ageing, the New South Wales Department of Health and the Children's Hospital at Westmead.

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Age-related risk of adverse events following yellow fever vaccination in Australia

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Abstract

Reports of six deaths internationally, including one from Australia, plus other cases of severe systemic adverse events following yellow fever (YF) vaccination have raised concern about the safety of YF vaccine, particularly among older vaccinees. We investigated the age-related reporting rates of adverse events following YF vaccination reported to the Australian Adverse Drug Reactions Advisory Committee for the period 1993 to 2002. The reporting rate of systemic adverse events leading to hospitalisation or death was significantly higher among vaccinees aged ≥65 years [reporting rate ratio (RRR) 8.95, 95% confidence interval (CI) 1.49–53.5] or ≥45 years (RRR 5.30, 95% CI 1.33–21.2) compared with younger YF vaccinees. The higher reporting rates among older vaccinees are similar to those identified in the United States of America. The data highlight the importance of assessing the destination-specific risk, especially for older travellers to yellow fever endemic areas, and careful monitoring of those who are vaccinated. *Commun Dis Intell* 2004;28:244–248.

Keywords: vaccination, yellow fever

Introduction

Until recently, the live attenuated 17D strain of yellow fever (YF) vaccine had a 50 year history as a safe and effective vaccine.[1,2] Mild adverse events, including mild headaches and fevers, occur in 2–5 per cent of vaccinees. Life-threatening systemic adverse events following YF vaccination are rare. Data from the United States of America (USA) suggest that viscerotropic disease (multiple organ involvement) occurs in 1 in 400,000 YF vaccinees, while in Brazil the estimate is 1 in 1.1 million vaccinees. Severe neurological disease is estimated to occur in <1 in 8 million YF vaccinees.[3]

In 1998, two reports to the US Vaccine Adverse Events Reporting System (VAERS) of multiple organ failure in elderly YF vaccinees led to a study to assess the age-related reporting rates to VAERS of serious systemic illnesses temporally associated with YF vaccination that led to hospitalisation or death.[5] The investigators identified three deaths among elderly YF vaccinees and estimated that the reporting rate of systemic illnesses leading to hospitalisation or death was 16 times higher among vaccinees aged ≥65 years compared with those aged 25–44 years.[5]

More recently, in 2001, a further three deaths among YF vaccinees in the USA, Brazil and Australia,[6–8] were reported in *The Lancet*. Common findings were viscerotropic disease, characterised by multiple organ failure and isolation of the vaccine strain of YF virus. That publication was followed by three additional case reports from Europe which described similar serious systemic adverse events among YF vaccinees.[9–11] The majority of deaths and serious adverse events following immunisation were in adults aged 45 years or older. The US Centers for Disease Control and Prevention (CDC) commenced active surveillance of serious adverse events following YF vaccination in mid-2001.[3,4]

The study reported here was undertaken following enquiries from the US CDC, the World Health Organization and the United Kingdom about age-related rates of serious systemic adverse events among YF vaccinees in Australia. The aim of the study was to estimate the age-specific reporting rates of systemic adverse events leading to hospitalisation or death among Australian YF vaccinees and compare these rates to those published for the USA. We also estimated the expected frequency of YF vaccine-associated viscerotropic disease (YFV-AVD) in Australia, based on the observed passive reporting rates of this in the USA and YF vaccine usage in Australia.

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Methods

Data sources

Cases of adverse events following YF vaccination were identified from the Adverse Drug Reactions Advisory Committee (ADRAC) database. This is a national database which contains spontaneous reports, from 1972 onwards, of adverse events following the administration of drugs or vaccines.[12] The number of doses of YF vaccine administered by age group was estimated from sales data supplied by the sole supplier of YF vaccine in Australia and from the age distribution of YF vaccine recipients recorded in the immunisation register of a national network of travel vaccination clinics.

Cases

All reports in the ADRAC database for the period 1993 to 2002 were reviewed where:

- YF vaccine was recorded as 'suspected' of being involved in the reported adverse event (with or without other vaccines or drugs);
- the report was assigned a causality rating[12] of 'certain', 'probable' or 'possible'; and
- the age of the person was recorded and was ≥15 years.

Cases were classified as having a 'systemic' adverse event or 'other' adverse event based on the timing of the onset of the reaction following immunisation, and the symptoms and signs listed in the database (Box). Classification was done independently by two of the authors (GL, MB) who were blinded to the age of person reported. Discrepancies were resolved by consensus before ages were revealed. The definitions of 'systemic' and 'other' adverse events were based on those used by USA researchers.[5] There was insufficient information recorded in the ADRAC database about the duration of symptoms to include duration as a component of the case definitions, so they were more sensitive than those used in the USA study.[5] Cases of 'systemic' adverse events were defined as 'serious' if hospitalisation or death was recorded either in the database or on the original report card. The original report card was reviewed if information about hospitalisation or death was missing from the database.

Denominator data

Only one company sells YF vaccine in Australia (Aventis-Pasteur). They provided the annual number of doses sold to the civilian sector for the four years 1999 to 2002. Prior to 1998, most YF vaccine sold in Australia was in five-dose vials. To estimate the number of individual doses administered during 1993 to 2002, it was assumed that there had been minimal wastage of single doses in the 1999 to 2002 period and that the rate of increase in doses administered over the six years 1993 to 1998 was similar to that for the period 1999 to 2002.

YF vaccine providers in Australia require a specific license, which is reviewed regularly. The majority of providers work in travel vaccination centres. The number of doses of YF vaccine administered to the age groups <15 years, 15–24 years, 25–44 years, 45–64 years and ≥65 years was obtained from the immunisation registry of a national network of clinics for the period 1999 to 2002. This was a large sample comprising 15 per cent of the total YF vaccine sales for 1999 to 2002 and is likely to be representative of YF vaccinees. The total number of doses administered to each age group for the 10-year period analysed (1993 to 2002) was estimated from the total sales estimates and the age-distribution data.

Data analysis

Age-specific adverse events reporting rates per 100,000 doses of YF vaccine were estimated. The reporting rate ratios (RRR) of 'serious systemic' adverse events were calculated using either the 25–44 year or the 15–44 year age group as the

Box. Categories of vaccine adverse events*

Systemic adverse events – onset within two weeks of vaccination

- Neurological:† new onset seizures, encephalitis, myelitis, altered mental status, focal cranial or peripheral neurological deficits, paraesthesia, vertigo, +/– headache.
- Multi-systemic:† myalgia, arthralgia, impaired hepatic function, respiratory distress, nausea, vomiting, impaired renal function, +/– fever.

Other adverse events – onset within two weeks of vaccination

- Mild non-specific:† headache or dizziness without other symptoms or signs.
- Hypersensitivity:† rash, urticaria, +/– fever; anaphylaxis, angioedema.
- Local reaction:† localised pain, swelling, erythema or warmth at the injection site.

* Cases that were reported with more than one category of reaction were only counted once, in the most serious reaction category.

† Examples of symptoms and signs.

referent group. Epi Info software[13] was used to calculate RRR values and 95 per cent confidence intervals (CI). The reporting rates per 100,000 vaccine doses and the RRR values estimated for Australia for 1993 to 2002 were compared with those published for the USA for the period 1990 to 1998.[5] Due to the lower number of cases identified in Australia, the published USA data were combined into broader age groups (15–44 years, ≥45 years, ≥65 years) and rates, RRR values and 95 per cent confidence intervals were recalculated for comparison with Australian data.

Results

Vaccine distribution

It was estimated that 210,656 doses of YF vaccine were administered to the civilian population in Australia in the 10 years 1993 to 2002. Approximately 4.3 per cent of YF vaccinees recorded on the travel clinic network immunisation register were aged ≥65 years, while 57.2 per cent were aged between 25 and 44 years (Table 1).

Adverse events reports

For the period 1993 to 2002, we identified 42 reports of adverse events following YF vaccination in the ADRAC database that met the criteria for review. Of these, 26 (62%) met the study definition of a 'systemic' adverse event with nine (21%) defined as 'serious systemic' adverse events (Table 1). One of these nine had died of YFV-AVD.[8] No cases of severe neurological disease were identified. YF vaccine was the only suspected vaccine or drug for 15 (36%) of the 42 reports. The most common vaccines administered at the same time as YF vaccine were typhoid (n=13), hepatitis A (n=12) and oral polio (n=10).

Age-specific adverse event reporting rates

There was an increasing trend in the reporting rates per 100,000 doses of YF vaccine for both 'systemic' adverse events and 'serious systemic' adverse events among vaccinees aged 45–64 years and ≥65 years compared with the 25–44 year reference group (Table 1). However, only the reporting rate of 'serious systemic' adverse events among people aged ≥65 years was significantly higher than that of the 25–44 year age group (RRR 8.95, 95% CI 1.49–53). Comparison of reporting rates for broader age groups revealed a significantly higher reporting rate of 'serious systemic' adverse event among vaccinees aged ≥45 years compared with those aged 15–44 years (RRR 5.30, 95% CI 1.33–21). The age-related RRR values showed similar patterns to that seen in the USA (Table 2).

Expected frequency of YFV-AVD

Based on the current Australian YF vaccine distribution data of approximately 24,000 doses per annum, and the reported rates of YFV-AVD in the USA of one in 400,000 doses, the expected frequency of YFV-AVD in Australia is one case in 16 years. The case reported in 2001 who died[8] is the only one recorded in the ADRAC database, which commenced in 1972.

Discussion

This study was prompted by international concern about the risk of serious adverse events caused by YF vaccine, particularly among the older vaccinees. It was found that, like the USA, there was a significantly higher reporting rate of serious systemic adverse events (leading to hospitalisation or death) temporally associated with YF vaccination among Australians aged ≥65 years and ≥45 years, compared with younger YF vaccinees.

The study had some limitations. Cases were identified through passive surveillance and are subject to the biases inherent in systems that rely on spontaneous reporting,[12] including the possibility of age-related reporting biases. The case definitions derived from the ADRAC data were more sensitive than those used in the USA study[5] because the duration of symptoms could not be used as part of the case definition. This contributed to the estimation of higher reporting rates compared with the USA, although this is unlikely to have impacted on the trends in age-related reporting rate ratios. The denominator data were estimates only and assumed that older YF vaccinees were as likely as younger YF vaccinees to attend travel vaccination clinics. Clinic data support this, showing that older travellers are more likely to attend for yellow fever vaccination than they are for other travel-related health matters such as typhoid vaccination or antimalarial prophylaxis. It was also found that the proportion of older YF vaccinees recorded on the travel clinic immunisation register differed according to the city of residence and reflected the population structure of the local region (e.g. there was a smaller proportion of older vaccinees at the Sydney clinic, and a higher proportion at the Adelaide clinic). If older YF vaccinees were less likely to attend travel vaccination clinics, the number of doses received by this age group would be higher than was estimated and would have resulted in lower age-related adverse event reporting than estimated.

As found in the USA study, YF vaccinees reported to ADRAC had often received other vaccines at the same time and these vaccines may have caused the reported reactions. To assess this, USA researchers estimated the age-specific reporting rates of serious

Table 1. Reporting rates per 100,000 vaccine doses and reporting rate ratios for adverse events following yellow fever vaccination, Australia, 1993 to 2002

Age (years)	Number of doses	% of doses	Systemic adverse events*				Serious systemic adverse events*			
			n	%	RRR†	(95% CI)‡	n	%†	RRR	(95% CI)‡
15–24	32,423	15.4	0	0.00	0.00	–	0	0.00	0.00	–
25–44	120,552	57.2	14	11.61	1.00§	–	3	2.49	1.00§	–
45–64	48,697	23.1	9	18.48	1.59	(0.69–3.68)	4	8.21	3.30	(0.62–9.90)
≥65	8,984	4.3	3	33.39	2.88	(0.83–10.0)	2	22.26	8.95	(1.49–53.5)
15–44	152,975	72.6	14	9.15	1.00§	–	3	1.96	1.00§	–
≥45	57,681	27.4	12	20.80	2.27	(1.05–4.91)	6	10.40	5.30	(1.33–21.2)
Total	210,656	100.0	26	12.34			9	4.27		

* See Box for definitions of adverse event categories; 'serious' adverse events were those leading to hospitalisation or death.

† Rate per 100,000 doses of vaccine.

‡ Confidence interval.

§ Reference age group for comparison.

Table 2. Reporting rates per 100,000 doses and reporting rate ratios for adverse events following yellow fever vaccination, United States of America, 1990 to 1998[5]

Age (years)	Number of doses	% of doses	Systemic adverse events*				Serious systemic adverse events*			
			n	%	RRR†	(95% CI)‡	n	%	RRR†	(95% CI)‡
15–24	189,991	13.2	3	1.58	1.01	(0.28–3.6)	2	1.05	3.70	(0.52–26)
25–44	702,783	48.7	11	1.57	1.00§	–	2	0.28	1.00*	–
45–64	442,605	30.7	12	2.71	1.73	(0.76–3.9)	5	1.13	3.97	(0.77–20)
≥65‖	108,307	7.5	9	8.31	5.31	(2.2–12.8)	5	4.62	16.2	(3.2–84)
15–44‖	892,774	61.8	14	1.57	1.00*	–	4	0.45	1.00*	–
≥45‖	550,912	38.2	21	3.81	2.43	(1.2–4.8)	10	1.82	4.04	(1.3–12.9)
Total	1,443,686	100.0	35	2.42			14	0.97		

* See Box for definitions of adverse event categories; 'serious' adverse events were those leading to hospitalisation or death.

† Rate per 100,000 doses of vaccine.

‡ Confidence interval.

§ Reference age group for comparison.

‖ Reporting rates and reporting rate ratio values for these age groups were calculated from published data.[5]

systemic adverse events following hepatitis A vaccination and found no significant differences across age groups.[5] We also estimated the age-specific RRR values for systemic and serious systemic adverse events following hepatitis A vaccination, using both the Australian age-specific YF vaccine distribution data and the USA hepatitis A distribution data as denominators, and found no increase in reporting rates among older hepatitis A vaccinees (data not shown).

Australia has not followed the USA[4] in implementing active surveillance of serious adverse events following YF vaccination. It was felt that serious and life-threatening reactions are likely to be reported by clinicians and so would be detected by the current passive surveillance system. However, the Therapeutic Goods Administration (the Australian regulatory authority) did request that the vaccine manufacturer amend the product insert. This now describes increased risks with YF vaccine in individuals aged ≥65 years and includes precautions for older individuals concerning pre-vaccination evaluation of health status and post-vaccination monitoring. A statement identifying the need to balance the risk of rare reactions in elderly travellers against the risk of YF infection is also included. This information is also available in the recently published 8th edition of the *Australian Immunisation Handbook*.[14]

YF is a serious and potentially fatal disease while the YF vaccine is highly efficacious and rarely causes serious adverse events.[1–3,14] The data from this study highlight the importance of assessing the destination-specific risk, especially for older travellers to YF endemic areas, and the need for appropriate monitoring of those who are vaccinated for possible serious adverse events.

Acknowledgements

We thank Dr Ian Boyd for providing ADRAC data for analysis and Paul Cohen of Aventis Pasteur for information about yellow fever vaccine sales in Australia. The National Centre for Immunisation Research and Surveillance is supported by the Australian Government Department of Health and Ageing, New South Wales Health and The Children's Hospital at Westmead.

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Planning for human papillomavirus vaccines in Australia

Report of a research group meeting

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Background

Human papillomavirus (HPV) is the most common viral sexually transmitted infection, with estimates that up to 75 per cent of people are infected at some time.[1] Whilst most infection resolves without symptoms, some human papillomavirus infections can persist and cause cancer. In particular, the role of HPV infection in causing cervical cancer, the second most common cause of cancer in women worldwide, is undisputed. HPV exists as over 200 types but only some of these types are oncogenic (causing cervical, other anogenital cancers, oral and laryngeal cancers.) Other common HPV types cause skin warts.

The recent development of vaccines to protect against infection with oncogenic HPV types holds promise for the primary prevention of both cervical cancer and its precursors. The vaccines are based upon the Australian discovery that the major HPV capsid protein L1 can self assemble into virus-like particles (VLPs) when independently expressed in cultured cells,[2] and induce high titres of type-specific and protective neutralising antibodies in animals. These VLPs lack any viral genetic information and are not infectious. VLP based vaccines against the cancer causing HPV types 16 and 18, and against types 6 and 11 (which cause genital warts) are in current clinical trials. Early results are promising, with vaccine providing 100 per cent protection (95% CI 90-100%) against persistent infection with HPV 16.[3] These vaccines are likely to become available in the next few years. According to a recent meta-analysis of worldwide prevalence data, vaccinating against HPV 16 and HPV 18 could prevent over 70 per cent of invasive cervical cancers worldwide.[4]

Human papillomavirus research meeting objectives

With a view to initiating discussion and research into the possible future role of HPV vaccines in Australia, on 12 December 2003, the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases brought together representatives from eight Australian research groups currently involved in HPV research and both vaccine manufacturers with current HPV vaccine candidates. The two main objectives of the meeting were:

1. to identify information needs and research priorities to inform national vaccination policy decisions; and
2. to foster links between research groups.

Current state of vaccine development

During the meeting, updates on vaccine development and trial results were discussed, with results to date indicating that the vaccines are effective in preventing persistent infection with targeted HPV types, induce high titres of protective antibody (Geometric Mean Titre 80-100 times those seen in natural infection) and are safe.

Epidemiology and burden of illness

Approximately 470,000 cases of cervical cancer and 235,000 deaths occurred worldwide in 2000.[5] Most of this disease occurs in the developing world. In Australia, cervical cancer is the eleventh most

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common cancer in women. Almost 800 new cases occur annually (incidence rate 2000, 7.1 per 100,000 women) with 262 deaths in 2001.[6] A disproportionate burden of cervical cancer is borne by Indigenous women and some ethnic groups within Australia. The age-standardised death rate from cervical cancer in Aboriginal women in Western Australia, South Australia, Queensland and the Northern Territory from 1998–2001 was over four times the rate for non-Indigenous women (11.4 per 100,000 compared to 2.5 per 100,000 non-Indigenous women).[6]

During 1999/2000 63 per cent of Australian women aged 20–69 years participated in the National Cervical Screening Program, resulting in the detection of approximately 200,000 low grade lesions, 14,000 high grade lesions, and 99 cases of micro-invasive cervical cancer. During this period 646 cases of invasive cancer and 249 cervical cancer deaths were documented.[6]

International prevalence surveys of HPV types in invasive cervical cancer have shown that the most common types of HPV found in cancers are types 16 and 18.[4,7] Geographical variation in prevalence of various HPV genotypes in cancers, as well as in HPV carriage of cytologically normal populations, has been noted. For example, HPV 18 is more common in cancers in Indonesia, whilst types 52 and 58 are more common in Asia. Three studies of genotypes in invasive cervical cancer in Australia determined using polymerase chain reaction (PCR) have been published. Chen, *et al* (1999) in Melbourne, screened tumour tissue of 186 women with invasive cervical cancer, for HPVs and found that 91.9 per cent were positive using PCR.[8] Of these, 54 per cent were HPV 16, 17 per cent HPV 18 and 21 per cent other types. In 1994, Thompson, *et al*, detected HPV using a different PCR in 89 of 103 cancers (86%) with HPV 16 in 65 per cent, HPV 18 in 18 per cent and HPV 31 in three per cent in women residing in Sydney.[9] More recently, Liu, *et al* (2003) detected HPV in 90 per cent of the tumours from 79 Sydney patients with invasive cancer, with HPV 16 comprising 55 per cent, HPV 18 thirteen per cent, and coinfection with HPV 16 and 18 in 11 per cent.[10] Thus HPV types causing cancer in Australia appear to reflect global consensus, although studies in Indigenous populations are yet to be undertaken.

Modelling

The continuing development of models for the natural history of HPV infection and transmission in populations, and the cost effectiveness and impact of HPV vaccination, were highlighted.[11,12,13] These models have been developed overseas but could form the basis of local models providing that Australian data were available. Local data requirements include HPV type specific prevalence estimates by age and sex and cervical intraepithelial neoplasia (CIN) status, local population structure and sexual mixing pattern data, screening program data (coverage, costs, efficacy), burden of disease data and costings and updated vaccine efficacy data.

Australian human papillomavirus research update

Participants at the meeting presented a diverse range of past, current and future research work in relation to HPV.

Professor Suzanne Garland (Department of Microbiology and Infectious Diseases, Women's and Children's Hospital Melbourne) outlined past projects conducted including HPV typing in high grade dysplasias and cervical cancers, research into sexual transmission by studying virgins, investigating self collected sampling methods, prevalence in Indigenous women, and in transplantation recipients.[14–17] Current research areas include: the detection of HPV DNA post elective treatment for dysplasia and as a marker of residual or recurrent disease (as compared to current standard of care); the use of p16 and the APOT (amplification of papillomavirus oncogene transcripts) assay to detect whether HPV DNA is integrated or episomal; and evaluation of various HPV typing methods such as the line-probe assay and the HPV DNA chip and micro-array. The research group is also taking part in a multi centre randomised double-blind placebo controlled trial of a quadrivalent prophylactic HPV vaccine.

Professor Ian Frazer (Centre for Immunology and Cancer Research, University of Queensland) updated participants on current knowledge regarding the natural history of HPV infection. He highlighted findings from large cohort studies describing the incidence of infection, the chance of cervical abnormalities given infection and the risk of progression and regression.[18,19] Over 99 per cent of oncogenic HPV infection will regress eventually. Older women with high risk HPV types have a greater chance of progression than younger women.[19]

Professor Frazer outlined his interest in preventing cervical cancer through preventing incident HPV infection (through vaccination) and his recent research into immunotherapy to produce resolution of CIN2/3 lesions. The Centre for Immunology and Cancer Research (CICR), in conjunction with the CSL Pharmaceuticals, has recently competed Phase 1 studies in this area of CerVax™ vaccine which is comprised of a recombinant bacterial

fusion of HPV 16 E6 and E7 proteins in 8M Urea with ISCOMATRIX® adjuvant. CICR has plans for ongoing vaccine immunotherapy trials and has recently been involved in trialling vaccination with HPV 6b L1 VLPs as a treatment for genital warts in China. CICR has taken part in a recent World Health Organization sponsored HPV serology standardisation exercise. It is also involved in ongoing collaborative basic research into HPV (animal models of cervical immunotherapy, studies on HPV uptake and presentation by dendritic cells, studies on mechanisms of innate resistance to HPV infection, siRNA therapy preclinical studies and polynucleotide vaccine preclinical studies.)

Mr Brian Brestovac (Division of Microbiology and Infectious Diseases, PathCentre) presented results from a HPV genotyping study of ThinPrep samples referred for cytology to PathCentre. Results were presented by cytology results, including 32 squamous cervical cancers and by region (Western Australia, Perth, Kimberley). Twenty-four different genotypes were identified with HPV 53 and 16 being the most common in Western Australia and differences in distribution between regions (e.g. HPV 52 only found in the Kimberley). HPV 16 was the most common genotype found in women with CIN or cancer. Mr Brestovac concluded that the distribution of HPV genotypes varies between geographical areas and that, whilst cancers are predominantly caused by HPV 16 and 18 in Western Australia, a large proportion of dysplastic lesions contain other HPV genotypes.

Professor Yvonne Cossart (Department of Infectious Diseases and Immunology, University of Sydney) highlighted HPV research undertaken with her principal collaborators, Barbara Rose and Carol Thompson. The department has produced 52 HPV related publications to date, encompassing three major research domains of natural history/epidemiology, virus genotypes and variants, and cell biology and host response. The group has produced four studies describing the genotypes that cause cervical cancer in Australia (dot-blot study of Zhang 1988,[20] historic cohort of cancers dating from 1922 onwards by Thompson, *et al* 1992,[21] 1994 study by Thompson[9] and modern cohort using PCR by Liu, *et al* 2003[10]). The cervical HPV prevalence and type distribution in Sydney women attending sexually transmitted diseases clinics and Family Planning Australia clinics has also been published. Two early studies using dot-blot methods typed HPV in genital warts but there have been no recent studies.[22,23] Recently, the department has been involved in HPV typing of laryngeal papillomas and of head and neck cancer (in collaboration with the International Agency for Research on Cancer). Other recent work has focused on cell biology and host response[24,25] and on intratypic variation in HPV 16. Other areas of research have involved skin carriage of HPV and viral loads in immunosuppressed and non-immunosuppressed individuals. Ongoing epidemiological work includes a study of women attending clinics in outer western Sydney for Pap smears and cervical dysplasia/cancer and risk factor (including HPV) analyses for South China and Australia.

Associate Professor Freddy Sitas (Director of the Research and Registers Division at the Cancer Council NSW) described the ongoing development of a study design, in collaboration with Associate Professor Dianne O'Connell (Cancer Epidemiology Research Unit, Cancer Council NSW), Dr Carol Thompson (Department of Infectious Diseases and Immunology, University of Sydney), Professor Valerie Beral of the Epidemiology Unit, Cancer Research UK, and others. The study aims to measure the association between CIN II/III and: smoking; current prolonged use of exogenous hormones (oral contraceptives, hormone replacement therapy); seroprevalence of the leading human papillomavirus subtypes (16 and 18); and long term 'persistence' of HPV (2 tests over 14 years). The study will be a nested case-control design of approximately 7,000 women from the New South Wales Pap Test Register cohort.

Dr Elizabeth Davey (Screening Test Evaluation Program, School of Public Health, University of Sydney) gave an overview of the research components of the Screening Test Evaluation Program (STEP) (diagnostic and screening accuracy, assessment of screening outcomes and informed decision-making.) The group has been involved in HPV research in relation to HPV testing as an adjunct to Pap screening. STEP is currently undertaking a trial of 400 women attending family planning clinics in New South Wales, Queensland and the Australian Capital Territory. The study is a three arm randomised trial with participants allocated to one of three management options, either (a) HPV DNA testing; (b) repeat Pap testing; or (c) a decision aid and choice of either management (a or b). The study aims to evaluate psychosocial and quality of life/utility outcomes of triage testing by HPV DNA testing among women with mild atypia on Pap smear. The study will evaluate the women's preferences for each management (HPV testing or repeat Pap) within the decision aid arm and psychosocial outcomes in those who make an 'informed choice'.

The chief investigator, Dr Kirsten McCaffery, also has research interest in the psychosocial issues around HPV vaccination. Such issues include the acceptability of the vaccine, parental consent, health education, reducing the stigma of HPV infection and the impact of vaccination on subsequent screening behaviour, safe sex and other risk behaviours. There are several potential impacts of HPV vaccination on cervical screening that will need to be considered. These include the effect on test performance (Pap, HPV tests), the effect of changes in disease prevalence on predictive values, possible alteration of disease process and the effect upon the cost-effectiveness of screening.

Ms Kerry-Ann O'Grady (Vaccine and Immunisation Research Group, Murdoch Childrens Research Institute and School of Population Health, University of Melbourne) described involvement in a quadrivalent HPV vaccine trial in Melbourne. The trial aimed to determine the immunogenicity, safety and end expiry specifications of the vaccine. The study recruited 150 adolescents through schools with study participation occurring at home visits. Sexual activity was an exclusion criterion and pregnancy testing occurred at every visit for all girls. Ms O'Grady discussed the challenges in the recruitment of the participants, particularly in relation to the sensitivity of the subject matter, privacy legislation, timing of the study in the school year, busy families and refusals. Ethical issues which the investigators had to address through detailed guidelines, related to dealing with HPV positive serology at baseline, and actions if, during the study, there was disclosure of abuse or sexual activity, or a positive pregnancy test. Future needs in relation to HPV vaccine trials identified included a need to address privacy laws, gain a greater understanding of the likely acceptability of these vaccines to the community and consider alternative implementation strategies for trials in this age group.

Determining the priorities

Small group discussions were held to determine future priorities for HPV research in Australia as it pertains to vaccination. Priorities determined were:

1. adequate local knowledge of age/sex/type specific burden of disease. This should particularly focus on women most at risk such as Indigenous women and women from certain ethnic groups and include the burden of disease from genital warts.
2. data collection for, and undertaking of, health economic analyses that would inform decisions about population use of HPV vaccines and about the target groups for vaccination (especially in relation to the impact on screening programs); and
3. the need to develop an understanding of communication and implementation issues relating to HPV immunisation of adolescents and the general population.

The key recommendations for future initiatives in HPV research were:

1. epidemiological data collection is a priority using modern assays/genotyping and representative sampling, preferably across Australia, but if necessary focusing on those groups with potentially most to benefit from vaccination (high risk groups).
2. measure type specific HPV prevalence now, and after vaccine introduction (replacement studies);
3. assess current burden of illness and costs from cervical cancer, genital warts and other HPV related diseases.
4. plan for impact of vaccination on cervical screening and disease burden including modelling. Start developing epidemiological and health economic models to assess where we should be focusing on getting 'harder' Australian data.
5. a focus on collaboration to ensure securement and efficient expenditure of adequate research funding;
6. education/communication strategy research;
7. utilise standardised serological test interpretation once available; and
8. ongoing assessment of the importance of sequence variation in L1 HPV 16 and 18.

The group plans to reconvene in 2004, following the February International Papillomavirus Conference, to discuss ongoing research, results and collaborations.

Acknowledgements

We would like to thank CSL Pharmaceuticals and Glaxo SmithKline for their support in facilitating this meeting. The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases is supported by the Australian Government Department of Health and Ageing, the NSW Health Department and The Children's Hospital at Westmead.

In attendance at the meeting were:

Mr Brian Brestovac, Division of Microbiology and Infectious Diseases, PathCentre (The Western Australian Centre for Pathology and Medical Research)

Professor Yvonne Cossart, Department of Infectious Diseases and Immunology, University of Sydney

Dr Elizabeth Davey and Ms Petra Macaskill, School of Public Health, University of Sydney

Professor Ian Frazer and Ms Olivia White, Centre for Immunology and Cancer Research, University of Queensland

Assoc. Professor Suzanne Garland and Dr Sepehr Tabrizi, Department of Microbiology and Infectious Diseases, Women's and Children's Hospital, Melbourne

Professor Lyn Gilbert, Centre for Infectious Diseases and Microbiology, Westmead Hospital

Ms Kerry Ann O'Grady, Vaccine and Immunisation Research Group, Murdoch Childrens Research Institute and the School of Population Health, University of Melbourne

Assoc. Professor Freddy Sitas, Research and Registers Division, The Cancer Council NSW

Dr Julia Brotherton, Professor Margaret Burgess and Assoc. Professor Peter McIntyre, National Centre for Immunisation Research and Surveillance

Dr John Anderson, Dr Lynne Conway, Dr Jane Greig and Dr Neil Formica, CSL Vaccines

Dr Damien Cramer, Ms Melanie Duiker and Dr Catherine Streeton, Glaxo SmithKline Biologicals

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Impact of *Haemophilus influenzae* type b (Hib) vaccination on Hib meningitis in children in Far North Queensland, 1989 to 2003

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Abstract

Over the four years 1989 to 1992 there were 28 cases of *Haemophilus influenzae* type b (Hib) meningitis in children aged under five years in Far North Queensland.[4] Thirteen (46%) of the cases were in Indigenous children, indicating that the rate of the disease was about 3.5 times greater in these children than in non-Indigenous children. However, no cases of Hib meningitis have occurred in Indigenous children in Far North Queensland in the 10 years following the addition of Hib vaccines to the Australian Standard Vaccination Schedule in 1993. There was only one case of Hib meningitis, in a (vaccinated) non-Indigenous child, between 1994 and 2003. About 70 cases of Hib meningitis in children were prevented by Hib immunisation in Far North Queensland between 1994 and 2003; possibly as many as five deaths and 12 cases with neurological sequelae were also prevented. *Commun Dis Intell* 2004;28:255–257.

Keywords: Haemophilus influenzae, *meningitis, vaccination*

Introduction

Prior to the introduction of *Haemophilus influenzae* type b (Hib) vaccines into the Australian Standard Vaccination Schedule in 1993, infections due to Hib were an important cause of morbidity and mortality, especially in young children. The most important risk factors for Hib disease in these children were attendance at child day-care, and the presence of young siblings in the home.[1]

The most common manifestation of invasive Hib disease was meningitis, which accounted for approximately 60 per cent of all invasive Hib disease. The overall Hib meningitis fatality was about 5 per cent, and up to 15 per cent of the survivors had neurological sequelae such as deafness and intellectual impairment.[1] Most cases of Hib meningitis occurred in children under 18 months of age. However, there were marked differences between the epidemiology of Hib meningitis in Indigenous and non-Indigenous children in northern and central Australia (and perhaps elsewhere). Not only was the incidence considerably higher, but also the mortality and neurological sequelae were considerably greater in Indigenous children.[2] Furthermore, of particular importance, the onset of Hib meningitis occurred at a much earlier age in Indigenous than the onset in non-Indigenous children.[3]

This report describes the Hib meningitis cases that occurred in children under five years of age in Far North Queensland (FNQ) over 15 years, 1989 to 2003 including the first 10 years following the introduction of Hib vaccines in 1993.

Methods

Surveillance for Hib meningitis in Far North Queensland dates back to 1989;[4] invasive Hib disease became a notifiable disease in Queensland several years later. Each invasive *Haemophilus influenzae* isolate is referred to the Queensland Health Scientific Services reference laboratory in Brisbane for serotyping. Detailed information is collected on each case of invasive Hib disease, including the patient's age and ethnicity, and if a child, from 1993 Hib vaccination status was collected.

The total population of children less than five years of age in Far North Queensland ascertained from the 2001 national census was approximately 16,500 children, 22 per cent of whom were Indigenous children.[5]

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Results

Over the four years 1989 to 1992 there were 28 cases of Hib meningitis in children under five years of age in FNQ.[4] Thirteen (46%) of the cases were in Indigenous children, indicating that the rate of the disease was about 3.5 times greater in these children than in non-Indigenous children. The average age of onset of Hib meningitis was 9.5 and 15.5 months in the affected Indigenous and non-Indigenous children respectively. Nearly 25 per cent of the Indigenous cases occurred before six months of age compared with 11 per cent in the non-Indigenous cases.

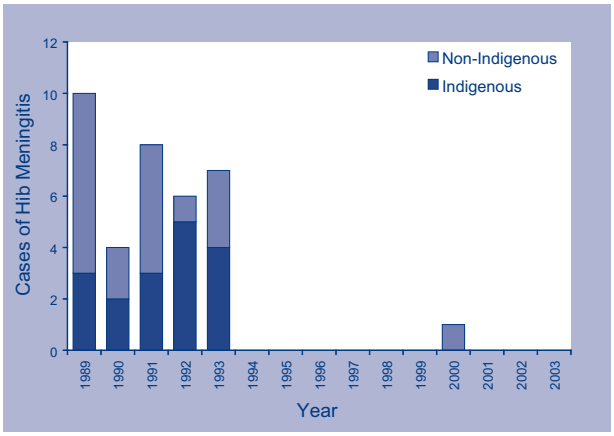
There were no cases of Hib meningitis in 1993 until April, when three cases occurred in unrelated Aboriginal children, all from remote communities, over a four-day interval. A Hib vaccine (PedvaxHIB; PRP-OMP), recently licensed for use in Australia, was immediately made freely available to all Indigenous children under two years of age in remote communities in FNQ. This local initiative commenced in May 1993; the last case (up to the present time) of Hib meningitis in an Indigenous child in FNQ occurred in June 1993 (Figure). This child was 10 months of age and was unimmunised at the time of illness.

The Commonwealth funded National Hib Immunisation Program began in mid-1993 for all children born in or after February 1993, and in September 1993 for older children. This program provided free Hib vaccination, for all Australian children under five years of age as part of the routine childhood immunisation program. Because of the epidemiological differences between the two populations of children, Indigenous children were given PRP-OMP vaccine, whereas non-Indigenous children were given a different vaccine (HibTITER; HbOC). However, from 2000, all children were given PRP-OMP.

Three cases of Hib meningitis occurred in non-Indigenous children in FNQ in 1993. No further cases occurred for the next seven years. However, a case occurred in a non-Indigenous child in September 2000; that (otherwise healthy) child was 11 months of age, and she had received the recommended three doses of HbOC vaccine at 2, 4 and 6 months. This case was the only case of Hib meningitis in a child under five years of age in FNQ in the 10 years, 1994 to 2003, following the implementation of the National Hib Immunisation Program.

Altogether, there were 35 cases of Hib meningitis in children aged under five years in FNQ between 1989 and 1993; an annual average of seven cases. Seventeen (49%) of these cases were in Indigenous children.

Figure. Cases of *Haemophilus influenzae* type b meningitis in children under five years of age in Far North Queensland, 1989 to 2003



Discussion

Hib vaccination has had a profound impact on the incidence of Hib meningitis, and very likely on the other less common forms of invasive Hib disease, in children in FNQ since 1993. The rapidity and extent of the impact was far more profound than expected from vaccine uptake in children in the region at the time. Surveys in the early 1990s showed that many children in child-care in Cairns,[6] and Indigenous children throughout FNQ,[7] were incompletely immunised by their second birthday. The rapidity in the decline of Hib meningitis was probably assisted by the reduction in nasopharyngeal carriage of the Hib bacterium in vaccinated children. This indirect effect reduces the transmission of the organism via respiratory droplets resulting in less exposure to the organism in unvaccinated children.[8]

Based on the epidemiology of Hib meningitis in FNQ in the years 1989 to 1993, it can be predicted that about 70 cases of Hib meningitis in children were prevented in FNQ over the 10 years 1994 to 2003. Using published information on the outcome of Hib meningitis in other parts of Australia, possibly as many as five deaths and 12 cases with neurological sequelae were also prevented. There is no reason to suggest that this success was not the same elsewhere in other parts of north Queensland, including the Townsville, Mackay and Mt Isa districts as well as FNQ. Taking into consideration the demography of the childhood population throughout north Queensland, with a total of approximately 43,480 children aged less than five years,[5] it can be estimated that about 150 cases of Hib meningitis in children were prevented by Hib vaccination throughout north Queensland over the 10 years 1994 to 2003.

With the exception of the one vaccine failure in 2000, this impact has been sustained over the decade. The vaccine failure reminds us that there is no such thing as a 100 per cent effective vaccine;[9] fortunately the child made a full recovery. It also reminds us that the Hib bacterium still exists in north Queensland, and indeed elsewhere in Australia, so there can be no suggestion of stopping Hib vaccination in the foreseeable future. To do so would invite an inevitable, and probably rapid, resurgence in Hib disease in young children.

During the 1990s allegations were made that Hib vaccination, when given early in life, increased the risk of developing type 1 diabetes. However, several very large epidemiological studies in Europe and the United States of America have failed to show any association between Hib vaccines and type 1 diabetes.[10] In essence, there are no recognised substantive safety concerns about Hib vaccines.

In conclusion, Hib vaccination is safe and extremely effective. Over the 10 years that Hib vaccines have been in widespread use, perhaps as many as 150 cases of Hib meningitis have been prevented in north Queensland. Indigenous children in the region are no longer at an increased risk of acquiring this disease; indeed, the risk to any child in north Queensland is now extremely low, and will remain so provided that the current high levels of vaccine uptake are sustained.

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Trends in potential exposure to Australian bat lyssavirus in South East Queensland, 1996 to 2003

Megan K Young,[1] Bradley J McCall[2]

Abstract

This study examined trends in notifications of potential exposure to Australian bat lyssavirus reported to the Brisbane Southside Public Health Unit, Australia between 1 November 1996 and 31 January 2003. Notification rates declined among all population groups and potential exposures were notified more promptly. Concern exists regarding possible under-reporting of potential exposure to Australian bat lyssavirus especially among volunteer bat carers. *Commun Dis Intell* 2004;28:258–260.

Keywords: lyssavirus; epidemiology; Australia

Introduction

Australian bat lyssavirus (ABL) is a member of the Rhabdoviridae family, possessing marked similarity to classic rabies virus on both serotyping and molecular sequencing.[1] To date, two cases of fatal ABL infection have been reported in Australia, one in 1996 and the second in 1998.[2] The epidemiology of potential exposure to ABL has been previously described.[3] The key feature, in a predominantly urban population in South East Queensland, was that potential exposures were likely to be the result of human-initiated contact by people with some professional or volunteer interest in caring for bats and/or flying foxes (53% of potential exposures). A lower proportion of potential exposures (35%) were reported by members of the general community.

Potential exposures to ABL continue to occur despite consistent information and reminders to the community about the dangers of handling flying foxes and insectivorous bats.[4] This paper examines population trends in potential exposure to ABL reported to the Brisbane Southside Public Health Unit (BSPHU) between 1 November 1996 and 31 January 2003.

Methods

Since 1 November 1996, all persons reporting a potential exposure to ABL have been asked to complete a standard questionnaire. The details of the questionnaire, methods of study and results until 31 January 1999, have been described.[3] However, the geographic boundaries of the area served by the BSPHU have changed since 1999. They now include South Brisbane (part of the Brisbane City Council Area), Logan, Redlands, Ipswich, Laidley, Boonah and Esk Local Government Areas (Figure) with an estimated resident population of 920,680 as at 30 June 2000.[5]

The time frame of this study was divided into period 1 (the initial study period from 1 November 1996 to 31 January 1999, which included the two human cases of ABL infection) and period 2 (1 February 1999 to 31 January 2003). To allow comparison of data across these time periods, the original study data was restricted to include only that related to people who resided within the current Brisbane Southside Public Health Unit boundaries. SPSS version 11.5 was used for analysis.[6]

Results

Two hundred and forty-six notifications were reported between 1 November 1996 and 31 January 2003. One hundred and thirty-six notifications of potential ABL exposure from the re-defined study area were reported to the BSPHU in period 1 (duration = 27 months), an average annual notification rate of 6.56 per 100,000. One hundred and ten notifications were reported in period 2 (duration = 48 months), an average annual notification rate of 2.98 per 100,000. The proportion of notifications from females fell from 60 per cent in period 1 to 46 per cent in period 2.

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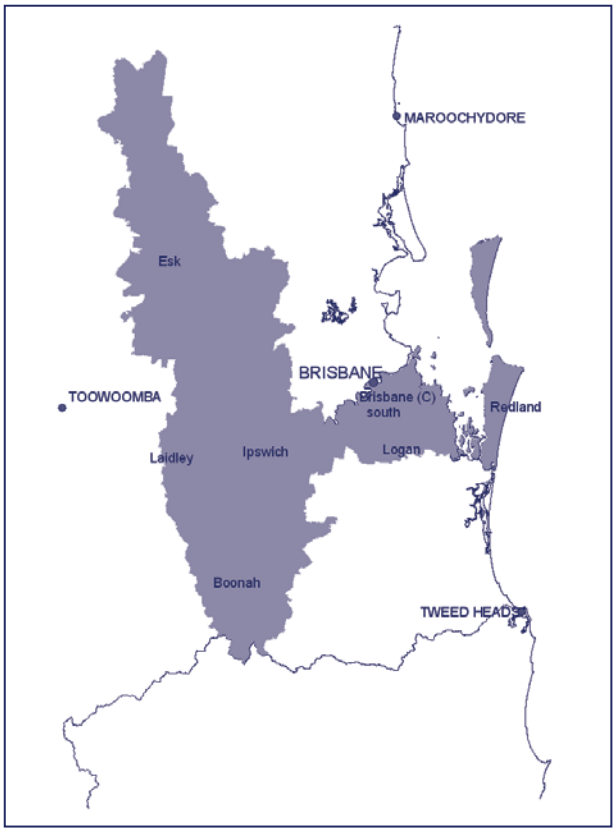
Figure. The geographical area covered by the Brisbane Southside Public Health Unit



There was no significant difference between the age distributions (χ^2 = 8.13, 6 df, p = 0.23), with the peak age group between 20 and 49 years in both time periods.

The median time interval between potential exposure and notification to the BSPHU fell from 14.5 days (range 0 to 3,636; 25th, 75th centiles: 2, 79.8) in period 1 to one day (range 0 to 1,860; 25th, 75th centiles: 0,3) in period 2.

The Table describes the circumstance of potential exposure to ABL, the treatment received and the history of previous vaccination in the two time periods. There was a decline in the average annual number of reported potential exposures for all population groups (professionals, volunteer bat carers and their family members, community members). However, the proportion of potential exposures reported by community members increased (from 40% to 71%). The proportion of potential exposures reported by professional handlers and volunteer bat carers fell substantially, with the greatest fall among volunteer bat carers (from 36% to 11%). Professional handlers and volunteer bat carers reporting potential exposures in period 2 were more likely to have been previously vaccinated. Only 16 per cent of professional and volunteer handlers reported no previous vaccination in period 2 compared to 86 per cent in period 1.

Table. Circumstance, treatment and history of previous vaccination of potential exposures to Australian bat lyssavirus for each time period, Brisbane Southside Public Health Unit area, 1 November 1996 to 31 January 2003*

	Period 1 (1/11/96 – 31/1/99) n=136		Period 2 (1/2/99 – 31/1/03) n=110	
Circumstance	**n**	**%**	**n**	**%**
Community member bat initiated contact	3	2.2	17	15.5
Community member intentionally handled bat	51	37.5	61	55.5
Professional handlers	17	12.5	10	9.0
Volunteer bat carers	49	36.0	12	10.9
Family member of volunteer bat carers	13	9.6	3	2.7
Treatment				
Nil (bat tested negative)	5	3.7	40	36.4
Course ceased (bat tested negative)	18	13.2	2	1.8
2 doses of vaccine	12	8.8	12	10.9
5 doses of vaccine	69	50.7	8	7.3
Rabies immunoglobulin and 5 doses of vaccine	31	22.8	47	42.7
Recommended treatment but declined	0	0.0	1	0.9
Previous vaccination				
Nil	121	89.0	85	77.3
Pre-exposure prophylaxis	3	2.2	10	9.1
Pre-exposure prophylaxis and booster/s	0	0.0	4	3.6
Previous post-exposure prophylaxis	10	7.4	6	5.5

* Percentages may not total 100 because of missing values.

Discussion

The notification rate of potential exposure to ABL fell markedly during the study. This rate may more accurately estimate baseline potential exposure rates since earlier rates were inflated by the large number of retrospective reports received after the initial recognition of this disease in humans in 1996. However, it is possible that a proportion of recent potential exposures are not being notified. This may arise from waning concern about the risks of bat bites or scratches among the community or medical profession, especially as no cases of human ABL infection have been reported since 1998.

Professional handlers and volunteer bat carers may be unwilling to notify potential exposures within their groups, a reluctance that has been communicated to both authors. This unwillingness may be an undesired consequence of the public health requirement to euthanase and test all bats involved in human potential exposures. Unwillingness to notify potential exposure may also reflect preformed opinions about the risk posed by scratches, the protection afforded by pre-exposure vaccination, or the level of risk associated with the clinical appearance of the bat. These suggestions are further supported by the finding that volunteer bat carers have the largest decrease in proportion of notifications from time period 1 to period 2 [36% (n=49) to 11% (n=12)], with a corresponding increase in community notifications [40% (n=54) to 71% (n=78)], despite no recognisable change to volunteer bat carer numbers in the Brisbane Southside area over recent years (personal communication, Allan McKinnon, Manager, Moggill Koala Hospital, Queensland Parks and Wildlife Services).

The reduction observed in notifications among females may also be explained by the fall in the proportion of volunteer bat carer notifications. The high proportion of females in period 1 was influenced by the high proportion of female volunteer bat carers in the study population.

The time between potential exposure and notification decreased substantially over the study, with fewer long-term retrospective reports of potential exposure in period 2. A corresponding decrease was observed in the proportion of people requiring treatment after their potential exposure. Post exposure treatment may be delayed for 48 hours pending the results of tests on the bat involved.[7] In period 2, potential exposures were notified more promptly. This allowed a greater proportion of the bats involved to be tested and the negative results to be obtained within the required 48 hours. This represented an important cost saving through the reduced use of rabies immune globulin (RIG), rabies vaccine and fewer doctors' visits.

Future public health messages should continue to emphasise the need for the community to maintain a safe distance from all bats or flying foxes, even if they are orphaned or distressed. Messages should reinforce that it is usually impossible for an untrained person to handle a bat without sustaining a bite or scratch, even if protective measures are used. Members of the public can be of most help to orphaned or injured bats by contacting a trained, vaccinated bat handler. Volunteer bat carers must also be aware of the potential risks associated with bites or scratches from apparently healthy looking bats, and seek medical advice regardless of their pre-exposure prophylaxis or the nature of the wound.

Acknowledgements

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Staff of Brisbane Southside Public Health Unit, Archerfield, Queensland

Allan McKinnon and staff at Wildlife Rangers Conservation Services, Queensland Parks and Wildlife Services, Moggill, Queensland

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Ross River virus and its vectors in Sorell Municipal Area, south-eastern Tasmania, January to March 2002

Greg J Robertson,[1] Stephen Doggett,[2] Owen Seeman,[3] Richard C Russell,[2] John Clancy,[2] John Haniotis[2]

Abstract

In 2002, Tasmania reported the largest number of Ross River virus (RRV) infections ever recorded for the state. Of the 117 cases, 37 lived in, or had visited, the Sorell Municipal Area, east of Hobart. In early 2002, a combination of spring tides and high summer rainfall produced extensive saltmarsh habitat in the Sorell region, resulting in unseasonably high densities of the mosquito *Ochlerotatus camptorhynchus*, recognised vector of RRV. Four isolates of RRV were identified from collections of adult mosquitoes. All four isolates were from *Oc. camptorhynchus,* collected near the Carlton River. This is the furthest south that RRV has been identified in Australia and the first identification from south-east Tasmania. The virus carriage rate in the mosquito vector populations were very high, with successive weekly minimum infection rates of 17.1, 3.0 and 11.1 per 1,000 *Oc. camptorhynchus* at Carlton River from mid-February to early March. The first isolation of RRV from mosquitoes coincided with the onset dates of the first human cases of RRV infection. *Commun Dis Intell* 2004;28:261–266.

Keywords: Ochlerotatus camptorhynchus, *Ross River virus*

Introduction

The number of cases of Ross River (RRV) disease have fluctuated in Tasmania between 8 and 117 during the period 1995–2002. The highest notifications were reported in 1996, 1999 and 2002, with 74, 67 and 117 notifications, respectively.[1]

Investigations into the dynamics of arboviral diseases within Tasmania have been limited to two previous studies. Both were undertaken on the east coast, largely focusing on the Coles Bay to Scamander region, and identified *Ochlerotatus camptorhynchus* and *Oc. flavifrons* as vectors of RRV for that region.[2,3]

Despite the presence of human cases of arbovirus disease further south in the Hobart region, there have been no investigations of the mosquito vectors and the relative disease risks in these south-eastern communities.

The Sorell Municipal Area is 25 km east of Hobart, and its population of 10,800 is located mostly in the coastal townships of Midway Point, Sorell, Dodges Ferry and Primrose Sands. Sorell Council has received notifications of cases of RRV infection since 1995. In 1999, following 30 cases of RRV and complaints of excessive local mosquito activity, Sorell Council commenced surveys of mosquito larval habitats and began adult trapping in several areas.

This surveillance was subsequently expanded throughout the municipal area, and in the summer of 2002, the Council decided to test field-collected adults for the presence of arboviruses to determine which local mosquitoes were likely RRV vectors. In order to determine the origin of the RRV infections, all local human cases were interviewed to establish the location of their residence and if they had travelled to, or outside of, the Sorell area in the 2–3 weeks before the onset of symptoms.

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Methods

Case investigation

Notifications of RRV were included in this study if they satisfied the Tasmanian case definition of RRV which required notification to the Director of Public Health, i.e. laboratory analysis of a blood sample satisfying one of the following criteria:

1. isolation of RRV from a sample;
2. detection of RRV by nucleic acid testing;
3. IgG seroconversion or a significant increase in antibody level, or a fourfold or greater increase in titre to RRV; or
4. detection of RRV specific IgM antibody titre.

Notifications of RRV infection in residents of Sorell Municipal Area, and in individuals that had travelled to the area within two weeks before the onset of symptoms were interviewed. The possible site of infection was determined by comparing residential address with proximity to known larval habitats. Demographic details of local cases were examined to determine if any particular group was at a greater risk. Infections acquired on the western side of Pittwater were not included in this study as that area is within the City of Clarence (which reported 50 of the 117 cases notified state-wide). The City of Clarence is located between Hobart and Sorell. Pittwater is an estuary extending from Richmond (City of Clarence) to Lewisham (Sorell Municipal Area). Pittwater contains the coastal towns of Midway Point and Sorell (Figure 1).

Habitat investigation and mosquito surveillance

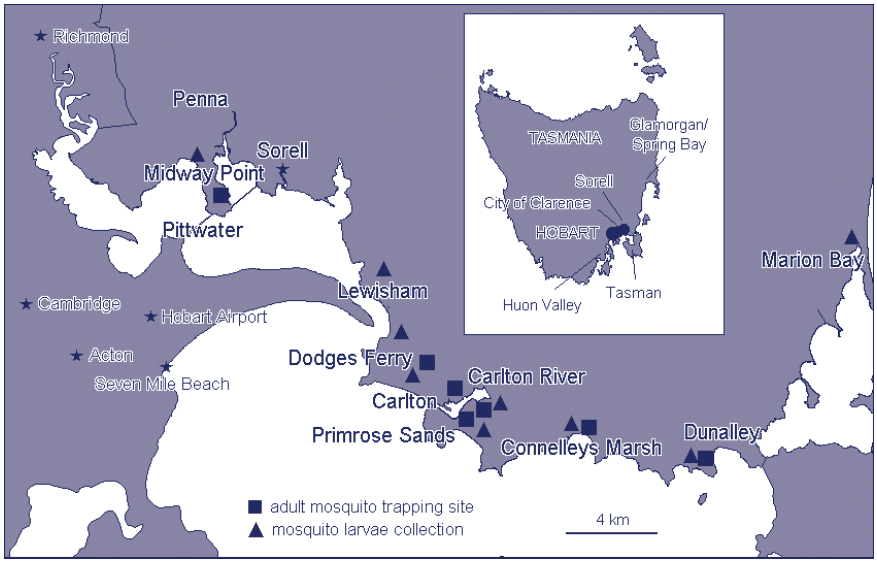
The purpose of the study was to determine which local mosquito species were the likely vectors of RRV.

Aquatic habitats were explored in the municipal area (west to east) at Penna, Midway Point, Lewisham, Dodges Ferry, Carlton, Primrose Sands, Connellys Marsh, Dunalley and Marion Bay (Figure 1). Mosquito larvae were collected and quantified by dip sampling techniques[4] and identified to species using larval identification keys.[5]

Adult mosquito populations were sampled with dry ice-baited EVS light traps[6] at seven sites; indicated in Figure 1. Traps were set late in the afternoon and collected early the following morning on 21 January, 20 February, 25 February, and 5 March 2002. Trapping days were selected considering climatic conditions and transport logistics for sending the mosquitoes to Sydney for virus isolation. All mosquitoes collected were sent for virus isolation testing.

Traps were set within coastal towns with the exception of the Carlton River site, which was set on the edge of the saltmarsh habitat. Areas where complaints about excessive numbers of mosquitoes had been received from local residents were also selected. Trapping focused around Carlton River where larval surveying revealed larger saltmarsh habitat areas. Trapping expanded to Dunalley, Connelly's Marsh, Dodges Ferry and Midway Point to establish the extent of distribution into residential areas.

Figure 1. Adult and larval sampling locations



Arbovirus isolation

The mosquito collections were transported live to the Institute of Clinical Pathology and Medical Research, Sydney, for processing. Mosquitoes were sorted and identified[4–8] on a refrigerated table, into pools of up to 25, according to species, sex, bloodfed, date and site.[9] Each pool was placed into a sterile plastic 5 mL tube containing 5 x 5 mm glass beads and 5 mL of cell growth media, macerated in a grinder/shaker for 20 minutes and centrifuged at 4,000 rpm and 4° C for 20 minutes, and a 50 UL aliquot of supernatant from each tube was inoculated into the cell lines C6/36, BHK and PSEK, as previously described.[9,10]

The presence of viruses was indicated by cell death. Presumptive viral isolates were tested by Fixed Cell Enzyme-Linked Immunosorbent Assay (FCE)[11] using specific monoclonal antibodies to detect alphaviruses (Ross River, Sindbis, Barmah Forest viruses) and flaviviruses (Murray Valley encephalitis, Kunjin, Stratford, Alfuy, Edge Hill, Kokobera). As RRV was detected in the mosquito samples, a RRV FCE screen was performed on later samples to rapidly identify virus. Minimum infection rates (MIRs) per 1,000 were calculated[12] per trap for the species that yielded virus.

Meteorological conditions

Rainfall data were recorded at the Park Beach Weather Station (Dodges Ferry), the closest weather station to Carlton River. Temperature data at Hobart Airport were obtained from the Bureau of Meteorology.

Results

Human cases

In 2002, a total of 117 cases of RRV were notified in Tasmania, with 19 of these resident in the Sorell Municipal Area.[13] Another 18 had travelled to the municipal area within two weeks prior to onset of symptoms and may have been infected during this period. The Department of Health and Human Services reported that more than 90 per cent of all cases notified were from single sample IgM results. Single sample IgM serology does not provide absolute certainty that infections occurred recently and cases notified should be regarded as presumptive.[14] However, in combination with clinical symptoms and RRV isolates from local mosquitoes, it is unlikely that a large number of previous infections are included within the 37 cases.

Of the 37 cases that had some association with the Sorell Municipal Area, 23 lived in, or had visited, the Carlton River, eight were from Pittwater, four were from Marion Bay and the other two cases were Sorell Municipal Area residents that had links with sites in the City of Clarence. RRV infection onset dates ranged from 22 February 2002 to 10 May 2002. Generally, infections linked to the Carlton River area occurred first and continued throughout the period, with cases from the Penna area occurring in late-March and those at Dunalley/Marion Bay in mid-April.

There were 20 males and 17 females with infections that could have been acquired locally. The average age of patients was 47 years (range 8 to 81); the majority (21) were in the 30–49 years age range, with three under 30 years and three over 70 years.

In addition to the cases reported in the Sorell Municipal Area another 50 RRV notifications were reported from the residents of the City of Clarence. Of these 50 cases, 22 lived in, or travelled to, the western Pittwater towns of Seven Mile Beach, Acton, Cambridge or Richmond.

Of the 117 cases in Tasmania, 105 lived in Southern Tasmania from the following Local Government Areas: Clarence (50), Sorell (19), Hobart (10), Glenorchy (7), Huon Valley (7), Kingborough (4), Tasman, (3), Brighton (3), and Glamorgan Spring Bay (2).[13] These areas are all situated on the south–east coast. Hobart, Glenorchy, Brighton, Clarence, and Kingborough are within 'Greater Hobart'. Huon Valley is 35 km south of Hobart, Glamorgan Spring Bay is on the east coast and Tasman is in the south east.

Weather conditions

Coastal regions of the Sorell Municipal Area usually receive a low annual average rainfall of 560 mm, and have an average maximum summer temperature of between 20 and 22° C. However, the spring and early summer months of 2001/02 were very wet, with a total rainfall of 450 mm. Rainfall between August 2001 and April 2002 was compared with long-term (since 1962) averages (Figure 2). The above average rainfall coincided with high tides, resulting in the saltmarsh at Carlton River holding water from early spring until March. In contrast, the saltmarsh totally dried out for extended periods (4–6 weeks) in the previous two summers.[15]

The summer months had below average monthly temperatures, with December 2001, January 2002 and February 2002 being 1.9, 1.7 and 0.8° C below average, respectively, although March and April were 1.5 and 1° C above average, respectively.

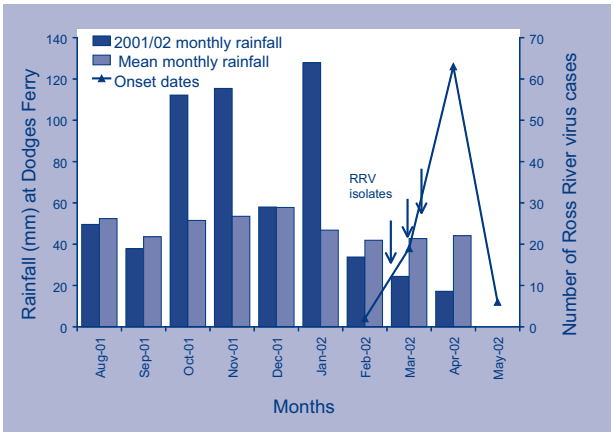
Mosquito fauna

Mosquito larvae identified included *Anopheles annulipes* s.l., *Culex australicus, Cx. fergusoni, Cx. globocoxitus, Cx. molestus, Cx. orbostiensis, Ochlerotatus alboannulatus,* and *Oc. camptorhynchus*.

Laval surveys found high numbers of *Oc. camptorhynchus* on the saltmarsh at Carlton River (n=257) at a density of >100 per larval dip on 21 February 2002. Surveys at other saltmarsh areas near Marion Bay, Penna, and Lewisham contained <5 per dip. Freshwater habitats had even lower numbers (<1 per dip).

Overall, there were 18 collections of adult mosquitoes. Four from the Carlton River (n=694) and Carlton (n=1,011), three from Dodges Ferry (n=336), two from Primrose Sands (n=143) and Midway Point (n=19), and one from Dunalley (n=8), Richmond (n=25) and Connelly's Marsh (n=11). A total of 2,247 adult mosquitoes were collected from five genera and 13 species (Table). *Ochlerotatus camptorhynchus* dominated the collections and comprised around 90 per cent of the total. Most other species appeared infrequently or rarely. Only two species collected as larvae were not trapped as adults; *Cx. fergusoni* and *Cx. orbostiensis*.

Figure 2. Rainfall compared with onset dates of cases of Ross River virus, Sorell and Clarence municipal residents/visitors



Viruses isolated

There were 153 mosquito pools processed and four RRV isolates obtained. All were from *Oc. camptorhynchus*, with three isolates coming from Carlton River at Primrose Sands and the other from Provence Drive at Carlton. Table 1 provides mosquito totals and details of the viral isolates. The MIRs varied from 3.0 to 17.1 per 1,000 and were highest at Carlton River, Primrose Sands.

Table. Mosquito species collected and numbers processed for virus isolation with viruses recovered and minimum infection rates of the mosquito vectors

Species	Collection dates with total adult numbers, virus isolations and minimum infection rates			
	21 January 2002	20 February 2002	25 February 2002	5 March 2002
Anopheles annulipes s.l.	3	0	4	1
Coquillettidia sp. nr. *linealis*	13	14	11	6
Culex australicus	6	0	3	0
Cx. fergusoni	0	0	0	0
Cx. globocoxitus	9	23	35	15
Cx. molestus	7	9	12	6
Cx. orbostiensis	0	0	0	0
Culiseta inconspicua	0	2	1	0
Ochlerotatus australis	0	1	3	0
Oc. alboannulatus	2	1	1	0
Oc. camptorhynchus	522	251	989	254
		1RRV* (MIR:17.1)	2RRV*,** (3.0, 7.3)	1RRV* (11.1)
Oc. nigrithorax	1	0	0	0
Oc. notoscriptus	0	3	18	8
Oc. rubrithorax	0	1	0	0
Tripteroides tasmaniensis	0	0	2	0

MIR = number of mosquitoes infected per 1,000 and based on pools of 25 (Chiang and Reeves 1962).

* Ross River virus isolate from Carlton River, Primrose Sands, Tasmania.

† Ross River virus isolate from Provence Drive, Carlton, Tasmania.

Discussion

The 117 RRV cases reported for Tasmania in 2002, exceeded the previous highest notification of RRV for Tasmania of 74 cases in 1996. The first cases of RRV reported for the Sorell Municipal Area were from the Carlton River locality (onset date 22 February) and coincided with the first collection of mosquitoes that yielded RRV (trapped 20 February 2002). Most human infections, based on onset date, occurred during March and April in the two months following the high rainfall and tides in January (Figure 2).

High tides and rainfall during spring and early summer provided extensive and sustained mosquito habitat, which allowed for the build-up of large vector populations compared to the previous year,[15] although previous preliminary studies focused on species identification and thus exact numerical comparisons of numbers of adults and larvae cannot be made. Carlton River was the most significant habitat area for the vector species *Oc. camptorhynchus*. Other larval populations of this species were at Marion Bay, Penna, Lewisham and Primrose Sands.

Several cases of RRV were linked to Penna and Marion Bay, which require further investigation. In addition, other saltmarsh areas in nearby Pittwater (not within Sorell Municipal Area), around Barilla Bay and the Coal River, may be significant habitat areas contributing to the 50 cases seen in the City of Clarence during the same period.

The Carlton River saltmarsh is, at its closest point, only 50–100 m from houses, and a large portion of residents of Primrose Sands, Carlton, Connelly's Marsh and Dodges Ferry live within 5 km of the saltmarsh. Carlton River is surrounded by pasture and forest, and these communities are habitats for macropods and other vertebrates[16] that may serve as vertebrate hosts for RRV and other arboviruses.[17,18]

Adult trapping in Primrose Sands, Carlton and Dodges Ferry revealed high numbers of the vector *Oc. camptorhynchus* compared to other species. This was consistent with the larval surveys, and reflected the large larval habitat at Carlton River (compared to freshwater habitats) and the lack of predators in the saltmarsh habitats. Other species from which RRV has been isolated elsewhere in Australia, such as *Coquillettidia* sp. nr. *linealis, Oc. notoscriptus, Oc. alboannulatus*, *Oc. rubrithorax, An. annulipes* s.l. and *Cx. australicus*[17] were present only in low numbers and did not yield any virus.

Ochlerotatus camptorhynchus is a major inhabitant of southern coastal Australia within saltmarsh habitats.[17] Adults can be active throughout the year, and may disperse widely from larval habitats.[8] They can be vicious biters, readily attacking humans and other animals, and will feed during the day, at dusk and after sunset. RRV has been isolated from this mosquito in all southern states of Australia except South Australia.[17,18,20] This species is presumably responsible for the transmission of the majority of RRV infections in southern coastal Australia.

Other mosquito species also may be locally significant as vectors of RRV. *Coquillettidia* sp. nr. *linealis*, a mosquito associated with permanent vegetated freshwater, has been shown to be a putative vector for RRV and other viruses from elsewhere in Australia.[17,18] *Ochlerotatus flavifrons*, the only species that yielded RRV in the inaugural Tasmanian mosquito/arbovirus investigations[2] was not found in this study. Further collections for virus isolation may elucidate whether *Coquillettidia* sp. nr. *linealis* or *Oc. flavifrons*, or other local mosquitoes, are involved in the transmission of RRV in the Sorell region.

The infection rates observed were relatively high and comparable to epidemic situations investigated elsewhere. During the Barmah Forest virus outbreak along the south coast of New South Wales in 1995 where there were 135 human cases, MIRs over three successive weeks during the peak of activity were 15, 27 and 4 per 1,000 *Oc. vigilax.*[19] During the outbreak of RRV in Western Australia in 1995/96 MIRs per 1,000 *Oc. camptorhynchus* were calculated for three sites: Peel Inlet (0.2, 1.1, 0.4, 2.2 & 3.5), Leschenault Inlet (10.5) and Busselton wetland (18 & 5.5),[20] and compared with the weekly successive MIRs (17.1, 3.0 and 11.1/1,000) observed in the present study. In 1991, arbovirus investigations in the Scamander region (north east coast of Tasmania), during a period when several local human cases were identified, yielded RRV from *Oc. camptorhynchus* but with lower MIRs (approx. 3.5/1000) for *Oc. camptorhynchus*.[3]

As no longitudinal data on mosquito infection rates within the Sorell region were available, it was not possible to determine if the high MIRs in 2002 reflected typical arbovirus activity. However, the level of virus activity detected, especially from Carlton River, suggests that at least occasionally there can be a considerable arboviral disease threat to the nearby human community from *Oc. camptorhynchus*.

Carlton River was confirmed as a significant habitat for the vector *Oc. camptorhynchus*, and epidemiological follow-up established that most cases of RRV were associated with the Carlton River, although cases did occur in the far west of the municipal area at Penna and in the far east at Marion Bay. Strategies to reduce the risk of RRV in south-eastern Tasmania will therefore require further investigation and cooperation from neighbouring councils and the Tasmanian government.

As there is no coordinated mosquito vector monitoring conducted outside of the Sorell municipal area, the source of RRV infections from residents of 'Greater Hobart' Huon Valley, and East Coast (Glamorgan Spring Bay and Tasman) is not conclusive.

The results of the study will form the basis for more comprehensive vector monitoring and control programs. The saltmarshes at Pittwater are contained within a Ramsar site (wetland of international importance) and Carlton River is 15 km from the Ramsar site. In both situations regulatory approval will be required to conduct any form of mosquito control.

The study has enabled targeted public health education information to be developed and warnings issued to residents in high risk areas. The Tasmanian Director of Public Health issued a warning in early summer 2001/02 to all residents. Following this in March 2002, Sorell Council was the only council to issue a specific warning to residents of the risk of RRV infection and to take measures to avoid being bitten by mosquitoes.

Acknowledgements

The Tasmanian Department of Health and Human Services, Public and Environmental Health Service provided the data on human cases.

Wayne Darby and Deborah Harding, Sorell Council, provided maps of the Sorell Municipal Area.

Dr Annette Broom, Arbovirus Laboratory, Department of Microbiology, University of Western Australia, kindly provided several of the monoclonal antibodies used in the Fixed Cell Enzyme-Linked Immunosorbent Assay.

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Scrub typhus in the Northern Territory: exceeding the boundaries of Litchfield National Park

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Abstract

Scrub typhus is recognised as an important differential diagnosis of fever, rash and sepsis in patients with a history of travel to Litchfield National Park in the Top End of the Northern Territory. All confirmed scrub typhus cases to date from the Northern Territory have visited the Park, but the presence of similar rainforest pockets elsewhere in the Top End suggested further infectious locations might be identified with increased tourism. We report a case of serologically confirmed *Orientia tsutsugamushi* infection in a man who had not been within Litchfield Park, but had visited another discrete Top End rainforest area. *Commun Dis Intell* 2004;28:267–269.

Keywords: scrub typhus, Orientia tsutsugamushi, *zoonoses, emerging infections, rainforest*

Scrub typhus often occurs in recognised 'mite islands', well-circumscribed foci where mite hosts (usually endemic rodents in Australia) are infected with the mite (*Leptotrombidium deliense* in Australia), which in turn is infected with *Orientia tsutsugamushi,* the rickettsial pathogen.[1]

Such foci are recognised in the high rainfall areas of Far North Queensland, the Torres Strait Islands,[2] and Litchfield National Park south of Darwin, Northern Territory, which opened to the general public in 1986.[1] Ten cases of scrub typhus, one fatal, have been confirmed serologically amongst people visiting Litchfield National Park since 1990 (Bart Currie, unpublished data), although cases had been suspected in the Top End of the Northern Territory from as early as 1937.[3] The 'Litchfield' strain of *O. tsutsugamushi* has been isolated and characterised as molecularly quite distinct from Australian strains found in north Queensland and from others in South East Asia.[3] A single case of scrub typhus has been reported from a remote location in the Kimberley area of Western Australia[4] and it was predicted that, as humans encroach on other remote rainforest locations in the tropical north, further foci with potential to transmit scrub typhus would be identified.[1]

Other emerging rickettsial infections in Australia include the recently identified Flinders Island spotted fever (*Rickettsia honei*) in Tasmania,[5] and the finding of a new endemic focus of Australian spotted fever (*Rickettsia australis*) south of Adelaide.[6]

A 40-year-old male was referred to the Royal Darwin Hospital with increasing headache, fever and malaise over 10 days. He had made a day trip for recreational shooting to the Woodcutters Mine reserve 12° 59' latitude, 131° 06' longitude, 70 km SSE of Darwin, 11 days prior to onset of symptoms. He had not been to Litchfield National Park. He did not have any other relevant travel history or infectious contacts, or past medical problems. Symptoms began with headache, and progressed over the next 10 days to include lethargy, fever, anorexia, vomiting, myalgia and rash. On examination, he was prostrate and febrile with a temperature of 39.9° C. He had a maculopapular rash affecting the upper arms and trunk, and conjunctival icterus and injection. He had crepitations and bronchial breath sounds bi-basally. He had a tender liver edge although the liver span was normal. He had a small healing wound 12 x 5 mm in diameter, in the left inguinal area, consistent with a healing eschar but without a central black crust. Laboratory tests revealed mixed pattern hepatitis with bilirubin 45 µmol/L

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(0 to 20 µmol/L), alkaline phosphatase 325 U/L (39 to 117 U/L), alanine transferase 223 U/L (5 to 44 U/L), gamma glutamyl transferase 391 U/L (0 to 66 U/L). Further results included C-reactive protein 222 mg/L (0 to 8 mg/L), white cell count 11.1 x 10^9/L (4 to 11 x 10^9/L), neutrophils 9.2 x 10^9/L (2 to 7.5 x 10^9/L), haemoglobin 118 x 10^9/L (135 to 185 g/L), platelets 201 x 10^9/L (150 to 450 x 10^9/L), creatinine 77 µmol/L (60 to 120 µmol/L) and creatine kinase 18 U/L (0 to 220 U/L). Cerebrospinal fluid contained 8 x 10^6 leucocytes per litre (95% mononuclear cells) with no erythrocytes. Chest radiography demonstrated bilateral lower lobe consolidation.

He was initially managed with intravenous ceftriaxone 2 g twice daily and oral ciprofloxacin 750 mg twice daily. On review by the Infectious Diseases Unit two days later he had not improved. Diagnosis of either scrub typhus or leptospirosis was considered likely, and ciprofloxacin was replaced with intravenous doxycycline 100 mg twice daily.

Within 24 hours he was afebrile, and he was discharged five days later. Mild headache and lethargy persisted, but he had recovered and returned to work after being reviewed six weeks later.

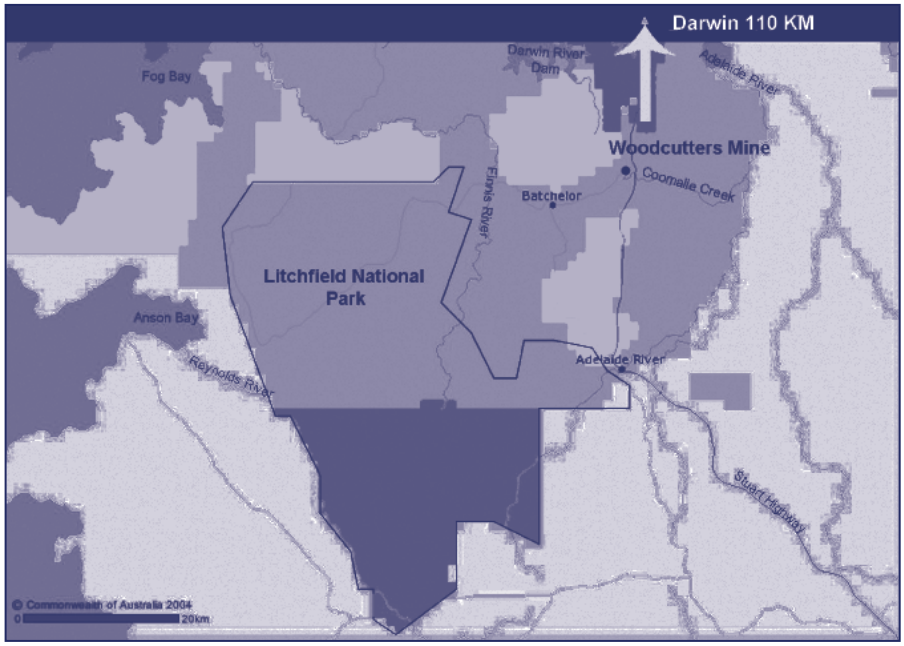
Indirect fluorescent antibody test for *O. tsutsugamushi* with sera processed in parallel demonstrated a significant rise in titre from <128 one week prior to hospital admission, to 1:512 four weeks later, confirming the diagnosis of scrub typhus. Blood cultures including culture for leptospirosis, and other serological tests including leptospirosis, HIV, hepatitis viruses, Epstein Barr virus and atypical respiratory pathogens were negative.

The Woodcutters Mine area is only 20 km from the boundary of Litchfield Park, but the rainforest habitats in the Woodcutters area are in the Coomalie Creek system which drains east to the Adelaide River system, while the Litchfield Park area drains from the elevated sandy and sandstone plateau of the Tabletop Range to the Reynolds River to the west and the Finnis River system to the east and north. Tributaries of the left branch of Coomalie Creek, which runs through the Woodcutters Reserve area, drain the same hills as some tributaries of the Finnis River, providing a close link to the two river systems. The upper reaches of these tributaries are however relatively steep, well drained, and sparsely vegetated, and dry soon after the finish of wet season rains.

The rainforest areas in Litchfield Park are primarily at the edge of the Tabletop range escarpment and are closely associated with creeks and seepage areas that drain from the escarpment to the Reynolds River or the Finnis River tributaries such as Florence Creek and Walkers Creek. These rainforest areas are discrete and characterised by emergent *Carpentaria acuminata* palms and are generally subject to perennial spring flow or seepage.

The rainforest areas in the Woodcutters area are botanically similar to Litchfield Park, and are also associated with perennial creek flow. A feature of the rainforest patches in both areas is the close proximity to perennially damp grassland and sedge areas that are probably an important element of the rodent—mite—*Orientia* ecology.[7]

Figure. Location of Woodcutters Mine reserve and Litchfield National Park, Northern Territory



Between the two locations there are however, large areas of open woodland savanna where dry season conditions with low humidity make a conjoining region of rodent—mite—*Orientia* cycles unlikely. The Woodcutters focus is therefore likely to be another discrete, stable location for potential scrub typhus transmission.

The severity of scrub typhus (50-60% mortality without antibiotics in some studies[8]), and failure to respond to conventional antibiotics, make early diagnosis of the illness and institution of appropriate treatment essential. The earlier Northern Territory fatality from scrub typhus acquired in Litchfield Park was associated with delayed diagnosis and treatment.[3] This case highlights the need to maintain a high index of suspicion for scrub typhus in the presence of a travel history to a high rainfall area anywhere in tropical northern Australia, especially remote rainforest areas, even though the region may not as yet be recognised as an area of *O. tsutsugamushi* transmission.

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Follow-up of communicable diseases reported among travellers on aeroplanes

From time to time health departments will be notified of patients who have been diagnosed with a communicable disease and have travelled in an aeroplane while infectious.

The risk of transmission to fellow travellers will vary according to the disease, the infectiousness of the case, the mode of transmission, the ventilation in the aeroplane, the dose of the exposure (which depends on duration and proximity), and the susceptibility of the other travellers.

For airborne infections, the risk may extend beyond travellers and crew on the aeroplane, and include people *en route* to and from the airport, and workers and travellers at the airport.

Aeroplane travellers could carry a variety of infections. These include those that may be airborne (such as influenza, tuberculosis, measles, and chickenpox), those spread by droplets or exposure to nasopharyngeal secretions (such as pertussis and meningococcal disease), and those spread by hands or fomites (such as rhinoviruses, or enteric infections).

The risk of infection after exposure is highly variable. Some of these infections are more serious than others. They have incubation periods that vary from a few days to weeks and longer. For some, post exposure interventions to prevent the development of the infection are available. For some, knowledge of exposure may lead to early medical attention should symptoms develop.

Public health aims to prevent disease. However, priorities must be set in balancing the resources required to prevent potential disease against the burden that disease is likely to have on the community.

In determining whether the contacts of an infectious aeroplane traveller should be contacted about their possible exposures, the following parameters should be considered:

- the risk of transmission;
- severity of the disease;
- existing recommendations about prevention;
- whether passengers that are likely to have been exposed can be readily identified;
- availability of an intervention; and
- the ability to trace other passengers in time to deliver an intervention.

The data available to guide when passengers should be contacted are patchy. There is some evidence that tuberculosis may be transmitted on long flights,[1] and the World Health Organization recommends that where a person with infectious tuberculosis has travelled on a commercial flight of more than eight hours duration in the previous three months, the airline company should inform others who were seated in the same cabin area as the infectious case, of the risk.[2] The US Centers for Disease Control and Prevention found no reports of secondary transmission of meningococcal disease among airline contacts[3] but nonetheless recommended that for flights of more than 8 hours, chemoprophylaxis be given to passengers in the seats directly beside a case. Subsequently, two passengers who travelled in different sections of the same aeroplane from Los Angeles to Sydney have been reported with serogroup B meningococcal disease in 2003.[4] Nonetheless, transmission of meningococcal infections between passengers remains very rare.

The Table provides an arbitrary summary of these parameters for selected diseases. It is likely that follow-up may only be worthwhile for those aeroplane passengers who have been seated in the same cabin area as a passenger diagnosed with infectious tuberculosis for more than eight hours or in the seats immediately next to a patient with meningococcal disease for more than eight hours. The value of following up contacts of other cases seems limited, except in the case of serious exotic diseases such as pneumonic plague or viral haemorrhagic fevers. These would need to be dealt with on a case-by-case basis.

Where significant exposures to an infectious disease have been identified among aeroplane travellers, then, depending on the risk to the travellers, options for alerting them may include public health workers contacting them directly, airline officials relaying messages, or issuing alerts through the news media.

It is important to prevent infections among travellers on aeroplanes from occurring in the first place. Potential travellers should:

1. seek medical clearance from a doctor before travelling if they have a fever with respiratory symptoms, or vomiting and diarrhoea;

2. ensure that they are immune to measles, mumps and rubella (i.e. all travellers older than one year and born since 1966 should have received two doses of measles, mumps and rubella vaccine, unless contraindicated);

3. check that they are protected from chickenpox, influenza and pertussis if they wish to avoid these infections (see the current edition of the *Australian Immunisation Handbook* for recommendations, available from http://immunise.health.gov.au/handbook.htm);

4. follow simple hygiene measures to minimise the spread of infections: covering coughs and sneezes with a disposable tissue, and wash hands with running water and soap regularly, especially after blowing the nose or using the toilet. Airlines may provide passengers who are coughing with a mask.

Note: Recommendations for following up contacts of passengers with measles are still under consideration by the communicable Diseases Network Australia. In recent years, transmission of measles on aeroplanes has rarely been documented.

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Endorsed by the Communicable Diseases Network Australia March 2004

Table. Should contacts of an infectious aeroplane traveller be contacted about their possible exposures to infectious diseases?

Parameter	Human influenza	Tuberculosis	Chickenpox	Meningococcal disease
Risk of infection	High	Moderate if smear positive case exposed >8 hours	Low among most adult travellers because of childhood infections	Low
Usual severity	Mild/variable	High	Low/variable	High
Existing recommendations for general population	Immunise if at risk of severe illness	No	Some	Early detection of symptoms and treatment
Passengers likely exposed to infection?	Whole plane	Adjacent seats	Whole plane	Adjacent seats
Post exposure prophylaxis available	Antiviral drugs	Screening, preventive therapy	Immunisation or varicella-zoster immunoglobin	Specific antibiotics
Trace travellers?	No	Seats in the same cabin area, if flight >8 hours	No	Seat beside if flight >8 hours

Parameter	Pertussis	Enteric infections	Quarantinable diseases
Risk of infection	Unclear: probably moderate if >8 hours flight	Low (few case reports), probably moderate for norovirus	Variable
Usual severity	Mild/variable	Variable	Variable
Existing recommendations for general population	Vaccination for children and some adults	Hand-washing	Variable
Passengers likely exposed to infection?	Adjacent seats	Variable/toilet users	Variable
Post exposure prophylaxis available	Specific antibiotics	No	Variable
Trace travellers?	No	Not usually	Yes for viral haemorrhagic fevers, plague

Role of the National Centre in HIV Epidemiology and Clinical Research in surveillance for HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia

The National Centre in HIV Epidemiology and Clinical Research (NCHECR) was established in 1986, as part of the Australian Government's response to the epidemic of human immunodeficiency virus (HIV) infection. The NCHECR's role was to provide national leadership in epidemiologic and clinical research on HIV infection and the acquired immune deficiency syndrome (AIDS), in support of the National HIV/AIDS Strategy.[1]

NCHECR receives core funding through the Australian Government Department of Health and Ageing (DOHA), and its work is overseen by a Scientific Advisory Committee. It is located on the campus of St Vincent's Hospital in Sydney, and is affiliated with the Faculty of Medicine at the University of New South Wales.

Under Australia's federal structure of government, state and territory health authorities have primary responsibility for surveillance for communicable diseases. In 1989, the state and territory health authorities and the NCHECR agreed to establish the National HIV Surveillance Committee, to provide national co-ordination and standardisation of HIV/AIDS surveillance activities. Under this framework, states and territories provide HIV/AIDS surveillance reports directly to the NCHECR.

In 1997, the NCHECR's terms of reference were extended to include epidemiologic monitoring of bloodborne viruses, sexually transmissible infections and other related infections, and their outcome. For these infections, the states and territories report cases to the National Notifiable Diseases Surveillance System, which is managed by the Surveillance and Epidemiology Section of the DOHA. NCHECR provides national coordination, as it does for HIV/AIDS.

In 2001, the National Viral Hepatitis Surveillance Committee and the Sexually Transmissible Infections Surveillance Committee were established as sub-Committees of the Intergovernmental Committee on AIDS, Hepatitis C and Related Diseases (IGCAHRD), which is, in turn, a sub-committee of the Communicable Diseases Network Australia. The National HIV Surveillance Committee was also made a sub-committee of IGCAHRD.

The role of the surveillance committees on HIV/AIDS, viral hepatitis and sexually transmissible infections is to standardise surveillance procedures across state and territory health jurisdictions in Australia, to develop new surveillance methods and analyses, to carry out quality control studies and to facilitate communication and feedback to people who directly contribute to surveillance activities. The surveillance committees include representation from each state and territory health authority, the Australian Government, organisations representing affected communities, specialist groups and the NCHECR. The committees meet at least twice per year, with an annual face-to-face meeting.

The NCHECR Surveillance Program coordinates national surveillance activities in its areas of responsibility through these surveillance committees. It provides secretariat support, and takes a leadership role in developing new initiatives in surveillance, following up surveillance outcomes and liaising with other expert groups where appropriate.

Developments in national surveillance for HIV/AIDS: a time line

Early 1980s

National surveillance for AIDS cases was established in the early 1980s, using the US Centers for Disease Control and Prevention (CDC) AIDS surveillance case definition and its revisions.[2] National AIDS surveillance provided information on the pattern of illness associated with advanced HIV infection and the pattern of HIV transmission in Australia. AIDS cases were notified to the National AIDS Registry with the namecode of the person with AIDS (consisting of the first two letters of the family name and the first two letters of the given name), to minimise duplicate notification while maintaining confidentiality.

National surveillance for HIV infection among blood donors was also established in the 1980s. Compulsory testing of blood donors for bloodborne viruses provides a unique opportunity for ongoing measurement of prevalence and incidence in a population subgroup at low risk of infection and for monitoring newly emerging patterns of transmission in Australia.

1989

National surveillance for cases of newly diagnosed HIV infection was established, providing a more recent indication of the pattern of HIV transmission than was available through national AIDS surveillance. State and territory health authorities provided monthly summaries of the number of cases of newly diagnosed HIV infection, broken down by sex, age group and HIV exposure category. However, very limited quality control of national HIV surveillance data could be carried out with tabulated data.

1990

Individual records of cases of newly diagnosed HIV infection, without namecode, were forwarded to the NCHECR to inclusion in the National HIV Database. While it was now possible to carry out some quality control studies, the lack of namecode meant that the extent of duplicate notification could not be accurately assessed.

In July 1990, the content and format of the quarterly *Australian HIV Surveillance Report* was substantially revised.[3] Counts of the number of new diagnoses of AIDS and HIV infection were published monthly in the *Australian HIV Surveillance Report* from August 1990 to December 1992. From January 1993, monthly counts of new HIV/AIDS diagnoses were published in *Communicable Diseases Intelligence.*

1991

National surveillance for newly acquired HIV infection was established, based on a prior negative or indeterminate HIV antibody test result in the 12 months prior to HIV diagnosis. Reports of newly acquired HIV infection provide information on the current pattern of HIV transmission.

Sentinel HIV surveillance through a network of public metropolitan sexual health clinics was established. HIV incidence and prevalence is monitored among people seen at the sexual health clinics, providing information on patterns of transmission of HIV in populations at higher risk of infection through sexual contact, primarily heterosexual contact, and other populations of special interest in HIV epidemiology such as female sex workers, people who have heterosexual contact overseas and injecting drug users.

National monitoring of HIV diagnoses among prison entrants in Australia was also established, providing information on HIV prevalence in a population subgroup at risk of infection primarily through injecting drug use.

1993

The Australian case definition for newly diagnosed HIV infection and AIDS was implemented.[4] Three illnesses were added to the CDC 1987 AIDS surveillance case definition. AIDS continued to be diagnosed on the basis of a diagnosis of one or more AIDS defining illnesses; like the European centres, Australia did not follow the US CDC, which expanded its AIDS surveillance case definition to include people with HIV infection whose CD4+ cell count was 200/µl or less.

The Sydney Men and Sexual Health cohort study was established among homosexually active men in Sydney, in collaboration with the National Centre in HIV Social Research, to monitor HIV incidence, risk behaviour for HIV infection and treatment uptake among men with diagnosed HIV infection.

National reporting of cases of newly diagnosed HIV infection by namecode was also introduced, facilitating more accurate reporting of trends in newly diagnosed HIV infection. State and territory health authorities gradually implemented notification of newly diagnosed HIV infection with namecode.

Collaboration commenced with the Australian Paediatric Surveillance Unit, providing information on the extent of perinatal exposure to HIV and perinatal HIV infection that was complementary to that available through national surveillance for newly diagnosed HIV infection. National surveillance for perinatal exposure to HIV also provides information on the prevalence of HIV infection among childbearing women and information on the use of interventions for reducing the risk of mother-to-child HIV transmission.

1994

A national program of assessment of self-report of exposure to HIV was established, for cases in which the exposure to HIV was attributed to sources other than male homosexual contact. An exposure assessment questionnaire was introduced to guide standardised sexual history taking, to provide information on the basis for exposure category classification and to record the doctor's assessment of the person's report of exposure to HIV.

1995

National reporting of Indigenous status among cases of newly diagnosed HIV infection and AIDS was established.

Sentinel surveillance for HIV and hepatitis C infection among people with a history of injecting drug use was initiated through a network of needle and syringe program sites.[5,6] Information on injecting risk behaviour and the prevalence of HIV and hepatitis C infection in program attenders has been made available annually through this surveillance system.

1996

The Gay Community Periodic Surveys were established in Sydney, in collaboration with the National Centre in HIV Social Research, providing information on risk behaviours for HIV infection and the use of antiretroviral treatment by men with diagnosed HIV infection.

Gay Community Periodic Surveys have also been carried out in Adelaide, Brisbane, Melbourne and Perth from 1998 and in Canberra from 2000.

The *Australian HIV Surveillance Report* was revised in preparation for publication of an annual report. Adjustment of the number of new HIV diagnoses for multiple reporting[7] and of AIDS cases and deaths for reporting delay was routinely incorporated in published analyses.[8]

1997

The first annual surveillance report, *HIV/AIDS and Related Diseases in Australia Annual Surveillance Report,* was published.[9] The *Annual Surveillance Report* provides a comprehensive analysis and interpretation of available national surveillance data on HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia.

1998

The Australian HIV Observational Database was established, providing information on the pattern of use of antiretroviral treatments for HIV infection among people seen through a network of hospitals, general practitioner sites and sexual health centres.

2001

The Australian AIDS Public Access Dataset was made publicly available to Australian and international health professionals, to facilitate analysis and interpretation of the occurrence of AIDS in Australia.

The Health in Men cohort study among homosexually active men in Sydney was established as a vaccine preparedness study. Incidence and risk behaviours for HIV infection and other sexually transmissible infections are monitored through the cohort.

2002

The Australian HIV Public Access Dataset was made publicly available.

Notification to the National HIV Database of information on country of birth among cases of newly diagnosed HIV infection was established.

2004

The NCHECR Surveillance Program became a collaborative unit of the Australian Institute of Health and Welfare, further strengthening its linkages in the public health system.

Current priorities in national surveillance for HIV infection and AIDS include the development and validation of specialised tests for identifying incident HIV infection, and their use in national surveillance for monitoring the pattern of HIV transmission. Improved estimates and projections of HIV/AIDS incidence in Australia in the era of antiretroviral treatment for HIV infection is another priority.

National surveillance for viral hepatitis

Information on the pattern of hepatitis C transmission, based on surveillance reports of newly acquired hepatitis C infection, was first published in *HIV/AIDS, Hepatitis C and Sexually Transmissible Infections in Australia Annual Surveillance Report 1999*.[10]

In 2000, information on the prevalence of hepatitis B surface antigen and hepatitis C in blood donors, made available through the Australian Red Cross Blood Service, was published in *HIV/AIDS, Viral Hepatitis and Sexually Transmissible Infections in Australia Annual Surveillance Report 2000*.[11]

Estimates of hepatitis C incidence among injecting drug users seen at the Kirketon Road Centre and the long-term outcome of hepatitis C infection, measured among cases of liver transplantation, recorded by the Australia and New Zealand Liver Transplant Register, were published in the *HIV/AIDS, viral hepatitis and Sexually Transmissible Infections in Australia Annual Surveillance Report 2001*.[12]

Information on the source of exposure to hepatitis C among cases of newly acquired hepatitis C, provided through state and territory health authorities, was published in *HIV/AIDS, Viral Hepatitis and Sexually Transmissible Infections in Australia Annual Surveillance Report 2002*.[13]

Current priorities in national surveillance for viral hepatitis include:

- enhancement of national surveillance for newly acquired hepatitis B and hepatitis C through more complete identification of cases and reporting of exposure category;
- improved estimates of hepatitis C incidence among people with a history of injecting drug use;
- establishment of a network for monitoring use of treatments for hepatitis C infection; and
- establishment of a network for monitoring the long-term outcome of hepatitis C infection.

Developments in national surveillance for sexually transmissible infections

The STI Surveillance Committee is currently working towards the completion of a comprehensive review of STI surveillance in Australia. The Committee has recently completed a report of current STI surveillance methods in the state and territory health jurisdictions, identifying a variety of needs in STI surveillance in the collection, quality control, analysis and reporting of routinely collected data. Acknowledging the importance of surveillance mechanisms outside routine case reporting, the Committee is also currently undertaking a review of the prevalence of chlamydia, the most common bacterial sexually transmissible infection in Australia and one of the most frequently reported notifiable infections in Australia. Following the completion of this review, the Committee will undertake similar studies of the prevalence of gonorrhoea and syphilis in Australia.

Future directions for the STI Surveillance Committee include both long and short-term goals. In the near future, the Committee will work with the states and territories to improve routine case reporting for selected bacterial STIs and, following the recommendations of the chlamydia review, develop methods for further assessing the prevalence and risk factors for chlamydia in Australia. In the longer term the Committee aims to develop a national plan for sexually transmissible infection surveillance for consideration by the Communicable Diseases Network Australia.

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A report from the Communicable Diseases Network Australia, January to March 2004

The Communicable Diseases Network Australia (CDNA) consists of communicable disease units from state and territory health departments and the Australian Government, and expert bodies and individuals in the specific areas of communicable disease epidemiology, clinical management, disease control and laboratory diagnosis. The CDNA provides national public health leadership and coordination on communicable disease surveillance, prevention and control, and offers strategic advice to governments and other key bodies on public health actions to minimise the impact of communicable diseases in Australia and the region.

Surveillance case definitions

CDNA has for some time been revising the 1994 National Health and Medical Research Centre's (NHMRC) document *Surveillance Case Definitions* and developing standard surveillance case definitions for the many new national notifiable diseases. From 1 January 2004, the *Interim Surveillance Case Definitions for the National Notifiable Disease Surveillance System* (NNDSS) have been available from http://www.cda.gov.au/surveil/nndss/dislist.htm for implementation. The definitions for over 60 national notifiable diseases were developed through a consensus approach and include clinical, laboratory and epidemiological evidence. All states and territories are to use these surveillance case definitions when notifying to the NNDSS.

National infection control guidelines

With extensive consultation and the assistance of the Australian Government Department of Health and Ageing and an expert advisory group, CDNA completed its review of the national infection control guidelines in 2003.

This review brings together the NHMRC documents *Infection Control in the Health Care Setting: Guidelines for the Prevention of Transmission of Infectious Diseases* (1996) and *Creutzfeldt-Jakob Disease and Other Human Transmissible Spongiform Encephalopathies: Guidelines on Patient Management and Infection Control* (1995). The Australian Health Ministers' Advisory Council, through the National Public Health Partnership, endorsed the new CDNA document *Infection Control Guidelines for the Prevention of Transmission of Infectious Diseases in the Health Care Setting* in January 2004.

The guidelines will be a useful resource to guide or implement infection control policy for health care establishments and individual health care workers. The scope of the guidelines is broad and applies to a wide range of health care establishments, including hospitals, (medical and dental) office practices, long-term residential care establishments, community nursing, emergency and first aid services.

Copies of the guidelines will be provided to hospitals, clinics, medical centres, surgeries, health authorities, professional organisations and aged care facilities. More information and a copy of the guidelines are available from http://www.icg.health.gov.au

Communicable diseases on aeroplanes

Health departments may be notified of patients who have been diagnosed with a communicable disease after having travelled in an aeroplane while infectious. To assist state and territory health departments in deciding whether potential contacts of an infectious aeroplane traveller should be notified about their possible exposures, CDNA has developed the document *Follow-up of Communicable Diseases Reported Among Travellers on Aeroplanes*, published in this issue of *Communicable Diseases Intelligence*, p. 270–271, and also available from the CDNA website. Recommendations for following up contacts of passengers with measles are still under consideration by CDNA.

Severe acute respiratory syndrome and highly pathogenic avian influenza

In fortnightly meetings of CDNA, members monitored global and regional activity of severe acute respiratory syndrome (SARS) and highly pathogenic avian influenza. During this period, CDNA developed the *Protocol for Isolation, Testing and Reporting of Possible Cases of Severe Acute Respiratory Syndrome (SARS)*, which is available from: http://www.health.gov.au/sars/guidelines/protocol.htm. The protocol defines criteria (clinical features and potential exposure) that must be met before testing a patient for SARS coronavirus is requested. The protocol also explains who should be contacted if a patient meets the specified criteria.

Changes to the management of meningococcal disease in Australia

The Communicable Diseases Network Australia (CDNA) has agreed to change its *Guidelines for the Early Clinical and Public Health Management of Meningococcal Disease in Australia,* June 2001, with regard to saliva.

Although salivary contact has in the past been regarded as a means of transmission of meningococci, there is little evidence to support this view. Indeed, the available evidence indicates that neither saliva nor salivary contact is important in the transmission of meningococci.

Saliva has been shown to inhibit the growth of meningococci. The inhibitory property is due to the presence of other bacteria in saliva, streptococci in particular.[1] As a result, meningococci can only rarely be isolated from saliva. In a study in the United Kingdom three swabs, one from the posterior nasopharyngeal wall, another from the tonsillar area, and the third from the front of the mouth, were taken from 258 college students and cultured for meningococci. In total, 32 per cent of the nasopharyngeal swabs and 19 per cent of the tonsillar swabs cultured *Neisseria meningitidis*. However, only one swab (0.4% of those collected from the front of the mouth) cultured the organism.[2]

Carriage of meningococci has not been convincingly shown to be associated with saliva contact. A case-control study of United Kingdom university students found no association between carriage of meningococci and sharing of drinks or cigarettes and a weak association with 'intimate kissing' (OR=1.4, CI from 1.0 to 1.8).[3] It is unclear whether carriage in these circumstances is due to saliva contact rather than to droplets shed during household-like (i.e. close and prolonged) contact.

Invasive meningococcal disease has not been shown to be associated with salivary contact. A case-control study from Auckland, New Zealand found no increased risk (p=0.07) of invasive meningococcal disease in children <8 years of age who had shared an item of food, drink or pacifier in the two weeks prior to hospitalisation.[4] A case-control study of college students in the United States of America found no association (in multi-variate analysis) between invasive meningococcal disease and kissing two or more contacts on the mouth in the month prior to the onset of the illness.[5] Clusters of invasive meningococcal disease in people who have had a low level of salivary contact (e.g. footballers who have shared drink bottles, churchgoers who have shared a communion cup) appear to be very rare. Although clusters have been described, for example, in association with sporting events[6] and sports clubs,[7] the reported details indicate that point-source salivary transmission was not involved. Secondary cases in situations where dribbling of saliva is common (e.g. child-care centres) are also rare.

As the available evidence does not support saliva or salivary contact as being important in the transmission of meningococci, chemoprophylaxis is not indicated for the following contacts of a case of invasive meningococcal disease unless they are either household-like, child-care or other very close contacts (e.g. sexual contacts):

- kissing contacts, even if mouth kissing was involved;
- food, drink (including drink bottle) sharing contacts;
- cigarette sharing, bong sharing contacts;
- communion cup, lip balm, wind instrument, referee's whistle sharing contacts; or
- any other similar low-level salivary contacts.

These revised recommendations are compatible with recently revised guidelines for the public health management of invasive meningococcal disease from the United Kingdom.[8]

The revised *Guidelines for the Early Clinical and Public Health Management of Meningococcal Disease in Australia* will be available from http://www.cda.gov.au/pubs/other/mening.htm. The CDNA and will continue to review these guidelines and, when necessary, post further amendments on the website.

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Workshop announcement

21st NRL Workshop on Serology!

The 21st National Serology Reference Laboratory Workshop on Serology will be held in Melbourne in August 2004. Since its inception the workshop has grown to become the premier event for scientists, regulators and manufacturers and distributors of diagnostic kits and reagents, to discuss advances and difficulties with serology and nucleic acid testing.

The 2004 Workshop will build on the strengths developed in previous conferences. The poster/oral presentations format and breakout sessions will once again provide a relaxed but informative environment for exchanging information and ideas. Participants can expect to be part of a vibrant learning and networking experience, through interactive sessions promoting discussion and debate on the issues that surround in vitro diagnostics. There will be interaction between clinicians, scientists, laboratory managers, blood transfusion specialists, regulators, quality assurers, pathologists, and manufacturers and distributors.

The Workshop will again feature prominent international speakers.

This year discussion on unusual diseases, difficult test interpretations, and situations where laboratory diagnoses are problematic are actively encouraged.

There remains some confusion surrounding the interpretation of HTLV and HIV-2 results. Participants are invited to submit abstracts relating to the diagnosis and management of these infections. They are particularly encouraged to discuss the clinical significance of immunoblot indeterminate reactivity.

The early diagnosis of infection continues to present problems for serologists and clinicians, and the presentation of posters on this subject is encouraged. Appropriate topics would include case studies relating to the confirmation of recent infections, and techniques for early diagnosis. Testing strategies and the interpretation of their results will also be of great interest.

Topics of interest include molecular testing including genotyping, the development and standardisation of assembled assays, and the analysis of quality control results from these assays.

The pathology industry is currently addressing the requirement to estimate the uncertainty of measurement for all quantitative tests. Discussion of this topic will be keenly sought and will help Workshop participants to expand their knowledge in this area.

Conference details

21st NRL Workshop on Serology
17-20 August 2004
Hilton on the Park
Melbourne Victoria

Workshop Secretariat

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Further information and registration forms are available on the NRL website at: www.nrl.gov.au

Communicable diseases surveillance

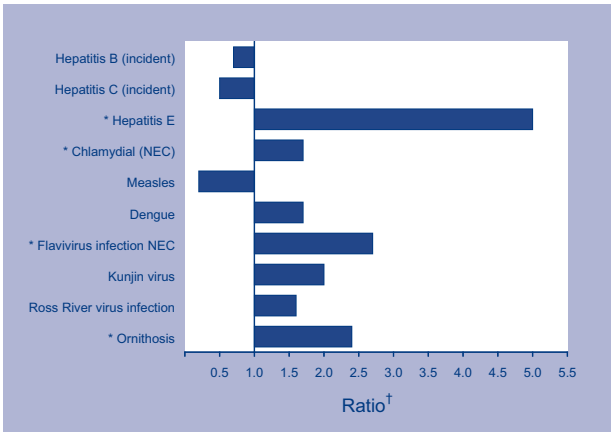
Highlights for 1st quarter, 2004

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

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

Figure 1 shows the changes in disease notifications with an onset in the first quarter of 2004, compared with a 5-year mean of the same period. Disease notifications outside the 5-year mean plus or minus two standard deviations are marked.

Figure 1. Selected* diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 January to 31 March 2004 with historical data*



* Selected diseases are chosen each quarter according to current activity.

† Ratio of current quarter total to mean of corresponding quarter for the previous five years.

‡ Notifications above or below the 5-year mean plus or minus two standard deviations for the same period.

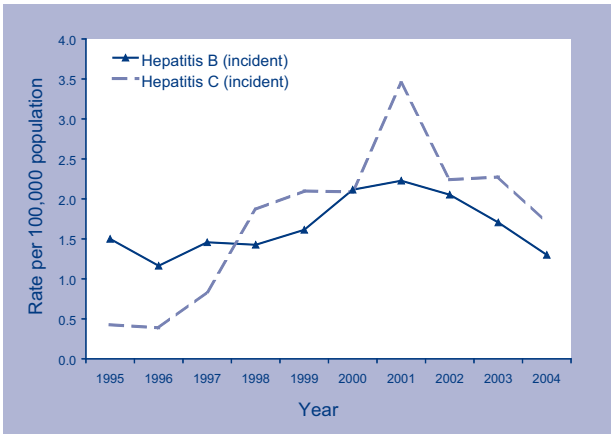
During the first quarter of 2004, there were increases in hepatitis E, *Chlamydia*, flavivirus (NEC) and ornithosis significantly above historical levels. Notifications of incident hepatitis B, incident hepatitis C and measles were lower than the five-year mean, but not outside the historical range of notifications for these diseases.

Bloodborne diseases

Incident hepatitis B and incident hepatitis C

Notification rates of incident hepatitis B and incident hepatitis C have continued to fall. There were 65 notifications of incident hepatitis B (compared to a 5-year average of 377 notifications for this quarter) and 67 notifications of incident hepatitis C (compared to an average of 472 for the quarter over the past 5 years). Trends in the national notification rates of incident hepatitis B and C are shown in Figure 2.

Figure 2. Notifications of incident hepatitis B and hepatitis C since 1995, by year of onset



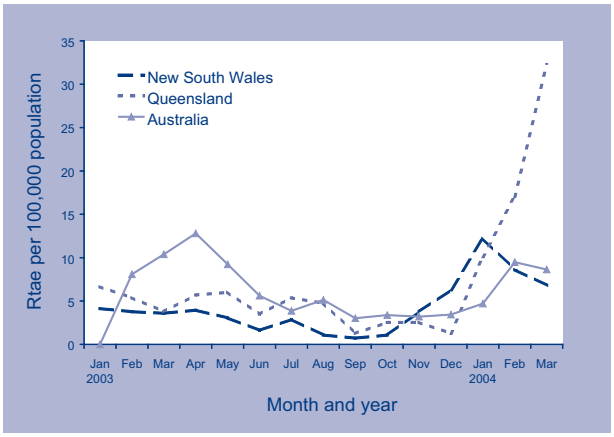
The rates of both diseases have fallen since peaking in 2001. Decreases in notifications are seen in all jurisdictions. It is unclear whether these declines represent a real decline in virus transmission or changes in testing practice or surveillance methods.

Gastrointestinal diseases

Cryptosporidiosis

Of the 503 notifications of cryptosporidiosis in the quarter, 188 were reported from Queensland and 150 from New South Wales. Although the rates of cryptosporidiosis were highest in the Northern Territory (84.7 per 100,000) and Queensland (19.8 per 100,000), the rates in New South Wales (9 per 100,000) were well above baseline (Figure 3) and the rate in January was the highest for the last three years. The NSW Health Department reported that investigations by public health units, including interviews with patients, had not identified any likely common sources of infection among cases. In Queensland, there was an increase in cryptosporidiosis in the Townsville area in February and March. Person-to-person transmission in public and private swimming pools and child-care centres was suspected.

Figure 3. Notification rates of cryptosporidiosis, New South Wales, Queensland and Australia, January 2003 to March 2004, by month of onset



Hepatitis E

There were 11 cases of hepatitis E reported in the quarter. Six cases were reported from Victoria, four from New South Wales and one from Queensland. On average over the past five years only two cases have been reported in first quarter.

Hepatitis E is the major enteric non-A, non-B hepatitis. Outbreaks have been reported from countries in South or Central Asia. Cases in Australia only occur among travellers returning from endemic areas. Travel histories of the 11 cases in the quarter revealed that all had travelled to Asian countries where hepatitis E is common (Bangladesh, India, Indonesia and China).

Shiga-like toxigenic *Escherichia coli*/verotoxin *E. coli*

Queensland reported two cases of shiga-like toxigenic *Escherichia coli* (STEC) infection presenting as haemolytic uraemic syndrome from a small rural town. One of the two cases, who were siblings, was confirmed as having an STEC infection. No common food source for this infection was identified.

Quarantinable diseases

Cholera

A single case of cholera was reported from Western Australia. The case was a 33-year-old man who had been holidaying in Bali, Indonesia. He was infected with *Vibrio cholerae* serogroup 01 and was treated without hospitalisation on his return to Western Australia.

Vaccine preventable diseases

Measles

In March, a mother and young child from New South Wales returned to Australia after acquiring measles while travelling in India. The cases were investigated by Public Health Unit (PHU) staff, who assessed the child to be in the highly infectious phase whilst returning home. The mother reported that she and the child had never received measles vaccine. Contacts at risk of infection in Australia included susceptible people who shared two flights to Brisbane, others at the airport, and other patients and staff at the medical clinic attended by the child. In response, NSW Health issued a media release warning other travellers to be alert for signs of measles, and the airline company agreed to contact passengers on the flight to alert them of the risk. Other social and health care contacts of the cases were contacted and offered immunisation or immunoglobulin.

Later in March the South Eastern Sydney Public Health Unit (SES PHU) investigated the case of a man in his twenties who developed measles after returning from Japan. Before the onset of his rash, he attended a concert at a local club. SES PHU traced close contacts at risk of infection, and issued local alerts to other patrons through the media and a sign at the club. Two weeks later three secondary cases in young adults who had attended the same concert were reported by alert clinicians. SES PHU found that these subsequent cases in turn had large numbers of contacts while infectious, including guests at a wedding, participants in a multi-day bike race, work and social contacts, and other patients sharing medical waiting rooms. A large number of people were also potentially exposed via public transport and other public venues and a general media alert was issued.

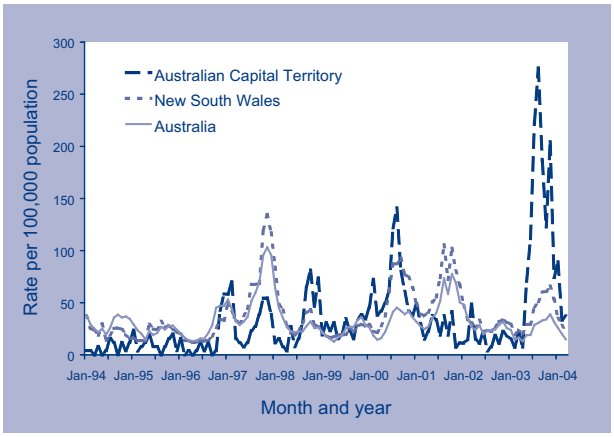
A total of 55 contacts between the ages of 0–38 years were identified. PHU staff contacted 49 of these. Of these, 44 were advised to attend an immunisation clinic provided the following day. Thirty-three of the 44 people contacted attended the clinic and received either the measles-mumps-rubella vaccine, normal human immunoglobulin (NHIG) or had serology tests for measles immunity. Remaining contacts over the age of 38 years as well as any people who accompanied them into the emergency department (ED) were contacted. Of 23 people contacted, four were advised to attend the ED for NHIG, of these two were found to be immunocompromised and two were within the susceptible age cohort.

Pertussis

There were 1,003 cases of pertussis reported to NNDSS in the first quarter of 2004, 468 of these were reported from New South Wales (rate 28 per 100,000) and 43 from the Australian Capital Territory (rate 53.3 per 100,000) (Figure 4). The number of notifications of pertussis have been high in the Australian Capital Territory since 2003, when 355 cases were reported, compared with 54 cases in 2002.

In response to the increase in notifications in the adolescent age group, the NSW Health Department sought advance funding from the Australian Government for the purchase of additional vaccines and will conduct a state-wide program commencing on 3 May 2004 to offer pertussis vaccine (DTPa) to all high school students.

Figure 4. Trends in notification rates of pertussis, New South Wales, the Australian Capital Territory and Australia, 1994 to 2004, by month of onset



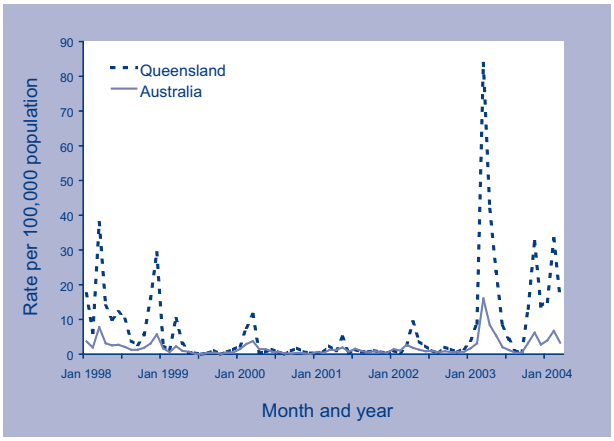
Vectorborne diseases

Dengue

During the quarter 204 of the 233 notifications of dengue were from Queensland, a rate of 21.5 cases per 100,000 population (Figure 5). This elevated rate was the product of three on-going outbreaks that began late in 2003 in the north Queensland towns of Cairns and Townsville and the Torres Strait islands. Up to the end of the quarter, more than 270 cases were recorded from the Torres Strait since the outbreak began in September 2003; more than 50 cases in Cairns and 30 cases in Townsville since October 2003. Several serotypes of dengue have been circulating in Queensland during these outbreaks. The first death from dengue haemorrhagic fever in Australia for 100 years was reported during the quarter. There have been more than 20 hospitalisations of cases of dengue haemorrhagic fever, a severe illness thought to be increased in populations exposed to different serotypes of the dengue virus.

The mosquito vector for dengue, *Aedes aegypti* was detected in Tennant Creek in the Northern Territory in February 2004. This species of mosquito has not been endemic in the Northern Territory for more than 50 years. Mosquito control activities including fogging in residential and public places, distribution of surface sprays, removal of water-filled receptacles and residual insecticide spraying were initiated along with public awareness campaigns. No human cases of dengue were reported in Tennant Creek up to the end of March.

Figure 5. Notification rate of dengue, Queensland and Australia, 1998 to 2004 (YTD)



Japanese encephalitis virus

Sentinel pigs tested positive for Japanese encephalitis virus (JEV) in Torres Strait and at Bamaga, Cape York. The latter is the first incursion into the Australian mainland since 1998, when there was a single human case of JEV infection in the Cape York region. Mosquito trappings are being conducted to ascertain whether the vectors are carrying the virus.

Murray Valley encephalitis virus

Sentinel chicken seroconversions for Murray Valley encephalitis virus (MVEV) were reported in the Northern Territory (Darwin), South Australia and Western Australia (Pilbara) in March. In early April a case of MVEV was confirmed in an infant in Central Australia. Although the arbovirus season is coming to an end with the onset of cooler weather, the Communicable Diseases Network Australia has recommended increased sentinel chicken surveillance for arboviruses in the coming season.

Ross River virus

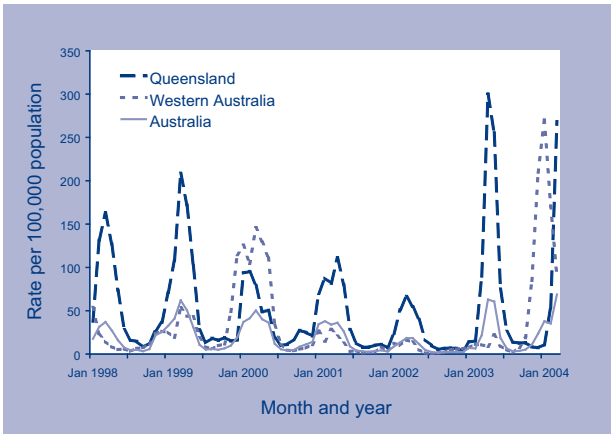
The outbreak of RRV, which began in the fourth quarter of 2003 in Western Australia, continued into the first quarter of 2004. Western Australia reported 522 cases with onset in the fourth quarter of 2003 and 844 cases reported in the first quarter of 2004 (compared to 27 and 46 cases for the fourth quarter in 2002 and first quarter of 2003, respectively). The majority of cases have been reported from the South-West region of the state in residents of, or visitors to, coastal areas stretching from Mandurah to Busselton. Transmission has also occurred across the Perth metropolitan area, particularly around the fringes.

The Swan coastal plain area contains two large estuarine systems (Peel-Harvey and Leschenault) and many other salt marshes and brackish wetland areas that are breeding sites of *Ochlerotatus camptorhynchus* and *Oc. vigilax*, the major vectors of RRV in the south-west of Western Australia. Historically, large epidemics occur every three or four years between October and March in South-West Western Australia, associated with spring or summer rains, high tides that flood salt marsh mosquito breeding areas, and low levels of immunity in kangaroos and other amplifying vertebrate animal species. Mosquito trapping this season revealed relatively low numbers of mosquitoes, but high levels of virus carriage.

Public health response to the epidemic has included local and state-wide media warnings including advice regarding mosquito avoidance, and local government mosquito control activities in mosquito breeding areas, especially those close to human population centres.

Increases in RRV were also seen in Queensland with 1,007 cases reported in the quarter of which 811 were reported with an onset in March. Cases were reported from across the State with north Queensland and the Darling Downs having a much higher number than their historical average.

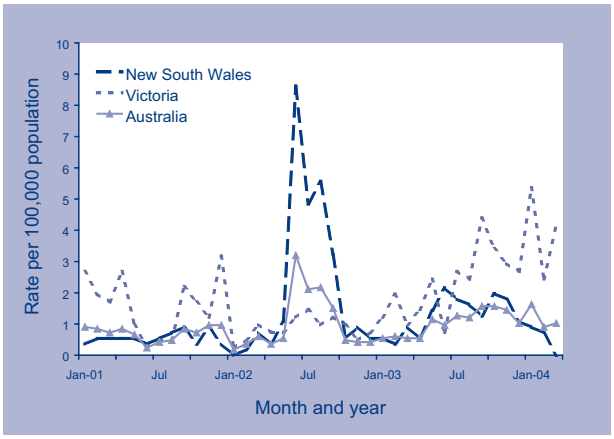
Figure 6. Notification rates for Ross River virus infections, Queensland, Western Australia and Australia, 1998 to 2004 (YTD)



Ornithosis

There were 60 cases of ornithosis reported in the quarter, which was 2.4 times the average for this period over the past five years. Forty-nine (82%) of these cases were reported from Victoria. Fourteen cases were linked to a duck processing plant in rural Victoria. In New South Wales eight cases were linked to a pet shop.

Figure 7. Trends in notifications of ornithosis, New South Wales, Victoria and Australia, 2001 to 2004 (YTD), by month of notification



Other bacterial infections

Legionellosis

There were 76 cases of legionellosis reported in the quarter, which was close to the historical average number of cases for this period of the year. Among the 20 cases reported from Victoria was a cluster of three cases of *Legionella pneumophila* serogroup 1. All three cases were male and aged more than 50 years.

Among the 22 cases reported from New South Wales were two clusters—one of *L. pneumophila* and the second of *L. longbeachae*.

In March 2004, investigations by the South Eastern Sydney Public Health Unit identified a possible linked cluster of six cases of Legionnaires' disease caused by *L. pneumophila*. The onset of illness for these cases ranged from late January to early March. They were aged between 23 and 65 years and four were male. Five reported movements in one small area of Oxford Street during their possible exposure period. Four also had movements in various other areas of the central business district (CBD). Another person with Legionnaires' disease due to *L. pneumophila* who was hospitalised in the Hunter Area, was also found to have stayed in the Sydney CBD for two days in this period.

SES PHU alerted local hospital and general practitioners, and worked with the Sydney City Council to assess the routine disinfection processes of nearby cooling towers and a large fountain. No source of the outbreak was identified. Sydney City Council wrote to the operators of all of the approximately 1,800 cooling towers in the CBD area to reinforce the need for careful assessment and disinfection of cooling towers. To identify any further related cases, all New South Wales public health units were advised of the cluster and requested to contact their local hospitals and laboratories for other possible cases of Legionnaires' disease and to question any suspected cases about their movements in the 10 days before onset of illness. NSW Health issued a state-wide media release to alert the public and building operators. By the end of March a further two possibly linked cases were identified, both with onset of illness in early March, but there were no reports of new linked cases acquired after the public health action was taken.

From January to March 2004, four cases of Legionnaires' disease due to *L. longbeachae* infection in the Illawarra and a fifth case in Sydney were reported. The patients were all aged over 60 years and three were men. Two of the cases died. Four of the five people reported using potting mix before the onset of their illness. *L. longbeachae* infection has previously been linked to gardening, particularly the use of potting mixes.

In response to this cluster of cases NSW Health released a media alert to again warn the public of the risk of Legionnaires' disease associated with gardening and potting mix. Reducing exposure to potting mix dust by following manufacturers instructions printed on the potting mix bags is vital in preventing infection from *Legionella* bacteria. People should avoid breathing in potting mix dust, wear gloves and a mask, and wash their hands immediately after handling potting mix or soil, especially before eating or drinking.

Invasive meningococcal disease

There were 89 cases of invasive meningococcal disease reported in the quarter. This level of disease is close to the historical average for the number of cases of this period. In Queensland among 20 cases reported in the quarter, six cases of serogroup B (of various serotypes) disease occurred in Indigenous children aged three months to 10 years. These six cases were spread across the State and had dates of onset from January to March. Three were vaccinated with the conjugate serogroup C vaccine and three were unvaccinated.

With contributions from:

Mark Bartlett, NSW Health Department

Craig Davis, Queensland Health

Trang Vu and Joy Gregory, Department of Human Services, Victoria

Gary Dowse and Carolien Giele, Health Department of Western Australia.

Tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 29,577 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 January and 31 March 2004 (Table 2). The notification rate of diseases per 100,000 population for each State or Territory is presented in Table 3.

There were 5,006 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 January to 31 March 2004 (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 jurisdictions except NT
Hepatitis C (incident)	All jurisdictions except Qld
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Hepatitis (NEC)	All jurisdictions except WA
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except NSW
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
SLTEC, VTEC	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Smallpox	All jurisdictions except ACT, NSW, Qld, SA
Tularemia	All jurisdictions except ACT, NSW, NT, Qld, SA
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis (unspecified)	All jurisdictions
Syphilis < 2 years duration	All jurisdictions except NSW, Qld, SA, Tas, Vic
Syphilis > 2 years duration	All jurisdictions except ACT, NT
Syphilis - congenital	All jurisdictions except ACT, NT

Disease	Data received from:
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease	All jurisdictions
Poliomyelitis	All jurisdictions
Rubella	All jurisdictions
Tetanus	All jurisdictions
Vectorborne diseases	
Barmah Forest virus infection	All jurisdictions
Flavivirus infection (NEC)	All jurisdictions
Dengue	All jurisdictions
Japanese encephalitis	All jurisdictions
Kunjin	All jurisdictions except ACT*
Malaria	All jurisdictions
Murray Valley encephalitis	All jurisdictions except ACT*
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Ornithosis	All jurisdictions
Other lyssaviruses (NEC)	All jurisdictions
Q fever	All jurisdictions
Other bacterial infections	
Creutzfeldt-Jakob disease	All jurisdictions except, ACT, NSW, NT, Qld, SA
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

* In the Australian Capital Territory, 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 2004, by date of notification*

Disease	State or territory								Total 1st quarter 2004[1]	Total 4th quarter 2003	Total 1st quarter 2003	Last 5 years mean 1st quarter	Year to date 2004	Last 5 years YTD mean	Ratio[†]
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis B (incident)	0	12	2	15	1	2	23	10	65	65	93	76	65	92	0.7
Hepatitis B (unspecified)	15	605	NN	212	60	23	391	114	1,420	1,464	1,628	1,654	1,420	1,738	0.8
Hepatitis C (incident)	1	6	NN	NN	15	2	14	29	67	87	124	72	67	130	0.5
Hepatitis C (unspecified)	63	1,711	64	782	153	70	766	272	3,881	3,887	3,866	4,525	3,881	4,627	0.8
Hepatitis D	0	4	0	0	0	0	0	0	4	6	6	4	4	5	0.9
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	18	16	0	0	3	0.0
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Campylobacteriosis[2]	96	NN	63	1,118	450	171	1,777	568	4,243	3,890	4,490	4,735	4,243	3,624	1.2
Cryptosporidiosis[‡]	5	150	42	188	13	0	54	51	503	189	520	581	503	1,050	0.5
Haemolytic uraemic sy0ome	0	2	0	0	0	0	1	0	3	6	5	3	3	5	0.6
Hepatitis A	0	48	5	4	6	1	18	21	103	96	118	112	103	227	0.5
Hepatitis E	0	4	0	1	0	0	6	0	11	1	2	11	11	2	5.0
Listeriosis	0	6	1	3	0	1	2	3	16	17	20	20	16	19	0.8
Salmonellosis	27	773	120	1,143	143	41	302	191	2,740	1,602	2,761	3,105	2,740	2,640	1.0
Shigellosis	0	27	26	17	31	1	13	36	151	83	153	175	151	155	1.0
SLTEC,VTEC[3]	0	0	0	5	4	0	3	0	12	9	19	14	12	18	0.7
Typhoid	0	13	0	4	1	0	6	3	27	12	19	33	27	26	1.1
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	1	1	0	0	1	1	1	1.3
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
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
Tularemia	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
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. Notifications of diseases received by State and Territory health authorities in the period 1 January to 31 March 2004, by date of notification,* *continued*

Disease	State or territory								Total 1st quarter 2004[1]	Total 4th quarter 2003	Total 1st quarter 2003	Last 5 years mean 1st quarter	Year to date 2004	Last 5 years YTD mean	Ratio†
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible diseases															
Chlamydial (NEC)	162	2,496	371	2,152	539	136	1,976	1,054	8,886	7,323	7,343	9,955	8,886	5,176	1.7
Donovanosis	0	0	1	1	0	0	0	0	2	1	7	2	2	7	0.3
Gonococcal infection[4]	9	353	372	267	65	4	308	315	1,693	1,450	1,768	1,879	1,693	1,590	1.1
Syphilis (unspecified)	6	0	64	0	3	5	0	59	137	183	234	145	137	400	0.3
Syphilis < two years duration	0	57	0	29	0	0	16	0	102	62	97	106	102	NA	NA
Syphilis > two years duration	0	279	0	41	0	0	67	0	387	313	207	417	387	NA	NA
Syphilis - congenital	0	0	0	0	0	0	0	0	0	2	2	0	0	2	0.0
Vaccine preventable disease															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Haemophilus influenzae type b	0	1	1	0	1	2	0	0	5	4	2	5	5	6	0.9
Influenza (laboratory confirmed)‡	0	46	1	22	10	0	16	10	105	256	98	117	105	90	1.2
Measles	0	6	0	0	1	0	4	0	11	17	22	12	11	45	0.2
Mumps	2	15	0	1	0	0	2	4	24	27	21	26	24	30	0.8
Pertussis	43	468	1	156	42	13	208	72	1,003	1,736	911	1,088	1,003	1,177	0.9
Pneumococcal disease (invasive)‡	9	100	17	60	40	2	70	17	315	526	299	376	315	260	1.2
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Rubella	0	5	0	1	0	0	0	0	6	2	36	7	6	56	0.1
Rubella - congenital	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0.0
Tetanus	0	0	0	1	0	0	0	0	1	0	2	1	1	2	0.6
Vectorborne diseases															
Barmah Forest virus infection	0	96	5	192	0	0	3	22	318	158	279	377	318	266	1.2
Dengue	3	9	10	204	3	1	2	1	233	203	346	246	233	139	1.7
Flavivirus infection NEC	0	4	1	54	0	0	4	0	63	27	20	77	63	24	2.7
Japanese encephalitis‡	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Kunjin virus‡	0	0	0	6	0	0	0	0	6	2	12	6	6	3	2.0
Malaria	5	20	5	56	2	3	14	5	110	138	181	129	110	213	0.5
Murray Valley encephalitis‡	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0.0
Ross River virus infection	2	235	182	1,007	19	13	50	844	2,352	658	584	2,881	2,352	1,433	1.6

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 January to 31 March 2004, by date of notification,* *continued*

Disease	State or territory								Total 1st quarter 2004[1]	Total 4th quarter 2003	Total 1st quarter 2003	Last 5 years mean 1st quarter	Year to date 2004	Last 5 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	0	0	0	0.0
Australian bat lyssavirus‡	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Brucellosis	0	0	0	3	0	0	1	0	4	2	4	6	4	7	0.6
Leptospirosis	0	10	0	54	0	0	0	0	64	22	47	67	64	76	0.8
Other lyssavirus (NEC)‡	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Ornithosis	0	9	0	0	2	0	49	0	60	67	28	62	60	25	2.4
Q fever	1	46	2	41	3	0	3	1	97	107	208	101	97	174	0.6
Other bacterial infections															
Creutzfeldt-Jakob disease	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
Legionellosis	0	22	0	9	9	1	20	15	76	93	86	87	76	80	1.0
Leprosy	0	1	0	1	0	0	0	0	2	0	2	2	2	1	1.4
Meningococcal infection	5	28	4	20	4	2	21	5	89	142	88	107	89	106	0.8
Tuberculosis	3	56	6	19	18	1	64	10	177	227	218	186	177	265	0.7
Total	457	7,723	1,366	7,889	1,638	495	6,274	3,735	29,577	25,181	26,992	33,591	29,577	26,074	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 *Escherichia coli* (SLTEC/VTEC).
4. Northern Territory, Queensland, South Australia , Victoria and Western Australia: includes gonococcal neonatal ophthalmia.

* 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 unit.

† Ratio = ratio of current quarter total to the mean of last 5 years.

‡ Notifiable from January 2001 only. Ratio and mean calculations are based the last two years.

NN Not notifiable

NEC Not elsewhere classified.

NA Not calculated as only notifiable for under 5 years.

Table 3. Notification rates of diseases by state or territory, 1 January to 31 March 2004. (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	0.7	4.0	1.6	0.3	1.7	1.9	2.0	1.3
Hepatitis B (unspecified)[†,‡]	18.6	36.2	NN	22.3	15.7	19.3	31.9	23.4	28.9
Hepatitis C (incident)	1.2	0.4	NN	NN	3.9	1.7	1.1	5.9	1.7
Hepatitis C (unspecified)	78.1	102.4	129.1	82.4	40.1	58.7	62.5	55.7	78.1
Hepatitis D	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1
Hepatitis (NEC)	0.0	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
Campylobacteriosis[2]	118.9	NN	127.0	117.8	117.8	143.4	144.9	116.4	128.8
Cryptosporidiosis	6.2	9.0	84.7	19.8	3.4	0.0	4.4	10.4	10.1
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Hepatitis A	0.0	2.9	10.1	0.4	1.6	0.8	1.5	4.3	2.1
Hepatitis E	0.0	0.2	0.0	0.1	0.0	0.0	0.5	0.0	0.2
Listeriosis	0.0	0.4	2.0	0.3	0.0	0.8	0.2	0.6	0.3
Salmonellosis	33.5	46.2	242.0	120.4	37.4	34.4	24.6	39.1	55.2
Shigellosis	0.0	1.6	52.4	1.8	8.1	0.8	1.1	7.4	3.0
SLTEC,VTEC[3]	0.0	0.0	0.0	0.5	1.0	0.0	0.2	0.0	0.2
Typhoid	0.0	0.8	0.0	0.4	0.3	0.0	0.5	1.0	0.6
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	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
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tularemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sexually transmissible diseases									
Chlamydial infection	200.7	149.3	748.2	226.7	141.2	114.0	161.1	216.0	178.9
Donovanosis	0.0	0.0	2.0	0.1	0.0	0.0	0.0	0.0	0.0
Gonococcal infection[4]	11.2	21.1	750.2	28.1	17.0	3.4	25.1	64.5	34.1
Syphilis (unspecified)	7.4	0.0	129.1	0.0	0.8	4.2	0.0	12.1	2.8
Syphilis < 2 years duration	0.0	3.4	0.0	3.1	0.0	0.0	1.3	0.0	2.1
Syphilis > 2 years duration	0.0	16.7	0.0	4.3	0.0	0.0	5.5	0.0	7.8
Syphilis - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3. Notification rates of diseases by state or territory, 1 January to 31 March 2004. (Rate per 100,000 population) , *continued*

Disease[1]	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	2.0	0.0	0.3	1.7	0.0	0.0	0.1
Influenza (laboratory confirmed)	0.0	2.8	2.0	2.3	2.6	0.0	1.3	2.0	2.1
Measles	0.0	0.4	0.0	0.0	0.3	0.0	0.3	0.0	0.2
Mumps	2.5	0.9	0.0	0.1	0.0	0.0	0.2	0.8	0.5
Pertussis	53.3	28.0	2.0	16.4	11.0	10.9	17.0	14.8	20.2
Pneumococcal disease	11.2	6.0	34.3	6.3	10.5	1.7	5.7	3.5	6.3
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.3	0.0	0.1	0.0	0.0	0.0	0.0	0.1
Rubella - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Vectorborne diseases									
Barmah Forest virus infection	0.0	5.7	10.1	20.2	0.0	0.0	0.2	4.5	6.4
Dengue	3.7	0.5	20.2	21.5	0.8	0.8	0.2	0.2	4.7
Flavivirus infection NEC	0.0	0.2	2.0	5.7	0.0	0.0	0.3	0.0	1.3
Japanese encephalitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus	0.0	0.0	0.0	0.6	0.0	0.0	0.0	0.0	0.1
Malaria	6.2	1.2	10.1	5.9	0.5	2.5	1.1	1.0	2.2
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	2.5	14.1	367.0	106.1	5.0	10.9	4.1	172.9	47.4
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	0.3	0.0	0.0	0.1	0.0	0.1
Leptospirosis	0.0	0.6	0.0	5.7	0.0	0.0	0.0	0.0	1.3
Other lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.5	0.0	0.0	0.5	0.0	4.0	0.0	1.2
Q fever	1.2	2.8	4.0	4.3	0.8	0.0	0.2	0.2	2.0
Other bacterial infections									
Creutzfeldt-Jakob disease	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Legionellosis	0.0	1.3	0.0	0.9	2.4	0.8	1.6	3.1	1.5
Leprosy	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Meningococcal infection	6.2	1.7	8.1	2.1	1.0	1.7	1.7	1.0	1.8
Tuberculosis	3.7	3.3	12.1	2.0	4.7	0.8	5.2	2.0	3.6

1. Rates are subject to retrospective revision.
2. Not reported for New South Wales because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
3. Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (SLTEC/VTEC).
4. Northern Territory, Queensland, South Australia, Victoria and Western Australia: includes gonococcal neonatal ophthalmia.

NN Not Notifiable.

NEC Not Elsewhere Classified.

Table 4. Virology and serology laboratory reports by state or territory[1] for the reporting period 1 January to 31 March 2004, and total reports for the year[2]

	State or territory								This period 2004	This period 2003	Year to date 2004[3]	Year to date 2003
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	0	0	0	3	2	0	2	0	7	14	7	14
Mumps virus	0	0	0	1	0	0	1	0	2	5	2	5
Rubella virus	0	0	0	4	0	0	2	0	6	8	6	8
Hepatitis virus												
Hepatitis A virus	0	3	1	2	3	0	1	0	10	17	10	17
Hepatitis D virus	0	1	0	2	0	0	0	0	3	6	3	6
Hepatitis E virus	0	0	0	0	0	0	5	0	5		5	
Arboviruses												
Ross River virus	0	11	10	383	14	3	8	2	431	212	431	212
Barmah Forest virus	0	7	1	73	1	0	2	0	84	77	84	77
Flavivirus (unspecified)	0	1	5	36	0	0	6	1	49	39	49	39
Adenoviruses												
Adenovirus not typed/ pending	0	35	1	21	76	0	30	0	163	213	163	213
Herpesviruses												
Cytomegalovirus	1	72	1	39	58	3	27	0	201	271	201	271
Varicella-zoster virus	0	46	3	267	106	1	15	1	439	419	439	419
Epstein-Barr virus	0	25	26	265	233	1	8	69	627	458	627	458
Other DNA viruses												
Poxvirus group not typed	0	0	0	0	0	1	0	0	1	1	1	1
Parvovirus	0	3	0	30	5	2	21	0	61	50	61	50
Picornavirus family												
Coxsackievirus A16	0	1	0	0	1	0	0	0	2	2	2	2
Echovirus type 11	0	1	0	0	0	0	0	0	1	1	1	1
Echovirus type 18	0	1	0	0	0	0	0	0	1		1	
Poliovirus type 1 (uncharacterised)	0	2	0	0	0	0	0	0	2	9	2	9
Poliovirus type 2 (uncharacterised)	0	1	0	0	0	0	0	0	1	1	1	1
Rhinovirus (all types)	0	57	0	0	9	0	1	0	67	124	67	124
Enterovirus type 71 (BCR)	0	2	0	0	0	0	0	0	2		2	
Enterovirus not typed/ pending	0	5	0	7	3	0	16	0	31	44	31	44
Picornavirus not typed	0	0	0	0	0	2	0	0	2	2	2	2
Ortho/paramyxoviruses												
Influenza A virus	0	3	0	6	24	0	5	0	38	103	38	103
Influenza B virus	0	0	0	0	12	0	0	0	12	27	12	27
Parainfluenza virus type 1	0	24	0	2	4	0	5	0	35	16	35	16
Parainfluenza virus type 2	0	0	0	0	2	0	0	0	2	19	2	19
Parainfluenza virus type 3	0	24	0	1	56	0	7	0	88	111	88	111
Respiratory syncytial virus	0	103	1	54	10	2	12	0	182	142	182	142

Table 4. Virology and serology laboratory reports by state or territory[1] for the reporting period 1 January to 31 March 2004, and total reports for the year,[2] *continued*

	State or territory								This period 2004	This period 2003	Year to date 2004[3]	Year to date 2003
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other RNA viruses												
HTLV-1	0	0	0	0	2	0	0	0	2	4	2	4
Rotavirus	0	6	0	0	45	2	22	0	75	59	75	59
Norwalk agent	0	0	0	0	0	0	66	0	66	28	66	28
Other												
Chlamydia trachomatis - A-K	0	1	0	0	0	0	0	0	1	–	1	–
Chlamydia trachomatis - L1-L3	0	0	0	0	0	0	1	0	1	–	1	–
Chlamydia trachomatis - not typed	7	143	5	589	381	9	9	1	1,144	1,231	1,144	1,231
Chlamydia pneumoniae	0	0	0	0	0	0	2	0	2	4	2	4
Chlamydia psittaci	1	0	0	2	2	0	54	0	59	17	59	17
Mycoplasma pneumoniae	0	49	8	139	89	1	63	6	355	201	355	201
Mycoplasma hominis	0	1	0	0	0	0	0	0	1	4	1	4
Coxiella burnetii (Q fever)	0	3	1	13	29	0	2	0	48	56	48	56
Streptococcus group A	0	1	0	82	0	0	42	0	125	145	125	145
Yersinia enterocolitica	0	1	0	0	0	0	0	0	1	2	1	2
Brucella abortus	0	0	0	0	1	0	2	0	3	1	3	1
Bordetella pertussis	4	12	0	18	33	0	88	0	155	139	155	139
Legionella pneumophila	0	0	0	0	4	0	16	0	20	43	20	43
Legionella longbeachae	0	1	0	0	6	1	8	0	16	10	16	10
Legionella species	0	0	0	0	0	0	4	0	4	3	4	3
Cryptococcus species	0	0	0	3	10	0	0	0	13	3	13	3
Leptospira species	0	0	0	12	1	0	0	0	13	6	13	6
Treponema pallidum	0	44	0	173	106	0	2	0	325	372	325	372
Entamoeba histolytica	0	0	0	0	0	1	3	0	4	3	4	3
Toxoplasma gondii	0	4	0	4	2	2	2	0	14	14	14	14
Echinococcus granulosus	0	0	0	0	4	0	0	0	4	5	4	5
Total	13	694	63	2,231	1,334	31	560	80	5,006	4,741	5,006	4,741

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

– No data received this period

Table 5. Virology and serology reports by laboratories for the reporting period 1 January to 31 March 2004*

State or territory	Laboratory	January 2004	February 2004	March 2004	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	40	36	121	197
	New Children's Hospital, Westmead	45	56	52	153
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	14	21	30	65
	South West Area Pathology Service, Liverpool	61	72	92	225
Queensland	Queensland Medical Laboratory, West End	482	996	857	2,335
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	431	427	473	1,331
Tasmania	Northern Tasmanian Pathology Service, Launceston	7	5	7	19
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Monash Medical Centre, Melbourne	11	18	4	33
	Royal Children's Hospital, Melbourne	90	95	78	263
	Victorian Infectious Diseases Reference Laboratory, Fairfield	79	104	94	277
Western Australia	PathCentre Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	15	50	43	108
Total		1,275	1,880	1,851	5,006

* The complete list of laboratories reporting for the 12 months, January to December 2004, 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

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 2004, nine conditions are being monitored, four of which are related to communicable diseases. These include influenza, gastroenteritis, varicella and shingles. There are two definitions for influenza for 2004. A patient may be coded once or twice depending on their symptoms. The definition for influenza 1 will include more individuals. Definitions of these conditions were published in Commun Dis Intell *2004;28:99–100.*

Data from 1 January to 31 March 2004 are shown as the rate per 1,000 consultations in Figures 8, 9, 10 and 11.

Figure 8. Consultation rates for influenza-like illness, ASPREN, 1 January to 31 March 2004, by week of report

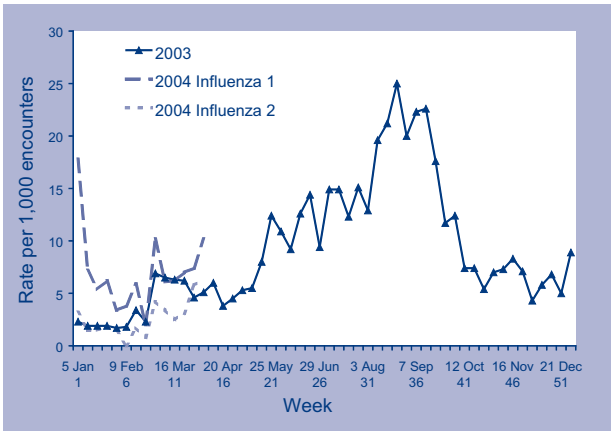


Figure 9. Consultation rates for gastroenteritis, ASPREN, 1 January to 31 March 2004, by week of report

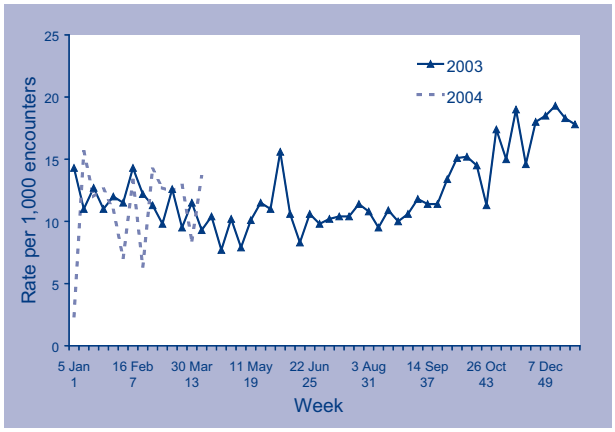


Figure 10. Consultation rates for varicella, ASPREN, 1 January to 31 March 2004, by week of report

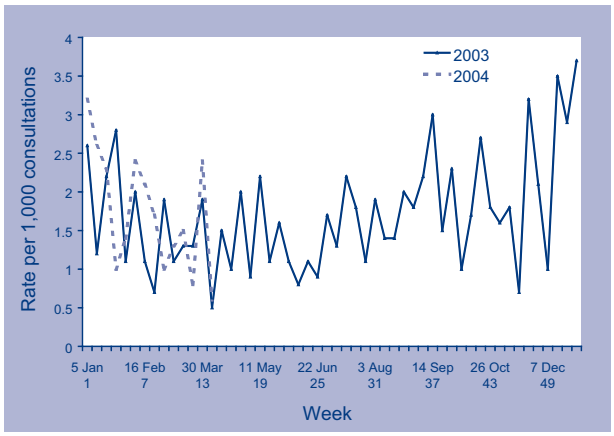
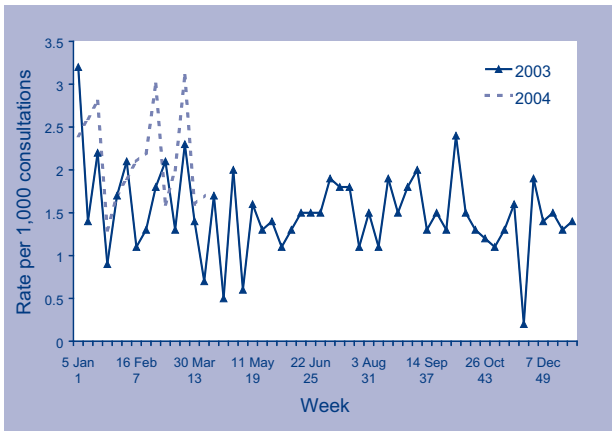


Figure 11. Consultation rates for shingles, ASPREN, 1 January to 31 March 2004, by week of report



Childhood immunisation coverage

Tables 10, 11 and 12 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR).

The data show the percentage of children fully immunised at 12 months of age for the cohort born between 1 October and 31 December 2002, at 24 months of age for the cohort born between 1 October and 31 December 2001, and at 6 years of age for the cohort born between 1 October and 31 December 1997 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.

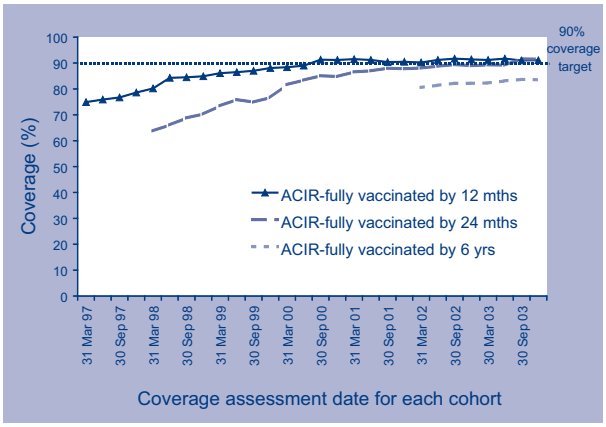
Immunisation coverage for children 'fully immunised' at 12 months of age for Australia increased marginally from the last quarter by 0.1 percentage points to 91.1 per cent (Table 10). There were substantial increases in 'fully immunised' coverage by state or territory in two jurisdictions, the Northern Territory (+3.5%) and the Australian Capital Territory (+4.8%), whilst all other jurisdictions experienced very little change in coverage. The Northern Territory and the Australian Capital Territory also experienced increases in coverage for diphtheria, tetanus, pertussis (DTP), poliomyelitis (OPV), and *Haemophilus influenzae* type b (Hib) vaccines. Significant changes in coverage in jurisdictions like the Northern Territory and the Australian Capital Territory, which have relatively small populations, are likely to be the result of a small number of children having a large impact on the coverage percentages.

National coverage for children 'fully immunised' at 24 months of age decreased marginally from the last quarter by 0.1 percentage points to 91.5 per cent (Table 11). Coverage for individual vaccines for Australia remained largely unchanged. DTP coverage remained high for this age group in all jurisdictions due to the removal of the 4th dose of DTP (due at 18 months) from the immunisation schedule from the December 2003 quarter onwards. The only other significant jurisdictional change in coverage for this age group was a decrease in 'fully immunised' coverage in Tasmania (–2.3%) with a decrease in DTP coverage (–1.1%), in contrast to the national trend.

Table 12 shows immunisation coverage estimates for 'fully immunised' and for individual vaccines at six years of age for Australia by state or territory. 'Fully immunised' coverage at six years of age for Australia decreased marginally by 0.2 percentage points from the previous quarter to 83.5 per cent with a significant decrease in coverage in the Northern Territory (–2.4%). Coverage for all individual vaccines at six years of age remained largely unchanged, except for the Northern Territory [DTP (–1.7%), OPV (–1.9%) and measles, mumps, rubella vaccine (–1.7%)] and in Tasmania [OPV (–1.1%)]. Coverage for vaccines assessed at six years is now at or close to 85 per cent in most jurisdictions, although coverage in Western Australia and the Northern Territory for this age group remains well below other jurisdictions.

Figure 12 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 six years, although the rate of increase has slowed over the past year for all age groups.

Figure 12. Trends in vaccination coverage, Australia, 1997 to 2003, 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.

Table 10. Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2002; assessment date 31 March 2004

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	1,001	21,594	860	11,907	4,193	1,444	15,240	6,016	62,255
Diphtheria, tetanus, pertussis (%)	93.8	92.5	90.0	92.6	92.7	93.1	92.7	90.2	92.4
Poliomyelitis (%)	93.9	92.4	90.0	92.4	92.5	92.9	92.7	90.2	92.3
Haemophilus influenzae type b (%)	95.6	94.1	94.4	94.9	95.3	95.6	94.7	93.5	94.5
Hepatitis B (%)	95.9	94.8	95.1	95.2	95.3	95.3	94.4	93.1	94.7
Fully immunised (%)	93.1	91.0	89.1	91.5	91.8	91.8	91.5	89.0	91.1
Change in fully immunised since last quarter (%)	+4.8	+0.1	+3.5	+0.2	+0.1	+0.1	-0.3	-0.1	+0.1

Table 11. Percentage of children immunised at 2 years of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2001; assessment date 31 March 2004*

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	966	21,040	862	12,186	4,314	1,500	15,236	6,053	62,157
Diphtheria, tetanus, pertussis (%)	95.2	95.6	97.0	95.5	96.1	95.3	95.9	94.7	95.6
Poliomyelitis (%)	94.2	94.5	96.8	94.7	95.3	96.3	95.1	93.8	94.7
Haemophilus influenzae type b (%)	91.2	92.7	94.2	93.7	94.2	94.6	93.9	91.9	93.3
Measles, mumps, rubella (%)	90.9	92.7	95.6	93.9	94.2	95.0	94.1	92.2	93.4
Hepatitis B(%)	94.8	95.1	98.0	95.3	96.0	97.1	96.1	94.5	95.5
Fully immunised (%)†	88.4	90.8	93.7	92.0	92.7	92.1	92.4	90.3	91.5
Change in fully immunised since last quarter (%)	-0.6	-0.0	+0.1	-0.5	-0.1	-2.3	+0.3	+0.5	-0.1

* The 12 months age data for this cohort was published in *Commun Dis Intell* 2003;27:302.

† 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 12. Percentage of children immunised at 6 years of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 1997; assessment date 31 March 2004*

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,032	22,306	795	12,782	4,655	1,558	16,144	6,395	65,667
Diphtheria, tetanus, pertussis (%)	86.1	85.3	81.8	84.5	84.8	86.3	87.2	81.5	85.2
Poliomyelitis (%)	86.1	85.3	83.0	84.5	85.3	86.2	87.2	81.6	85.3
Measles, mumps, rubella (%)	85.5	84.2	82.8	84.1	84.6	85.2	87.2	81.2	84.7
Fully immunised (%)†	84.1	83.2	80.0	83.0	83.3	84.1	85.9	79.6	83.5
Change in fully immunised since last quarter (%)	-0.6	+0.2	-2.4	-0.6	-0.3	-0.9	-0.3	-0.6	-0.2

* 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.

Gonococcal surveillance

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

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various States and Territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5 per cent or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatment.[1] Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as TRNG. Tetracyclines are however, not a recommended therapy for gonorrhoea in Australia. Comparability of data is achieved by means of a standardised system of testing and a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. For more information see Commun Dis Intell *2004;28:100.*

Reporting period 1 January to 31 March 2004

The AGSP laboratories received a total of 993 isolates in this quarter of which 915 underwent susceptibility testing. The total received numbered slightly less than the 1,051 isolated or referred in 2003. About 32 per cent of this total was from New South Wales, 28 per cent from Victoria, 13.5 per cent from Queensland, 11.9 per cent from the Northern Territory and 8.9 per cent from Western Australia. About five per cent of all isolates were from South Australia, but susceptibility data are not yet available from that centre. Isolates from other centres were few. Where comparisons of data in this quarter were made with those from the same period in 2003, South Australian data were excluded from both sets of data.

Penicillins

Figure 13 shows the proportions of gonococci fully sensitive (MIC ≤ 0.03 mg/L), less sensitive (MIC 0.06 – 0.5 mg/L), relatively resistant (MIC ≥ 1 mg/L) or else penicillinase producing (PPNG) aggregated for Australia and by state or territory. A high proportion of those strains classified as PPNG or else resistant by chromosomal mechanisms fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

In this quarter 20.9 per cent of all isolates examined were penicillin resistant by one or more mechanisms—nine per cent PPNG and 11.9 per cent by chromosomal mechanisms (CMRNG). The number of PPNG increased to 82 from the 67 seen in the same period in 2003, as did the number of CMRNG to 109 from 99. The proportion of all strains resistant to the penicillins by any mechanism ranged from 2.7 per cent in the Northern Territory to 33 per cent in Victoria.

The highest proportion of PPNG was found in Western Australia where the 17 PPNG accounted for 20 per cent of all isolates. Thirty-four PPNG, representing 10.8 per cent of all isolates, were found in New South Wales, 20 (7.3%) in Victoria and eight (6%) in Queensland. Three PPNG were found in the Northern Territory. More isolates were resistant to the penicillins by separate chromosomal mechanisms and CMRNG were especially prominent in Victoria (72 isolates, 26% of all gonococci tested). Twenty-two CMRNG were present in New South Wales (7% of NSW isolates), seven in Queensland and eight in Western Australia. No CMRNG were detected in the Northern Territory.

Ceftriaxone

Two isolates with decreased susceptibility to ceftriaxone were detected in New South Wales.

Spectinomycin

All isolates were susceptible to this injectable agent.

Figure 13. Categorisation of gonococci isolated in Australia, 1 January to 31 March 2004, by penicillin susceptibility and region

FS Fully sensitive to penicillin, MIC ≤0.03 mg/L.

LS Less sensitive to penicillin, MIC 0.06–0.5 mg/L.

RR Relatively resistant to penicillin, MIC ≥1 mg/L.

PPNG Penicillinase producing *Neisseria gonorrhoeae.*

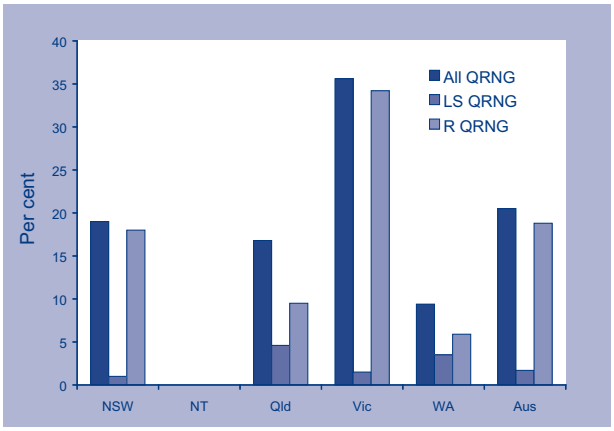
Quinolone antibiotics

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

The total number (188) and proportion (20.5%) of all QRNG were both substantially higher than the corresponding figures in the first quarter of 2003 (108 isolates,11.5%). The majority of QRNG (172 of 188, 91%) exhibited higher-level resistance.

QRNG were again widely distributed. The highest number (94) and proportion (36%) were found in Victoria. In New South Wales there were 60 QRNG (19% of isolates), in Queensland 16 (12%) and in Western Australia 8 (9)% (Figure 14). No QRNG detected in the Northern Territory.

Figure 14. The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 January to 31 March 2004, by jurisdiction



LS QRNG Ciprofloxacin MICs 0.06–0.5 mg/L.

R QRNG Ciprofloxacin MICs ≥1 mg/L.

High level tetracycline resistance

The number (107) and proportion (11.7%) of high level tetracycline resistance (TRNG) detected decreased somewhat from the 2003 figures. TRNG represented between 10 per cent (Victoria and Queensland) and 26 per cent of isolates Western Australia. Two TRNG were present in the Northern Territory.

Reference

1. Management of sexually transmitted diseases. World Health Organization 1997; Document WHO/GPA/TEM94.1 Rev.1 p 37.

Meningococcal surveillance

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

The reference laboratories of the Australian Meningococcal Surveillance Programme report data on the number of laboratory confirmed cases confirmed either by culture or by non-culture based techniques. Culture positive cases, where a Neisseria meningitidis is grown from a normally sterile site or skin, and non-culture based diagnoses, derived from results of nucleic acid amplification assays and serological techniques, are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions. Data contained in the quarterly reports are restricted to a description of the number of cases per jurisdiction, and serogroup, where known. A full analysis of laboratory confirmed cases of IMD is contained in the annual reports of the Programme, published in Communicable Diseases Intelligence.

Laboratory confirmed cases of invasive meningococcal disease for the period 1 January to 31 March 2004, are included in this issue of Communicable Diseases Intelligence *(Table 6).*

Table 6. Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 January to 31 March 2004, by jurisdiction and serogroup

Jurisdiction	Serogroup					
	B	C	Y	W135	ND	All
Australian Capital Territory	0 (1)	2				2 (1)
New South Wales	19 (11)	5 (7)	1 (2)	1	5 (4)	31 (24)
Northern Territory	3 (1)					3 (1)
Queensland	12 (9)	7 (7)			2 (2)	21 (18)
South Australia	4 (4)	0 (1)				4 (5)
Tasmania	2	0 (1)			2 (1)	4 (2)
Victoria	9 (5)	4 (13)	2		1 (3)	16 (21)
Western Australia	4 (6)	1 (2)				5 (8)
Australia	53 (37)	19 (31)	3 (2)	1 (0)	10 (10)	86 (80)

ND Not diagnosed.

Numbers in parentheses are the laboratory confirmed diagnoses of invasive meningococcal disease made in the same period in 2003.

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 2002, over 1,000 clinicians participated in the surveillance of 14 conditions through the APSU, with an overall response rate of 96 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 *2004;28:101.*

The results for the period 1 January to 31 December 2003 are shown in Table 7.

Table 7. Confirmed cases of communicable diseases reported to the Australian Paediatric Surveillance Unit between 1 January and 31 December 2003*

Condition	Previous reporting period Jan–Dec 2002	Current reporting period Jan–Dec 2003*
Acute flaccid paralysis	30	15
Congenital cytomegalovirus		
confirmed (< 3 weeks of age)	9	6
suspected (3–52 weeks of age)	8	3
Congenital rubella	3†	3
Perinatal exposure to HIV	25	14
HIV infection		1‡
Neonatal herpes simplex virus infection	11	8
Hepatitis C virus infection	commenced 2003	11

* Surveillance data are provisional and subject to revision

† Two imported cases i.e. children born to mothers who had rubella in Indonesia. One child was born in Indonesia, one child born in Australia. The third infant was born in Victoria in 2001, but was not notified to the APSU until 2002. The parents were Fijian, it is not known where the mother acquired her infection.

‡ HIV infection through heterosexual contact

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 *2004;28:99.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 October to 31 December 2003, as reported to 31 March 2004, are included in this issue of Communicable Diseases Intelligence *(Tables 8 and 9).*

Table 8. New diagnoses of HIV infection, new diagnoses of AIDS, and deaths following AIDS occurring in the period 1 October to 31 December 2003, by sex and state or territory of diagnoses

	Sex	State or territory								Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2003	This period 2002	Year to date 2003	Year to date 2002
HIV diagnoses	Female	0	8	0	5	2	0	4	1	20	26	84	89
	Male	0	79	1	25	12	0	47	7	171	213	758	744
	Sex not reported	0	4	0	0	0	0	0	0	4	0	1	5
	Total[1]	0	91	1	30	14	0	51	8	195	240	853	839
AIDS diagnoses	Female	0	0	0	0	0	0	1	0	1	3	9	16
	Male	0	23	2	4	1	0	13	1	44	45	175	195
	Total[1]	0	23	2	4	1	0	14	1	45	48	185	212
AIDS deaths	Female	0	0	0	0	0	0	1	0	1	3	8	7
	Male	1	11	0	3	1	0	3	1	20	22	71	75
	Total	1	11	0	3	1	3	4	1	21	25	79	82

1. Totals include people whose sex was reported as transgender.

Table 9. Cumulative diagnoses of HIV infection, AIDS, and deaths following AIDS since the introduction of HIV antibody testing to 31 December 2003 and reported by 31 March 2004, by sex and state or territory

	Sex	State or territory								
HIV diagnoses	Female	29	723	15	209	80	7	291	159	1,513
	Male	243	12,335	120	2,363	796	85	4,645	1,055	21,642
	Not reported	0	240	0	0	0	0	22	0	262
	Total[1]	272	13,325	135	2,580	876	92	4,976	1,221	23,477
AIDS diagnoses	Female	10	216	1	56	30	4	89	34	440
	Male	93	5,024	41	941	380	47	1,817	403	8,746
	Total[1]	103	5,255	42	999	410	51	1,916	439	9,215
AIDS deaths	Female	6	127	0	38	20	2	58	22	273
	Male	72	3,429	26	617	256	31	1,343	276	6,050
	Total[1]	78	3,565	26	657	276	33	1,409	299	6,343

1. Totals include people whose sex was reported as transgender.

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 *NEPSS 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.*

Interpret historical quarterly mean counts cautiously – these may be affected by outbreaks and surveillance artefacts such as newly recognised 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, facsimile: +61 3 9625 2689.

Reports to the National Enteric Pathogens Surveillance System of Salmonella infection for the period 1 January to 31 March 2004 are included in Tables 13 and 14. Data include cases reported and entered by 28 April 2004. 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 *2004;28:101–102.*

1 January to 31 March 2004

The total number of reports to NEPSS of human *Salmonella* infection increased to 2,703 in the first quarter of 2004, more than double the count in the fourth quarter of 2003, and 10 per cent more than in the comparable first quarter of 2003. The incidence of human salmonellosis typically peaks in the first few months of each year. Case counts to 28 April 2004 are approximately 95 per cent of the expected final counts for the quarter.

During the first quarter of 2004, the 25 most common *Salmonella* types in Australia accounted for 1,857 cases, 69 per cent of all reported human *Salmonella* infections.

Twenty-one of the 25 most common *Salmonella* infections in the fourth quarter of 2003 were amongst the 25 most commonly reported in the previous quarter.

Among the most common salmonellae in the nation, there were typical widespread seasonal increases in counts of *S*. Typhimurium phage type 135 and *S*. Typhimurium phage type 9.

S. Typhimurium phage type 170 (and the similar *S*. Typhimurium phage type 108) were uncommon before emerging as a common human pathogen during 2000 and 2001, and are now among the most common salmonellae in New South Wales, Victoria and South Australia.

Reports of *S*. Typhimurium phage type 197 and *S*. Typhimurium phage type U290 increased markedly during 2002, particularly in the south-eastern states, and remain elevated.

Reports of other common salmonellae with counts exceeding historical averages include *S*. Typhimurium phage type 12 (particularly in New South Wales), and *S*. Virchow phage type 8 and *S*. Hvittingfoss (both in Queensland).

Acknowledgement

We thank scientists, diagnostic and reference laboratories, State and Territory health departments, and the Australian Government Department of Health and Ageing for their contributions to NEPSS.

Table 13. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 January to 31 March 2004, as reported to 28 April 2004

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total all *Salmonella* for quarter	28	796	110	1,088	128	47	319	187	2,703
Total contributing *Salmonella* types	17	121	41	114	47	13	84	69	225

Table 14. Top 25 *Salmonella* types identified in Australia, 1 January to 31 March 2004, by state or territory

National rank	*Salmonella* type	State or territory								Total 1st quarter 2004	Last 10 years mean 1st quarter	Year to date 2004	Year to date 2003
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
1	*S.* Typhimurium 135	4	80	2	59	5	0	18	32	200	229	200	407
2	*S.* Typhimurium 170	5	117	2	16	0	1	55	0	196	57	196	205
3	*S.* Typhimurium 9	3	48	0	30	7	1	53	2	144	177	144	128
4	*S.* Typhimurium 12	1	113	0	19	3	0	3	1	140	13	140	42
5	*S.* Virchow 8	0	12	1	109	0	2	15	0	139	71	139	69
6	*S.* Saintpaul	0	9	10	90	5	0	5	10	129	122	129	124
7	*S.* Birkenhead	1	19	0	85	0	0	1	0	106	92	106	81
8	*S.* Typhimurium 197	1	14	0	58	0	0	13	0	86	9	86	79
9	*S.* Chester	1	14	5	41	4	0	5	8	78	63	78	113
10	*S.* Hvittingfoss	0	8	0	53	1	0	1	3	66	25	66	34
11	*S.* Waycross	0	12	0	49	0	0	0	0	61	40	61	30
12	*S.* Typhimurium U290	1	29	0	5	1	1	13	3	53	7	53	36
13	*S.* Infantis	3	24	2	5	3	0	11	3	51	48	51	71
14	*S.* Anatum	0	7	3	26	2	0	2	5	45	31	45	33
15	*S.* Muenchen	0	4	8	19	0	1	1	9	42	58	42	61
16	*S.* Mississippi	0	1	1	2	1	34	3	0	42	35	42	40
17	*S.* Singapore	1	12	1	15	9	0	0	1	39	20	39	29
18	*S.* Typhimurium 108	0	10	5	0	24	0	0	0	39	6	39	16
19	*S.* Aberdeen	0	0	0	34	0	0	0	0	34	38	34	35
20	*S.* Typhimurium 126	0	6	0	7	1	0	12	5	31	31	31	24
21	*S.* Agona	0	9	1	14	0	0	4	3	31	19	31	20
22	*S.* Typhimurium 4	0	24	0	1	0	0	4	0	29	20	29	25
23	*S.* Potsdam	0	5	1	14	1	1	3	1	26	21	26	22
24	*S.* Typhimurium RDNC	0	6	0	11	4	1	1	2	25	36	25	18
25	*S.* Havana	0	5	6	0	6	0	1	7	25	18	25	21

Overseas briefs

ProMED-mail

This material has been summarised from information provided by ProMED-mail (http://www.promedmail.org). A link to this site can be found under 'Other Australian and international communicable diseases sites' on the Communicable Diseases Australia homepage.

Avian influenza, human — East Asia

Viet Nam

Source: BBC News online, 20 March 2004 (edited)

A 12-year-old boy has died from avian influenza virus infection in Viet Nam, taking this year's death toll in East Asia to 24. Viet Nam has been the worst-hit country, with 16 deaths now reported, whereas eight people have died in Thailand. It is not clear whether this child was a previous laboratory-confirmed case of avian influenza A (H5N1) virus infection or is a new case only diagnosed at death. Prior to this report, the number of confirmed cases was 34; 12 in Thailand and 22 in Viet Nam.

Thailand

Source: World Health Organisation (WHO), CSR, Disease Outbreak News, 17 March 2004 (edited)

The Ministry of Public Health in Thailand has confirmed another case of human infection with avian influenza A (H5N1) virus. The case, which was fatal, was a 39-year-old woman from Ayudhaya Province. She developed symptoms on 1 March 2004, was hospitalised on 3 March, and died on 12 March 2004. To date, Thailand has reported 12 confirmed cases, eight of them fatal.

Avian influenza A (H7) virus, human — Canada

Source: World Health Organization (WHO), CSR, Disease Outbreaks News, 5 April 2004 (edited)

The first human case of avian influenza A (H7) virus infection in British Columbia arose in a person who was involved in the culling of infected birds on 13–14 March 2004. On 13 March 2004, he may have been accidentally exposed in the eye. On 16 March, he reported conjunctivitis and nasal discharge. Treatment with oseltamivir — an antiviral drug active against influenza A viruses — began on 18 March. On 30 March, Health Canada concluded that this case was caused by avian influenza A (H7) virus and informed the World Health Organization (WHO) on 31 March. The patient has recovered fully.

On 2 April 2004, WHO was informed by Health Canada of a second poultry worker in British Columbia infected with avian influenza A (H7) virus infection. This worker developed conjunctivitis on 25 March after close contact with infected birds. He was treated with oseltamivir and his symptoms resolved.

Based on this epidemiological information provided by Health Canada, WHO today raised the global pandemic preparedness level from 0.1 to 0.2 for the Canadian outbreak. Global pandemic preparedness levels are dictated by the epidemiological situation for each local event. Level 0.2 means that more than one human case caused by a new subtype of influenza virus has been identified in the local event.

Preparedness levels have been organised into a matrix established by WHO in 1999 (WHO Influenza Pandemic Preparedness Plan). When a preparedness level is raised to 0.2, affected countries are advised to step up their surveillance in people exposed to affected poultry, to organise special investigations to better understand the new virus, to advise people at risk to wear protected clothing, and to consider the use of antivirals and normal human influenza vaccine.

Severe acute respiratory syndrome — world-wide

Source: Centers for Disease Control, Traveller's' Health, 10 February 2004 (edited)

The Centers for Disease Control and Prevention (CDC) have been working closely with the World Health Organization (WHO) and other international partners to track cases of severe acute respiratory syndrome (SARS). Since 16 December 2003, the Chinese Ministry of Health has reported four cases of SARS (three confirmed and one probable). All four cases are from Guangdong, the same province where the first case of SARS in 2003 was confirmed in a 32-year-old man and where the first cases of SARS were identified in November 2002. All four patients have recovered from their illness and have been discharged from the hospital. None of their contacts have developed SARS-like illness. The source of infection of the four patients is not known; however, SARS coronavirus (SARS-CoV), the virus that causes SARS, has been collected from cages that housed civet cats in the restaurant where one of the patients worked. The Chinese Ministry of Health continues to work in close collaboration with WHO to broaden the investigation and enhance surveillance throughout China.

BSE update 2004

Source: BSE in Europe, updated 29 March 2004 (edited)

During 2003, decreased incidence of recorded BSE cases, compared to 2002, was seen in most countries. The exceptions were Portugal, Spain, Japan, Poland, and the Czech Republic.

The number of BSE cases in Germany seems to be increasing in 2004 after a steep decline during 2003. In the first quarter of 2004, Germany counted 14 cases; in the same period of 2003 the number of confirmed cases was only seven. Since the previous update (23 February 2004), one new case each was observed in Japan and Slovenia. Poland has observed two new cases: according to an AFP report of 27 March 2004, a 13th BSE case was discovered there 'during a routine test at a slaughterhouse'. This new case is not included in the above table.

For additional information, the reader is referred to the Office International des Epizooties table, last updated on 24 March 2004, available from: http://www.oie.int/eng/info/en_esbmonde.htm.

CJD (new var.), blood supply — United Kingdom

Source: Eurosurveillance Weekly, 2004;8, 18 March 2004 (edited)

On 16 March 2004, the Department of Health in England announced that people who have received a blood transfusion in the United Kingdom (UK) since 1 January 1980 will no longer be able to donate blood. This additional donor selection criterion will be implemented by all four of the UK Blood Services (UKBS), including the National Blood Service, on 5 April 2004.

BSE confirmed cases update, 23 February 2004

Country	2001	2002	2003	2004	Total since 1987
UK	1,175	1,104	611	57	182,528
Austria	1	0	0	0	1
Belgium	46	38	15	6	124
Canada	0	0	1	0	1
Czech Republic	2	2	4	1	9
Denmark	6	3	2	0	13
Finland	1	0	0	0	1
France	274	239	137	20	906
Germany	125	106	54	14	312
Greece	1	0	0	0	1
Ireland	246	333	182	47	1,391
Israel	0	1	0	0	1
Italy	50	36	31	1	120
Japan	3	2	4	2 (1)	11
Liechtenstein	0	0	0	0	2
Luxembourg	0	1	0	0	2
Netherlands	20	24	19	4	75
Portugal	110	86	133	17	879
Poland	0	4	5	3	12
Slovakia	5	6	2	1	14
Slovenia	1	1	1	1	4
Spain	82	127	167	26	421
Switzerland	42	24	21	0	453
United States	0	0	1*	0	1

* The data from some countries may include exceptional imported cases or exclude exported cases which were found positive in the countries of destination.

This additional precautionary measure to safeguard the blood supply is being taken in the light of the first possible transmission of variant Creutzfeldt-Jakob disease by blood transfusion, which was reported in December 2003. The transfusion occurred in 1996; the blood donor was well at the time but developed symptoms of vCJD in 1999 and died the following year. The recipient was diagnosed with vCJD in 2003.

This is a highly precautionary approach, and the benefit of receiving a blood transfusion when needed far outweighs any possible risk of contracting vCJD. To date there has been only one possible case of vCJD being transmitted by blood, yet the UKBS issue over 2.5 million units of blood every year.

As of 1 March 2004 there have been 146 definite and probable cases of vCJD in the UK, one case each in the Republic of Ireland, Italy, the United States of America, Canada, and Hong Kong, and six cases in France. The eventual number of individuals within the UK population likely to develop vCJD remains uncertain; estimates range from the current numbers up to 540. It is not known how many current or past blood or tissue donors may develop vCJD in the future.

Hepatitis C virus, blood supply — South Korea

Source: Reuters Health online, 29 March 2004 (edited)

South Korea's Red Cross mishandled donor information and circulated blood donated by hepatitis virus carriers, infecting nine people, government auditors said. The Board of Audit and Inspection (BAI) called on the Korea National Red Cross to punish officials responsible for shipping blood donated by hepatitis virus carriers to hospitals and pharmaceutical companies for five years up until January 2004.

A BAI audit conducted at the end of 2003 found that 76,677 units of blood received from donors who had been infected with hepatitis C virus had been distributed for transfusions or research by the Red Cross, a board official said. The South Korean branch of the international agency also put into circulation 228 units of blood donated by 99 people who had been suspected of carrying human immunodeficiency virus, but who later tested negative for the virus, the official said. Nine people were found to have been infected with hepatitis during February 2004 after receiving blood transfusions from the Red Cross.

Cholera update

Mali

Source: Afrol News, Africa, 5 March 2004 (edited)

The cholera epidemic along River Niger in Mali is increasing, according to local media. More than 2,000 cholera cases have been reported and some 117 Malians have died. Cholera started spreading in August 2003 in Mali. It was associated with the release of sewage into River Niger from the outskirts of Bamako, the Malian capital. In the following months, the water-borne disease was reported in several cities and towns several hundreds of kilometres down-river. According to the World Health Organization, there is a total number of 13 clusters affected by the current cholera outbreak. The widespread nature of the epidemic makes it difficult to control.

Togo

Source: Afrol News, Africa, 1 March 2004 (edited)

A cholera epidemic in the Togolese capital, Lome, has so far caused 661 cases and 37 deaths, according to the Togolese Red Cross. While the epidemic is still on the increase, the Red Cross is heading information and awareness work to prevent a greater disaster in the city of 700,000 inhabitants. There is fear of the epidemic escalating as the rains are increasing.

Together with local health authorities and the WHO, Red Cross volunteers are now conducting a mass awareness campaign through radio and public institutions such as schools. Other activities to control the spread of the epidemic include active case surveillance and referring infected persons to special clinics established to treat cholera patients.

Mozambique

Source: WHO Disease Outbreak, 23 March 2004 (edited)

The Ministry of Health of Mozambique has reported a total of 15,237 cases and 85 deaths between 20 December 2003 and 18 March 2004 in seven provinces. Maputo city is the worst-affected area with 9,522 cases and 37 deaths. The Ministry of Health declared a cholera emergency on 9 January 2004. WHO and other agencies, including UNICEF, Médecins sans Frontières, World Food Programme, the United States Agency for International Development, and the Italian Government are supporting local health authorities in containing the outbreak.

South Africa (Eastern Cape)

Source: SABC News, 24 March 2004 (edited)

Thirteen people in the Eastern Cape have died from cholera and more than 100 had to be treated in hospital for the disease after a recent outbreak. The provincial government has promised the affected communities emergency medical resources to stop the spread of the disease. The disease has claimed many lives over the past three years in rural Transkei, due to a lack of clean water and sanitation.

Zambia

Source: AllAfrica.com, 27 March 2004 (edited)

Cholera has broken out in Lusaka at State Lodge, the police camp housing staff and families of presidential security staff at the presidential lodge, with four people currently undergoing treatment at various cholera centres. The cause of the outbreak was due to a sewer pipe that burst and contaminated the drinking water in the police camp.

Anthrax, cattle — Australia (Victoria)

From: Chief Veterinary Officer, Department of Primary Industries Victoria 16 March 2004 (edited)

Anthrax has been confirmed as the cause of death of seven cattle that died over a four-day period (10–14 March 2004) on a farm (Farm 1) in the Goulburn Valley. A single case occurred on an adjacent farm (Farm 2) during the same period. There have been earthworks between both farms associated with water drainage channels that may account for the incident. The incident has occurred in an area where anthrax has occurred previously and where occasional cases are not unexpected. Farm 1 has never recorded a case; Farm 2 had a case back in 1997.

The following response actions have been taken in accordance with Victorian and national protocols for anthrax control (starting immediately from the time anthrax was suspected):

(i) Quarantine has been applied to both farms; the quarantine will remain in place until 42 days after vaccination or 21 days after the last case, whichever is the later date.

(ii) All carcasses have been incinerated on-site.

(iii) All cattle on both farms were vaccinated on 11 March 2004, and

(iv) local agencies and key industry contacts have been advised of the situation.

No cattle have been moved from either property during the past 20 days, so there is no need to trace cattle movements and no implications for meat safety. Both properties are outblocks used to hold young heifers prior to first calving. These were not milking cattle; there are no implications for milk or dairy products. Action taken thus far means there are no public health implications or issues in relation to the safety of livestock, meat, or dairy products from the area.

Smallpox vaccination, secondary/ tertiary transfer

Source: MMWR Morb Mortal Wkly Rep 2004;53:103–105, 13 February (edited)

In December 2002, the Department of Defense (DoD) began vaccinating military personnel as part of the pre-event vaccination program.[1] Because vaccinia virus is present on the skin at the site of vaccination, it can spread to other parts of the body (autoinoculation) or to contacts of vaccinees (contact transfer).

To prevent autoinoculation and contact transfer, DoD gave vaccinees printed information that focused on handwashing, covering the vaccination site, and limiting contact with infants.[1,2] This report describes cases of contact transfer of vaccinia virus among vaccinated military personnel since December 2002; findings indicate that contact transfer of vaccinia virus is rare. Continued efforts are needed to educate vaccinees about the importance of proper vaccination-site care in preventing contact transmission, especially in household settings.

DoD conducts surveillance for vaccine-associated adverse events by using automated immunisation registries, military communication channels, and the Vaccine Adverse Events Reporting System. Contact transfer cases are defined as those in which vaccinia virus is confirmed by viral culture or polymerase chain reaction (PCR) assays. Other cases are classified as suspected on the basis of lesion description and reported linkage to a vaccinated person three to nine days before lesion development.

During the period December 2002 to January 2004, a total of 578,286 military personnel were vaccinated; 508,546 (88%) were male, and 407,923 (71%) were primary vaccinees (received smallpox vaccination for the first time). The median age of vaccinees was 29 years (range: 17 to 76). Among vaccinees, cases of suspected contact transfer of vaccinia were identified among 30 persons: 12 spouses, eight adult intimate contacts, eight adult friends, and two children in the same household. The sources of suspected contact transfer were all male service members who were primary vaccinees. Except for six male sports partners, all infected contacts were female.

Vaccinia virus was confirmed in 18 (60%) of the 30 cases by viral culture or PCR. Sixteen of the 18 confirmed cases involved uncomplicated infections of the skin; two involved the eye.[3] None resulted in eczema vaccinatum or progressive vaccinia. Twelve of the 18 confirmed cases were among spouses or adult intimate contacts. The observed rate of contact transfer was 5.2 per 100,000 vaccinees overall or 7.4 per 100,000 primary vaccinees.

Among 27,700 smallpox-vaccinated DoD health-care workers, no transmission of vaccinia from a vaccinated health-care worker to an unvaccinated patient or from a vaccinated patient to an unvaccinated health-care worker has been identified.

Two of the 18 confirmed cases of transfer of vaccinia virus resulted from tertiary transfer. One involved a service member, his wife, and their breast-fed infant; the other involved serial transmission among male sports partners.

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Dengue/DHF update 2004

Indonesia

Source: The Jakarta Post, Samarinda, East Kalimantan, 24 March 2004 (edited)

The East Kalimantan provincial government imposed a state of emergency in the province, as the number of people with dengue fever had reached an alarming level. As of 19 March 2004, a total of 1,028 people in the province had been hospitalised with dengue fever since January; 18 of them have died. In the same period in 2003, 1,951 people contracted dengue fever, 31 of them died.

Venezuela

Source: Ministry of Health, Venezuela, 23 March 2004 (edited)

In the first nine weeks of 2004 there were 8,848 cases of dengue, with a peak of 1,211 cases in the third epidemiological week 18–25 January 2004. There was a total of 634 dengue haemorrhagic fever cases reported (7% of total) and three deaths. The population under 15 years represented 50 per cent of total cases. The most affected age group was 15–24 years with 23.11 per cent of cases. The accumulated morbidity rate was 32.2 per 100,000 population. Circulating serotypes were DEN–1, DEN–2, DEN–3 and DEN–4.

El Salvador

Source: Diario El Mundo, El Salvador, 17 March 2004 (edited)

El Salvador's Ministry of Health reported laboratory confirmation of 37 dengue fever cases. This represents a considerable increase compared with the two previous years.

In 2004, 455 cases of dengue have been reported (in 2003 for the same period there were only 269 cases). There are now more cases than in 2002, a year which was considered as epidemic for dengue fever in El Salvador. Dengue haemorrhagic fever case numbers are still low. This year only 19 cases have been reported, compared with 24 in 2003. The current national larval index is 5.9 per cent, although there are places with up to 18 per cent.

The incidence rate is high in comparison with the last epidemic year (2002). Current incidence rate is 7.1 per 100 000 population. In 2002 this figure was 5.6 per 100,000 population.

Viet Nam

Source: AFP, 18 March 2004 (edited)

Health authorities in Vietnam expressed concern over the growing number of people infected with dengue fever, the mosquito-borne disease that has killed seven people this year in the country. The Hanoi-based Institute of Hygiene and Epidemiology reported that 4,199 people had been infected since the beginning of 2004, a 90 per cent increase compared to the same period of 2003. The worst-affected areas were in the southern Mekong Delta region, where moist and humid conditions provide ideal breeding conditions for mosquitoes. The region accounted for over 90 per cent of the cases and all the deaths.