Australian Government
Department of Health and Ageing

# Communicable Diseases Intelligence

Volume 32 Number 1 Quarterly report March 2008

## Annual reports



## Articles



## Short reports



*Continued on back cover*

Communicable Diseases Intelligence

Volume 32 Issue 1 March 2008

Table of contents, *continued*

## Quarterly reports



## Administrative

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* 2008;32:xx–xx.

*Communicable Diseases Intelligence* contributes to the work of the Communicable Diseases Network Australia (http://www.health.gov.au/cdna)



## Website

http://www.health.gov.au/cda

## Subscriptions and contacts

*Communicable Diseases Intelligence* is produced every quarter by:
Surveillance Branch
Office of Health Protection
Australian Government Department of Health and Ageing
GPO Box 9848, (MDP 6)
CANBERRA ACT 2601;
Telephone: +61 2 6289 2717
Facsimile: +61 2 6289 2600
Email: cdi.editor@health.gov.au

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

## Disclaimer

Opinions expressed in *Communicable Diseases Intelligence* are those of the authors and not necessarily those of the Australian Government Department of Health and Ageing or the Communicable Diseases Network Australia. Data may be subject to revision.

Printed by Union Offset, Canberra

Annual reports

# Tuberculosis notifications in Australia, 2006

Paul W Roche, Vicki Krause, Anastasios Konstantinos, Ivan Bastian with input from, Ral Antic, Lynne Brown, Amanda Christensen, Sandra Gebbie, Mark Hurwitz, Avner Misrachi and Justin Waring as members of the National Tuberculosis Advisory Committee, for the Communicable Disease Network Australia

## Abstract

The National Notifiable Disease Surveillance System received 1,201 tuberculosis (TB) notifications in 2006, of which 1,142 were new cases and 59 were relapses. The incidence of TB in Australia was 5.8 cases per 100,000 population in 2006 up from 5.3 per 100,000 in 2005, but still below 6 per 100,000 as it has been since 1985. Eighty-five per cent of TB notifications in 2006 were in people born outside Australia. The incidence in people born overseas and Indigenous Australians were 20.7 and 6.6 cases per 100,000 population, respectively. By contrast, the incidence of TB in the non-Indigenous Australian-born population was 0.9 cases per 100,000 population. Household or other close contact was reported as the most common risk factor for TB infection. The number of cases of TB reported in health care workers increased in 2006; these were mostly in health care workers born in TB–endemic countries and there were no reports of TB transmission in Australian health care settings. Outcome data of the 2005 TB cohort indicates that treatment success was attained in more than 95% of cases. Progress towards TB elimination in Australia will rely on continued TB awareness, maintenance of high standards of TB diagnostic and control practices, and promoting regional and global TB control activities. *Commun Dis Intell* 2008;32:1–11.

Keywords: disease surveillance, tuberculosis

## Introduction

In 2007, the World Health Organization (WHO) reported that although tuberculosis (TB) is still a major cause of death worldwide, the global epidemic 'is on the threshold of decline'.[1] In 2005, 8.8 million TB cases and 1.6 million deaths attributable to TB were reported worldwide. However, WHO data show that the TB incidence rates were stable or declining in all regions, even while the total numbers of cases continue to increase slowly due to increased case load in Africa, the Eastern Mediterranean and South East Asia. These findings if confirmed, suggest that the Millennium Development Goals Target 8: 'to have halted by 2015 and begun to reverse the incidence of major diseases' may have been attained. WHO TB case detection targets (70%) and successful treatment targets (85%) have been met in the WHO Western Pacific Region, which includes Australia and in the neighbouring South East Asian region. This is important for TB control in Australia, since a large proportion of TB cases diagnosed in Australia are people born in these regions.

Approximately 1.7% of all TB cases worldwide have multi-drug-resistant (MDR) disease (i.e. *Mycobacterium tuberculosis* resistant to at least isoniazid and rifampicin).[2] In 2006, extensively drug-resistant tuberculosis (XDR-TB; defined as MDR-TB plus resistance to any fluoroquinolone and at least one injectable agent: kanamycin, amikacin or capreomycin) emerged. The US Centers for Disease Control and Prevention (CDC) and the WHO reported results of an international survey that found 2% of more than 17,000 isolates collected between 2000 and 2004 were XDR-TB, with cases occurring in 17 countries and the prevalence increasing over the study period.[3] In the KwaZulu-Natal region of South Africa, between 2005 and 2006, 53 cases of XDR-TB were detected, of whom 55% had not been previous treated; 44 cases were HIV-infected and 52 died.[4] The spread of XDR-TB globally has been fuelled by the HIV epidemic, inadequate public health systems, limited access to high-quality laboratory resources, and a neglect of infection control measures.[5]

The South African experience provides lessons for TB control efforts in Australia. Some of these themes will be addressed in this report. For example, while HIV-TB co-infection has not been a major problem in Australia, determining the HIV status of all Australian TB patients has not been achieved.[6] Notifications of TB in Australian health care workers (HCWs) recruited from countries with a high TB incidence continue to be monitored. Data on MDR-TB in patients from Papua New Guinea accessing Queensland TB services in the Torres Strait are also provided as an example of how TB control in the region can impact public health control of TB in Australia.

## Methods

### Data collection

TB is a notifiable disease in Australia. Medical practitioners, public health laboratories and other health professionals are legally required to report cases of TB to the state and territory health authorities. Information on notified cases for 2006 was collated by jurisdictions and sent electronically to the National Notifiable Diseases Surveillance System (NNDSS) managed by the Australian Government Department of Health and Ageing. Records were dispatched in a de-identified format to ensure confidentiality. The National Tuberculosis Advisory Committee (NTAC), as a sub-committee of the Communicable Diseases Network Australia, was responsible for determining the dataset collected in 2006 and for data reporting to NNDSS. Key data fields in the TB dataset that are analysed in this report are listed in Table 1, with a brief description of each variable. TB drug susceptibility data on bacteriologically confirmed cases is collected, analysed and reported by the Australian Mycobacterial Reference Laboratory Network in an accompanying report.[7]

### Data processing and quality control

Data on all TB notifications reported in 2006 were received by September 2007. Updated information on the outcomes of treatment of patients notified in 2005 was received by December 2007. Data received from the jurisdictions were examined for completeness and accuracy. Any invalid or missing entries were returned to the jurisdictions for review and correction.

Almost all cases of TB in Australia are reported to the surveillance system. Reasons for the high level of reporting include the presence of effective TB screening programs, a high standard of health care, and specialised and multi-disciplinary TB services in each jurisdiction. The terms 'notification rate' and 'incidence' are therefore used interchangeably in this report.

Additional data were provided from the Queensland Tuberculosis Control centre related to the incidence of tuberculosis among Papua New Guinea (PNG) citizens in the Torres Strait Treaty Zone within which Indigenous people on both sides of the border are allowed free movement for traditional practices. The data for 2007 are incomplete, as full

**Table 1. Description of key data fields in the enhanced tuberculosis data set of the National Notifiable Disease Surveillance System used in this report***

| Data field | Description |
|---|---|
| TB outcomes | Options are:<br>• Cured (bacteriologically confirmed- pulmonary cases only)<br>• Completed treatment (80% of standard regimen completed)<br>• Interrupted treatment for less than two months (but still completed)<br>• Died of TB during treatment phase<br>• Died of other cause during treatment phase<br>• Defaulter (failed to complete treatment)<br>• Treatment failure (completed treatment but failed to be cured)<br>• Transferred out of Australia during treatment phase |
| Indigenous status | Whether notified case is Indigenous (Aboriginal and/or Torres Strait Islander) Australian by descent, community acceptance or self-identification |
| Selected risk factors | Options are:<br>• Household member or close contact with a TB patient<br>• Currently/recently residing in a correctional facility within last five years<br>• Currently/recently residing in an aged care facility within last five years<br>• Currently/previously employed in an institution within last five years<br>• Currently/previously employed in the health industry within last five years<br>• HIV status (positive or negative)<br>• Past residence (3 months or more) in a high risk country (as defined by the Australian Government Department of Immigration and Citizenship) |

* Other data collected on each case included country of birth, length of residence in Australia (for overseas-born cases), site of tuberculosis disease.

case ascertainment for the year and completion of drug susceptibility testing had not been finalised at the time of retrieval of information.

### Case definitions

TB cases were classified as new or relapsed. A new case required a diagnosis accepted by the Director of TB Control (or equivalent) in the relevant jurisdiction, based on laboratory or clinical evidence, and in the absence of any previous treated or untreated TB diagnosis. Laboratory evidence includes either the isolation of *Mycobacterium tuberculosis* complex (*M. tuberculosis, M. bovis or M. africanum)* from a clinical specimen by culture; or nucleic acid amplification testing indicating *M. tuberculosis* complex, except where it is likely to be due to previously treated or inactive disease. Microscopy and culture remain mainstays of TB laboratory diagnosis and provide the capacity for assessing the level of risk for transmission and drug susceptibility testing. Clinical evidence is a diagnosis made by a clinician experienced in tuberculosis and includes clinical follow-up assessment, with or without supporting radiology.

A relapsed TB case was defined as a case of active TB diagnosed bacteriologically, radiologically or clinically, having been considered inactive or quiescent following previous treatment (as deemed by the state or territory Director of Tuberculosis). Relapses refer to re-treatment cases of which some may be reinfections rather than a true relapse of prior disease. Relapse cases are sub-divided into relapse after full or partial treatment, in Australia or overseas.

### National Performance Indicators

The performance criteria for the National Performance Indicators were set by NTAC in 2002 and reviewed in 2003. In TB annual reports before 2005, the performance criteria for incidence in people born overseas applied to people who have been living in Australia for more than five years. In this report the criteria have been applied to all cases regardless of length of residence.

### Population estimates for 2006

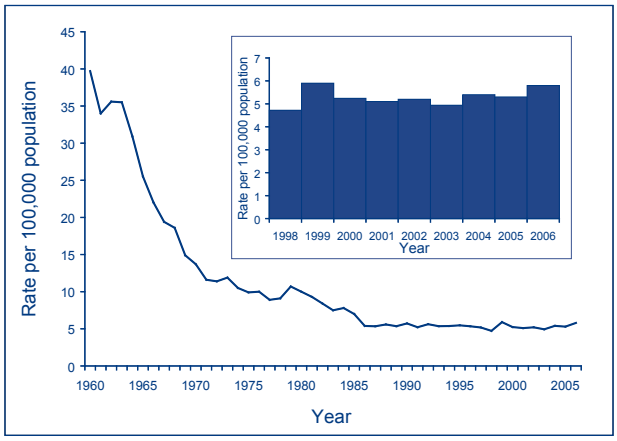
The rates presented in this report were calculated using population data produced by the Australian Bureau of Statistics. The estimated resident population as at 30 June 2006, in each state and territory and in Australia as a whole, was used as the denominator in crude rate calculations. Estimates of the Indigenous Australian population were based on projections from the 2001 census estimate of the Indigenous population in Australia. Data on Migration 2005-6 (ABS Catalogue No: 3412.0) were used to calculate incidence rates of TB in people born overseas.

## Results

### Tuberculosis notification rates

The total number of cases reported across Australia in 2006 was 1,201 (5.8 cases per 100,000 population). This was 129 (12%) cases more than that reported in 2005 (1,072 and 5.3 cases per 100,000 population, Figure 1). In 2006 there were 1,142 new cases and 59 relapses. Of the 59 relapsed cases, 12 relapsed after full treatment in Australia, two following partial treatment in Australia, 26 following full treatment overseas and 19 following partial treatment overseas.

**Figure 1. Incidence rates for tuberculosis notifications, Australia, 1960 to 2006**




### Tuberculosis notifications by state or territory

New South Wales reported the largest number of TB cases (464) however the highest rate was recorded in the Northern Territory (15.5 cases per 100,000 population, Table 2).

TB notification rates by jurisdiction are shown in Figure 2. In 2006, rates were the highest for five years in New South Wales, South Australia, and Western Australia. When compared with 2005 totals, increases were seen in Western Australia (91% increase, 54 additional cases), South Australia (56% increase, 26 additional cases), Queensland (27% increase, 30 additional cases), the Northern Territory (18% increase, 5 additional cases) and New South Wales (4.9% increase, 22 additional cases). The number of cases and rates in the other three jurisdictions in 2006 were lower than in 2005.

### Tuberculosis in non-Indigenous Australian-born population

Indigenous status was reported for all Australian-born patients. The incidence of TB in non-Indigenous Australians for 2006 was 0.9 cases per

100,000 population (140 cases), which was a small increase on the 122 cases (0.8 per 100,000) reported in 2005 (Figure 3 and Table 3).

### Tuberculosis in Indigenous Australians

The TB incidence rate in the Indigenous Australian-born population (6.6 cases per 100,000 population; 33 cases) was an increase of six cases

**Figure 2. Tuberculosis notification rates, Australia, 2002 to 2006, by state or territory**

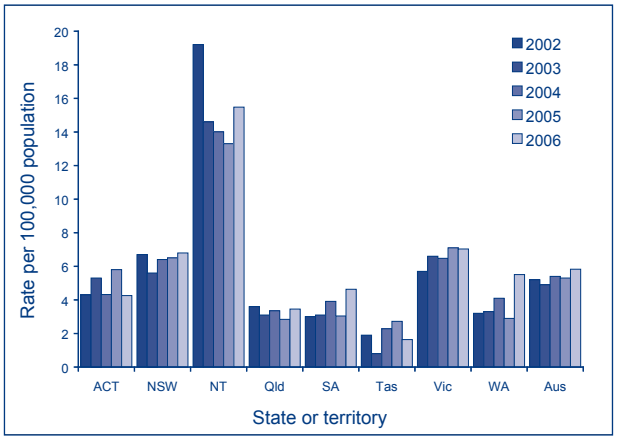



**Figure 3. Tuberculosis incidence rates, Australia, 1991 to 2006, by indigenous status and country of birth**

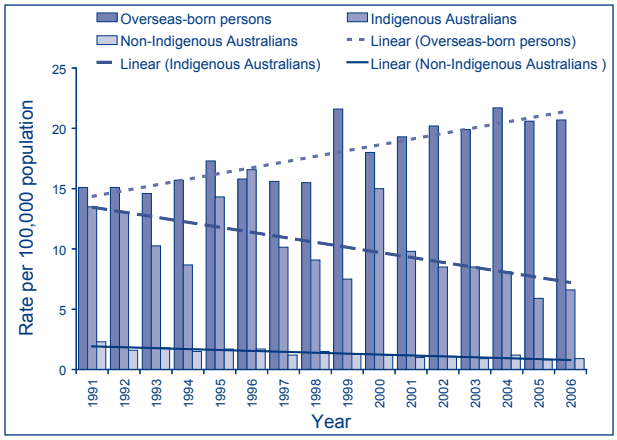



**Table 2. New and relapsed cases and rates per 100,000 population, Australia, 2006, by state or territory**

| | New cases | New cases rate | Relapse cases | Relapse case rate | Total notifications | Total rate |
|---|---|---|---|---|---|---|
| ACT | 14 | 4.3 | 0 | 0.0 | 14 | 4.3 |
| NSW | 437 | 6.4 | 27 | 0.4 | 464 | 6.8 |
| NT | 29 | 14.0 | 3 | 1.5 | 32 | 15.5 |
| Qld | 129 | 3.2 | 11 | 0.3 | 140 | 3.5 |
| SA | 67 | 4.3 | 5 | 0.3 | 72 | 4.6 |
| Tas | 8 | 1.6 | 0 | 0.0 | 8 | 1.6 |
| Vic | 349 | 6.9 | 9 | 0.2 | 358 | 7.0 |
| WA | 109 | 5.3 | 4 | 0.2 | 113 | 5.5 |
| Australia | 1,142 | 5.5 | 59 | 0.3 | 1,201 | 5.8 |

**Table 3. Tuberculosis notifications and incidence rates in all Australian-born, Australia, 2006, by state or territory**

| | Indigenous | Indigenous rate | Non-Indigenous Australian-born | Non-Indigenous rate | Total Australian-born | Total rate |
|---|---|---|---|---|---|---|
| ACT | 0 | 0.0 | 2 | 0.8 | 2 | 0.8 |
| NSW | 4 | 2.7 | 44 | 0.9 | 48 | 0.9 |
| NT | 9 | 14.7 | 2 | 2.1 | 11 | 7.1 |
| Qld | 13 | 9.3 | 18 | 0.6 | 31 | 1.0 |
| SA | 1 | 3.6 | 14 | 1.2 | 15 | 1.3 |
| Tas | 1 | 5.4 | 4 | 1.1 | 5 | 1.4 |
| Vic | 2 | 6.5 | 41 | 1.1 | 43 | 1.1 |
| WA | 3 | 4.2 | 15 | 1.0 | 18 | 1.2 |
| Australia | 33 | 6.6 | 140 | 0.9 | 173 | 1.1 |

over the 27 cases reported in 2005 (5.9 per 100,000). The crude TB incidence rate in Indigenous Australians in 2006 was seven times the rate in non-Indigenous Australian-born people.

### Tuberculosis notifications in the overseas-born population

In 2006, country of birth was reported for 1,200 of the 1,201 cases. One thousand and twenty-seven (85.5%) cases were overseas-born. The rate of notification, 20.7 cases per 100,000, was similar to rates in this population in the previous two years (20.6 and 21.7 per 100,000 population in 2005 and 2004 respectively, Figure 3). Rates of TB in the overseas born have shown an increase since 1991. Table 4 shows the cases and rates ranked by estimated rate in the Australian resident population for country of birth. While the highest rates were among those born in Somalia, Bangladesh and Ethiopia, these represent a relatively small number of cases in a small resident population. The largest numbers of TB cases were in those born in India, Vietnam, Indonesia, the Philippines and China as in previous years (Table 4).

Data on the year of arrival was available for 938 of the 1,027 overseas-born cases in 2006. Four hundred and sixteen (44%) of the 2006 cases presented within two years of arrival in Australia and 794 (85%) within 20 years of arrival (Figure 4).

The Australian immigration status was available on overseas-born cases of TB from all states and territories excluding New South Wales and the Australian Capital Territory. The majority of the 477 (59%) cases were permanent residents, 14% were refugees

**Table 4. Notification of tuberculosis and estimated rate per 100,000 population for selected countries of birth, Australia, 2006**

| Country of birth | New | Relapse | Total cases | Estimated resident population 2006* | Estimated Rate per 100,000 population in Australia 2006 | WHO incidence rate per 100,000 2005† |
|---|---|---|---|---|---|---|
| Somalia | 20 | 2 | 22 | 5,431 | 405 | 224 |
| Bangladesh | 20 | 2 | 22 | 13,751 | 160 | 227 |
| Ethiopia | 12 | 0 | 12 | 7,516 | 160 | 344 |
| Papua New Guinea | 34 | 3 | 37 | 26,302 | 141 | 250 |
| Sudan | 34 | 6 | 40 | 29,282 | 137 | 228 |
| Indonesia | 68 | 3 | 71 | 67,952 | 104 | 239 |
| India | 148 | 5 | 153 | 153,579 | 100 | 168 |
| Vietnam | 120 | 3 | 123 | 180,352 | 68 | 175 |
| Pakistan | 11 | 2 | 13 | 19,768 | 66 | 181 |
| Cambodia | 18 | 0 | 18 | 28,175 | 64 | 506 |
| Afghanistan | 11 | 1 | 12 | 21,140 | 57 | 168 |
| Zimbabwe | 11 | 0 | 11 | 21,142 | 52 | 601 |
| Philippines | 65 | 3 | 68 | 135,619 | 50 | 291 |
| Thailand | 14 | 1 | 15 | 32,747 | 46 | 142 |
| China‡ | 60 | 8 | 68 | 203,143 | 33 | 100 |
| Sri Lanka | 22 | 0 | 22 | 70,908 | 31 | 60 |
| South Korea | 12 | 2 | 14 | 49,141 | 28 | 96 |
| Egypt | 10 | 0 | 10 | 38,782 | 26 | 25 |
| Hong Kong | 14 | 2 | 16 | 76,303 | 21 | 75 |
| United Kingdom | 27 | 1 | 28 | 1,153,264 | 2 | 14 |
| Other overseas born | 242 | 10 | 252 | 2,622,566 | 10 | |
| Total overseas born | 973 | 54 | 1,027 | 4,956,863 | 20.7 | |
| Australia | 168 | 5 | 173 | 15,648,625 | 1.1 | |
| Total§ | 1,141 | 59 | 1,201 | 20,605,488 | 5.8 | |

* The estimated resident population (ERP) at June 2006 (ABS 3412) except for Somalia ERP 2005.

† Rates from the World Health Organization 2007 Global Tuberculosis Report.

‡ China excludes Hong Kong SAR and Taiwan.

§ Country of birth unknown in one case.

**Figure 4. Notifications of tuberculosis in the overseas-born population, Australia, 2006, by year of arrival in Australia**

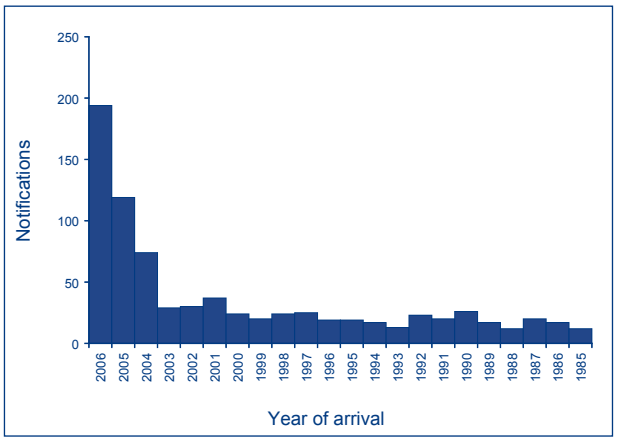
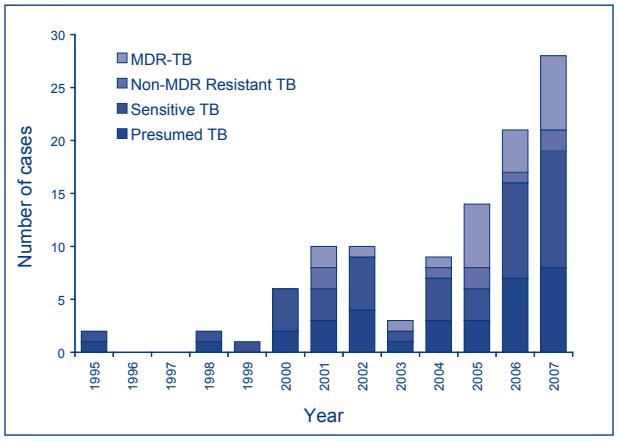



**Figure 5. Number of tuberculosis cases and susceptibilities among Papua New Guinea nationals accessing Queensland health facilities in the Torres Strait Treaty Zone, 1995 to 2007***




* Data provided by A Konstantinos, Director, Queensland TB Control Centre.

or humanitarian entrants and 10% were overseas students. Unauthorised entrants, made up only 6% nationally but 85% of cases (n=17) reported in the Northern Territory. These were illegal fishermen detained by Australian Customs diagnosed with TB and commenced on TB treatment. The majority of refugees (56/68, 82%), overseas visitors (20/31, 96%), and overseas-born students (47/51, 92%) diagnosed with TB arrived in Australia between 2002 and 2006. All unauthorised entrants diagnosed with TB in 2006 had arrived within the past 12 months. By contrast, overseas-born permanent residents diagnosed with TB in 2006 had arrived in Australia between 1940 and 2006.

There was an increased number of notifications of TB in Queensland in 2006 among Papua New Guinea nationals accessing health care in the Torres Strait Islands treaty zone. Twenty-one cases, four with MDR-TB, were diagnosed in 2006. The number of cases (including MDR-TB cases) continued to increase in 2007 (Figure 5).

### Tuberculosis notifications by age and sex

Information on the sex of TB cases was available for all cases and age was identified in all but two cases (1 non-Indigenous Australian-born and 1 overseas-born case). The male to female ratio in TB notifications was 1.5:1 in non-Indigenous Australian-born TB cases, 1:1 in Indigenous cases and 1.2:1 in overseas-born cases.

One of the most important measures of TB control is the incidence in children aged less than 15 years because these cases represent recent TB infection. TB was notified in 62 children aged less than 15 years in 2006, slightly lower than the number (65) reported in 2005. These were 21 Australian-born children and 41 children born overseas. Of the 21 Australian-born children, three were identified as Indigenous.

The NTAC target performance indicator for rates of TB in children aged less than 15 years is less than 0.1 cases per 100,000 population for all groups. The overall notification rate for the less than 15 years age group in 2006 was 1.6 cases per 100,000 population. The rate was highest in overseas-born children (19.8 cases per 100,000 population) and remained low in the non-Indigenous (0.5 cases per 100,000 population) and Indigenous Australian-born children (1.7 cases per 100,000 population, Table 5).

The age group incidence rates for TB in overseas-born, Indigenous Australian-born and non-Indigenous Australian-born populations are shown in Figure 6 and Table 5. TB incidence in the overseas-born population showed three peaks: one among infants aged less than five years; a second among young adults (15–34 years) and a third peak in the over 65 year age group. TB rates among Indigenous and non-Indigenous Australians showed increasing rates throughout adult life with the highest TB rates in those aged 65 years or more.

The age adjusted rate for Indigenous people was 18.1 per 100,000 (crude 6.6 per 100,000); 28.5 per 100,000 (crude 20.7 per 100,000)for overseas-born; and 0.9 per 100,000 (crude 0.9 per 100,000) in the non-Indigenous Australian-born.

## Table 5. Tuberculosis notifications and estimated incidence rate, Australia, 2006,* by age group, indigenous status and country of birth

| Age group | Indigenous Australian-born | | Non-Indigenous Australian-born | | Overseas-born | |
|---|---|---|---|---|---|---|
| | n | Rate | n | Rate | n | Rate |
| 0–4 | 1 | 1.7 | 9 | 0.8 | 13 | 60.8 |
| 5–14 | 2 | 1.8 | 9 | 0.4 | 28 | 13.9 |
| Subtotal < 15 years | 3 | 1.7 | 18 | 0.5 | 44 | 19.8 |
| 15–24 | 6 | 6.4 | 8 | 0.3 | 139 | 30.5 |
| 25–34 | 3 | 4.5 | 21 | 1.0 | 254 | 35.6 |
| 35–44 | 5 | 10.6 | 7 | 0.3 | 180 | 20.1 |
| 45–54 | 7 | 25.6 | 9 | 0.5 | 138 | 15.3 |
| 55–64 | 5 | 41.0 | 14 | 1.0 | 78 | 9.6 |
| 65+ | 4 | 60.4 | 62 | 3.5 | 197 | 65.7 |

* One non-Indigenous Australian-born and one overseas-born case had no age information.

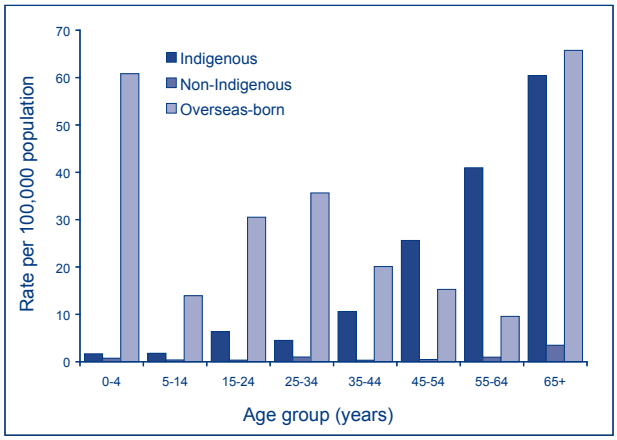
### Tuberculosis and selected risk factors

Information on risk factors for TB disease, excluding HIV, were reported in 301 cases (Table 6).

Household or other close contact with a TB patient was the most common risk factor in all three patient groups. Sixty-five cases of TB were reported in people who had previously worked or were currently working in a health care setting; 54 of these were overseas-born. Past residence of three months or longer in duration in a TB high risk country (defined as more than 12.5 cases per 100,000) was reported in 29 cases, including 18 Australian-born cases.

The number of TB cases reported in health care workers has risen from 34 in 2001 to 65 in 2006. More importantly, among health care workers notified with TB, the proportion born overseas has increased from 47% (16 of 34 cases) in 2001 to 83% (54 of 65 cases) in 2006 (Figure 7). At diagnosis, most health care workers were or had been working in the previous 12 months in an Australian health care setting. None of the cases were deemed to have acquired TB in an Australian health care setting, nor were there any reports of TB transmission to patients from health care workers in Australia in 2006.

## Figure 6. Tuberculosis incidence in Australian-born and overseas-born population, 2006, by age group



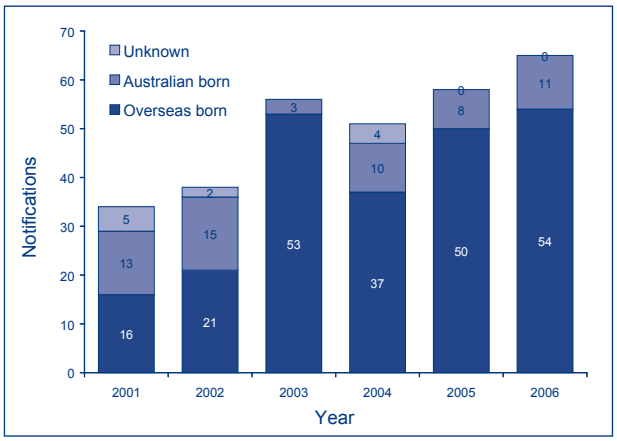

## Table 6. Selected risk factors* in tuberculosis notifications, Australia, 2006, by indigenous status and country of birth

| Risk factor | Indigenous | Non-Indigenous | Overseas-born | Total |
|---|---|---|---|---|
| Household or other close contact with TB | 5 | 33 | 139 | 177 |
| Currently or recently resident in correctional facility | 1 | 1 | 3 | 5 |
| Currently or recently residing in aged care facility | – | 3 | 2 | 5 |
| Currently or recently employed in an institution | – | 3 | 17 | 20 |
| Currently or previously employed in health industry | 1 | 10 | 54 | 65 |
| Past residence in high risk country | – | 18 | 11† | 29 |

* Excludes HIV status (see below); includes multiple risk factors.

† Excludes overseas born in tuberculosis high risk countries (incidence > 12.5 cases/100,000).

**Figure 7. Trends in cases of tuberculosis reported in health care workers, Australia, 2001 to 2006, by country of birth**




### Tuberculosis and HIV status

Information on HIV status was reported in 423 of the 1,201 cases (35.2%). Eleven people were identified with HIV infection at the time of diagnosis. All were overseas-born. In 2006, the proportion of cases with HIV status reported was similar to that in 2005.

### Anatomical site of disease

The anatomical site of TB infection was recorded in 1,168 (97.2%) cases. Of these, 591 (50.6%) cases of notified cases had pulmonary disease only, a further 113 (9.7%) cases had pulmonary disease and disease at an extrapulmonary site. Pulmonary TB was reported in 78% of the Australian-born cases and 55% of the overseas-born cases. 464 (39.7%) cases had extrapulmonary disease only. The sites of disease in new and relapse cases are shown in Table 7.

### Treatment outcomes of 2005 tuberculosis patient cohort

Treatment outcome data for all TB cases reported in 2005 were received by December 2007 (Table 8). Treatment success, including those with bacteriologically confirmed cure and those who completed treatment, was reported in more than 95% of non-Indigenous Australian born and overseas-born cases with assessable outcomes. In contrast, treatment success was reported in only 81% (17 of 21 with assessable outcomes, $p<0.05$) of Indigenous TB patients. This lower treatment success rate was due to a small number of cases (n=4) with poor outcomes (Table 8). There were no treatment failures.

### National Performance Indicators

Performance criteria for incidence (less than 1 per 100,000) were met only for the crude incidence rates in non-Indigenous Australian-born cases (Table 9). Incidence rates in under 15 year age groups exceeded the performance criteria (less than 1 case per million) in all groups. The completeness of HIV data collection remains well below the goal of 100%. Outcome reporting met the target of 100% for the 2005 patient cohort and while overall treatment success rate was met in the non-Indigenous and overseas-born, the treatment success rate in Indigenous cases (81.1%) was below the goal of >95%.

## Discussion

In 2006, notification rates of TB in Australia remained low despite increases in some jurisdictions associated with more reports of TB in those born overseas. Increased notifications of TB were reported in unauthorised entrants such as Indonesian fishermen who were detained, diagnosed and started

**Table 7. New and relapsed tuberculosis cases, Australia, 2006, by site of disease**

| Site | New | Relapse | Total | Per cent of cases |
|---|---|---|---|---|
| Total pulmonary disease | 658 | 46 | 704 | 60.3 |
| Pulmonary only | 551 | 40 | 591 | 50.6 |
| Pulmonary plus other sites | 107 | 6 | 113 | 9.7 |
| Extrapulmonary only | 455 | 9 | 464 | 39.7 |
| Pleural | 67 | 0 | 67 | 5.7 |
| Lymph nodes | 165 | 6 | 171 | 14.6 |
| Bone/joint | 35 | 3 | 38 | 3.3 |
| Genito/urinary | 29 | 0 | 29 | 2.5 |
| Milliary | 16 | 1 | 17 | 1.5 |
| Meningeal | 24 | 0 | 24 | 2.1 |
| Peritoneal | 12 | 0 | 12 | 1.0 |
| Other | 59 | 2 | 61 | 5.2 |

## Table 8. Tuberculosis treatment outcomes, Australia, 2005, by population group

| Outcomes | Indigenous | | Non-Indigenous Australian-born | | Overseas-born | | Total cases | |
|---|---|---|---|---|---|---|---|---|
| **Assessable outcomes** | **n** | **% assessable** | **n** | **% assessable** | **n** | **% assessable** | **n** | **% assessable** |
| Treatment success | 17 | 81.0 | 108 | 95.6 | 795 | 95.7 | 920 | 95.3 |
| Cured (bacteriologically confirmed)* | 5 | 23.8 | 6 | 5.3 | 41 | 4.9 | 52 | 5.4 |
| Completed treatment | 12 | 57.1 | 102 | 90.3 | 754 | 90.7 | 868 | 89.9 |
| Interrupted treatment† | 1 | 4.8 | 0 | 0.0 | 5 | 0.6 | 6 | 0.6 |
| Died of tuberculosis | 1 | 4.8 | 3 | 2.7 | 9 | 1.1 | 13 | 1.3 |
| Defaulted‡ | 2 | 9.5 | 2 | 1.8 | 9 | 1.1 | 13 | 1.3 |
| Failure§ | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| Not followed up, outcome unknown | 0 | 0.0 | 0 | 0.0 | 13 | 1.6 | 13 | 1.3 |
| Total assessable | 21 | 100.0 | 113 | 100.0 | 831 | 100.0 | 965 | 100.0 |
| **Non-assessable outcomes** | **n** | **% total** | **n** | **% total** | **n** | **% total** | **n** | **% total** |
| Transferred out of Australia | 0 | 0.0 | 1 | 0.8 | 44 | 4.8 | 45 | 4.2 |
| Died of other causes | 3 | 12.5 | 14 | 10.9 | 35 | 3.8 | 52 | 4.8 |
| Still under treatment | 0 | 0.0 | 1 | 0.8 | 10 | 1.1 | 11 | 1.0 |
| Total | 24 | | 129 | | 920 | | 1,073 | |

* Cured is defined as the bacteriologically confirmed cure of smear or culture positive pulmonary cases.

† Interrupted treatment means treatment interrupted for two months or more but completed.

‡ Defaulted means failed to complete treatment.

§ Failed means treatment completed but failed to be cured.

## Table 9. National tuberculosis performance indicators, performance criteria and the current status of tuberculosis in Australia, 2005 and 2006

| National tuberculosis Performance Indicator<br>Annual incidence of TB (per 100,000 population) | Performance criteria | 2005 | 2006 |
|---|---|---|---|
| **Crude incidence** | | | |
| Indigenous Australians | <1 | 5.9 | 6.6 |
| Non-Indigenous Australian-born persons | <1 | 0.8 | 0.8 |
| Overseas-born persons | * | 20.8 | 20.7 |
| Relapse cases initially treated in Australia | <2% treated cases | 1.4 | 0.9 |
| **Incidence in children <15 years, by risk group (per 100,000 population)** | | | |
| Indigenous Australian children | <0.1 | 0.6 | 1.7 |
| Non-Indigenous Australian-born children | <0.1 | 0.7 | 0.5 |
| Overseas-born children | * | 18 | 19.8 |
| Collection of HIV status in tuberculosis cases (% of cases with data collected) | 100% over next 3 years | 37 | 35 |
| **Treatment outcome measures (%)** | | | |
| Cases evaluated for outcomes† | 100 | 100 | TBA |
| Cases that have treatment completed and are cured | >90 | 95.3 | TBA |
| Cases recorded as treatment failures† | <2 | 0 | TBA |

* Performance criteria currently under review.

† Evaluation of outcomes of 2005 patient cohort re-assessed in October 2007.

TBA To be assessed: 2006 patient cohort outcomes to be reported in 2007 annual report.

on treatment for TB before deportation from Western Australia and the Northern Territory. In Queensland, Papua New Guinean nationals accessing health care in the Torres Strait Islands Treaty Zone contributed to an increase in that jurisdiction. In South Australia, several factors contributed to the increase in TB notifications, including 10 TB notifications in health care workers. These three groups are the focus of increased surveillance by tuberculosis services. Australian TB services continue to provide high quality services as evidenced by very high treatment success rates, low rates of relapse, the complete absence of treatment failure and low case fatality rate.

The number of TB notifications in health care workers has increased since 2001 and the proportion of these cases who were born overseas has also increased. These increases are linked to increasing recruitment of HCWs from countries with a high TB incidence in recent years. Ninety-five per cent of overseas-born HCWs diagnosed with TB in Australia since 2001 come from TB 'high risk' countries as defined by the Australian Government Department of Immigration and Citizenship (TB incidence > 12.5 per 100,000). In 2006, there were no reports of HCWs infecting patients, nor any proven cases of health care workers acquiring TB in an Australian health care setting. NTAC is developing a set of recommendations for screening and assessment of HCWs for TB prevention and control.

It is well recognised that those with HIV are more likely to become infected with *M. tuberculosis,* more likely to progress to active TB if *M. tuberculosis* infected, and more likely to have extrapulmonary disease than non-HIV infected. While the overlap of TB and HIV infection is still considered low in the Australian population, knowing the TB patient's HIV status allows for a best standard of care. In 2006, however the HIV status was reported for only 35.2% of Australia's TB cases, similar to the 37% reported for the 2005 TB cases. Emerson and Post observed that in the seven years, 1999 to 2005, only 21% of Australian notified TB cases had reported HIV status. They advocate an HIV test be offered, despite a low perceived risk for HIV infection, to all people with TB, with appropriate pretest discussion.[8] In the United States of America it took 10 years (1993 to 2003) to increase the HIV testing of TB patients from 35% to 68%.[9] It is hoped that those managing TB cases in Australia will heed the recommendation for HIV testing in a timely manner.

Since 1986, the Australian Mycobacterium Reference Laboratory Network (AMRLN) have collected data on bacteriologically confirmed TB cases, including drug susceptibility testing (DST) results, and have published annual reports which complement the data in this report. The NNDSS and AMRLN datasets will be combined during the next 12 months. By combining the demographic and clinical information in the NNDSS dataset with the DST results provided by the AMRLN more detailed analyses will be possible. For example, future reports will provide drug resistance rates among new and re-treatment cases (rather than a single combined resistance rate). Drug resistance rates will also be computed for migrants and refugees from particular high-incidence countries (e.g. Indonesia, Somalia, Sudan), where MDR-TB rates are currently unknown. This information may assist Australian doctors to recognise patients at increased risk of drug-resistant disease and will be informative for international TB control organisations, such as the WHO. Of note regarding MDR-TB in Australia, a recent publication from NTAC, *The NTAC Multi-drug Resistant Tuberculosis (MDR-TB) Information Paper (October 2007),* provides background information and advice on avoiding the production of MDR-TB, on the detection of MDR-TB, and approaches to treatment and management of cases and contacts on MDR-TB.[10]

Increased notifications of TB noted in some jurisdictions in 2006 were attributed to increased notification of TB in overseas-born residents. The failure to control TB within neighbouring nation states can pose direct public health threats to Australia as exemplified in the Treaty Zone between the outer Torres Strait Islands of Queensland and the various villages of the South Fly District of the Western Province of Papua New Guinea. Multi-drug-resistant tuberculosis has been detected among Papua New Guinean nationals accessing health care in the Torres Strait Islands within the treaty zone.[11] In 2006, 21 cases of TB (4 with MDR-TB) were diagnosed among these visitors; a significant contribution to the 129 new cases notified in Queensland that year. This has increased from the 14 (6 MDR) TB cases in 2005 and 9 (1 MDR) in 2004, and 2007 data show the number continues to increase among these visitors (A Konstantinos, personal communication).

Clearly, supporting effective TB control in this region would significantly decrease the risk of transmission of TB (including MDR-TB) across this border. With the high proportion of MDR-TB (10 of 35 cases diagnosed in 2005 and 2006), it is also important that the public health principles of TB management are maintained as failure to do so could bring not only MDR-TB but XDR-TB directly to Australia's doorstep. Therefore it is in Australia's interest to have regional and global involvement in TB control.

## Author details

Paul Roche[1]
Vicki Krause[2]
Anastasios Konstantinos[3]
Ivan Bastian[4]
Ral Antic[5]
Lynne Brown[6]
Amanda Christensen[7]
Sandra Gebbie[8]
Mark Hurwitz[9]
Avner Misrachi[10]
Justin Waring[11]

1. Surveillance Policy and Systems Section, Department of Health and Ageing, Canberra, Australian Capital Territory
2. Centre for Disease Control, Department of Health and Community Services, Darwin, Northern Territory
3. Director, Queensland TB Control Centre, Queensland Health, Brisbane, Queensland
4. Clinical Microbiology Consultant, Institute of Medical and Veterinary Science, Adelaide, South Australia
5. Director of Thoracic Medicine and South Australian TB Services, Royal Adelaide Hospital, Adelaide, South Australia
6. Program Manager, TB Control, Department of Human Services, Melbourne, Victoria
7. Manager, NSW TB Program, Communicable Diseases Branch, NSW Health Department, North Sydney, New South Wales
8. Director, Communicable Disease Management and Health Risk Policy, Australian Government Department of Health and Ageing, Canberra, Australian Capital Territory
9. Director Thoracic Medicine, The Canberra Hospital, Garran, Australian Capital Territory
10. Senior Medical Advisor, Communicable Diseases Prevention Unit, Department of Health and Human Services, Hobart, Tasmania
11. Head, Tuberculosis Services, Perth Chest Clinic, Perth, Western Australia

Corresponding author: Dr Paul Roche, Australian Government Department of Health and Ageing, MDP 14, GPO Box 9848, CANBERRA ACT 2601. Telephone +61 2 6289 2708. Facsimile: +61 2 6289 2600. Email: paul.roche@health.gov.au

## References

1. World Health Organization. Global Tuberculosis Control: surveillance, planning, financing. Geneva: World Health Organization; 2007. Report No. WHO/HTM/TB/2007.376.
2. WHO/IUATLD global project on anti-tuberculosis drug resistance surveillance. Anti-tuberculosis drug resistance in the world: third global report 1999–2002. Geneva: World Health Organization; 2004. Report No. WHO/HTM/TB/2004.343.
3. Wright A, Bai G, Barrera L, Boulahbal F, Martin-Casabona N, Gilpin C, et al. Emergence of *Mycobacterium tuberculosis* with extensive resistance to second-line drugs – world wide, 2000 – 2004. *MMWR Morb Mortal Wkly Rep* 2006;55:301–305.
4. Gandhi NR, Moll A, Sturm AW, Pawinski R, Govender T, Lalloo U, et al. Extensively drug-resistant tuberculosis as a cause of death in patients co-infected with tuberculosis and HIV in a rural area of South Africa. *Lancet* 2006;368:1575–1580.
5. Raviglione MC, Smith IM. XDR tuberculosis—implications for global public health. *N Engl J Med* 2007;356:656–659.
6. Roche PW, Bastian I, Krause V, National Tuberculosis Advisory Committee. Tuberculosis notifications in Australia, 2005. *Commun Dis Intell* 2007;31:71–80.
7. Lumb R, Bastian I, Gilpin C, Jelfs P, Keehner T, Sievers A. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2006. A report of the Australian Mycobacterium Reference Laboratory Network. *Commun Dis Intell* 2008;32:12–17.
8. Emerson CR, Post JJ. To routinely offer testing for HIV infection in all cases of tuberculosis: a rational clinical approach? *Med J Aust* 2008;188:162–163.
9. Marks S, Magee E, Robison V. Reported HIV status of tuberculosis patients—United States, 1993–2005. *MMWR Morb Mortal Wkly Rep* 2007;56:1103–1106.
10. National Tuberculosis Advisory Committee. Multi-drug-resistant tuberculosis. *Commun Dis Intell* 2007;31:406–409.
11. Gilpin CM, Simpson G, Vincent S, O'Brien TP, Knight TA, Globan M, et al. Evidence of primary transmission of multi-drug-resistant tuberculosis in the Western Province of Papua New Guinea. *Med J Aust* 2008;188:148–152.

# Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2006 A report of the Australian Mycobacterium Reference Laboratory Network

Richard Lumb, Ivan Bastian, Chris Gilpin, Peter Jelfs, Terillee Keehner, Aina Sievers

## Abstract

In 2006, the Australian Mycobacterium Reference Laboratory Network identified 905 bacteriologically confirmed cases of disease caused by members of the *Mycobacterium tuberculosis* complex. The annual reporting rate was 4.4 cases per 100,000 population. Of the 905 isolates, 903 were *Mycobacterium tuberculosis* and two were *Mycobacterium bovis*. Fourteen children aged under 10 years (male n=5, female n=9) had bacteriologically confirmed tuberculosis. A total of 100 (11.1%) isolates of *M. tuberculosis* were resistant to at least one first-line anti-tuberculosis agent. Resistance to at least H and R (defined as multi-drug resistant – MDR) was detected in 22 (2.4%) *M. tuberculosis* isolates. Of the 22 MDR-TB isolates, 17 were from the respiratory tract (sputum n=11 bronchoscopy n=5, nasogastric aspirate n=1), three from lymph node, one from a sacral mass, and one sterile site fluid. Smear-positive specimens from the MDR-TB cases were found in sputum (n=6), lymph node (n=2), and one each of bronchoscopy and nasogastric aspirate specimens. The country of birth was known for all 100 cases with a drug-resistant isolate; 10 of whom were born in Australia. The 90 overseas-born cases with drug-resistant disease were from 27 countries. Two Australian-born cases had MDR-TB; one had worked extensively in the Philippines; the other was a contact of a known MDR-TB case. No cases of extensively drug-resistant TB (XDR-TB) were identified in 2006. However, an on-going review of laboratory data identified one case of XDR-TB in 2004. *Commun Dis Intell* 2008;32:12–17.

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

## Introduction

Several events in 2007 have highlighted the importance of a well resourced, quality assured laboratory service to national tuberculosis (TB) control program in low– and high-income countries. For example, the expanding outbreak in South Africa of extensively drug-resistant TB (XDR-TB; defined as MDR-TB with additional resistance to a fluoroquinolone and a second-line injectable agent) has emphasised that culture and susceptibility testing facilities are necessary in low-income countries where HIV and drug-resistant TB are endemic.[1] The case of a lawyer from the United States of America who travelled on several international flights whilst diagnosed purportedly with XDR-TB but who was subsequently confirmed to 'only' have multidrug-resistant TB (MDR-TB), has reinforced the importance of timely and accurate culture and drug susceptibility test (DST) results in high income countries.[2]

Laboratories and laboratory networks are a fundamental component of TB control, providing testing for diagnosis, surveillance and treatment monitoring at every level of the health-care system. New technologies that provide rapid detection, identification and drug susceptibility testing of *Mycobacterium tuberculosis* have contributed to the decline of TB disease prevalence.[1] Australia has been fortunate to have five Mycobacterium reference laboratories overseeing and supporting a network of public and private pathology laboratories providing high-quality mycobacteriology diagnostic services.

There are two sources of TB-related data for Australia. Since 1991, the National Notifiable Diseases Surveillance System (NNDSS) has provided statistics on TB notifications reported to public health authorities in Australia's states and territories, and includes cases that were identified on the basis of clinical and epidemiological information or on non-bacteriological laboratory investigations. The Australian Tuberculosis Reporting Scheme has been conducted by the Australian Mycobacterium Reference Laboratory Network (AMRLN) since 1986. Statistics compiled by the AMRLN relate to cases of bacteriologically confirmed tuberculosis. This AMRLN report describes the bacteriologically confirmed TB diagnoses for the year 2006.

## Methods

The data are based on clinical specimens that were culture-positive for *Mycobacterium tuberculosis* complex (MTBC). No information on infections due to the bacille Calmette-Guérin strain of

*Mycobacterium bovis* is included in the present report. Isolates of MTBC were referred to one of the five laboratories comprising the AMRLN for species identification and drug susceptibility testing. Comparable methodologies are used in the reference laboratories. Relapse cases, as defined by the *National Strategic Plan for TB Control in Australia Beyond 2000* prepared by the National TB Advisory Committee,[3] were included in the laboratory data as laboratories are generally unable to differentiate relapse cases from new cases. Data include temporary visitors to Australia, illegal aliens or persons detained in Australia in correctional services facilities, and asylum seekers.

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

- demography: patient identifier, age, sex, HIV status and state of residence;
- specimen: type, site of collection, date of collection and microscopy result;
- isolate: *Mycobacterium* species and results of drug susceptibility testing;
- nucleic acid amplification testing results; and
- drug-resistant isolates: patient country of origin, and history of previous TB treatment to determine whether resistance was initial or acquired.

Data from contributing laboratories were submitted in standard format to the AMRLN coordinator for collation and analysis. Duplicate entries (indicated by identical patient identifier and date of birth) were deleted prior to analysis. Rates were calculated using mid-year estimates of the population for 2006 supplied by the Australian Bureau of Statistics.[4]

For each case, the nature of the first clinical specimen that yielded an isolate of MTBC was used to record the nominal site of disease. Culture-positive specimens collected at bronchoscopy or by gastric lavage were counted as pulmonary disease. Patients with isolates recovered from multiple sites were counted as pulmonary disease (the most important category for public health purposes) if a sputum, gastric aspirate, bronchoscopy, or lung biopsy specimen was culture-positive.

Drug resistance among new cases (proxy for primary resistance) was defined as the presence of resistant isolates of *M. tuberculosis* in patients, who, in response to direct questioning, denied having received any prior anti-TB treatment (for more than one month) and, in countries where adequate documentation is available, for whom there is no evidence of such a history. Drug resistance among previously treated cases (proxy for acquired resistance) is defined as the presence of resistant isolates of *M. tuberculosis*, in cases who, in response to direct questioning, admit having been treated for one month or more or, in countries where adequate documentation is available, for whom there is evidence of such a history.[5]

## Results

There were 905 bacteriologically confirmed cases of tuberculosis in 2006, representing an annual rate of 4.4 per 100,000 population. State-specific reporting rates varied from 1.8 (Tasmania) to 12.9 (Northern Territory) cases per 100,000 population (Table 1).

### Causative organism

Almost all isolates were identified as *M. tuberculosis* (n=903), the remaining isolates being *Mycobacterium bovis* (n=2).

**Table 1. Bacteriologically confirmed cases of tuberculosis in Australia, 1996 and 2004 to 2006, cases and rate per 100,000 population, by state or territory**

| State or territory | 2006 | | 2005* | | 2004* | | 1996* | |
|---|---|---|---|---|---|---|---|---|
| | n | Rate | n | Rate | n | Rate | n | Rate |
| New South Wales† | 342 | 4.8 | 346 | 4.9 | 308 | 4.4 | 341 | 5.3 |
| Northern Territory | 27 | 12.9 | 24 | 11.9 | 21 | 10.5 | 23 | 12.6 |
| Queensland | 120 | 3.0 | 91 | 2.3 | 88 | 2.3 | 90 | 2.7 |
| South Australia | 51 | 3.3 | 36 | 2.3 | 43 | 2.8 | 28 | 1.9 |
| Tasmania | 9 | 1.8 | 10 | 2.1 | 8 | 1.7 | 3 | 0.6 |
| Victoria | 263 | 5.2 | 261 | 5.2 | 262 | 5.3 | 214 | 4.7 |
| Western Australia | 93 | 4.5 | 42 | 2.1 | 57 | 2.9 | 51 | 2.9 |
| Total | 905 | 4.4 | 810 | 4.0 | 787 | 3.9 | 750 | 4.1 |

* Data from previous reports of the Australian Mycobacterium Reference Laboratory Network.

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

### Distribution by gender, age and site of disease

Complete information for gender and age were available for 902 (99.7%) of all patients; 392 (43.5%) were from females, 511 (56.7%) were from males, and gender was unknown for three cases. Fourteen children aged under 10 years (male n=5, female n=9) had bacteriologically confirmed tuberculosis (lymph node n=5, gastric aspirate n=2, sputum n=2, bronchoscopy n=1, cerebrospinal fluid n=2, biopsy n=2).

The site of disease was dependent upon age and gender. The overall male:female ratio was 1.3:1. For respiratory isolates, the male:female percentage was 1.7:1. For TB lymphadenitis, the female:male percentage was 1.7:1. For males, there were two distinct peak age groups in bacteriologically-confirmed rates: a rise to 8.2 cases of TB per 100,000 population in the 25–29 year age group and a second peak in elderly males aged more than 75 years (>13.0 cases per 100,000 population). The age distribution of female cases was similar with 7.5 and 9.3 bacteriologically confirmed TB cases per 100,000 population in the 25–29 years and >84 years age groups, respectively. The median age group for patients with bacteriologically confirmed disease was 30–34 years for males and 35–39 years for females.

The predominant culture-positive specimen type was sputum (n=438, 48.4%); a further 122 (13.5%) were obtained from bronchoscopy, and five were from lung biopsies (Table 2). Fifty-nine pleural specimens (38 fluid, 21 biopsy/tissue) were culture-positive. Of these 59 pleural specimens, five biopsy specimens and one pleural fluid was smear-positive. The most commonly encountered extrapulmonary culture-positive specimen was lymph tissue (n=163, 18.0%) followed by pleural (n=59, 6.5%), peritoneal (n=14, 1.5%), bone/joint (n=29, 3.2%), and genitorurinary tract (n=28, 3.1%).

**Table 2. Site of specimens smear– and culture-positive for *Mycobacterium tuberculosis* complex, 2006**

| | n | Smear positive (%)* |
|---|---|---|
| Sputum | 438 | 241 (55.8) |
| Bronchoscopy | 122 | 36 (29.5) |
| Lymph node | 163 | 43 (26.5) |
| Pleural | 59 | 6 (10.2)† |
| Genito-urinary | 28 | ND‡ |
| Bone/joint | 29 | ND‡ |
| Peritoneal | 14 | ND‡ |
| Skin | 3 | ND‡ |
| Cerebrospinal fluid | 7 | ND‡ |

* Based on specimens that reported a microscopy result and excludes (i) microscopy not performed or (ii) result unknown.

† 5/6 smear positive specimens were pleural biopsies.

‡ Percentage of specimens smear positive not calculated due to the small number of cases.

### Association with HIV

The AMRLN database recorded the HIV status of only 110 (12.2%) patients. Four patients were identified as HIV-seropositive.

### Microscopy

Results of microscopy were available for 889 of 905 (98.2%) specimens. Microscopy was not performed on 14 specimens and no results were provided for the remaining two specimens. For specimens where smear results were available, 241 of 438 (55.8%) sputum and 36 of 122 (29.5%) bronchoscopy specimens respectively were positive (Table 2). Of 59 pleural specimens (21 biopsy and 38 fluids) that were culture positive for *M. tuberculosis*, five biopsies and one fluid specimen was smear-positive. Lymph node specimens were smear-positive in only 43 of 163 (26.5%) patients.

### Drug susceptibility testing

Results of *in vitro* drug susceptibility testing were available for all 905 isolates for isoniazid (H), rifampicin (R), ethambutol (E), and for 904 of 905 isolates for pyrazinamide (Z). A total of 100 (11.1%) isolates of *M. tuberculosis* were resistant to at least one of these anti-tuberculosis agents. Results of testing for streptomycin (S) were available for 308 of 905 (34.0%) isolates with 46 demonstrating resistance to at least S; 11 had mono-resistance, 17 were resistant to S and H, 15 MDR-TB strains were also S-resistant, and there were three cases of S/E resistance. Resistance to at least H and R (defined as MDR) was detected in 22 (2.4%) isolates. All of the MDR isolates were *M. tuberculosis* (Table 3). Of the 22 MDR-TB isolates, 17 were from the respiratory tract (sputum n=11 bronchoscopy n=5, nasogastric aspirate n=1), three from lymph node, one from a sacral mass, and one fluid (site not stated). Six of the MDR-TB positive sputum specimens were smear-positive, one bronchoscopy specimen and the nasogastric aspirate, and two lymph node specimens.

Five patients with MDR-TB were from the Papua New Guinea–Torres Strait Islands (TSI) cross-border region who access health services in outer TSI and are eligible to receive treatment in Australia. MDR-TB was also isolated from patients born in India (n=6), Australia (n=2), and Indonesia (n=2) with a single case each from England, Lebanon,

**Table 3. Drug resistance patterns in multi-drug-resistant strains, Australia 1995 to 2006**

| Resistance pattern (standard drugs)* | 2006 | 2005 | 2004 | 2003 | 2002 | 2001 | 2000 |
|---|---|---|---|---|---|---|---|
| H+R only | 16 | 5 | 7 | 4 | 8 | 8 | 3 |
| H+R+E | 1 | 3 | 2 | 2 | 1 | 1 | 1 |
| H+R+Z | 0 | 1 | 1 | 1 | 1 | 3 | 3 |
| H+R+E+Z | 5 | 3 | 2 | 0 | 2 | 0 | 1 |
| XDR-TB | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| Total (%) | 22 (2.4) | 12 (1.5) | 12 (1.5) | 7 (0.9) | 12 (1.7) | 12 (1.6) | 8 (1.0) |

| Resistance pattern (standard drugs)* | 1999 | 1998 | 1997 | 1996 | 1995 |
|---|---|---|---|---|---|
| H+R only | 2 | 2 | 6 | 10 | 3 |
| H+R+E | 1 | 1 | 1 | 1 | 1 |
| H+R+Z | 1 | 2 | 5 | 4 | 1 |
| H+R+E+Z | 0 | 1 | 2 | 0 | 0 |
| XDR-TB | 0 | 0 | 0 | 0 | 0 |
| Total (%) | 4 (0.5) | 6 (0.9) | 14 (1.9) | 15 (2.0) | 5 (0.7) |

* The streptomycin result was not considered for this table.

† H = isoniazid, R = rifampicin, E = ethambutol, Z = pyrazinamide

Nigeria, the Philippines, Somalia, Thailand, and Uzbekistan. The English-born patient had been a health care worker in South Africa. Of the two Australian-born cases, one had worked extensively in the Philippines, and the other was a cousin to an Indian case of MDR-TB.

Mono-resistance to isoniazid (H) was detected in 53 isolates, three isolates were resistant to ethambutol (E) alone, and one isolate was resistant to pyrazinamide (Z) alone. No rifampicin mono-resistance was observed. Ninety-two isolates demonstrated resistance to H at a concentration of 0.1 mg/L. Of these, 66 (71.2.0%) demonstrated resistance to H at the higher level of 0.4 mg/L. Among MDR-TB strains, 19/22 (86.4%) demonstrated H resistance at the higher concentration (0.4 mg/L). Forty of 100 (40.0%) specimens culture-positive for drug-resistant strains, including 33 of 68 (48.5%) sputum or bronchoscopy specimens, were smear-positive for acid fast bacillus. The two *M. bovis* isolates, which are inherently resistant to pyrazinamide, were not included in the above results.

### New case or previously treated, and country of birth

Of the 100 *M. tuberculosis* isolates resistant to at least one of the standard drugs (H,R,E,Z), 80 were from new cases, 14 were from previously treated cases, and treatment information was not available for four cases. The country of birth was known for all cases with a drug-resistant isolate; 10 were born in Australia. The 90 overseas-born cases with drug-resistant disease were from 27 countries, 46 (51.1%) were from four countries: India (n=16). Vietnam (n=11), China (n=10), and the Philippines (n=9).

## Discussion

The AMRLN has collected data on bacteriologically confirmed cases of TB since 1986. The results for each year have been published in peer reviewed journals.[6–19] Data from 2006 broke new ground for: (i) the greatest number of bacteriologically confirmed cases of TB/cases per 100,000 population (905/4.4%); (ii) the number of isolates with drug resistance to at least one anti-tuberculous drug; and (iii) the number/percentage of MDR-TB isolates (22/2.4%). Since the AMRLN began collecting data in 1986, the number of bacteriologically confirmed cases per 100,000 population has remained stable at between a low of 3.5 (1992) and a previous high of 4.1 (1996).

Technological advances in laboratory equipment such as automated broth-based culture systems have certainly reduced the time to culture positivity and may have increased the total number of cases.[20] The radiometric broth-based culture system was introduced into Australia in the late 1980's and was the mainstay culture system into the early 2000s. All AMRLN laboratories are now using a non-radiometric automated broth culture system for primary culture.

Drug-resistant TB has emerged as a global problem that threatens TB control programs in many countries. In so many ways, Australia has been the 'lucky country' and the national TB control programs have achieved enviable success. None of the MDR-TB cases from 2006 have been acquired through treatment within Australia, a tribute to the continued high quality of Australian TB clinical services. The finding of 2.4% MDR-TB isolates is the highest recorded since data collection began in 1986. The significance or otherwise of the 2006 data will depend upon future findings but they must not be ignored.

The spectre of MDR-TB makes pre– and post-arrival screening of overseas-born persons even more critical. In particular, health care workers (HCWs) require monitoring; as emphasised by the detection in 2006 of MDR-TB in an English-born HCW who had worked previously in South Africa and similar instances of MDR-TB in overseas-born HCWs in previous reports. Australia also has a role to assist national TB control program and TB laboratory networks in our region.

The 2005 report discussed the global emergence of extensively drug-resistant TB (XDR-TB) and reported that a review of AMRLN data found no cases of XDR-TB.[19] Subsequently, an on-going review of Australian laboratory records found that one confirmed XDR-TB case in an overseas-born person had been identified in 2004 (personal communication, Dr M Hurwitz, Director, Thoracic Unit, The Canberra Hospital). The retrospective diagnosis of XDR-TB has been confounded in Australia and other countries by a change in the definition of XDR-TB in 2006,[21] by changes in laboratory technologies, and by revisions to the critical breakpoints for defining resistance to individual drugs. Retrospective and prospective surveillance of laboratory data will continue in the Australian setting.

The National TB Advisory Committee and the AMRLN have produced complementary publications for over 15 years focusing on the epidemiological and clinical information from the NNDSS database, and on DST results from bacteriologically-confirmed cases in the AMRLN dataset, respectively. This article will be the last standalone MRLN publication because the two TB databases will be combined before the end of 2008 so that more detailed analyses can be performed. For example, Australia will finally be able to report separate 'primary' and 'acquired' drug resistance rates when the clinical information in the NNDSS database, which identifies 'new' and 're-treatment' cases, is combined with the DST results in the AMRLN database. Furthermore, combining the two Australian TB databases will allow drug resistance rates to be calculated by the country of origin for overseas-born patients. These analyses may identify particular migrant groups at increased risk of drug-resistant disease. Australian doctors might consider adding additional agents to the initial treatment regimens of these patients until individual DST results become available. These calculations will also provide WHO and national TB programs with surrogate estimates of the drug resistance rates for some countries where quality assured drug susceptibility testing facilities are not widely available (e.g. Indonesia, Vietnam, Somalia, Sudan, Papua New Guinea).

## Acknowledgements

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

- Institute of Medical and Veterinary Science, Adelaide, South Australia
- Queensland Health Pathology Services, Herston Hospitals Complex, Herston, Queensland
- Victorian Infectious Diseases Reference Laboratory, North Melbourne, Victoria
- PathWest Laboratory Medicine WA – QEIIMC, Hospital Avenue, Nedlands, Western Australia
- Institute of Clinical Pathology and Medical Research, Westmead Hospital, Westmead, New South Wales

Additional information and support from Ms Amanda Christensen, Dr Ral Antic, Dr Vicki Krause, Ms Lynne Brown, Dr Anastasios Konstantinos, and Dr Christine Drummond is gratefully acknowledged.

## Author details

Richard Lumb[1,2]
Ivan Bastian[1,2]
Chris Gilpin[2]
Peter Jelfs[2]
Terillee Keehner[2]
Aina Sievers[2]

1. Infectious Diseases Laboratories, Institute of Medical and Veterinary Science, Adelaide, South Australia
2. Australian Mycobacterium Reference Laboratory Network

Corresponding author: Mr Richard Lumb, Chief Medical Scientist, Mycobacterium Reference Laboratory, Infectious Diseases Laboratories, Institute of Medical and Veterinary Science, PO Box 14, Rundle Mall, ADELAIDE SA 5000. Telephone: +61 8 8222 3579. Facsimile: +61 8 8222 3543. E-mail: richard.lumb@imvs.sa.gov.au

## References


1. Ridderhof JC, van Deun, A, Kam KM, Narayanan PR, Aziz MA. Role of laboratories and laboratory systems in effective tuberculosis programmes. *Bull World Health Organ* 2007;85:354–359.
2. Division of Tuberculosis Elimination, Centers for Disease Control and Prevention. Extensively drug-resistant tuberculosis. Available from: http://www.cdc.gov/tb/XDRTB/default.htm Accessed December 2007.
3. Communicable Diseases Network Australia. *National Strategic Plan for TB Control in Australia Beyond 2000*. Commonwealth Department of Health and Ageing, Canberra, July 2002.
4. Australian Bureau of Statistics. Australian Demographic Statistics, June Quarter 2006.
5. World Health Organization. Anti-tuberculosis drug resistance in the world. Third global report. WHO/HTM/TB/2004.343.
6. Dawson S, Anargyros P, Blacklock Z, Chew W, Dagnia H, Gow B, et al. Tuberculosis in Australia: an analysis of cases identified in reference laboratories in 1986–88. *Pathology* 1991;23:130–134.
7. Dawson D, Cheah DF, Chew F, Haverkort F, Lumb R, Sievers AS. Tuberculosis in Australia, 1989–1992. Bacteriologically confirmed cases and drug resistance. *Med J Aust* 1995;162:287–290.
8. Curran M, Dawson D, Cheah D. Laboratory surveillance of *Mycobacterium tuberculosis* isolates in Australia, 1992. *Commun Dis Intell* 1994;18:337–339.
9. Curran M, Dawson D. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 1993: report of the Australian Mycobacterium Reference Laboratory Network. *Commun Dis Intell* 1995;19:343–345.
10. Dawson D. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 1994 and 1995: report of the Australian Mycobacterium Reference Laboratory Network. *Commun Dis Intell* 1997;21:245–249.
11. Dawson D. Tuberculosis in Australia; bacteriologically confirmed cases and drug resistance, 1996: report of the Australian Mycobacterium Reference Laboratory Network. *Commun Dis Intell* 1998;22:183–188.
12. Dawson D. Tuberculosis in Australia; bacteriologically confirmed cases and drug resistance, 1997: report of the Australian Mycobacterium Reference Laboratory Network. *Commun Dis Intell* 1999;23:349–353.
13. Dawson D. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 1998-1999: report of the Australian Mycobacterium Reference Laboratory Network. *Commun Dis Intell* 2001;25:261–265.
14. Lumb R, Bastian I, Dawson D, Gilpin C, Haverkort F, James G, Sievers A. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2000: report of the Australian Mycobacterium Reference Laboratory Network. *Commun Dis Intell* 2002;26:226–233.
15. Lumb R, Bastian I, Dawson D, Gilpin C, Haverkort F, James G, Sievers A. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2001: report of the Australian Mycobacterium Reference Laboratory Network. *Commun Dis Intell* 2003;27:173–180.
16. Lumb R, Bastian I, Chew W, Gilpin C, Haverkort F, Sievers A. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2002: report of the Australian Mycobacterium Reference Laboratory Network. *Commun Dis Intell* 2003;27:458–464.
17. Lumb R, Bastian I, Crighton T, Gilpin C, Haverkort F, Sievers A. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2003: report of the Australian Mycobacterium Reference Laboratory Network. *Commun Dis Intell* 2004;28:474–480.
18. Lumb R, Bastian I, Crighton T, Gilpin C, Haverkort F, Sievers A. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2004: report of the Australian Mycobacterium Reference Laboratory Network. *Commun Dis Intell* 2006;30:102–108.
19. Lumb R, Bastian I, Gilpin C, Jelfs P, Keehner T, Sievers A. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2005. *Commun Dis Intell* 2007;31:80–86.
20. Cruciani M, Scarparo C, Malena M, Bosco O, Serpelloni G, Mengoli C. Meta-analysis of BACTEC MGIT 960 and BACTEC 460 TB , with or without solid media for detection of mycobacteria. *J Clin Microbiol* 2004;42:2321–2325.
21. World Health Organization. Extensively drug-resistant tuberculosis (XDR-TB): recommendations for prevention and control. *Wkly Epidemiol Rec* 2006;45:430–432. Available from: http://www.who.int/wer

# Invasive pneumococcal disease in Australia, 2006

Paul W Roche, Vicki Krause, Heather Cook, with input from Jenny Barralet David Coleman, Amy Sweeny, James Fielding, Carolien Giele, Robin Gilmour, Ros Holland, Riemke Kampen, as members of the Enhanced Invasive Pneumococcal Disease Surveillance Working Group and with laboratory data supplied by Mitchell Brown, Lyn Gilbert, Geoff Hogg, Denise Murphy, for the Pneumococcal Working Party of the Communicable Diseases Network Australia

## Abstract

Enhanced surveillance for invasive pneumococcal disease (IPD) was carried out in all Australian states and territories in 2006 with comprehensive comparative data available since 2002. There were 1,445 cases of IPD notified to the National Notifiable Diseases Surveillance System in Australia in 2006; a notification rate of 7 cases per 100,000 population. The rates varied between states and territories and by geographical region with the highest rates in the Northern Territory, the jurisdiction with the largest proportion of Indigenous people. Invasive pneumococcal disease was reported most frequently in those aged 85 years or over (30.8 cases per 100,000 population) and in children aged one year (26.5 cases per 100,000 population). There were 130 deaths attributed to IPD resulting in an overall case fatality rate of 9%. The overall rate of IPD in Indigenous Australians was 4.3 times the rate in non-Indigenous Australians. The rate of IPD in the under two years population continued to fall in 2006, but the rate in Indigenous children (73 cases per 100,000 population) was significantly greater than in non-Indigenous children (21 cases per 100,000 population). The rates of disease caused by serotypes in the 7-valent pneumococcal conjugate vaccine (7vPCV) decreased between 2002 and 2006 by 78% in children aged under two years as a result of the introduction of a universal childhood 7vPCV immunisation program. Significant decreases in IPD caused by 7vPCV serotypes also occurred in the 2–14 years and 65 years or over age groups. Rates of disease caused by non-7vPCV in the same periods were little changed. Serotypes were identified in 94% of all notified cases, with 43% of disease caused by serotypes in the 7vPCV and 85% caused by serotypes in the 23-valent polysaccharide pneumococcal vaccine (23vPPV). The number of invasive pneumococcal isolates with reduced penicillin susceptibility remains low and reduced susceptibility to third generation cephalosporins is rare. *Commun Dis Intell* 2008;32:18–30.

Keywords: disease surveillance, pneumococcal disease, *Streptococcus pneumoniae*, *vaccination*

## Introduction

Since 2001 Australia has had a comprehensive surveillance system collecting data on all cases of invasive pneumococcal disease (IPD) in children aged less than five years and on most cases in adults. Surveillance data collected includes data on vaccination status, risk factors and clinical presentation of the patient and the serotype and antibiotic susceptibility of the pneumococcal isolate.

Surveillance has documented the impact of the 7-valent pneumococcal conjugate vaccine (7vPCV) immunisation programs for Indigenous children from July 2001 and the universal 7vPCV childhood immunisation program and 23-valent pneumococcal polysaccharide vaccine (23vPPV) immunisation program for adults aged 65 years or over, from January 2005 (Table 1).

In 2007, evidence began to emerge in a defined geographic area, of an increase in incidence of IPD in Alaskan Native children with high levels of 7vPCV vaccination caused by non-7vPCV serotypes.[1] An increase in disease caused by the non-7vPCV serotype, 19A, with a significant proportion resistant to multiple antibiotics, has been reported among children in Massachusetts.[2] Maintaining post-immunisation surveillance in Australia is essential to detect such changes in the epidemiology of IPD and to direct further reduction strategies.

## Methods and materials

### Case definition

A case of IPD was defined as the isolation from or the detection by nucleic acid test (NAT) in blood, cerebrospinal fluid (CSF) or other sterile site of *Streptococcus pneumoniae*.

### Data collection

Invasive pneumococcal disease has been a notifiable disease in some Australian states and territories for several years. In 2001, IPD was made notifiable in all states and territories and data are forwarded to the National Notifiable Diseases Surveillance System

## Table 1. Recommendations and funding initiatives for pneumococcal vaccination in Australia

| Vaccine | 23-valent polysaccharide vaccine | 7-valent conjugate vaccine |
|---|---|---|
| Pneumococcal serotypes | 1, 2, 3, 4, 5, 6B, 7F, 8, 9N, 9V, 10A, 11A, 12F, 14, 15B, 17F, 18C, 19A, 19F, 20, 22F, 23F, 33F | 4, 6B, 9V, 14, 18C, 19F, 23F |
| Target populations | All individuals aged 65 years or over to receive a single dose of vaccine with a booster five years later*<br>Aboriginal and Torres Strait Islander people aged 50 years or over to receive a single dose of vaccine with a booster five years later†<br>Aboriginal and Torres Strait Islander people aged between 15 and 49 years at high risk to receive a single dose of vaccine and appropriate booster(s)‡<br>Children who have underlying chronic illnesses predisposing to invasive pneumococcal disease (including asplenia and immunocompromised)§<br>Immunocompetent individuals with chronic illness including chronic cardiac, renal or pulmonary disease, diabetes and alcohol-related problems<br>Individuals with cerebrospinal fluid leaks‖<br>Tobacco smokers¶<br>As a booster dose at 18 to 24 months of age following a primary course of 7vPCV in Aboriginal and Torres Strait Islander children in regions of high incidence**<br>As a booster dose at 4 to 5 years of age following a primary course of 7vPCV in children at risk because of predisposing medical conditions** | Children at 2, 4 and 6 months of age.††<br>Children born between 1 January 2003 and 31 December 2004.‡‡<br>Additional booster dose for children in specific high-risk groups.** |

* Funded in Victoria from 1998, Funded nationally from 2005.

† Targeted funded programs in north Western Australia, Far North Queensland and the Northern Territory from 1995, Funded nationally from 1999.

‡ Funded nationally from 1999. Funded for all children aged 15 years or over in the Northern Territory from 1999.

§ Targeted funded programs for high risk aged over two years in north Western Australia and the Northern Territory from 1986. Recommended nationally for children aged over two years (pre-July 2001) and children aged over five years from July 2001.

‖ Recommended nationally for children aged over two years (pre-July 2001) and children aged over five years from July 2001.

¶ Recommended nationally from 2003.

** Funded nationally from July 2001.

†† Funded nationally for Indigenous children from July 2001 and all children from 2005.

‡‡ Funded nationally as a catch-up program in 2005.

(NNDSS). Since this required changes to state and territory public health legislation, the data in 2001 were incomplete in some states and territories, but were complete for all jurisdictions from 2002. NNDSS data in 2006 comprised core data, which is a set of data collected on all cases of all notifiable diseases, as well as 'enhanced' data specific for IPD. Data are continuously cleaned and updated and totals may vary from previous years.

### Clinical presentation

Clinical presentations were coded as pneumonia, meningitis, bacteraemia, other or unknown. Pneumonia was defined as blood culture or NAT positive for *S. pneumoniae* with clinical and/or radiological signs of pneumonia. Meningitis was defined as the detection of *S. pneumoniae* in the CSF and/or blood with supportive clinical findings. Bacteraemia was defined as the detection of *S. pneumoniae* in blood with no localising signs. 'Other' presentations included detection of *S. pneumoniae* in pleural, peritoneal or joint fluid. More than one clinical presentation could be recorded for each case.

### Risk factors

The national surveillance working party defined risk factor categories for IPD. They include prematurity, (less than 37 weeks gestation), congenital or chromosomal abnormality, anatomical or functional asplenia, being immunocompromised, chronic illness, childcare attendee, previous episode of IPD and other (e.g. a smoker). Other risk factors defined by jurisdictions were also collected. More than one risk factor could be recorded for each case.

### Antibiotic resistance

Antibiotic susceptibility results are reported from the patient's treating institutions and classified as sensitive, intermediate resistance or resistant. In some cases the results are from referral laboratories. Reduced susceptibility includes intermediate and fully resistant results.

### Vaccination

The definitions of vaccination status, vaccination confirmation and vaccine failure used in this report are described in Table 2. Vaccine coverage data (7vPCV) were provided by the Australian Childhood Immunisation Register (ACIR). The ACIR records details of vaccinations given to children under the age of seven years who live in Australia.

### Populations under surveillance

There were different populations under enhanced surveillance in jurisdictions in 2006 (Table 3).

Data were analysed by date of diagnosis, which was the earliest of the dates recorded in NNDSS (date of onset, specimen date, notification date or notification received date).

### Data analysis

The notification rates presented in this report were calculated using population data from the Australian Bureau of Statistics (ABS). The Estimated Resident Population (ABS 3201.0) in each state and territory and in Australia as a whole, as at 30 June 2006, was used as the denominator in rate calculations. Estimates of the Indigenous Australian population were based on projections from the 2001 census.

## Table 2. Definitions of vaccination status and vaccine failure used in this report

| Category | Definition |
|---|---|
| Fully vaccinated – aged < 15 years | Those that have completed the primary course of the relevant vaccine(s) required for their age, indigenous status, geographical location and/or other risk factor(s) according to the most recent edition of the *Australian Immunisation Handbook,* at least two weeks prior to disease onset with at least 28 days between doses of vaccine.<br>This includes the following;<br>• a child that received a vaccine as 'catch up' and therefore does not require a full three dose primary schedule. Providing they have had the number of doses required for the age they were at first dose they should be considered fully vaccinated.<br>• a child <15 years who received at least one 23vPPV vaccine at aged over five years and they are not yet due a subsequent dose of 23vPPV.<br>NB: A young child who has had all the required doses for their age but is not old enough to have completed the primary course would not be assessed as fully vaccinated. |
| Fully vaccinated – aged ≥ 15 years | Those that have had the number of doses of 23vPPV required for their age, indigenous status, geographical location and/or other risk factor(s) according to the most recent edition of the *Australian Immunisation Handbook,* at least two weeks prior to disease onset with at least 28 days between doses of vaccine.<br>NB: This is calculated on the age they were when they had their first dose of 23vPPV aged at least ≥ 15 years. |
| Partially vaccinated – aged < 15 years | Those that have received at least one dose, but not *all* the recommended doses of the relevant vaccine(s) required for their age, indigenous status, geographical location and/or other risk factor(s) according to the most recent edition of the *Australian Immunisation Handbook,* at least two weeks prior to disease onset with at least 28 days between doses of vaccine.<br>This includes the following;<br>• a child who is too young to have completed their primary course;<br>• a child that is overdue (>8 weeks) for a subsequent dose of their primary course;<br>• a child that is overdue for a booster dose of the relevant vaccine. |
| Partially vaccinated – aged ≥ 15 years | Those that have been vaccinated with at least one dose of 23vPPV but the time frame for a subsequent dose is outside the recommended schedule according to the *Australian Immunisation Handbook.* |
| Not vaccinated – all ages | Those that have never received a pneumococcal vaccine. |
| Vaccination validation | Written confirmation of vaccination through the Australian Childhood Immunisation Register, state or territory immunisation register or health record. |
| Vaccine failure | A fully vaccinated person (as defined above) with disease due to a serotype found in the corresponding vaccine |

### Table 3. Enhanced invasive pneumococcal disease surveillance data collection by states and territories in 2006

| Age group | Jurisdictions |
|---|---|
| Under 5 years | Australian Capital Territory, New South Wales, Queensland (South Brisbane Public Health Unit only), |
| Over 50 years | New South Wales |
| All ages | Northern Territory, Queensland (except South Brisbane Public Health Unit), Tasmania, South Australia, Victoria, Western Australia |

The ABS calculated projections based on assumptions about future births, deaths and migrations in the Indigenous population and a 'low' and 'high' estimate were reported. The 'low' estimate has been used in this report, consistent with the reporting of other national communicable diseases.

The significance of differences in rates and proportions was calculated using the Chi-square test with Yates correction.

## Results

There were 1,445 notifications of IPD to NNDSS in 2006; a 14% decrease on the number of notifications in 2005 with declines in all jurisdictions of between 7.2% in Victoria and 37% in the Australian Capital Territory. The number of notifications and notification rate per 100,000 population are shown in Table 4. The Northern Territory continued to have the highest notification rate (27.1 per 100,000 population) while Victoria had the lowest (5.4 per 100,000).

When notification rates of IPD were examined by geographical distribution, variation within states and territories was apparent (Map).

The number of notifications of IPD was greater in winter months with the peak number of notifications in August (210 notifications). The effect of season was more evident in the distribution of cases aged five years or more compared with younger children (Figure 1).

### Table 4. Notifications, rates and demographics of invasive pneumococcal disease cases Australia, 2006, by state or territory

| | State or territory | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW* | NT | Qld | SA | Tas | Vic | WA | Australia |
| Notifications | 19 | 566 | 56 | 253 | 104 | 41 | 276 | 130 | 1,445 |
| Rate per 100,000 | 5.8 | 8.3 | 27.1 | 6.2 | 6.7 | 8.4 | 5.4 | 6.3 | 7.0 |
| Male:female ratio | 1.7:1 | 1.3:1 | 1.7:1 | 1.4:1 | 1.1:1 | 1.3:1 | 1.3:1 | 1.1:1 | 1.3:1 |
| **Notifications aged < 5 years** | | | | | | | | | |
| Total | 8 | 62 | 11 | 46 | 15 | 5 | 31 | 18 | 196 |
| Indigenous | 0 | 3 | 10 | 5 | 1 | 0 | 0 | 7 | 26 |
| Non-Indigenous | 4 | 58 | 1 | 35 | 14 | 5 | 30 | 11 | 158 |
| Unknown | 4 | 1 | 0 | 6 | 0 | 0 | 1 | 0 | 12 |
| **Notifications aged 5 to 64 years** | | | | | | | | | |
| Total | 8 | 286 | 37 | 138 | 50 | 25 | 128 | 82 | 754 |
| Indigenous | 0 | 1* | 33 | 22 | 3 | 0 | 3 | 46 | 108 |
| Non-Indigenous | 1 | 131* | 4 | 106 | 47 | 24 | 117 | 36 | 466 |
| Unknown | 7 | 154* | 0 | 10 | 0 | 1 | 8 | 0 | 180 |
| **Notifications ≥ 65 years** | | | | | | | | | |
| Total | 3 | 218 | 8 | 69 | 39 | 11 | 117 | 30 | 495 |
| Indigenous | 0 | 1 | 4 | 1 | 0 | 0 | 1 | 0 | 7 |
| Non-Indigenous | 1 | 215 | 4 | 59 | 39 | 11 | 108 | 30 | 467 |
| Unknown | 2 | 2 | 0 | 9 | 0 | 0 | 8 | 0 | 21 |

* Under the *NSW Public Health Act 1991,* all diagnoses of invasive pneumococcal infection are notifiable by laboratories. Aboriginality is not typically provided by laboratories at the time of notification. New South Wales Public Health staff undertake enhanced surveillance on cases aged less than five years and 50 years or over. Aboriginality for cases aged 5 to 49 years is likely to be incomplete and valid inferences cannot be made from this data.

**Figure 1. Notifications of invasive pneumococcal disease, Australia, 2006, by month of report and age group**

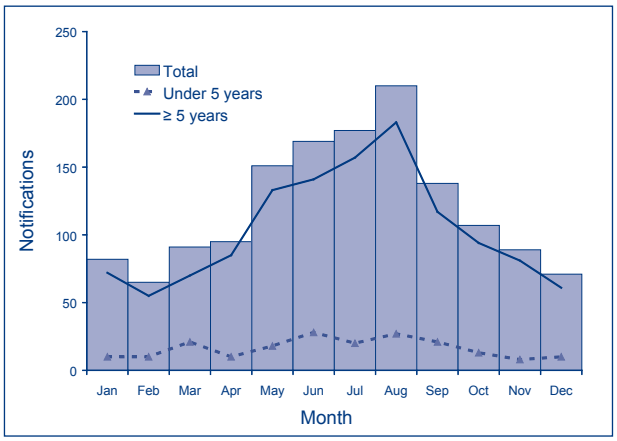
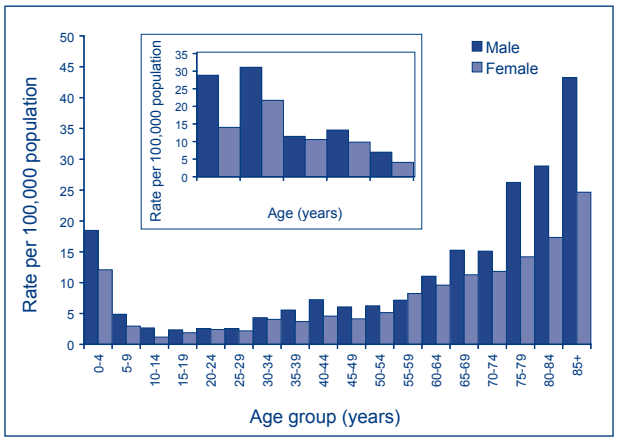



**Figure 2. Notification rates of invasive pneumococcal disease, Australia, 2006, by age group and sex**

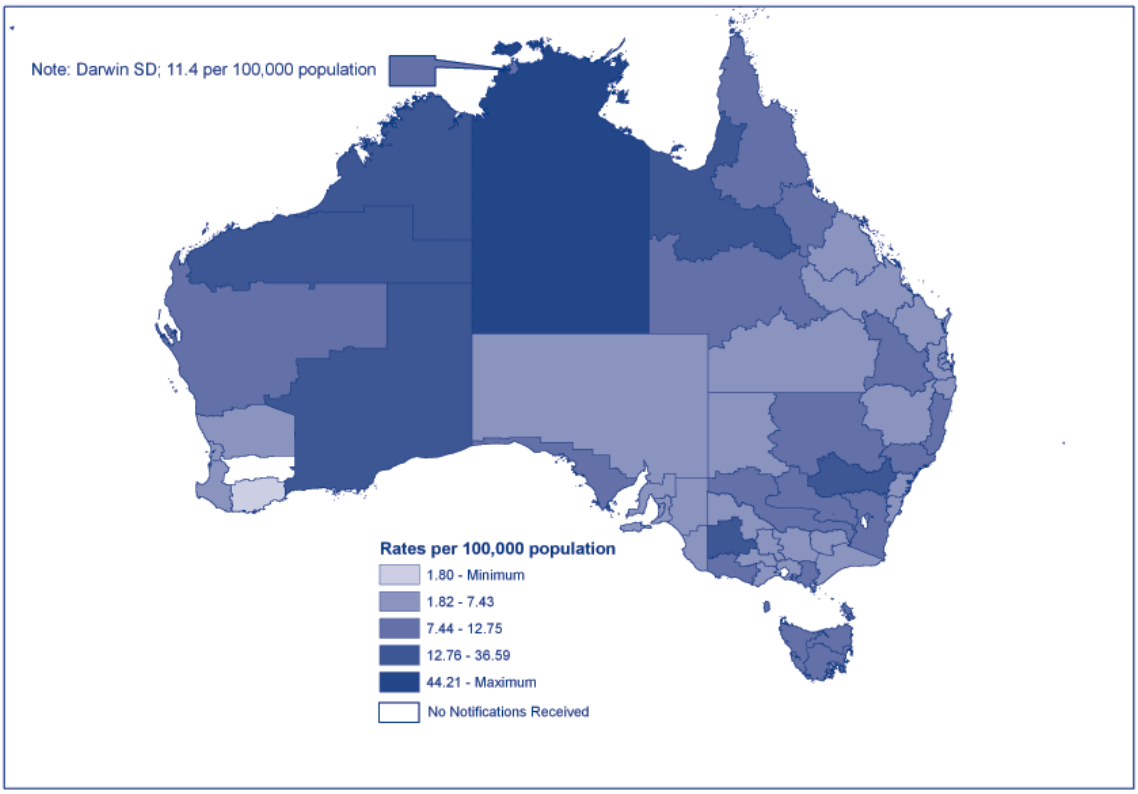



The highest rates of IPD disease in 2006 were among the elderly aged 85 years or over (30.8 cases per 100,000 population) and in children aged one year (26.5 cases per 100,000 population, Figure 2). In all age groups there were more male than female cases (overall male to female ratio 1.3:1).

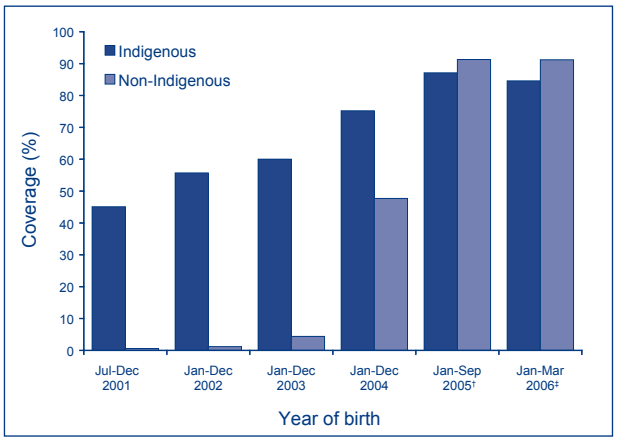
There were continued declines in notification rates between 2005 and 2006, in the under five year age group (from 24 to 15 per 100,000) and in children aged one year (from 36.5 to 26.5 per 100,000) reflecting the impact of the introduction of the universal 7vPCV immunisation program in 2005.

In 2006, the proportion of children aged 12 months immunised with three doses of 7vPCV was 84.6% in Indigenous children and 91.2% in non-Indigenous children. The proportion of children who are fully vaccinated against pneumococcal disease has increased steadily since 2001 (Figure 3).

**Map. Notification rates of invasive pneumococcal disease, Australia, 2006, by Statistical Division of residence**



### Figure 3. The proportion of children aged 12 months fully vaccinated with 7vPCV,* Australia, 2001 to 2006, by indigenous status

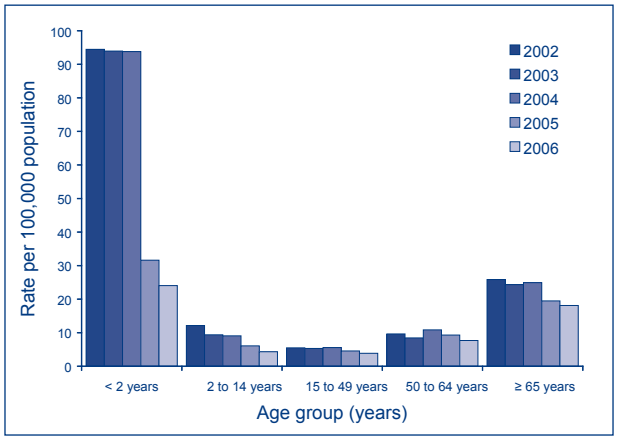



* Source: The Australian Childhood Immunisation Register.

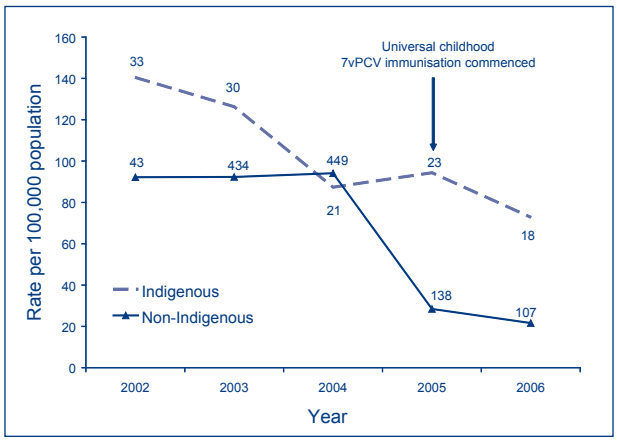
† 2005 data assessed for cohort born between 1 January and 30 September 2005 only.

‡ 2006 data assessed for the cohort born between 1 January 2006 and 31 March 2006 and assessed at 30 June 2007.

### Figure 4. Notification rate of pneumococcal disease, Australia, 2002 to 2006, by age group




### Figure 5. Notification rates of invasive pneumococcal disease in Indigenous and non-Indigenous children aged less than two years, Australia, 2002 to 2006




An examination of trends in rates of IPD in different age groups from 2002 to 2006 is shown in Figure 4. The rates in children aged under two years declined by 75% ($p<0.0001$) and in adults aged 65 years or over by 30% ($p<0.0001$) over the five-year period. Rates of IPD in other age groups not specifically targeted for pneumococcal immunisation also declined – there was a 65% ($p<0.0001$) reduction in the 2–14 year age group; a 30% ($p<0.0001$) decline in the 15–49 year age group and a 20% decline ($p<0.01$) in the 50–64 year age group.

## Rates in Indigenous people

In 2006, Indigenous status was reported in 1,232 (85%) notifications. New South Wales and the Australian Capital Territory continue to have the highest proportion of incomplete reporting. There were 141 cases of IPD among Indigenous people (9.7% of all cases). This represents a rate of 28 cases per 100,000 in the Indigenous population—a rate 4.3 times that seen in the non-Indigenous population (6.5 per 100,000).

## Rates in children

Rates in Indigenous children aged less than 2 years fell from 94 per 100,000 in 2005 (23 cases) to 73 cases per 100,000 population (18 cases) in 2006. In non-Indigenous children in the same age group rates fell from 28.7 cases per 100,000 population in 2005 (138 cases) to 21 cases per 100,000 population(107 cases) in 2006 (Figure 5).

Figure 6 shows the annual rates in single year age groups in children aged two years or under. In 2006, the rates of IPD in Indigenous children aged less than one year (0 to 11 months) fell from 97.6 in 2005 to 48.1 per 100,000 (from 12 to 6 cases). In 2006, the number of notifications and rate in Indigenous one year olds (12 cases, 98 per 100,000) and two year olds (5 cases, 41.5 per 100,000) were similar to those in 2005 (11 cases, 91.1 per 100,000 and 6 cases, 54 per 100,000 respectively). None of these declines in rates reached statistical significance.

In non-Indigenous children, rates fell in all three age groups: in those aged less than one year from 23.3 to 19.4 cases per 100,000 population; (57 to 51 cases); in the one year age group from 33.7 to 21.9 per 100,000; (81 to 56 cases) and in the two year age group from 27.5 to 9.1 cases per 100,000 (population 61 to 25 cases, Figure 6). All these declines in rates were highly statistically significant.

**Figure 6. Rates of invasive pneumococcal disease in children aged two years and under, Australia, 2002 to 2006, by indigenous status and single year age group**

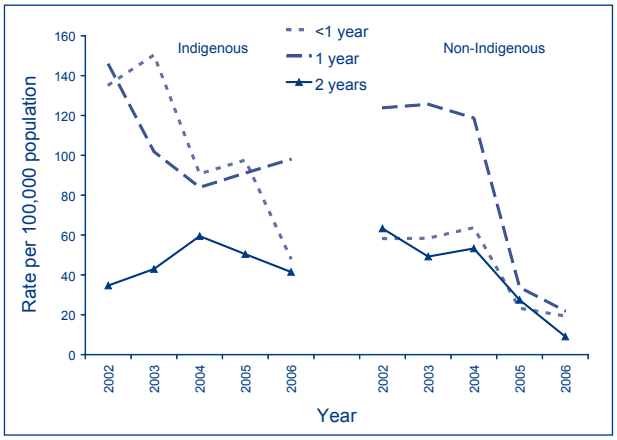
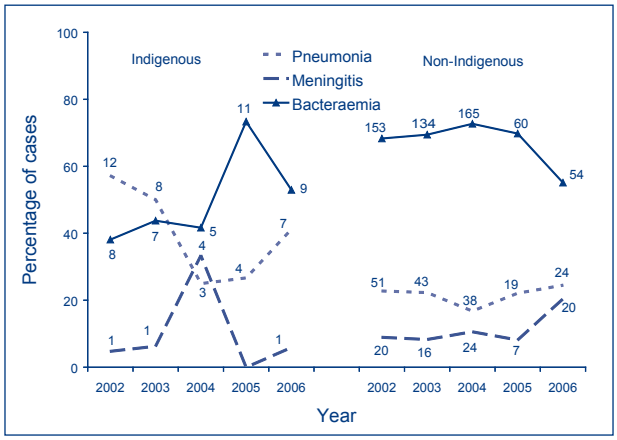



**Figure 7. Changes in clinical presentation of invasive pneumococcal disease in cases aged less than two years, 2002 to 2006, by indigenous status**




## Clinical presentations of invasive pneumococcal disease

Clinical presentation was reported on 1,172 (81%) cases. More than one clinical presentation could be reported. Seven hundred and fifty-one (64%) were pneumonia, 307 (26%) were bacteraemia, 101 (8.6%) were meningitis and 33 were other sterile site presentations (3%). Trends in clinical presentations of IPD in Indigenous and non-Indigenous children aged less than two years are shown in Figure 7. The relative proportions of pneumonia, bacteraemia and meningitis were similar in both groups in 2006.

## Deaths in invasive pneumococcal disease cases

There were 130 deaths recorded among IPD cases in Australia in 2006, a case fatality rate of 9% (Table 5). While the overall number of deaths increased by only two over the total in 2005, the case fatality rates were higher in all categories because of the lower number of cases. The case fatality rate in those aged 65 years or over (17.4%) was significantly higher than in children aged less than five years (3.1%, $p<0.0001$).

A case fatality rate for Indigenous and non-Indigenous cases was not calculated because of concerns about the completeness of indigenous status reporting particularly in the 5–64 year age group in New South Wales and the Australian Capital Territory.

Deaths are likely to be under reported as enhanced data are not collected in some jurisdictions for age groups between 5 and 50 years or 5 and 64 years. Further details of the six children aged less than five years whose deaths were associated with IPD are shown in Table 6. The four unvaccinated children all had 7vPCV serotype disease, while the two children fully vaccinated for age had non-7vPCV serotype disease.

**Table 5. Deaths and case fatality rates for invasive pneumococcal disease, Australia, 2006, by age, indigenous status and state or territory**

| | State or territory | | | | | | | | Australia |
|---|---|---|---|---|---|---|---|---|---|
| | ACT* | NSW* | NT | Qld | SA | Tas | Vic | WA | |
| Cases | 19 | 566 | 56 | 253 | 104 | 41 | 276 | 130 | 1,445 |
| Deaths | 2 | 66 | 5 | 10 | 3 | 3 | 29 | 12 | 130 |
| Deaths in under 5 years | 0 | 3 | 0 | 0 | 0 | 0 | 1 | 2 | 6 |
| Case fatality rate under 5 years | 0.0 | 4.8 | 0.0 | 0.0 | 0.0 | 0.0 | 3.2 | 11.1 | 3.1 |
| Deaths in ≥65 years | 1 | 53 | 1 | 5 | 3 | 1 | 17 | 5 | 86 |
| Case fatality rate ≥65 years | 33.3 | 24.3 | 12.5 | 7.2 | 7.7 | 9.1 | 14.5 | 16.7 | 17.4 |
| Deaths in Indigenous people | 0 | 1 | 5 | 4 | 0 | 0 | 0 | 3 | 13 |
| Deaths in non-Indigenous people | 2 | 65 | 0 | 6 | 3 | 3 | 29 | 9 | 117 |

* Limited indigenous identity data on cases aged between 5 and 64 years in Australian Capital Territory and New South Wales.

### Risk factors for invasive pneumococcal disease

Recognised risk factors were recorded in 47 (24%) of 196 cases aged less than five years. Ten (38%) Indigenous children aged less than five years with IPD had risk factors, compared with 37 non-Indigenous children (23%, p=ns, Table 7). Attending childcare was identified as an IPD risk factor in a higher proportion of non-Indigenous children than Indigenous children ($p<0.05$), while other risk factors such as exposure to smoke were more common in Indigenous children ($p<0.001$).

### Pneumococcal serotypes causing disease in Australia

Pneumococcal serotypes were identified for isolates from 1,361 (94%) of all notified cases in 2006. Of these, 587 (43.1%) were serotypes in the 7vPCV and 1,154 (84.8%) were serotypes in the 23vPPV (Table 8).

The proportion of 7vPCV serotypes in cases of IPD in the Northern Territory (14.8%) and Western Australia (39%) were significantly lower than the proportion of the national total (43.1%). The proportion of 23vPPV serotypes in the Northern Territory (64.8%) was also significantly lower than the proportion in the national total (84.8%, Table 8).

An examination of the rates of IPD disease caused by 7vPCV serotypes in Indigenous children aged less than two years showed a decline in rates (from 54.2 to 12 per 100,000) between 2002 and 2006. In non-Indigenous children, rates of 7vPCV serotype disease, which fell from 73.4 per 100,000 to 15 cases per 100,000 population between 2004 and 2005, continued to decline in 2006 to 6.7 per 100,000. Rates of disease caused by non-7vPCV serotypes in the same periods were little changed for both Indigenous and non-Indigenous children—62.6 to 59.8 (2002–2006) and 17.6 to 13.5 (2004–2006 respectively, Figure 8).

**Table 6. Characteristics of childhood deaths from invasive pneumococcal disease, Australia, 2006**

| Patient | Sex | Age (mos) | Indigenous status | Serotype | Vaccination status | Vaccine | Risk factors |
|---|---|---|---|---|---|---|---|
| **Deaths preventable by 7vPCV** | | | | | | | |
| 1 | Male | 1.5 | Unknown | 19F | Unvaccinated | Nil | Unknown |
| 2 | Male | 21.6 | Indigenous | 14 | Unvaccinated | Nil | Unknown |
| 3 | Female | 46.0 | Non-Indigenous | 18C | Unvaccinated | Nil | Unknown |
| 4 | Male | 53.2 | Non-Indigenous | 4 | Unvaccinated | Nil | No risk factors |
| **Deaths not preventable by 7vPCV** | | | | | | | |
| 5 | Female | 6.6 | Non-Indigenous | 7F | Fully for age | 7vPCV | Unknown |
| 6 | Male | 13.8 | Indigenous | 33F | Fully for age | 7vPCV | Premature chronic illness |

**Table 7. Risk factors* for invasive pneumococcal disease in children aged less than five years with invasive pneumococcal disease, Australia, 2006, by indigenous status**

| Risk factor | Indigenous n=26 | Non-Indigenous n=158 | Significance of difference |
|---|---|---|---|
| Premature birth | 2 | 7 | ns |
| Congenital abnormality | 0 | 6 | ns |
| Asplenia | 0 | 0 | |
| Immunocompromised | 1 | 4 | ns |
| Chronic illness | 1 | 3 | ns |
| Child-care attendee | 0 | 12 | ns |
| Previous episode of IPD | 1 | 4 | ns |
| Other† | 7 | 4 | p<0.0001 |
| No risk factors | 16 | 121 | |

* Child could have more than one risk factor.

† Other risk factors include exposure to smoke.

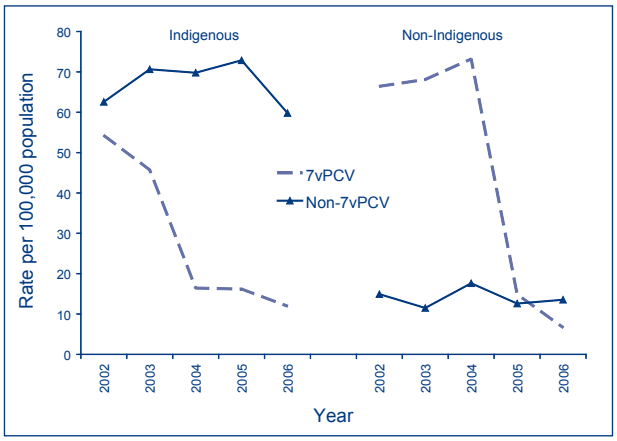
## Table 8. Number and proportion* of pneumococcal serotypes in cases of invasive pneumococcal disease covered by the 7-valent and 23-valent pneumococcal vaccines, Australia, 2006, by state or territory

| | State or territory | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Total |
| 7v serotypes | 8 | 242 | 8§ | 109 | 43 | 16 | 113 | 48‡ | 587 |
| % | 42.1 | 46.9 | 14.8 | 45.0 | 43.9 | 40.0 | 41.9 | 39.3 | 43.1 |
| 23V serotypes | 14 | 446 | 35§ | 206 | 82 | 34 | 235 | 102 | 1,154 |
| % | 73.7 | 86.4 | 64.8 | 85.1 | 83.7 | 85.0 | 87.0 | 83.6 | 84.8 |
| Total serotyped | 19 | 516 | 54 | 242 | 98 | 40 | 270 | 122 | 1,361 |

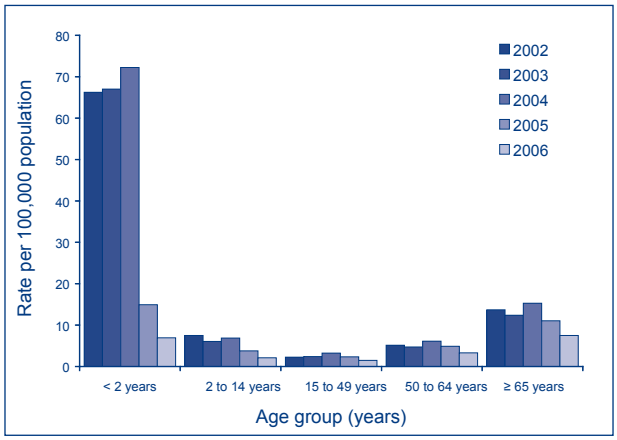
* As a proportion of serotyped isolates, including untypable isolates (2 in New South Wales, 3 in Victoria, 1 in Queensland and 1 in the Northern Territory).

Significantly lower proportion of 7vPCV and 23vPPV serotypes compared with national total (‡ p<0.05, § p<0.0001).

## Figure 8. Notification rates of 7-valent and non-7-valent serotypes causing cases of invasive pneumococcal disease in children aged less than two years, 2002 to 2006, by indigenous status




## Figure 9. Rates of invasive pneumococcal disease caused by 7-valent pneumococcal vaccine serotypes, 2002 to 2006, by age group




The total population rates of IPD caused by 7-valent vaccine serotypes fell 89.6% between 2002 and 2006 in the under two years age group (66.2 to 6.9 per 100,000), 82% in the 2–14 years age group (7.5 to 2.1 per 100,000) and 46% in the 65 years or over age group (13.7 to 7.5 per 100,000). All these declines were statistically significant. There were smaller declines in the rates in the 15–49 years (32%, 2.2 to 1.5 per 100,000) and 50–64 years age group (36%, 5.1 to 3.3 per 100,000, Figure 9).

A recent study of Alaskan Native children who received 7vPCV from 2000 has identified the emergence of replacement pneumococcal disease caused by non-7vPCV strains, in particular serotypes 7F and 19A.[1] Figure 10 shows trends in the rates of IPD in Indigenous and non-Indigenous children caused by serotypes 6A, 7F and 19A between 2002 and 2006.

Serotype 7F disease remains infrequent in non-Indigenous children (less than 5 cases in all years) and in Indigenous children (2 cases in all years except 2004, Figure 10). Rates of disease caused by serotype 19A increased in Indigenous children from 1.7 per 100,000 (1 case) in 2002, to 11.9 per 100,000 (7 cases) in 2004, and declined to four cases in both 2005 and in 2006 (6.6 per 100,000). Disease caused by serotype 19A increased minimally in non-Indigenous children from 2 per 100,000 (24 cases) in 2002 to 3.2 per 100,000 (39 cases) in 2006 (p=0.07). Serotype 6A has become the most common non-vaccine serotype in Indigenous children in 2006 (8.3 per 100,000, 5 cases) and the second most common in non-Indigenous children (0.9 per 100,000, 11 cases).

Rates of IPD caused by 23vPPV serotypes in Indigenous adults aged 50 years or over due to 23vPPV serotypes increased slightly between 2002 and 2006 from 9 to 16 cases (18.5 to 28.8 per 100,00). At the

**Figure 10. Rates of invasive pneumococcal disease due to serotypes 19A, 6A and 7F in Indigenous and non-Indigenous children aged less than five years, 2002 to 2006**

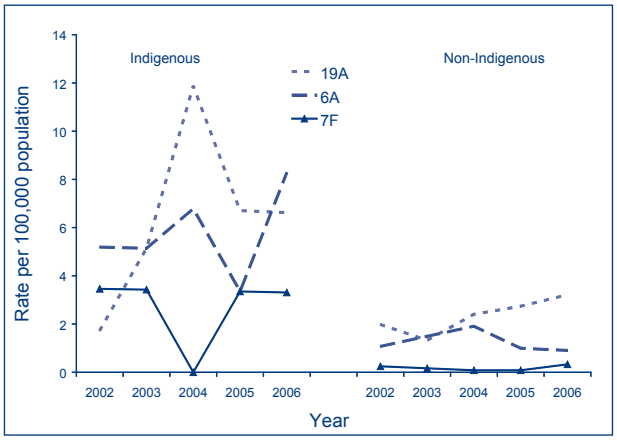
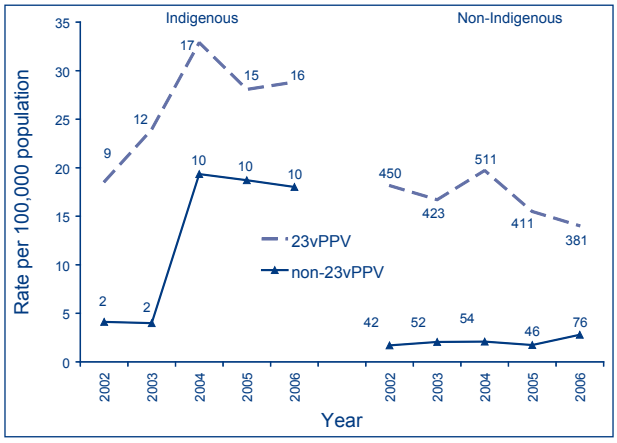



**Figure 11. Notification rates of 23-valent and non-23-valent serotypes causing cases of invasive pneumococcal disease in Indigenous adults (aged more than 50 years) and non-Indigenous adults (aged 65 years or over), 2002 to 2006**




same time rates of disease caused by non-23vPPV serotypes increased from 4 to 18 per 100,000 (from 2 to 10 cases).

In non-Indigenous adults aged 65 years or over rates of IPD caused by 23vPPV serotypes decreased slightly between 2005 and 2006 (from 15.5 to 14 per 100,000) and rates of non-23PPV serotype disease remained low (1.7 per 100,000 in 2005 and 2.8 per 100,000 in 2006, Figure 11).

### Antibiotic resistance in invasive pneumococcal disease

The penicillin susceptibility was tested in 1,351 isolates and ceftriaxone/cefotaxime susceptibility was tested in 1,046 isolates (Table 9).

A total of 143 (10.6%) isolates had reduced susceptibility to penicillin, which was lower than the number and proportion of isolates with reduced penicillin susceptibility in 2005 (176 isolates, 12%). Thirty isolates (2.9%) had reduced susceptibility to ceftriaxone/cefotaxime in 2006, which was also lower than the number and proportion of isolates with reduced susceptibility to ceftriaxone/cefotaxime reported in 2005 (44 isolates, 3.8%).

Of the 143 isolates with reduced susceptibility to penicillin, 138 were serotyped. Ninety-three (67.4%) isolates with reduced penicillin susceptibility were serotypes in the 7vPCV and 132 (95.7%) were serotypes in the 23vPPV. Of the penicillin insensitive isolates: 36 were serotype 19A; 31 were serotype 9V; and 26 were serotype 19F, accounting for 67.4% of isolates with reduced penicillin susceptibility and with known serotypes.

**Table 9. *Streptococcus pneumoniae* susceptibility to penicillin and ceftriaxone/cefotaxime, Australia, 2006, by state or territory**

| | | State or territory | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Antibiotic | Description | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Australia |
| Penicillin | Resistant | 0 | 15 | 1 | 12 | 1 | 0 | 3 | 0 | 32 |
| | Intermediate | 1 | 29 | 4 | 26 | 14 | 0 | 21 | 16 | 111 |
| | Sensitive | 17 | 450 | 50 | 220 | 86 | 40 | 237 | 108 | 1,208 |
| | Total tested | 18 | 494 | 55 | 258 | 101 | 40 | 261 | 124 | 1,351 |
| | % reduced susceptibility | 5.6 | 8.9 | 9.1 | 14.7 | 14.9 | 0.0 | 9.2 | 12.9 | 10.6 |
| Ceftriaxone | Resistant | 0 | 6 | 0 | 2 | 0 | 0 | 1 | 0 | 9 |
| | Intermediate | 0 | 11 | 1 | 3 | 2 | 0 | 4 | 0 | 21 |
| | Sensitive | 18 | 348 | 43 | 231 | 40 | 39 | 172 | 125 | 1,016 |
| | Total tested | 18 | 365 | 44 | 236 | 42 | 39 | 177 | 125 | 1,046 |
| | % reduced susceptibility | 0.0 | 4.7 | 2.3 | 2.1 | 4.8 | 0.0 | 2.8 | 0.0 | 2.9 |

Of the 30 isolates with reduced susceptibility to ceftriaxone/cefotaxime in 2006, 27 were serotyped. Twenty-five (92.6%) were serotypes in the 7vPCV and 26 (96.3%) were serotypes in the 23vPPV. There were 13 serotype 19F and 7 9V serotypes which together accounted for 74% of the ceftriaxone/cefotaxime insensitive isolates where serotype was known.

### Vaccination status

Vaccination data were available for 94% of children with IPD aged less than five years and 82% of those aged 65 years or over. Seventy-two per cent of children aged less than five years with a known vaccine history were recorded as having received at least one dose of 7vPCV with 32 developing 7vPCV serotype disease. The 17 vaccine failures (based on those children who had received all the required doses of 7vPCV according to their age at 1st dose) were aged between 10 and 17 months with 19F being the most common serotype (9 cases), four cases due to 6B, two due to 18C and one case due to serotypes 23F and 4. Only one of these children was Indigenous. Three cases had risk factors for IPD disease recorded. There were no deaths among the 7vPCV vaccine failures.

Of the 403 IPD cases reported in adults aged 65 years or over with available vaccine history, 210 had received at least one vaccine. A total of 131 cases developed disease due to a 23vPPV serotype following vaccination within the recommended time frame. Of the 754 cases in persons aged between 5 and 64 years, 396 (53%) had vaccine data available. The majority (314) of these were reported as not vaccinated. A total of 42 cases of 23vPPV serotype disease occurred in persons aged 5–64 years who were fully vaccinated with 23vPPV. A large proportion of vaccine failures in cases of IPD aged more than five years were associated with one or more identified risk factors (Figure 12).

## Discussion

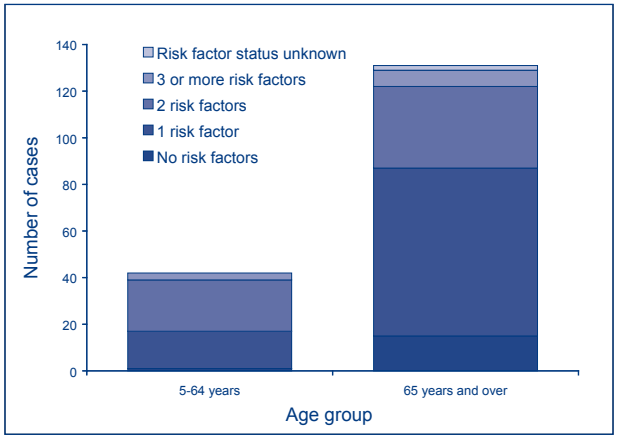
In 2006, rates of IPD due to 7vPCV serotypes continued to fall in all age groups, particularly in those targeted for immunisation. Non-vaccine serotype disease has remained unchanged, specifically in those aged less than two years. Overall rates of IPD with reduced antimicrobial susceptibility have remained low and stable.

Considerations for optimal immunisation schedules for IPD in Australia are ongoing under the auspices of the Australian Technical Advisory Group on Immunisation (ATAGI). ATAGI will examine the optimal 7vPCV schedule for high-risk Indigenous children; examine evidence for various 7vPCV schedules in the first two years of life; review the direct and indirect effects of the 7vPCV immunisation program; review data on the immunologic responses to a primary 7vPCV schedule followed by a 23vPPV boost in infants; and estimate the impact of the 7vPCV with a 23vPPV booster schedule on IPD in eligible Indigenous children. ATAGI's recommendations on the use of 23vPPV in this group are expected to be made early in 2008.

Serotypes associated with disease in populations with high levels of 7vPCV immunisation coverage attracted a lot of attention in 2006/07. In Alaskan Native children, rates of IPD fell between 1995 and 1998 after the introduction of the 7vPCV. This trend was reversed between 2001 and 2006 when the incidence increased by 82%, driven by a 140% increase in disease caused by non-vaccine serotypes. Serotype 19A represented 28% of the non-vaccine type disease. These increases were not seen in non-Native Alaskan children.[1] In Massachusetts, serotype 19A has emerged as a dominant serotype in all children aged less than 18 years, increasing from 10% of serotypes in this age group in 2001/02 to 40% in 2006. While overall rates of IPD fell in the same period by 70%, the emergence of the 19A serotype was significant because of the large proportion in 2006, were resistant to penicillin (61%), ceftriaxone (24%), and other antibiotics.[2,3]

In Australia, disease due to 19A serotype, as well as that due to 7F and 6A in the Indigenous under 5 years population has been minimal with no trends emerging. In the non-Indigenous under five years age group there has been a minimal increase in 19A serotype disease however, with recognition of natural serotype fluctuation over time, continued observation is prudent. Continued comprehensive surveillance of IPD cases will be essential to monitor the serotypes, drug resistance and the prevalence of modifiable risk factors.

**Figure 12. Number of risk factors for cases of vaccine failure over five years, Australia, 2006, by age group**



## Acknowledgements

The contribution of Mark Bartlett, Communicable Diseases Branch, Department of Health, New South Wales and Lisa Hall, Communicable Disease Unit, Queensland is gratefully acknowledged. We thank the following laboratories for their support of Pneumococcal Laboratory Surveillance.

**ACT**

ACT Pathology

**New South Wales**

Central Coast Pathology
CIDM, ICPMR
Concord Hospital
Douglass Hanley Moir
Hunter Area Pathology
Laverty Pathology
Nepean Hospital
PaLMS
Royal Prince Alfred Hospital
SEALS
St George
St Vincents Hospital
SWAPS
Sydney Adventist Hospital
The Children's Hospital, Westmead
THE Pathology
Wollongong Hospital

**Northern Territory**

Alice Springs Hospital
Gove District Hospital
Katherine Hospital
Private laboratories in the Northern Territory
Royal Darwin Hospital, Department of Microbiology
Tennant Creek Hospital

**Queensland**

Communicable Diseases Unit, Brisbane
Private Pathology Laboratories throughout Queensland
Queensland Health Pathology Laboratories and the Microbiology Discipline Working Party
Tropical Public Health Unit, Cairns

**South Australia**

Clinipath Laboratories
Institute for Medical and Veterinary Science Laboratories
South Path Microbiology and Infectious Diseases
The Gribbles Group
Women's and Children's Hospital, Adelaide

**Tasmania**

Launceston General Hospital (Northern Tasmanian Pathology Service)
North West Pathology
Royal Hobart Hospital

**Victoria**

Microbiology Diagnostic Unit, Public Health Laboratory is grateful to the following laboratories who have been identified as having contributed isolates to the reported data-set.

Alfred Hospital Pathology Service
Austin Hospital Pathology Service
Ballarat Health Services (Base campus) Pathology Service
Bendigo Health Pathology Service
Box Hill Hospital Pathology Service
Dorevitch Pathology Mayne Health (Heidelberg)
Echuca Hospital Pathology Service
Forensicare – Victorian Institute of Forensic Medicine
Geelong Hospital Pathology Service (Pathcare)
Gippsland Pathology Service Sale (and Traralgon)
Goulburn Valley Health (Shepparton) Pathology Service
Gribbles Pathology (Melbourne)
Melbourne Pathology
Mildura Base Hospital Pathology Service
Monash Medical Centre (Clayton) Pathology Service
Northern Hospital (Epping) Pathology Service
Royal Childrens Hospital (Parkville) Pathology Service
Royal Melbourne Hospital (Parkville) Pathology Service
Saint Frances Xavier Cabrini Hospital Pathology Service
South West Healthcare (Warnambool) Pathology Service
St John of God Health Care Ballarat Pathology Service
St John of God Health Care Mildura Pathology Service
St Vincents Hospital (Melbourne) Ltd Pathology Service
Wimmera Base Hospital (Horsham) Pathology Service

From the Microbiology Diagnostic Unit, Public Health Laboratory, Ms Janet Strachan contributed to testing, Dr Mark Veitch and Ms Sally Bodenham to data management.

**Western Australia**

We would like to acknowledge the Vaccine Impact Surveillance Network, which is funded by the Meningitis Centre of Western Australia and The Telethon Institute for Child Health Research.

Clinipath Department of Microbiology

Fremantle Hospital Department Microbiology

PathWest

Princess Margaret and King Edward Memorial Hospitals Department Microbiology

Royal Perth Hospital Department Microbiology

St John of God Pathology Department Microbiology

Western Diagnostic Pathology Department Microbiology

## Author details

Paul Roche[1]
Vicki Krause[2]
Heather Cook[2]
Jenny Barralet[3]
David Coleman[4]
Amy Sweeny[3]
James Fielding[5]
Carolien Giele[6]
Robin Gilmour[7]
Ros Holland[8]
Riemke Kampen[9]
Mitchell Brown[10]
Lyn Gilbert[10]
Geoff Hogg[11]
Denise Murphy[12]

1. Surveillance Policy and Systems Section, Department of Health and Ageing, Canberra, Australian Capital Territory
2. Centre for Disease Control, Department of Health and Community Services, Darwin, Northern Territory
3. Communicable Disease Unit, Queensland Health, Brisbane, Queensland
4. Communicable Disease Prevention Unit, Department of Health and Human Services, Hobart, Tasmania
5. Communicable Disease Control Unit, Department of Human Services, Melbourne, Victoria
6. Communicable Disease Control Directorate, Department of Health, Perth, Western Australia
7. Communicable Diseases Branch, Department of Health, Sydney, New South Wales
8. Communicable Disease Control Branch, Department of Health, Adelaide, South Australia
9. Communicable Diseases Control Unit, ACT Health, Canberra, Australian Capital Territory
10. Centre for Infectious Diseases and Microbiology, Institute of Clinical Pathology and Medical Research, Westmead, New South Wales
11. Microbiological Diagnostic Unit, University of Melbourne, Melbourne, Victoria
12. Queensland Health Pathology & Scientific Services, Brisbane, Queensland

Corresponding author: Dr Paul Roche, Surveillance Policy and Systems Section, Office of Health Protection, Department of Health and Ageing, GPO Box 9848 (MDP14), CANBERRA ACT 2601. Telephone +61 2 6289 2708. Facsimile +61 2 6289 2600. Email: paul.roche@health.gov.au

## References

1. Singleton RJ, Hennessey TW, Bulkow LR, Hammitt LL, Zulz T, Hurlburt DA, et al. Invasive pneumococcal disease caused by nonvaccine serotypes among Alaska native children with high levels of 7-valent penumococcal conjugate vaccine coverage. *JAMA* 2007;297:1784–1792.
2. Lett SM, De Maria A, Huot H, Wesson J, Karumuri S, Truong L, et al. Emergence of antimicrobial-resistant serotype 19A *Streptococcus pneumoniae* – Massachusetts, 2001–2006. *MMWR Morb Mortal Wkly Rep* 2007;56:1077–1080.
3. Pelton SI, Huot H, Finkelstein JA, Bishop CJ, Hsu KK, Kellenberg J, et al. Emergence of 19A as virulent and multidrug resistant pneumococcus in Massachusetts following universal immunization of infants with pneumococcal conjugate vaccine. *Medscape* 2007.

# Communicable Diseases Network Australia National Arbovirus and Malaria Advisory Committee annual report, 2006–07

Conan Liu, Kylie Begg, Cheryl Johansen, Peter Whelan, Nina Kurucz, Lorna Melville, and the National Arbovirus and Malaria Advisory Committee

## Abstract

This report describes the epidemiology of mosquito-borne disease in Australia for the mosquito-borne disease season 1 July 2006 to 30 June 2007, which was moderately low compared to previous seasons. Ross River virus (RRV) infections (55%), Barmah Forest virus (BFV) infections (29%) and overseas acquired malaria (11%) were the most common mosquito-borne diseases reported in 2006–07. The number, proportion and rate of national BFV notifications were the second highest on record since 1998–99. The Northern Territory reported the highest BFV notification rate this season. BFV notification rates were the highest in the 40–59 year age groups when compared to other age groups. The number, proportion and rate of RRV notifications were moderately low this season compared with previous seasons. The highest RRV rate was reported by Western Australia from the Kimberley region. The highest age-specific RRV notification rate was observed in the 40–59 year age groups. Locally acquired dengue virus notifications were low this season compared to previous seasons, with a small outbreak of dengue serotype 3 in 39 cases confined to the greater Townsville region. There were 640 notifications of malaria in 2006–07 of which none were reported as locally acquired. This was the third highest number of malaria notifications since 2001. *Plasmodium falciparum* was reported as the infecting species in 47% of the malaria notifications and *Plasmodium vivax* for 40% of cases. Young adolescents and adults in the 15–29 year age group had the highest number of cases accounting for 32% of notifications. Sentinel chicken surveillance data for flaviviruses and sentinel pig surveillance data for Japanese encephalitis virus are also reported. *Commun Dis Intell* 2008;32:31–47.

Keywords: arbovirus; Barmah Forest virus, chikungunya, dengue, disease surveillance; epidemiology, flavivirus, Japanese encephalitis, Kunjin, malaria, mosquitoes, Murray Valley encephalitis virus, Ross River virus, vectorborne disease

## Introduction

This report describes the epidemiology of nationally notifiable mosquito-borne disease in Australia for the season 1 July 2006 to 30 June 2007.

The eight notifiable mosquito-borne diseases under national surveillance include the alphaviruses (Barmah Forest virus and Ross River virus), the flaviviruses (dengue, Japanese encephalitis, Kunjin, Murray Valley encephalitis and flavivirus not elsewhere classified), and malaria.

Alphaviruses are ribonucleic acid (RNA) viruses which cause disease epidemics characterised by fever, rash and polyarthritis. In Australia, Barmah Forest virus (BFV) infection and Ross River virus (RRV) infection are the alphaviruses of major public health significance. There are a variety of mosquito vectors which facilitate the transmission of these viruses in diverse environments (freshwater habitats, coastal regions, salt marshes, floodwaters, established wetlands and urban areas).[1,2] At this time, the alphavirus chikungunya virus has not become established in Australia despite its increased activity in southern Asia and the Indian Ocean over the past year, and its occasional diagnosis in returning travellers.

Flaviviruses are single-stranded RNA viruses, some of which are associated with epidemic encephalitis in various regions of the world. In Australia, the flaviviruses of public health importance are the dengue viruses (DENV) with frequent seasonal outbreaks,[3–6] Japanese encephalitis virus (JEV) with occasional outbreaks,[7–12] and sporadic cases of Murray Valley encephalitis virus (MVEV) or Kunjin virus (KUNV) infections.[13] The International Committee for Taxonomy of Viruses refers to Kunjin as a strain of West Nile virus (WNV)[14] and the Australian Kunjin strains are phylogenetically located in the WNV lineage 1, clade B.[15]

Malaria is caused by infection with a protozoan blood parasite from the genus *Plasmodium* that has been transmitted by a species of mosquito from the genus *Anopheles*. Malaria was eradicated from Australia in 1981 and Australia was certified malaria-free by the World Health Organization in

1983,[16] but the region of northern Australia above 19°S latitude in particular, remains receptive to malaria transmission. Since 1981, malaria acquired in Australia has been rare, but there have been several documented reports of outbreaks[17,18] and sporadic locally acquired cases in Queensland,[19,20] malaria acquired in the Torres Strait,[21] and the artificial induction of malaria by blood transfusion.[22]

## Methods

Eight nationally notifiable mosquito-borne diseases were analysed for the seasonal period 1 July 2006 to 30 June 2007. Historical data from 2001, and in some cases from 1991, are also included for comparison. Data were extracted by diagnosis date from the National Notifiable Diseases Surveillance System (NNDSS) on 21 September 2007 and finalised with state and territory public health surveillance managers.

Epidemic curves by state or territory were produced for each of the eight diseases. Notifiable mosquito-borne disease activity is shown compared with a five-year mean for the same period, by jurisdiction. The rolling monthly mean was calculated for the mosquito-borne disease activity for the equivalent month over five years. The number of notifications and annual or annualised notification rates for locally acquired mosquito-borne disease were calculated using the December 2006 population estimates from the Australian Bureau of Statistics (ABS), for each year. Age– and sex-specific notification rates were calculated using age and sex population estimates for each jurisdiction.

A survey was conducted with state and territory surveillance managers and public health laboratories about imported cases of chikungunya virus infections during 2006–2007.

The geographical distribution of selected diseases was mapped using ArcGIS (ESRI, Redlands, CA, USA). Maps were based on the postcode of residence of each notification aggregated to the appropriate Statistical Division, and rates were calculated using the number of notifications (numerator) divided by the estimated 2006 ABS populations for each division (denominator).

Sentinel chicken surveillance data for flaviviruses and sentinel pig surveillance data for Japanese encephalitis virus are reported.

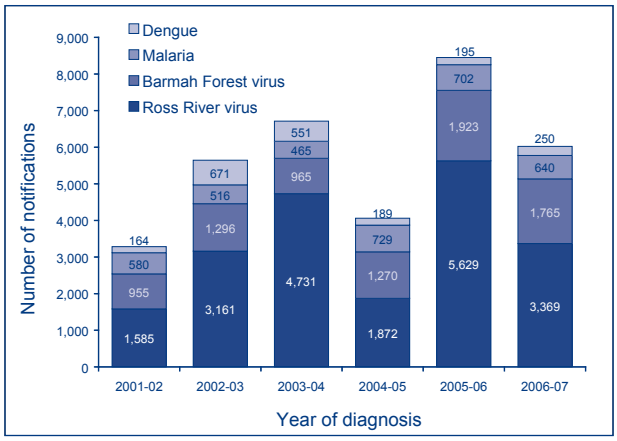
## Results

### Alphaviruses

During this reporting period, there were 5,134 alphavirus notifications (BFV and RRV) reported in Australia, which was a moderate season when compared with other seasons since 1995–96 (mean: 4,933, range: 612–8,422 notifications). The highest alphavirus season on record was observed in 1995–96. During 2005–06, Australia experienced the second highest alphavirus season on record with 7,552 notifications.

During 2006–07, RRV infections accounted for 55% (n=3,369) of notifications (Figure 1), which as a proportion of total mosquito-borne disease activity is moderately low when compared to previous seasons (range: 47–73%). The number and proportion of BFV notifications (n=1,765, 29%) were the second highest on record since 1998–99 and the BFV notification activity for 2006–07 was 1.4 times the five-year mean.

**Figure 1. Notifications of select mosquito-borne diseases, Australia, 1 July 2001 to 30 June 2007, by season of onset**



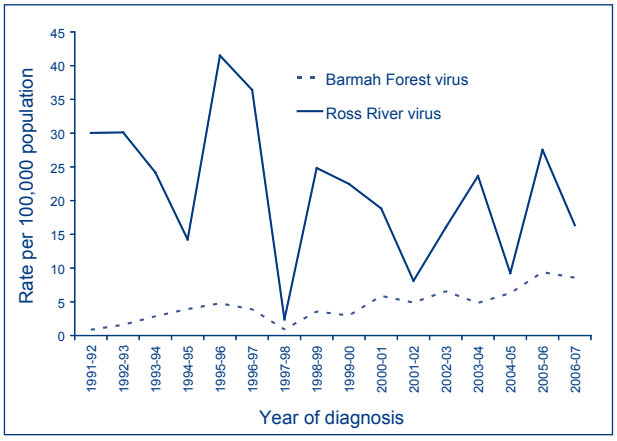

The crude annual BFV notification rate in 2006–07 of 8.6 cases per 100,000 population was the second highest notification rate since reliable reporting of this disease commenced in 1991 (Figure 2). The crude annual RRV notification rate was moderately low in 2006–07 (16.4 cases per 100,000 population) when compared to 2005–06 and other seasons in which there was significantly higher epidemic activity (1991–93, 1995–97, 1998–00, 2003–04).

### Barmah Forest virus infections

Figure 3 shows the epidemic curves of BFV notifications by month and season since 2002–03 with a rolling mean of BFV activity for the same month over five years. During 2006–07, there were 1,765 BFV notifications, the majority of which were reported by Queensland (n=875, 50%) and New South Wales (n=590, 33%). The peak number of notifications was observed in April 2007 (n=245) and was smaller than the previous peak month in 2003 and 2006. The most striking feature of the BFV season in 2006–07 was the unusually high and sig-

nificant notification activity occurring outside of the peak seasonal months, with notifications exceeding two standard deviations above the five year monthly rolling mean from July to October 2006. New South Wales, the Northern Territory, Queensland, and South Australia notified the majority of activity from June to October 2006, with between 25%–47% of their jurisdictional total for the season reported during this quarter. One explanation is that the laboratory-based notifications diagnosed from July to September 2006 may be the result of late onset or late presentations from possible exposure during the epidemic peak months in February–May 2006. Another possible explanation may be that climatic conditions were favourable for earlier than usual breeding of vectors leading to an earlier period for transmission.

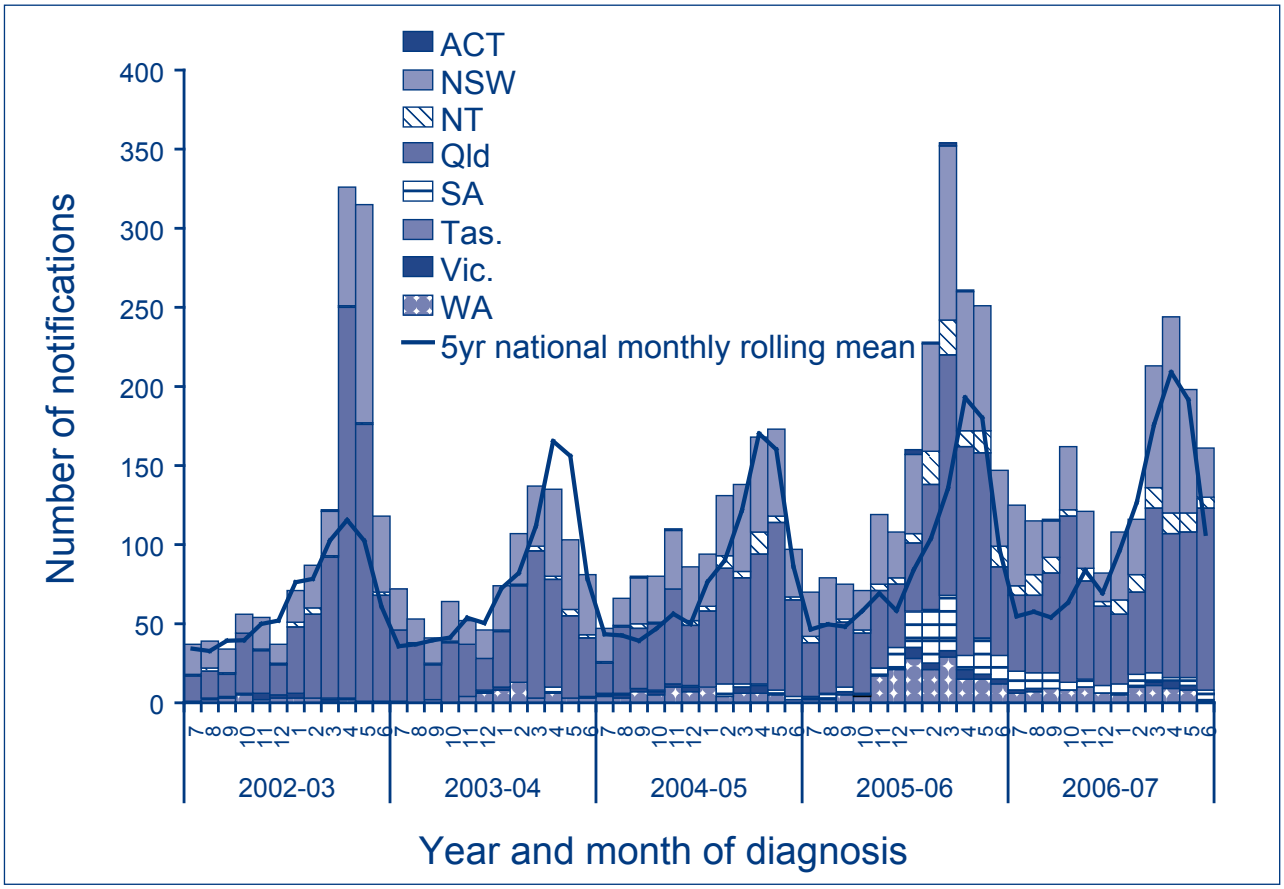
**Figure 2. Crude annual notification rates for Barmah Forest virus and Ross River virus, Australia, 1 July 1991 to 30 June 2007, by season of diagnosis**




During 2006–07, all jurisdictions except Tasmania and Victoria reported BFV activity above the five-year mean (Figure 4). The Northern Territory and South Australia notified more than two times the five-year mean and notifications from these jurisdictions have been increasing since the 2004–05 season (Figure 3).

Table 1 and Map 1 show that the highest rates of BFV notifications in Statistical Divisions during 2006–07 were reported from the Northern Territory (74.4 per 100,000 population), which excludes Darwin, Palmerston and Darwin rural areas (as there are only two Statistical Divisions in the Northern Territory). There is a higher rate of alphavirus notification in the Darwin rural area when compared with Darwin, with Palmerston having an intermediate rate (Peter Whelan, personal communication).

**Figure 3. Epidemic curves of Barmah Forest virus infection notifications, Australia, 1 July 2002 to 30 June 2007, by month and season of diagnosis**



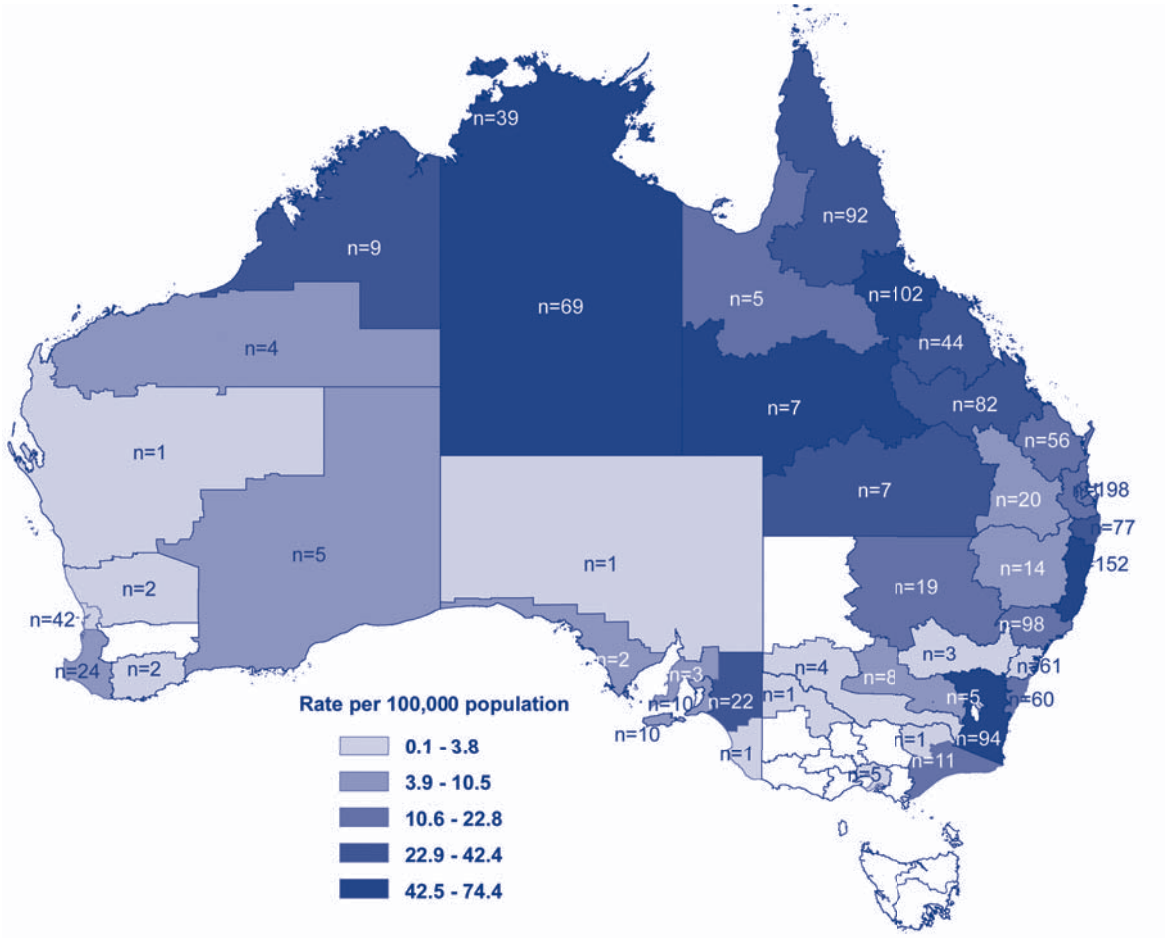
Moderately high rates of BFV notification in Australia were reported from the Central West and Northern Statistical Divisions in Queensland, and the Mid-North Coast Statistical Division in New South Wales. The second highest BFV notification rate in New South Wales (45.9 notifications per 100,000 population) was reported in the South Eastern Statistical Division, and this was linked to

**Table 1. Highest notification rates of Barmah Forest virus infection in select jurisdictions, Australia, 1 July 2006 to 30 June 2007, by Statistical Division of residence**

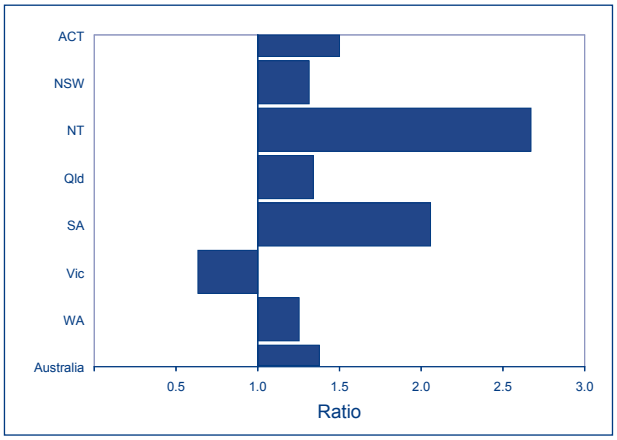
| Notifying jurisdiction | Statistical Division | Rate (per 100,000) | Notifications (n) | ABS population estimate |
|---|---|---|---|---|
| Northern Territory | Northern Territory* | 74.4 | 69 | 92,733 |
| Queensland | Central West | 57.6 | 7 | 12,155 |
| New South Wales | Mid-North Coast | 51.1 | 152 | 297,409 |
| Queensland | Northern | 48.4 | 102 | 210,943 |
| New South Wales | South Eastern | 45.9 | 94 | 204,854 |
| Queensland | Fitzroy | 42.4 | 82 | 193,182 |
| Queensland | Far North | 37.7 | 92 | 243,948 |
| Northern Territory | Darwin | 34.2 | 39 | 113,955 |
| New South Wales | Richmond-Tweed | 33.8 | 77 | 227,815 |
| South Australia | Murray Lands | 31.9 | 22 | 69,066 |
| Victoria | East Gippsland | 13.1 | 11 | 84,222 |

* Excludes Darwin, Palmerston and Darwin rural area.

**Map 1. Notifications and notification rates of Barmah Forest virus infection, Australia, 1 July 2006 to 30 June 07, by Statistical Division of residence**



### Figure 4. Ratio of Barmah Forest virus infection notifications to mean of previous five years, Australia, 1 July 2006 to 30 June 2007, by state or territory*

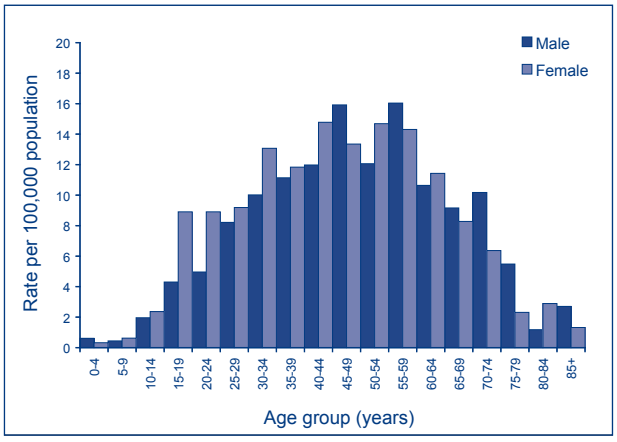



* Note: Tasmania not shown with only one Barmah Forest virus infection notification in 2005–06.

the largest documented outbreak of BFV along the South Coast, particularly the Eurobodalla region, which peaked in April 2007.[23]

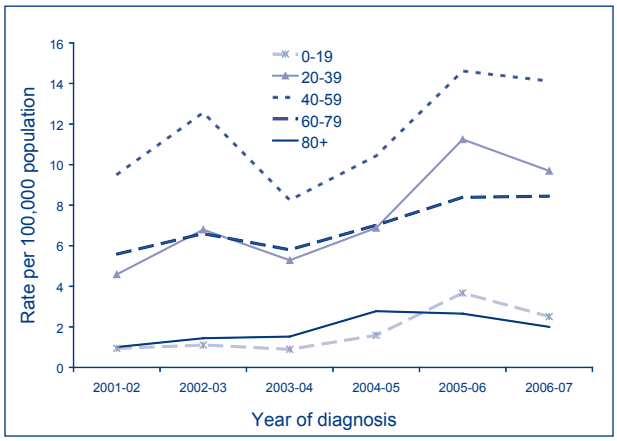
The national notification rate of BFV was highest in the 55–59 year age group (Figure 5). The highest notification rates in males were in the 55–59 year age group (16.5 cases per 100,000 population) and females in the 40–44 year age group (14.8 cases per 100,000 population).

### Figure 5. Notification rate for Barmah Forest virus infections, Australia, 1 July 2006 to 30 June 2007, by age group and sex




With the exception of the 60–79 year age group, the age-specific rates of BFV notifications in 2006–07 decreased slightly when compared to 2005–06 (Figure 6). The BFV age-specific notification rates for all age groups in 2006–07 were still above the five-year mean.

### Figure 6. Trends in Barmah Forest virus infection notification rates, Australia, 1 July 2001 to 30 June 2007, by age group




## Ross River virus infections

During 2006–07, Australia experienced a moderately low RRV season (n=3,369) when compared to the previous five seasons (mean=3,395), similar to the low activity observed during the 2004–05 season. The majority of RRV notifications during 2006–07 were reported by Queensland (n=1,753, 52%), New South Wales (n=674, 20%) and Western Australia (n=397, 12%).

The monthly notifications of RRV were above the five-year monthly rolling mean at the start of the season in October and November 2006, but the numbers of notifications dropped well below the five-year monthly mean from December 2006 to April 2007 (Figure 7).

In contrast to last season, only four jurisdictions (the Australian Capital Territory, New South Wales, the Northern Territory and South Australia) reported RRV activity above the five-year mean for their jurisdiction (Figure 8).

Table 2 and Map 2 show that the highest rates of RRV notifications in Australia during 2006–07 were reported from the Kimberley region of Western Australia (242.6 notifications per 100,000 population), the Northern Territory (170.4 notifications per 100,000 population) and the Central West Statistical Division in Queensland (156.3 notifications per 100,000 population). Moderately high rates of RRV notification were reported in the Mackay, Far North and North West Statistical Divisions in Queensland, and the Wide Bay Burnett, North Western and Mid-North Coast Statistical Divisions in New South Wales.

## Table 2. Highest notification rates of Ross River virus infection in select jurisdictions, Australia, 1 July 2006 to 30 June 2007, by Statistical Division of residence

| Notifying jurisdiction | Statistical Division | Rate (per 100,000) | Notifications (n) | ABS population estimate |
|---|---|---|---|---|
| Western Australia | Kimberley | 242.6 | 87 | 35,865 |
| Queensland | South West | 191.9 | 52 | 27,095 |
| Northern Territory | Northern Territory* | 170.4 | 158 | 92,733 |
| Queensland | Central West | 156.3 | 19 | 12,155 |
| Queensland | Northern | 147.9 | 312 | 210,943 |
| Queensland | Fitzroy | 102.0 | 197 | 193,182 |
| Northern Territory | Darwin | 92.1 | 105 | 113,955 |
| Queensland | Mackay | 71.9 | 109 | 151,572 |
| Queensland | North West | 66.6 | 23 | 34,558 |
| New South Wales | North Western | 63.7 | 76 | 119,276 |
| Queensland | Far North | 57.8 | 141 | 243,948 |
| New South Wales | Mid-North Coast | 55.5 | 165 | 297,409 |
| Queensland | Wide Bay-Burnett | 44.7 | 118 | 264,201 |
| South Australia | Murray Lands | 40.5 | 28 | 69,066 |
| Victoria | Mallee | 22.5 | 21 | 93,415 |

* Excludes Darwin, Palmerston and Darwin rural area.

## Map 2. Notifications and notification rates of Ross River virus infections, Australia, 2006–07, by Statistical Division of residence

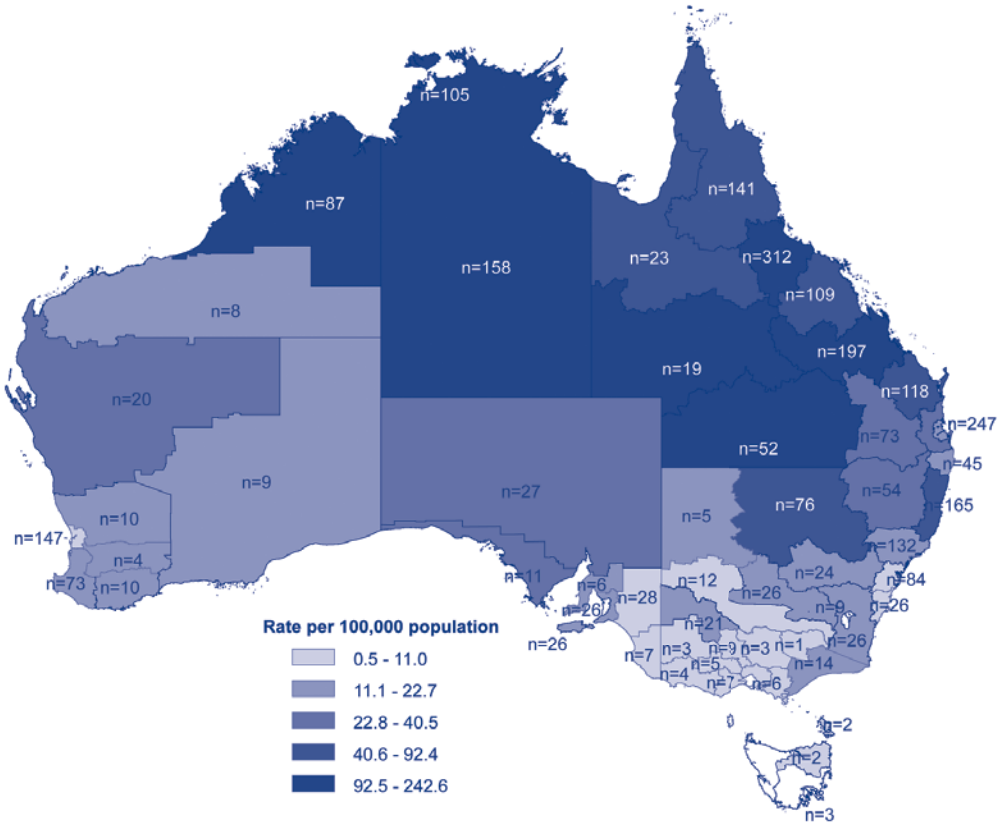


**Figure 7. Epidemic curves of Ross River virus infection notifications, Australia, 1 July 2002 to 30 June 2007, by month and season of diagnosis**

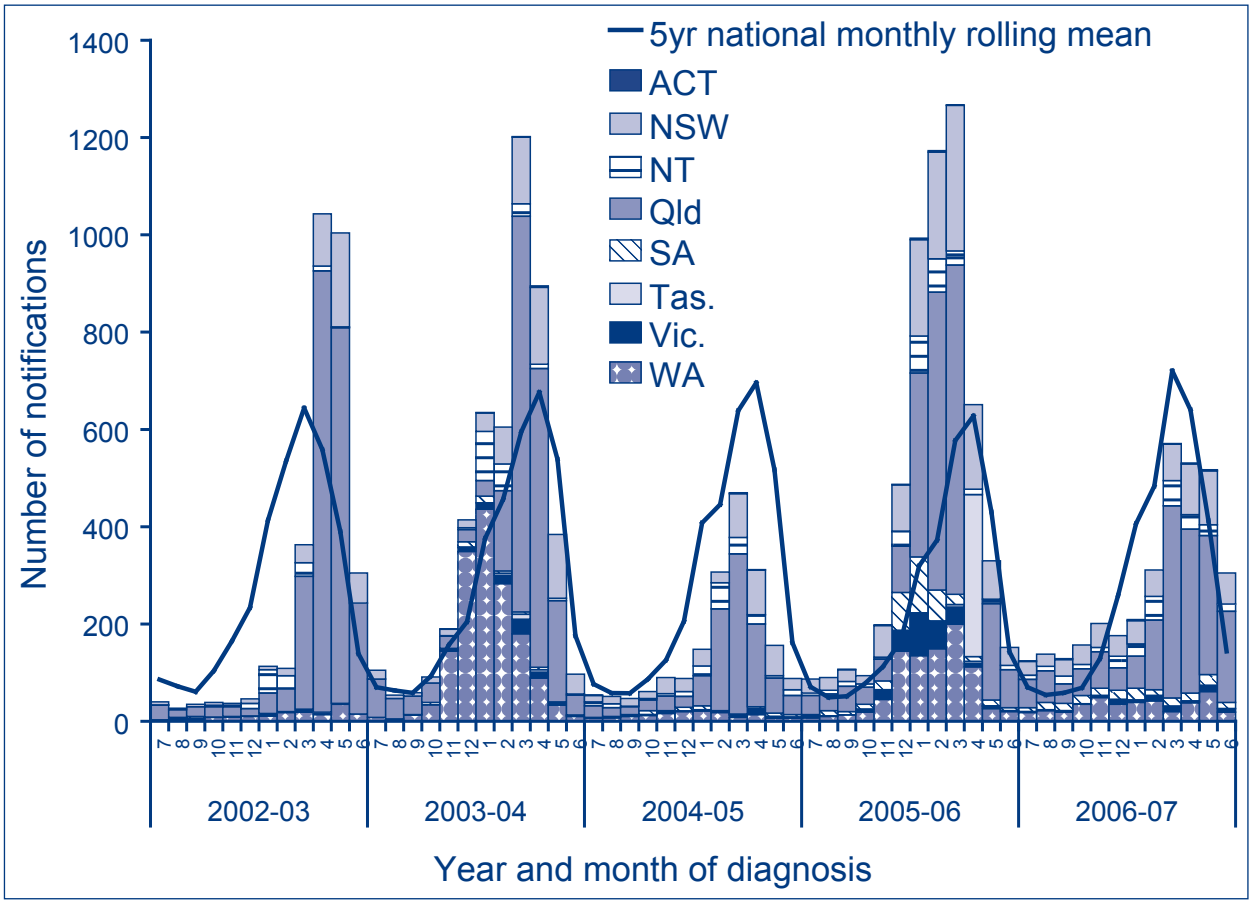



**Figure 8. Ratio of Ross River virus infection notifications to mean of previous five years, Australia, 1 July 2006 to 30 June 2007, by state or territory**

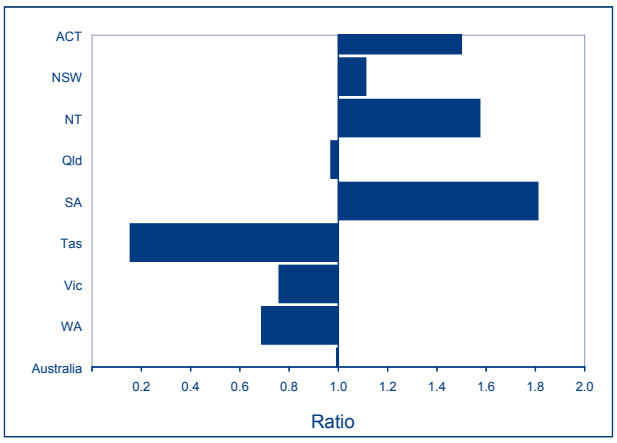
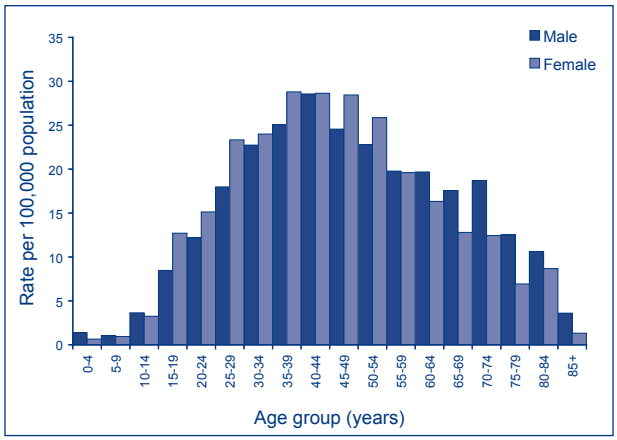


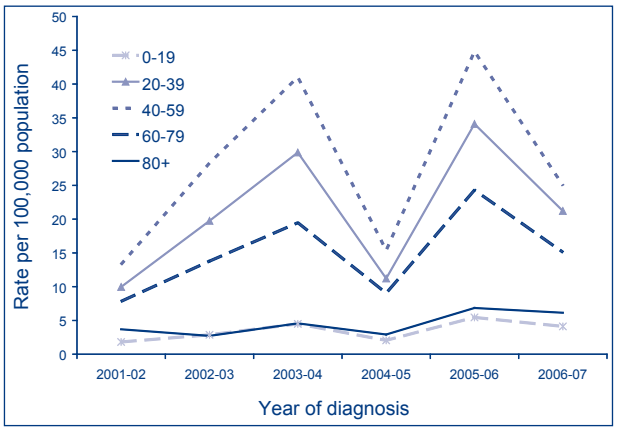

**Figure 9. Notification rate for Ross River virus infections, Australia, 1 July 2006 to 30 June 2007, by age group and sex**




The rate of national notifications for RRV was highest in the 40–44 year age group (Figure 9). Females in the 35–39 year age group (28.6 cases per 100,000 population) and males in 40–44 year age group (28.6 cases per 100,000 population) had the highest national age– and sex-specific notification rates.

The age-specific notification rates for RRV were lower for all age groups when compared to last season. The overall pattern of the susceptible age groups has not changed, with the 20–39 and 40–59 age groups recording the highest age-specific notification rates (Figure 10).

**Figure 10. Trends in Ross River virus infections notification rates, Australia, 1 July 2001 to 30 June 2007, by age group**




## Chikungunya virus infections

In January and March 2005 there was a large epidemic of chikungunya virus infection on Comoros and Reunion Island, in the south-west Indian Ocean. Chikungunya virus subsequently spread to Mauritius, the Seychelles and Madagascar in 2006. Epidemics were also reported in India during 2005–2007 with well over a million cases from 13 states. The largest outbreaks were reported from Kerala state. Epidemic activity also occurred in Sri Lanka and the Maldives in 2006. At the time of writing there have been several large outbreaks reported from Indonesia and Malaysia.

The Northern Territory reported the first known imported cases of chikungunya virus infection (n=2) in 2004 (Peter Markey, personal communication), one of which was reported in a 30-year-old woman who most probably acquired the infection in East Timor.[24] Table 3 shows that there have been at least 30 cases of imported chikungunya infections diagnosed in Australia since 2006, with the majority detected by PathWest Laboratory Medicine, (Western Australia) (n=14); the Institute of Clinical Pathology and Medical Research, and Westmead Hospital, (New South Wales) (n=7); and the Victorian Infectious Diseases Reference Laboratory.[25] The majority of chikungunya virus infections were acquired in Sri Lanka (n=10), India (n=7) and Mauritius (n=6). There were no reports of cases imported from Indonesia during 2006–2007.

The National Arbovirus and Malaria Advisory Committee has recently developed a provisional national case definition for chikungunya virus infection (Appendix), which is pending endorsement from the Communicable Diseases Network Australia and Public Health Laboratory Network. It is important to note that at the time of writing, chikungunya is not nationally notifiable but is notifiable in New South Wales and the Northern Territory under the disease category arbovirus not elsewhere classified. The provisional case definition attempts to distinguish past and recent chikungunya infections from endemic Australian alphaviruses such as Barmah Forest virus and Ross River virus. As chikungunya is most closely related to the o'nyong-nyong virus and is a member of the Semliki Forest antigenic complex,[26] it is important to be aware of false-positive reactions with other arboviruses, particularly when travellers may be arriving from countries with endemic alphaviruses.

## Flaviviruses

Table 4 shows human notifications of flavivirus infections from 1 July 2006 to 30 June 2007, by state or territory. There were 276 notifications of human

**Table 3. Number of imported chikungunya cases to Australia (n=30), 2006–2007, by state or territory**

| Notifying jurisdiction | Cases | Year of diagnosis | Laboratory tests | Source | Country of acquisition |
|---|---|---|---|---|---|
| Western Australia | 14 | 2006 (n=9)<br>2007 (n=5) | High HI, IgM pos (9), high rise HI, IgM pos (3), IgM only (2) | David Smith, PathWest Laboratory | India (4), Mauritius (3), Sri Lanka (2), China (1), overseas (2), unknown (2) |
| Victoria | 8 | 2006 (n=5), 2007 (n=3) | PCR, then sequencing | Johnson et al,[25] Druce et al,[27] Liu et al,[28] James Fielding DHS | Sri Lanka (6), Mauritius (1), India (1) |
| New South Wales | 7 | 2006 (n=4)<br>2007 (n=3) | Culture negative, negative for BFV, RRV, DENV, flavivirus by ELISA and neutralisation, IgM pos for chikungunya by IFA, high neutralisation titres and clinical histories | Linda Hueston, ICPMR | Mauritis (2) Seychelles (2) India (1) Sri Lanka (2) |
| Northern Territory | 1 | 2007 (n=1) | DENV negative, flavivirus and chikungunya positive | Peter Markey, NT CDC DHCS | India (1) |

flavivirus infection during 2006–07, the majority of which were locally acquired and imported DENV notifications.

The Sentinel Chicken Programme is a surveillance network involving New South Wales, the Northern Territory, Victoria and Western Australia, and is designed to detect flavivirus activity (including the endemic arboviruses MVEV and KUNV).[29]

**Table 4. Number of flavivirus notifications, 1 July 2006 to 30 June 2007, Australia, by state or territory**

| State or territory | Notifications | | | |
|---|---|---|---|---|
| | DENV | Flavi NEC | KUNV | MVEV |
| ACT | 2 | 0 | 0 | 0 |
| NSW | 72 | 0 | 0 | 0 |
| NT | 15 | 0 | 0 | 0 |
| Qld | 113 | 22 | 0 | 0 |
| SA | 12 | 0 | 0 | 0 |
| Tas | 0 | 0 | 0 | 0 |
| Vic | 8 | 3 | 0 | 0 |
| WA | 28 | 1 | 0 | 0 |
| Australia | 250 | 26 | 0 | 0 |

*Northern Territory*

The Northern Territory sentinel chicken program commenced in January 1992 and replaced an earlier program run by the Australian Quarantine and Inspection Service (AQIS). Sentinel chicken flocks in the Northern Territory are maintained, bled and analysed for flavivirus infection in a combined program between the Northern Territory Department of Health and Community Services, the Northern Territory Department of Primary Industry, Fisheries and Mines (DPIFM), and volunteers.

Sentinel chicken flocks are presently at Darwin urban (Leanyer), Darwin rural (Howard Springs), Beatrice Hill (Coastal Plains Research Station), Kakadu (Jabiru), Katherine, Nhulunbuy, Tennant Creek, Alyangula, Nathan River and Alice Springs (Ilparpa and Arid Zone Research Station).

DPIFM officers or volunteers usually bleed flocks once a month and the samples are sent to the DPIFM for specific testing for MVEV and KUNV. Sometimes for operational reasons, chickens are not bled during a schedule month and hence seroconversion shown in the next bleed could have occurred in the previous month. When chickens from a flock show new antibodies to MVEV during a prime risk period, a media warning is issued for the region for the risk period. These warnings advise the public of the need to take added precautions to avoid mosquito bites. Chickens are replaced at least annually and more frequently if birds die or if a large proportion seroconvert. They are well positioned to detect flavivirus activity near the principal towns of the Northern Territory and hence provide timely and accurate indication of risk to people in those towns.

In the 2006–07 season, MVEV activity was detected in Howard Springs in November, Adelaide River in June, Nhulunbuy in October and February, Katherine in February and May, Tennant Creek in April and Nathan River in May. The MVEV total seroconversions this season (n=11) was slightly less compared to last season (n=15), with most seroconversions this reporting period (n=3) occurring in Katherine and Nathan River, followed by the Nhulunbuy flock (n=2). Most seroconversion this season occurred in May (n=4), which is the month when the long-term seroconversion peak occurs, followed by February (n=3).

There were no seroconversions in the two Alice Springs flocks, most probably due to low seasonal vector numbers. In addition, the successful effluent swamp drainage and better effluent management from nearby sewage facilities in the Ilparpa area, have led to an overall reduction in vector numbers near the Alice Springs outskirts during summer. There were also no seroconversions in the Leanyer and the Alyangula flock. However, the Alyangula flock was last bled in February 2007, due to operational issues.

No human cases of MVEV disease were reported in the Northern Territory in 2006–07 and the last reported case was in March 2005, when a 3-year-old boy from a community in Arnhem Land had a relatively mild illness and made a complete recovery.

Kunjin virus activity was present throughout the Northern Territory, with seroconversions to KUNV in Darwin (Howard Springs) in June; Darwin (Leanyer) in April, May and June, Adelaide River in June; Nhulunbuy in October; Katherine in August, February, March and May; Tennant Creek in April; Jabiru in May and June; and Nathan River in August. The virus activity from July to October is probably a result of activity extending from the last arbovirus season rather than an indication of early activity this season. There has been a trend over the last 10 years to increasing numbers of seroconversions to KUNV, with this season's total (n=24) nearly double the number from last season (n=13) and the highest since the program started in 1992. Most seroconversions occurred in the Leanyer (n=6), Katherine (n=6) and Jabiru (n=5) flocks. Seroconversions mostly occurred this season in

May (n=9) and June (n=6), while the long term peak is also in May in the Northern Territory. The high number of seroconversions to KUNV was most likely due to significant late wet season rain in the Top End, leading to an extended period of relatively high *Culex* vector numbers.

The Northern Territory did not report any human cases of KUNV infection this season. The last reported KUNV case from the Northern Territory was in a 23-year-old female from Alice Springs in May 2001.

*Western Australia*

Sentinel chicken flocks in Western Australia are maintained, bled and analysed for specific antibodies to MVEV and KUNV in a combined program between The University of Western Australia, the Western Australian Department of Health (WA DOH), local governments and community volunteers. Twenty-eight sentinel chicken flocks are located at major towns and communities in the Kimberley, Pilbara, Gascoyne, Goldfields, Midwest and Central Coastal regions of Western Australia. Environmental health officers or trained volunteers take blood samples from the chickens each fortnight from December to June (the major MVEV 'risk' season) and monthly at other times. Samples are tested by the Arbovirus Surveillance and Research Laboratory at The University of Western Australia.[30] Sometimes for operational reasons, chickens are not bled fortnightly and a seroconversion detected in the next bleed may have occurred earlier.

With the exception of August, rainfall was generally above average and temperatures were usually average to above average in northern Western Australia between July and December 2006. Seasonal monsoonal rains occurred in January. The monsoonal period quietened in February before becoming active again in March and April, and a series of tropical cyclones also caused heavy widespread rainfall and flooding in the Kimberley, Pilbara and Gascoyne regions. Heavy rainfall in north-eastern parts of the Kimberley region continued into the late wet season in May and June.

A total of 3,405 serum samples from the 27 flocks located in Western Australia were screened for antibodies to flaviviruses during 2006–07. Seroconversions to flaviviruses were detected in 68 (2.0%) of the samples. Thirty-six seroconversions detected between July and September 2006 were associated with a prolonged period of activity extending from the 2005–06 wet season, possibly due to continued rainfall in northern Western Australia in July 2006. The majority of these seroconversions were due to MVEV activity (n=22), and to a lesser degree KUNV activity (n=7).

The first activity associated with the 2006–07 wet season was detected in January when KUNV was simultaneously detected at Kununurra in the north-east Kimberley region and in the Ophthalmia sentinel chicken flock near Newman in the Pilbara region. MVEV activity was first detected in April at Wyndham and Halls Creek in the north-east and south-east Kimberley region, respectively, and in the sentinel chicken flock at Marble Bar in the north-east Pilbara region in June 2007. A total of 32 flavivirus seroconversions were associated with the 2006–07 wet season. Most of these flavivirus seroconversions were due to KUNV activity (56%), whilst MVEV accounted for 25% of the flavivirus seroconversions. Seroconversions continued in Kimberley and Pilbara sentinel chicken flocks beyond June 2007, possibly facilitated by continued late wet season rainfall in northern Western Australia in May and June 2007. No flavivirus activity was detected south of Newman in the Pilbara region in 2006–07. The level of MVEV activity was substantially lower than the previous year, however the level of activity of KUNV in sentinel chickens was greater, particularly in the Kimberley region. Unidentified flavivirus infections were detected at several locations in the Kimberley and Pilbara regions. These are possibly due to activity of other flaviviruses that are occasionally isolated from mosquitoes collected in northern Western Australia.

The WA DOH initially issued health warnings of increased risk of KUNV to residents and visitors to northern Western Australia on 15 February 2007, following seroconversions to KUNV in the east Kimberley and east Pilbara regions. An additional warning was issued on 3 May 2007 advising residents and travellers to the north-east Kimberley region of the increased risk of MVEV after seroconversions to MVEV were detected in sentinel flocks in this region. A third warning was issued on 25 June 2007 following continued detections of MVEV and KUNV in the Kimberley and east Pilbara regions, including the first activity for the season at Marble Bar. No locally-acquired flavivirus human cases were reported from Western Australia during the 2006–07 season.

*New South Wales*

Samples from four sentinel chicken sites were tested weekly for KUNV and MVEV antibodies in New South Wales over a six month period in 2006–07.[23] There were no seroconversions to MVEV or KUNV during this period. There were no human cases reported from New South Wales for either MVEV or KUNV. The last reported case of KUNV from New South Wales was notified in May 2001 in a 58-year-old female from Griffith. There have been no recorded cases of MVEV to date in NNDSS from New South Wales.

*Victoria*

Samples from sentinel chicken flocks located throughout northern inland Victoria (10 sites along the Murray River, Map 3) were tested weekly for flavivirus antibodies from 6 November 2006 to early March 2007. No KUNV or MVEV activity was detected in any of the samples. There were no human cases of KUNV or MVEV reported from Victoria during 2006–07. The last reported case of KUNV infection in Victoria was in October 2004. There have been no recorded cases of MVEV in NNDSS from Victoria.

*Queensland*

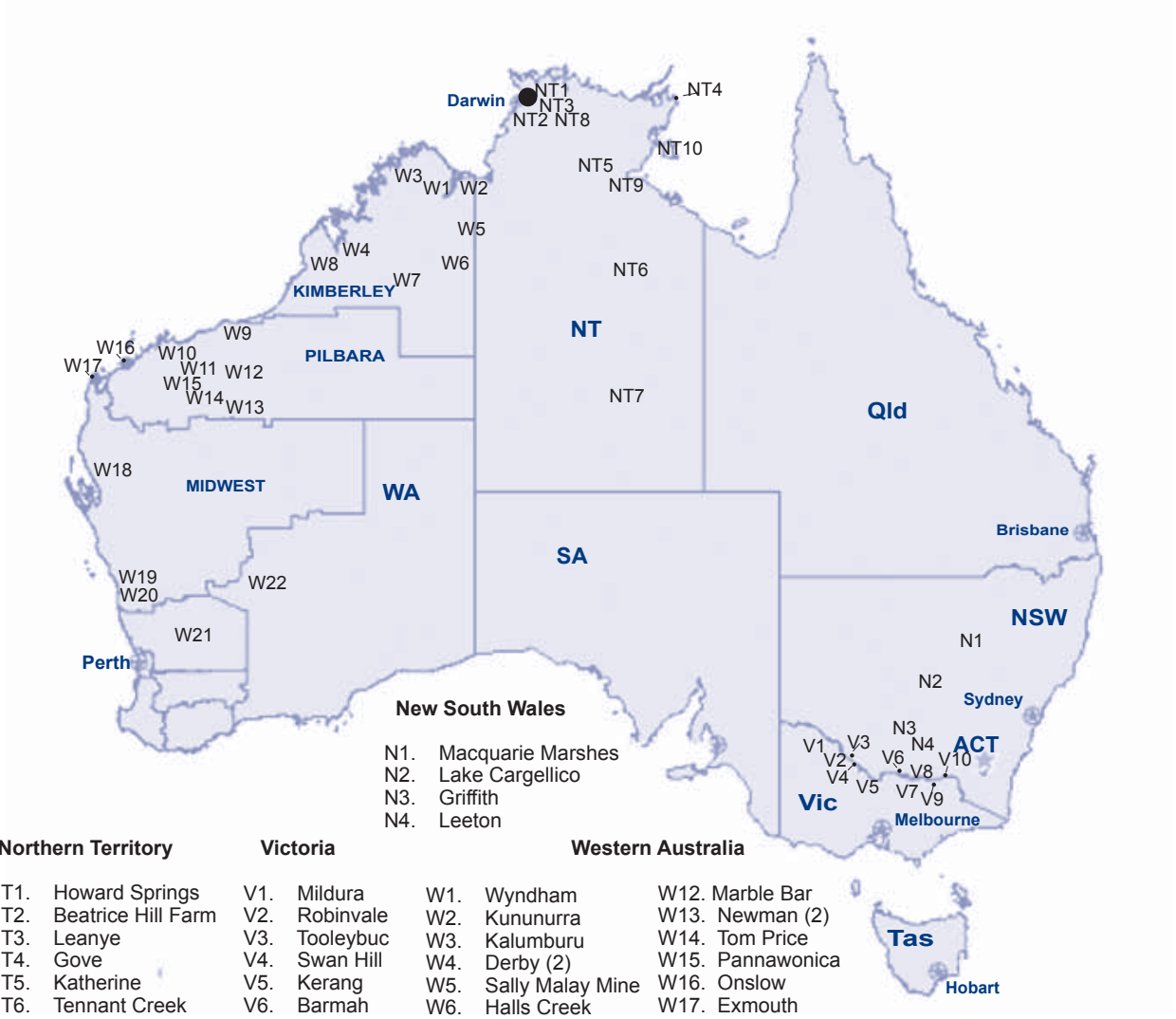
There were no sentinel chicken flocks in Queensland during 2006–07 although flocks were maintained in 2002–03. There were no cases of KUNV or MVEV reported by Queensland during 2006–07. The last reported cases of KUNV from Queensland were three sporadic cases notified in July 2004, December 2004, and February 2005. The last reported MVEV case from Queensland was in a 3-year-old boy from Mount Isa in 2001.

**Japanese encephalitis virus infections**

Japanese encephalitis virus appears nearly annually in the Torres Strait in far northern Queensland and surveillance has involved the use of sentinel pigs that develop detectable viraemia and antibody titres to JEV.

AQIS, through its Northern Australia Quarantine Strategy program, conducted monitoring for JEV for the 2007 wet season using sentinel pigs at Injinoo airport, Northern Peninsula Area, Cape York. The five sentinel pigs did not seroconvert

## Map 3. Sentinel chicken testing sites, Australia 2006–07



and there was no evidence of transmission of JEV to the mainland in 2007 (based on results of testing at Queensland Health Scientific Services and the CSIRO Australian Animal Health Laboratory).

There were no human cases of JEV in Australia during 2006–07. The last reported JEV case was in February 2004, when Queensland Health notified that a 66-year-old male acquired JEV from Papua New Guinea. There have been nine other cases of JEV reported to NNDSS since 1995, although JEV was not nationally notifiable until 2001. Four of these notifications were reported in Torres Strait Islanders from the Badu Island community, two of which were fatal (1995). Another locally-acquired JEV case was reported in a resident from the mouth of the Mitchell River, Cape York Peninsula, Queensland in 1998. The remaining four cases were reported as acquired from overseas countries.

### Flavivirus infections (not elsewhere classified)

There were 26 flavivirus (not elsewhere classified or NEC) notifications during the 2006–07 season, the majority of which were reported by Queensland. Of the 26 flavivirus NEC notifications, five were due to Kokobera infection with the virus unidentified for the remaining 21 notifications. The importation status was reported in four notifications with two acquired in Australia, and one each from Uruguay and Indonesia. The country of acquisition was not reported for the remaining 22 notifications.

### Dengue virus infections

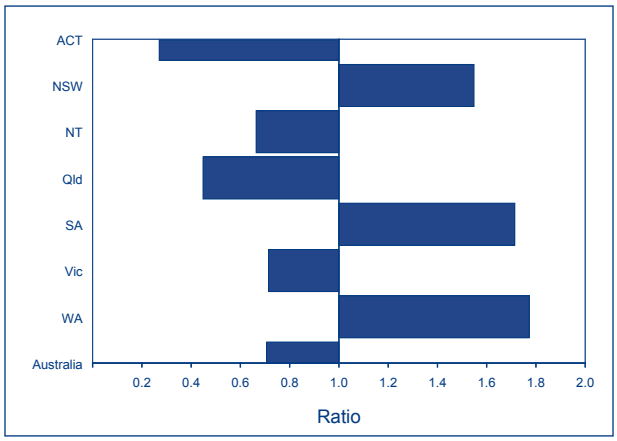
There were 250 notifications of DENV infections during the 2006–07 season. Table 5 shows that the cases were mainly from Queensland (n=113, 45%), New South Wales (n=72, 29%) and Western Australia (n=28, 11%).

**Table 5. Number of dengue notifications, Australia, 1 July 2006 to 30 June 2007, by state or territory**

| Notifying jurisdiction | n | % of total |
|---|---|---|
| ACT | 2 | 1 |
| NSW | 72 | 29 |
| NT | 15 | 6 |
| Qld | 113 | 45 |
| SA | 12 | 5 |
| Tas. | 0 | 0 |
| Vic. | 8 | 3 |
| WA | 28 | 11 |
| Total | 250 | 100 |

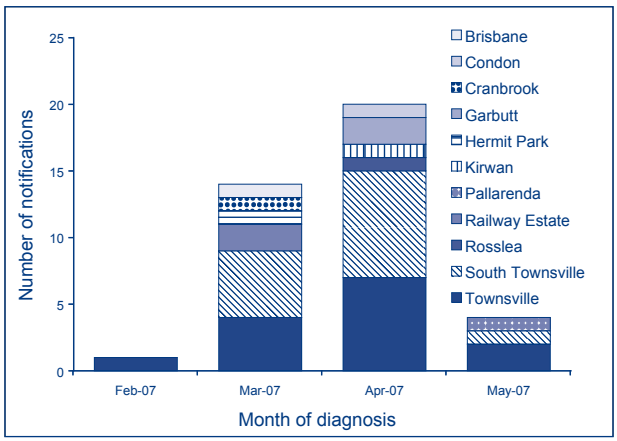
Notifications from New South Wales, South Australia, Victoria and Western Australia exceeded the five-year mean in each jurisdiction (Figure 11), reflecting an increase in imported cases of dengue virus from overseas countries or from Queensland.

**Figure 11. Ratio of dengue virus infection notifications to mean of previous five years, Australia, 1 July 2006 to 30 June 2007, by state or territory**




Queensland reported that of the 113 DENV notifications, only 39 were acquired locally. Figure 12 shows that the local outbreak of DENV this season began in February and peaked in April 2007. The notifications were reported in residents mainly from the Townsville area. One resident of Brisbane acquired the infection in Townsville (Christine Selvey, personal communication.) The DENV serotype for this outbreak was Type 3 in 67% of notifications (n=26 of 39) and the serotype was not typed in the remaining 13 cases.

**Figure 12. Epidemic curve of locally-acquired dengue notifications (n=39), 1 July 2006 to 30 June 2007, by month of diagnosis and residential location**



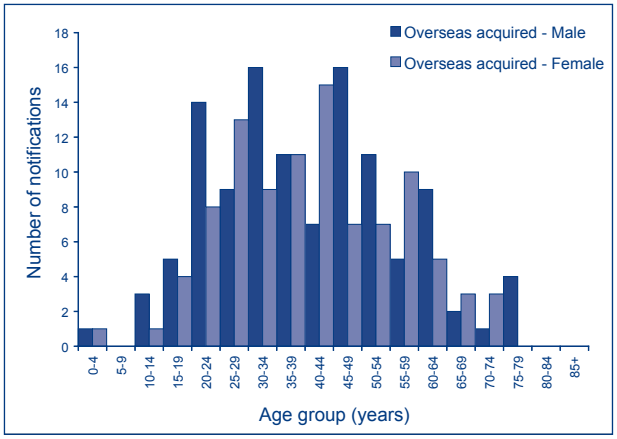
For DENV notifications which were acquired overseas (Table 6), the country of acquisition was reported to NNDSS in 20% of notifications (n=42 of 211). Indonesia, the Philippines, and Thailand were the most frequently reported country in which DENV infection was acquired. The most commonly reported dengue serotype acquired overseas was Type 1 (n=25), Type 2 and 3 (n=16 each). The dengue serotype was not supplied in 72% of the overseas-acquired dengue notifications (n=152 of 211).

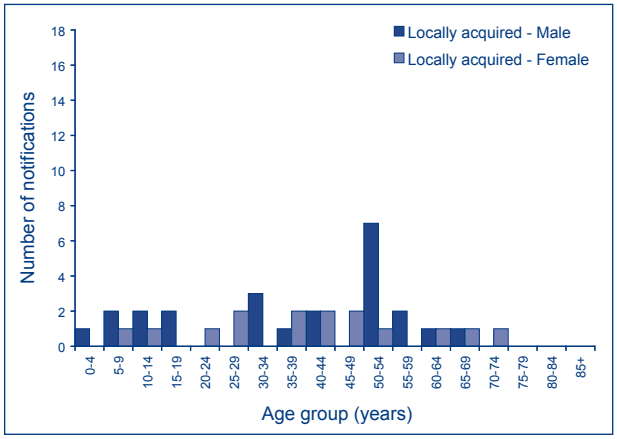
Figure 13 shows that imported DENV notifications in Australia were most frequently reported across the 20–49 year age groups (n=136, 64%) whereas in locally acquired cases from Queensland, notifications were scattered across age groups with a significant peak in the 50–54 year age group (n=8, 10%), which were mostly male (n=7).

## Table 6. Dengue notifications, Australia, 1 July 2006 to 30 June 2007, by serotype and country of acquisition

| Country of acquisition | Total | Serotype | | | | |
|---|---|---|---|---|---|---|
| | | Not typed | Type 1 | Type 2 | Type 3 | Type 4 |
| Country unknown | 169 | 138 | 14 | 8 | 8 | 1 |
| Australia | 39 | 13 | 0 | 0 | 26 | 0 |
| Indonesia | 7 | 4 | 1 | 1 | 0 | 1 |
| Philippines | 6 | 1 | 1 | 1 | 3 | 0 |
| Thailand | 6 | 1 | 0 | 3 | 2 | 0 |
| Cook Islands | 4 | 0 | 4 | 0 | 0 | 0 |
| India | 3 | 2 | 0 | 1 | 0 | 0 |
| Papua New Guinea | 3 | 2 | 0 | 1 | 0 | 0 |
| Fiji | 2 | 0 | 2 | 0 | 0 | 0 |
| Samoa | 2 | 2 | 0 | 0 | 0 | 0 |
| Sri Lanka | 2 | 1 | 1 | 0 | 0 | 0 |
| Bangladesh | 1 | 0 | 0 | 1 | 0 | 0 |
| Cambodia | 1 | 0 | 0 | 0 | 1 | 0 |
| Mexico | 1 | 0 | 1 | 0 | 0 | 0 |
| Pakistan | 1 | 0 | 0 | 0 | 1 | 0 |
| Singapore | 1 | 1 | 0 | 0 | 0 | 0 |
| South America | 1 | 0 | 0 | 0 | 1 | 0 |
| Vietnam | 1 | 0 | 1 | 0 | 0 | 0 |
| Total | 250 | 165 | 25 | 16 | 42 | 2 |

## Figure 13. Dengue notifications, locally acquired and imported cases, 1 July 2006 to 30 June 2007, by age group and sex

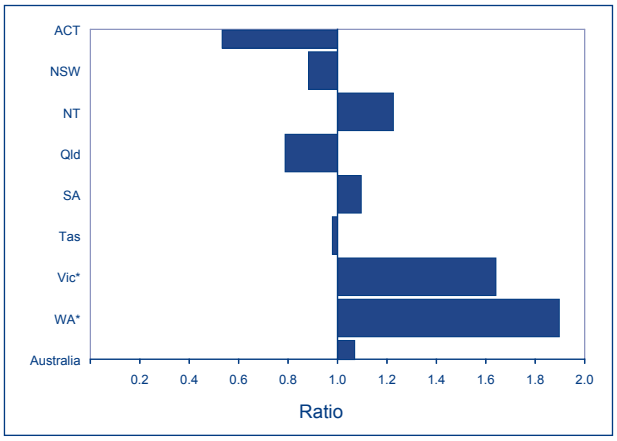


## Malaria

There were 640 notifications of overseas-acquired malaria in Australia in the period 1 July 2006 to 30 June 2007 with no reports of locally-acquired malaria. The majority of malaria notifications were reported by Queensland (28%, n=177, Table 7), Victoria (20%, n=177), New South Wales (19%, n=119) and Western Australia (17%, n=107). The number of malaria notifications reported from Victoria and Western Australia exceeded two standard deviations above their five-year average (Figure 14).

### Figure 14. Ratio of malaria notifications to mean of previous five years, Australia, 1 July 2006 to 30 June 2007, by state or territory

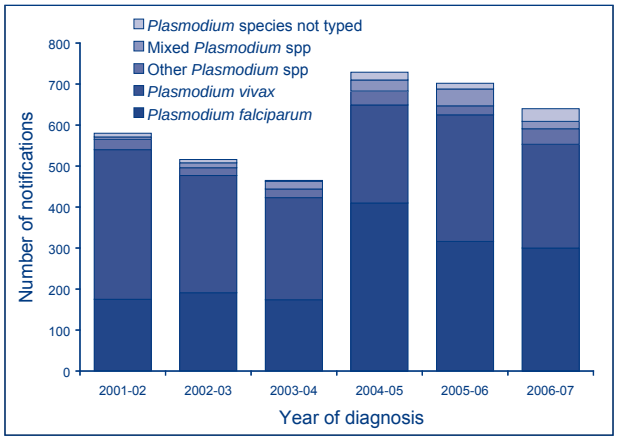



* Above two standard deviations.

Figure 15 shows that the 2006–07 reporting period was the third largest for malaria notifications since 2001–02.

Overall, malaria notifications were highest in the young adult 20–24 year age group (Figure 16). This trend was also observed in 2005–06 and in years prior to 2004–05 (Figure 17).

### Figure 15. Number of notifications of malaria, Australia, 2000 to 2006, by year of onset




### Figure 16. Number of imported malaria notifications, Australia, 1 July 2006 to 30 June 2007, by age group and sex

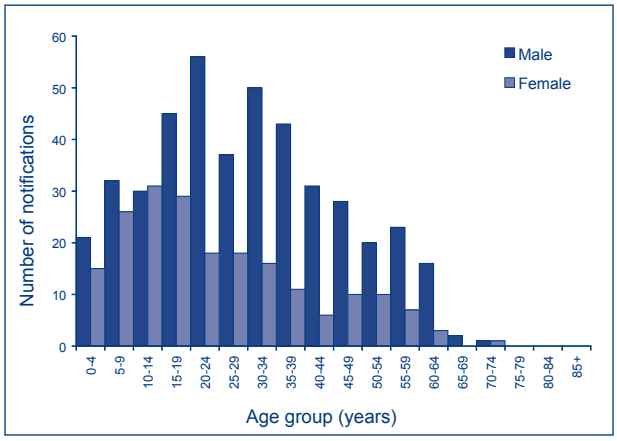



Figure 17 shows that since 2000–01 there has been a steady increase in the proportion of children under the age of 15 years notified with malaria, starting at 7% in 2000–01 and peaking in 2004–05 at over 30% (n=260). In 2006–07 the proportion of notifications in children aged under 15 years has dropped slightly to 25% but this proportion remains the second highest for this age group over

### Table 7. Malaria notifications in Australia, 1 July 2006 to 30 June 2007, by parasite type

| Parasite type | Type % | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Australia |
|---|---|---|---|---|---|---|---|---|---|---|
| *Plasmodium falciparum* | 47 | 2 | 48 | 36 | 77 | 21 | 13 | 40 | 63 | 300 |
| *Plasmodium vivax* | 40 | 5 | 56 | 14 | 87 | 6 | 5 | 69 | 11 | 253 |
| Other *Plasmodium* spp | 6 | 1 | 9 | 0 | 10 | 1 | 0 | 9 | 8 | 38 |
| Mixed *Plasmodium* spp | 3 | 0 | 1 | 0 | 3 | 2 | 0 | 9 | 3 | 18 |
| *Plasmodium* species not typed | 5 | 0 | 5 | 1 | 0 | 2 | 1 | 0 | 22 | 31 |
| Total | 100 | 8 | 119 | 51 | 177 | 32 | 19 | 127 | 107 | 640 |

**Figure 17. Trends in the age distribution of malaria notifications, Australia, 1 July 2000 to 30 June 2006, by age group**

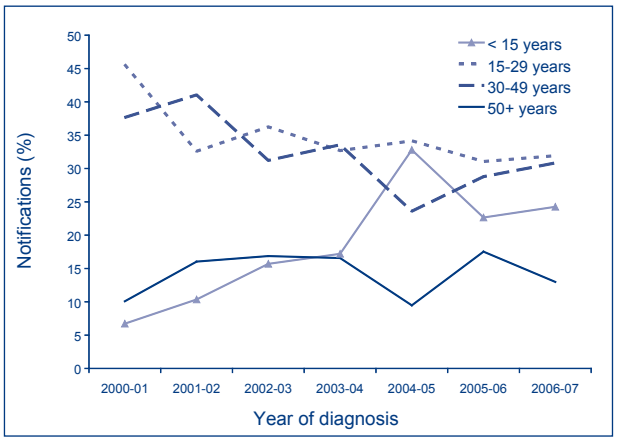
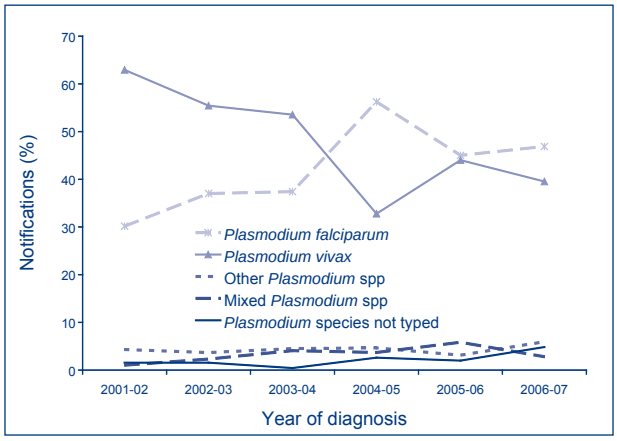



**Figure 18. Trends in malaria notifications, by infecting species and year of onset**




the past seven years. This trend in malaria notifications from young children has been discussed elsewhere and is related to refugee arrivals.[29]

The infecting *Plasmodium* species were reported for 95% of malaria notifications in 2006–07 (Table 8). The majority of the 640 malaria notifications were due to *P. falciparum* (47%, n=300) and *P. vivax* (40%, n=253) while other *Plasmodium* species and mixed *Plasmodium* species infections accounted for 6% and 3% respectively.

Figure 18 shows that in 2006–07 the proportion of notifications due to *P. falciparum* malaria (47%) increased slightly from last year (45%) and the number of *P. falciparum* malaria notifications was up 1.4 times from the five-year mean for the same species.

## Acknowledgements

The National Arbovirus and Malaria Advisory Committee members are (in alphabetical order): Bart Currie, Peter Daniels, Julie Hall, Rogan Lee, Mike Lindsay, Conan Liu, John Mackenzie, Rodney Moran, Scott Ritchie, Richard Russell, Christine Selvey, David Smith, Greg Smith, James Walker, Peter Whelan, Craig Williams, with Susan Barker and Phil Wright from the Secretariat.

We would also like to thank:

Alison Milton and Stefan Stirzaker, Office of Health Protection, Australian Government Department of Health and Ageing

State and territory public health communicable disease surveillance managers

Sentinel reports were provided by:

Cheryl Johansen and technical staff in the Arbovirus Surveillance and Research Laboratory and the Mosquito-Borne Disease Control Branch, Western Australian Department of Health

The New South Wales Arbovirus Surveillance and Mosquito Monitoring Program, Institute of Clinical Pathology and Medical Research, University of Sydney and Westmead Hospital

**Table 8. Malaria notifications in Australia, 1 July 2001 to 30 June 2007, by parasite type**

| Infecting species | Year of diagnosis | | | | | | Last 5 year mean | Ratio 06/07 5 year mean |
|---|---|---|---|---|---|---|---|---|
| | 2001–02 | 2002–03 | 2003–04 | 2004–05 | 2005–06 | 2006–07 | | |
| *Plasmodium falciparum* | 175 | 191 | 174 | 410 | 316 | 300 | 253.2 | 1.2 |
| *Plasmodium vivax* | 365 | 286 | 249 | 239 | 309 | 253 | 289.6 | 0.9 |
| Other *Plasmodium* spp | 25 | 19 | 21 | 34 | 22 | 38 | 24.2 | 1.6 |
| Mixed *Plasmodium* spp | 6 | 12 | 19 | 27 | 41 | 18 | 21.0 | 0.9 |
| *Plasmodium* species not typed | 9 | 8 | 2 | 19 | 14 | 31 | 10.4 | 3.0 |
| Total | 580 | 516 | 465 | 729 | 702 | 640 | | |

Peter Whelan, Nina Kurucz, Northern Territory Department of Health and Community Services and Lorna Melville, Department of Primary Industry, Fisheries and Mines

Elizabeth Birbilis and Joe Azuolas, Victorian Department of Human Services and Victorian Department of Primary Industries

James Walker, Northern Australia Quarantine Strategy, AQIS, and Scott Ritchie, Tropical Public Health Unit Network – Cairns, Queensland Health

## Author details

Conan Liu[1]
Kylie Begg[1]
Cheryl Johansen[2]
Peter Whelan[3]
Nina Kurucz[3]
Lorna Melville[4]

1. Surveillance Policy and Systems Section, Australian Government Department of Health and Ageing, Canberra, Australian Capital Territory
2. Arbovirus Surveillance and Research Laboratory, Discipline of Microbiology and Immunology, The University of Western Australia, Western Australia
3. Medical Entomology, Communicable Disease Control, Northern Territory Department of Health and Community Services, Northern Territory
4. Virology, Department of Primary Industry, Fisheries and Mines, Northern Territory

Corresponding author: Mr Conan Liu, Surveillance Policy and Systems, Office of Health Protection, Australian Government Department of Health and Ageing, GPO Box 4898, MDP 14, CANBERRA ACT 2601. Telephone: +61 2 6289 2712. Facsimile: +61 2 6289 7791. Email: conan.liu@health.gov.au

## References

1. Russell RC. Ross River virus: ecology and distribution. *Annu Rev Entomol* 2002;47:1–31.
2. Russell RC, Dwyer DE. Arboviruses associated with human disease in Australia. *Microbes Infect* 2000;2:1693–1704.
3. Hanna JN, Ritchie SA, Hills SL, Pyke AT, Montgomery BL, Richards AR, et al. Dengue in north Queensland, 2002. *Commun Dis Intell* 2003;27:384–389.
4. Hanna JN, Ritchie SA, Merritt AD, van den Hurk AF, Phillips DA, Serafin IL, et al. Two contiguous outbreaks of dengue type 2 in north Queensland. *Med J Aust* 1998;168:221–225.
5. Hanna JN, Ritchie SA, Phillips DA, Serafin IL, Hills SL, van den Hurk AF, et al. An epidemic of dengue 3 in Far North Queensland, 1997–1999. *Med J Aust* 2001;174:178–182.
6. Hanna JN, Ritchie SA, Richards AR, Taylor CT, Pyke AT, Montgomery BL, et al. Multiple outbreaks of dengue serotype 2 in north Queensland, 2003/04. *Aust N Z J Public Health* 2006;30:220–225.
7. Hanna JN, Ritchie SA, Phillips DA, Lee JM, Hills SL, van den Hurk AF, et al. Japanese encephalitis in north Queensland, Australia, 1998. *Med J Aust* 1999;170:533–536.
8. Hanna JN, Ritchie SA, Phillips DA, Shield J, Bailey MC, Mackenzie JS, et al. An outbreak of Japanese encephalitis in the Torres Strait, Australia, 1995. *Med J Aust* 1996;165:256–260.
9. Mackenzie JS. Emerging zoonotic encephalitis viruses: lessons from Southeast Asia and Oceania. *J Neurovirol* 2005;11:434–440.
10. Mackenzie JS, Broom AK, Hall RA, Johansen CA, Lindsay MD, Phillips DA, et al. Arboviruses in the Australian region, 1990 to 1998. *Commun Dis Intell* 1998;22:93–100.
11. Johansen CA, van den Hurk AF, Pyke AT, Zborowski P, Phillips DA, Mackenzie JS, et al. Entomological investigations of an outbreak of Japanese encephalitis virus in the Torres Strait, Australia, in 1998. *J Med Entomol* 2001;38:581–588.
12. Van Den Hurk AF, Johansen CA, Zborowski P, Phillips DA, Pyke AT, Mackenzie JS, et al. Flaviviruses isolated from mosquitoes collected during the first recorded outbreak of Japanese encephalitis virus on Cape York Peninsula, Australia. *Am J Trop Med Hyg* 2001;64:125–130.
13. Brown A, Bolisetty S, Whelan P, Smith D, Wheaton G. Reappearance of human cases due to Murray Valley encephalitis virus and Kunjin virus in Central Australia after an absence of 26 years. *Commun Dis Intell* 2002;26:39–44.
14. Büchen-Osmond C. Index to Classification and Taxonomy of Viruses Database, version 3, based on the 7th ICTV Report and subsequent up-dates. 2001 onwards. National Center for Biotechnology Information, National Library of Medicine, National Institutes of Health. Available from: http://www.ncbi.nlm.nih.gov/ICTVdb/index.htm Accessed on June 2005.
15. Bakonyi T, Ivanics E, Erdelyi K, Ursu K, Ferenczi E, Weissenbock H, et al. Lineage 1 and 2 strains of encephalitic West Nile virus, central Europe. *Emerg Infect Dis* 2006;12:618–623.
16. World Health Organization. Synopsis of the world malaria situation in 1981. *Wkly Epidemiol Rec* 1983;58:197–199.
17. Hanna JN, Ritchie SA, Eisen DP, Cooper RD, Brookes DL, Montgomery BL. An outbreak of *Plasmodium vivax* malaria in Far North Queensland, 2002. *Med J Aust* 2004;180:24–28.
18. Musgrave IA. Malarial outbreak in Queensland. *Med J Aust* 1987;146:278.
19. Brookes DL, Ritchie SA, van den Hurk AF, Fielding JR, Loewenthal MR. *Plasmodium vivax* malaria acquired in Far North Queensland. *Med J Aust* 1997;166:82–83.
20. Jenkin GA, Ritchie SA, Hanna JN, Brown GV. Airport malaria in Cairns. *Med J Aust* 1997;166:307–308.
21. Merritt A, Ewald D, van den Hurk AF, Stephen Jr S, Langrell J. Malaria acquired in the Torres Strait. *Commun Dis Intell* 1998;22:1–2.
22. Stickland JF, Roberts AN, Williams V. Transfusion-induced malaria in Victoria. *Med J Aust* 1992;157:499–500.
23. Doggett S, Clancy J, Haniotis J, Russell RC, Hueston L, Marchetti M, et al. The New South Wales Arbovirus Surveillance and Mosquito Monitoring Program Annual Report 2006–2007: Department of Medical Entomology, Institute of Clinical Pathology and Medical Research, Westmead Hospital; 2007.

24. Whelan P, Spencer E, Currie B. An imported case of chikungunya in the Northern Territory and a summary of the ecology of the disease. *The Northern Territory Disease Control Bulletin* 2004;11:19–22.

25. Johnson DF, Druce JD, Chapman S, Swaminathan A, Wolf J, Richards JS, et al. Chikungunya virus infection in travellers to Australia. *Med J Aust* 2008;188:41–43.

26. Pialoux G, Gauzere BA, Jaureguiberry S, Strobel M. Chikungunya, an epidemic arbovirus. *Lancet Infect Dis* 2007;7:319–327.

27. Druce JD, Johnson DF, Tran T, Richards MJ, Birch CJ. Chikungunya virus infection in traveler to Australia. *Emerg Infect Dis* 2007;13:509–510.

28. Liu C, Johansen C, Kurucz N, Whelan PI. Communicable Diseases Network Australia: National Arbovirus and Malaria Advisory Committee annual report 2005–06. *Commun Dis Intell* 2006;30:411-429.

29. Broom AK, Azuolas J, Hueston L, Mackenzie JS, Melville L, Smith DW, et al. Australian encephalitis: Sentinel Chicken Surveillance Programme. *Commun Dis Intell* 2001;25:157–160.

30. Johansen C, Avery V, Dixon G, Geerlings K, Power S, McFall S, et al. The University of Western Australia Arbovirus Surveillance and Research Laboratory Annual Report: 2006–2007: Discipline of Microbiology and Immunology, The University of Western Australia; 2007.

## Appendix. Provisional case definition for chikungunya virus infection (Communicable Diseases Network Australia and Public Health Laboratory Network endorsement still pending)

***Reporting***

*Only confirmed cases should be notified*

***Confirmed case***

*A confirmed case requires either:*

*Laboratory definitive evidence*

*OR*

*Laboratory suggestive evidence AND epidemiological evidence AND clinical evidence.*

***Laboratory definitive evidence***

1. *Isolation of chikungunya virus*

*OR*

2. *Detection of chikungunya virus by nucleic acid testing*

***Laboratory suggestive evidence***

1. *Seroconversion or a significant rise in antibody level or a fourfold or greater rise in titre to chikungunya virus, in the absence of a corresponding change in antibody levels to Ross River virus or Barmah Forest virus*

*OR*

2. *Detection of chikungunya virus-specific IgM,[1] in the absence of IgM to Ross River virus or Barmah Forest virus*

***Epidemiological evidence***

1. *A history of travel to a chikungunya affected area[2] within the two weeks prior to the onset of illness*

***Clinical evidence***

1. *Arthralgia OR myalgia OR rash*

**Note**

1. An IgG level should always accompany and IgM as this may help to interpret the significance of a single IgM. If IgM only is detected it is important that a convalescent sample is collected to test for seroconversion.
2. These may vary but include (at the time of writing) parts of Africa (including South Africa, Uganda, Congo, Nigeria, Ghana, Zimbabwe, Senegal, Burkina Faso, the Central African Republic, Cameroon, Guinea-Bissau), South East Asia (including Cambodia, Indonesia, Malaysia, Philippines and Timor Leste), islands in the Indian Ocean (La Réunion, Madagascar, Mauritius, Mayotte, Seychelles) and the Indian-sub-continent (including Pakistan and India).

# Surveillance of antibiotic resistance in *Neisseria gonorrhoeae* in the WHO Western Pacific Region, 2006

The WHO Western Pacific Gonococcal Antimicrobial Surveillance Programme

## Abstract

The World Health Organization Western Pacific Region Gonococcal Antimicrobial Surveillance Programme (WHO WPR GASP) examined approximately 8,400 isolates of *Neisseria gonorrhoeae* from 16 countries for resistance to relevant antibiotics in 2006. Antimicrobial resistance was at record levels in the region and poses major problems for the management of this important disease. High rates of resistance to penicillins and quinolones persisted. Gonococci 'non-susceptible' to third generation cephalosporins were found in several centres. There were infrequent instances of spectinomycin resistance. *Commun Dis Intell* 2008;32:48–51.

Keywords: disease surveillance, *Neisseria gonorrhoeae*, Western Pacific Region

## Introduction

The impact of increasing antimicrobial resistance (AMR) in *Neisseria gonorrhoeae* (GC), in terms of rendering treatment for gonorrhoea ineffective for infected individuals and compromising wider disease control efforts for gonococcal disease, is all-too familiar in the World Health Organization (WHO) Western Pacific Region (WPR). This undesirable outcome is increasingly evident in many parts of the world where the progressive loss of therapeutic effectiveness of the penicillins, tetracyclines, macrolides (including azithromycin), spectinomycin and, most recently, quinolone antibiotics is well documented over many years.[1] The WHO recommends that use of an antibiotic for routine treatment be discontinued when therapeutic failure reaches a level of 5%.[2] Currently in the WHO WPR, rates of gonococcal resistance to some of the above antibiotics are well in excess of 50%. The WPR Gonococcal Antimicrobial Surveillance Programme (WPR GASP) has documented the emergence and spread of this resistance in gonococci at a regional level from 1992 onwards[3,4] to provide information for action and to optimise the antibiotic treatment for gonorrhoea.

Because of the progressive loss of effectiveness of other antibiotics, in parts of the WPR treatment for gonorrhoea has become increasingly reliant on use of injectable (ceftriaxone) and oral (cefixime, ceftibuten, cefpodoxime, cefdinir and cefropozan) later-generation cephalosporins. Worryingly, the appearance and spread of GC 'non-susceptible' to these later-generation cephalosporins (and usually also resistant to other antibiotics[4,5]) has been documented over several years and in an increasing number of countries in the WPR, and is sometimes accompanied by evidence of treatment failure.[6]

This report provides an analysis of antimicrobial resistance in *N. gonorrhoeae* in the WHO WPR derived from the results of the WPR GASP surveillance for 2006.

## Methods

The methods used by the WHO WPR GASP have been published[3] and provide full details of the source of isolates, sample populations, laboratory test methods and quality assurance programs used to generate data. These methods were unaltered in 2006.

## Results

A total of 8,374 isolates of *N. gonorrhoeae* were examined for susceptibility to one or more antibiotics in 16 participating countries in 2006.

### Quinolone antibiotics

Table 1 shows the distribution of quinolone-resistant *N. gonorrhoeae* (QRNG) in 14 countries that examined a total of 7,954 isolates in 2006. The proportion of QRNG in most of these centres was high to very high. The exceptions were New Caledonia where there were no QRNG detected, and Papua New Guinea where only a single QRNG was found. Very high rates of quinolone resistance (more than 80% of isolates tested) were present in Brunei, China, Hong Kong, Japan, Korea, Laos and Vietnam. The proportion of QRNG found in isolates from Malaysia (62%), the Philippines (69%) and Singapore (70%) was also high, while those from Australia (38%) and New Zealand (14%) while lower, were still substantial. When compared to earlier data, these rates were in general as high or higher as rates in previous years although a lower rate was noted in New Zealand.

**Table 1. Quinolone resistance in 7,954 strains of *Neisseria gonorrhoeae* isolated in 14 countries in the WHO Western Pacific Region, 2006**

| Country | Number of isolates | Less susceptible | | Resistant | | All QRNG | |
|---|---|---|---|---|---|---|---|
| | | n | % | n | % | n | % |
| Australia | 3,850 | 42 | 1.2 | 1,413 | 36.7 | 1,455 | 37.8 |
| Brunei | 208 | 50 | 24 | 120 | 57.7 | 170 | 81.7 |
| China | 1,134 | 61 | 5.4 | 1,068 | 94.2 | 1,129 | 99.6 |
| Hong Kong SAR | 1,622 | 32 | 2 | 1,554 | 95.8 | 1,586 | 97.8 |
| Japan | 211 | 16 | 7.6 | 160 | 75.8 | 176 | 83.4 |
| Korea | 47 | 7 | 14.9 | 35 | 74.5 | 42 | 89.4 |
| Lao PDR | 9 | | | | | 9 | 100 |
| Malaysia | 29 | 5 | 17.2 | 13 | 44.8 | 18 | 62 |
| New Caledonia | 93 | 0 | 0 | 0 | 0 | 0 | 0 |
| New Zealand | 284 | 0 | 0 | 39 | 13.7 | 39 | 13.7 |
| Papua New Guinea | 53 | 0 | 0 | 1 | 1.8 | 1 | 1.8 |
| Philippines | 42 | 0 | 0 | 29 | 69 | 29 | 69 |
| Singapore | 160 | 13 | 8.2 | 99 | 61.8 | 112 | 70 |
| Vietnam | 212 | 54 | 25.5 | 120 | 56.6 | 174 | 82.1 |

## Cephalosporins

Strains 'non-susceptible' to ceftriaxone and/or other later generation cephalosporins were again reported amongst isolates from Australia and Brunei and were especially prominent in China. The correlation between clinical outcomes and *in vitro* tests for 'resistance' to this group of antibiotics has not been reliably performed to date so that published parameters for decreased sensitivity to ceftriaxone and oral agents remains arbitrary. Because of this and some other methodological issues, minimal inhibitory concentrations (MIC) values are not directly comparable between different centres.

## Spectinomycin

Only very small numbers of spectinomycin resistant gonococci have been reported in recent years in WPR GASP surveys and these sporadically, and this continued to be the case in 2006.

## Penicillins

Resistance to penicillins has been widespread and at high levels for many years in the WPR, and may be the result of penicillinase production (PPNG) or aggregation of a number of chromosomally mediated mechanisms (CMRP). These mechanisms may co-exist in the one strain. Table 2 shows the penicillin susceptibility of 8,374 gonococci isolated in 16 WHO WPR centres in 2006. Because the penicillins have not been used for treatment of gonorrhoea for many years in some WPR countries, only testing for PPNG is performed in these centres. For example, China found 48% PPNG, but did not test for chromosomally mediated resistance. Once again penicillin resistance was widespread and, although less than that for quinolone antibiotics in most centres, was present in a high proportion of isolates in most countries. Rates of resistance to the penicillins above 50% in decreasing order of magnitude, were Laos, 100% (in a small sample), the Philippines 78%, Korea 69%, Brunei 64%, Hong Kong 63% and Singapore 59%. Rates ranging between 34% in Malaysia and Australia, 30% in Vietnam to 23% in Japan and 21.5% in New Zealand, were also recorded. In previous reports, some Pacific Island states have consistently reported low levels of penicillin resistance. In Fiji, 12% of 409 gonococci were penicillin resistant in 2006 with 7% of all isolates being PPNG. Papua New Guinea currently monitors AMR in GC every two years, and 43% of isolates tested from a number of centres in 2006 were PPNG. In Tonga, 9% of 32 gonococci tested were penicillin resistant, but no penicillin resistance was observed in New Caledonia.

## Tetracyclines

These antibiotics are still widely available in the WPR. Approximately 7,650 isolates in 10 countries in 2006 were examined for one particular form of resistance, namely, the high-level plasmid-mediated form referred to as TRNG (Table 3). The highest rates of TRNG were reported from Singapore (77%), Hong Kong (49%), China (35%) and the Philippines (31%). Only low numbers were present in Japan and New Caledonia. Low proportions of TRNG (at or around 10%) were found in Korea and Australia, and slightly higher rates were found in New Zealand (25%) and Vietnam (16.5%).

**Table 2. Penicillin resistance in 8,374 strains of *Neisseria gonorrhoeae* isolated in 16 countries in the WHO Western Pacific Region, 2006**

| Country | Number of isolates | PPNG | | CMRP | | All Pen R | |
|---|---|---|---|---|---|---|---|
| | | n | % | n | % | n | % |
| Australia | 3,850 | 342 | 9 | 964 | 25 | 1,306 | 34 |
| Brunei | 308 | 119 | 38.6 | 79 | 25.6 | 198 | 64.2 |
| China | 1,013 | 483 | 47.7 | | | | |
| Fiji | 409 | 30 | 7.3 | 20 | 4.9 | 50 | 12.2 |
| Hong Kong SAR | 1,622 | 553 | 34.1 | 464 | 28.6 | 1,017 | 62.7 |
| Japan | 211 | 2 | 0.9 | 46 | 21.8 | 48 | 22.7 |
| Korea | 47 | 5 | 10.6 | 30 | 63.8 | 35 | 74.5 |
| Lao PDR | 9 | | | | | 9 | 100 |
| Malaysia | 29 | | | | | 10 | 34.4 |
| New Caledonia | 93 | 0 | 0 | 0 | 0 | 0 | 0 |
| New Zealand | 284 | 3 | 1 | 58 | 20.4 | 61 | 21.5 |
| Papua New Guinea | 53 | 23 | 43.4 | | | 53 | 43.4 |
| Philippines | 42 | 21 | 50 | 12 | 28.5 | 33 | 78.5 |
| Singapore | 160 | 79 | 49.4 | 16 | 10 | 95 | 59.4 |
| Tonga | 32 | 2 | 6 | 1 | 3 | 3 | 9 |
| Vietnam | 212 | 65 | 30.7 | 1 | 0.5 | 66 | 30.2 |

**Table 3. High-level tetracycline resistance in 7,654 strains of *Neisseria gonorrhoeae* isolated in 10 countries in the WHO Western Pacific Region, 2006**

| Country | Number of isolates | Number of TRNG | % TRNG |
|---|---|---|---|
| Australia | 3,850 | 462 | 12 |
| China | 1,133 | 399 | 35.2 |
| Hong Kong SAR | 1,622 | 793 | 48.9 |
| Japan | 211 | 2 | 0.9 |
| Korea | 47 | 4 | 8.5 |
| New Caledonia | 93 | 2 | 2.1 |
| New Zealand | 284 | 71 | 25 |
| Philippines | 42 | 13 | 31 |
| Singapore | 160 | 123 | 76.8 |
| Vietnam | 212 | 35 | 16.5 |

## Discussion

The now well-documented picture of increasing antimicrobial resistance in *Neisseria gonorrhoeae* to many of the antibiotics used for treatment of gonorrhoea in the WHO WPR was reinforced by data obtained in this survey. As in previous surveys,[3,4] there were considerable differences in rates of resistance to several important groups of antibiotics across the WHO WPR, with a high proportion of both penicillin and quinolone resistance amongst isolates tested in north Asia, but much lower rates of penicillin resistance and little if any quinolone resistance present in Pacific Island states.

While the situation in regard to resistance to penicillins and quinolones was little changed, concerns continue over increasing numbers of isolates 'non-susceptible' to later-generation cephalosporins. This has been manifested as a rise in the MICs for these cephalosporins,[7] and at least some of this increase in MICs has been shown to be due to alterations in PBP2, the site of action of these antibiotics.[8–10] Particular attention has been paid to 'mosaic' *penA* genes that encode these PBP2 alterations. In gonorrhoea caused by GC with altered PBP2, treatment failures have been reported following therapy with a number of oral cephalosporins,[6] but so far not with the injectable agent ceftriaxone. However, there is much that is still unclear regarding these GC that are cephalosporin 'non-susceptible' in regard to the resistance mechanisms involved, the differential effect of these mechanisms on the various cephalosporin agents and how these phenomena affect treatment outcomes. Further clarification regarding the correlations between laboratory and clinical findings, and the extent of spread of GC with these alterations in the region is required before the longer term implications that arise from these events can be fully elucidated.

Surveys of this kind suffer from a number of limitations. The most obvious of these is that low sample numbers only are available in some centres, for several reasons, and the resource limitations that restrict capacity for testing, even when isolates are available. Other antibiotics, such as azithromycin, are being used either as a primary treatment or as adjunctive treatment for other pathogens, but sub-

stantive data on emerging azithromycin resistance in WPRO is not available. Some incomplete data suggest that azithromycin resistance is present, albeit in low proportions. Despite these limitations, in the absence of other data sources, and when conducted over extended periods under the same conditions, this series provides reasonably reliable guides to AMR trends in the region. The difficulties associated with surveillance in the WHO WPR GASP have increased and become more complex over time, but the ongoing need to meet these challenges is shown in the continuing problems posed by AMR in GC. Further, given the past history of the spread of antibiotic resistant GC from the WPR to other parts of the world,[1] there is a high likelihood that, unless better disease control becomes a reality, they will continue to spread well beyond the region.

## Acknowledgements

Members of the WHO Western Pacific Gonococcal Antimicrobial Surveillance Programme in 2006: JW Tapsall and EA Limnios, Australia; Hjh Mahami Hj Abu Bakar, Brunei Darussalam; Yin Yue Ping, China; EM Buadromo and S Singh, Fiji; J Lo, Hong Kong; Yuko Watanabe and M Tanaka, Japan; K Lee and Y Chong, South Korea; T Phouthavane, Lao PDR; I-Ching Sam, Malaysia; R Goursaud, New Caledonia; T Bathgate and M Brokenshire, New Zealand; Tony Lupiwa, Papua New Guinea; CC Carlos, Philippines; Cecilia Ngan and Marianne Yuen, Singapore, M Fakahau, Tonga, Le Van Hung and Le Thi Phuong, Vietnam

## Author details

Correspondence: Associate Professor John Tapsall, WHO Collaborating Centre for STD, Department of Microbiology, The Prince of Wales Hospital, RANDWICK NSW 2031. Telephone: +61 2 9298 9084. Facsimile: +61 2 9398 4275. Email: j.tapsall@unsw.edu.au

## References

1. Tapsall J. Antibiotic resistance in *Neisseria gonorrhoeae* is diminishing available treatment options for gonorrhea: some possible remedies. *Expert Rev Anti Infec Ther* 2006;4:619–628.
2. Management of sexually transmitted diseases. World Health Organization 1997; Document WHO/GPA/TEM94.1 Rev.1 p 37.
3. WHO Western Pacific Region Gonococcal Surveillance Programme. Surveillance of antibiotic susceptibility of *Neisseria gonorrhoeae* in the WHO Western Pacific Region 1992–4. *Genitourin Med* 1997;73:355–361.
4. The WHO Western Pacific Gonococcal Antimicrobial Surveillance Programme. Surveillance of antibiotic resistance in *Neisseria gonorrhoeae* in the WHO Western Pacific Region, 2005. *Commun Dis Intell* 2006;30:430–433.
5. Tanaka M, Nakayama H, Huruya K, Konomi I, Irie S, Kanayama A, et al. Analysis of mutations within multiple genes associated with resistance in a clinical isolate of *Neisseria gonorrhoeae* with reduced ceftriaxone susceptibility that shows a multidrug-resistant phenotype. *Int J Antimicrob Agents* 2006;27:20–26.
6. Yokoi S, Deguchi T, Ozawa T, Yasuda M, Ito S, Kubota Y, et al. Threat to cefixime treatment for gonorrhea. *Emerg Infect Dis* 2007;13:1275–1277.
7. Tanaka M, Nakayama H, Tunoe H, Egashira T, Kanayama A, Siaka T, et al. A remarkable reduction in the susceptibility of *Neisseria gonorrhoeae* isolates to cephems and the selection of antibiotic regimens for the single-dose treatment of gonococcal infection in Japan. *J Infect Chemother* 2002;8:81–86.
8. Ameyama, S, Onodera S, Takahata M, Minami S, Maki N, Endo K, et al. Mosaic-like structure of penicillin-binding protein 2 gene (*penA*) in clinical isolates of *Neisseria gonorrhoeae* with reduced susceptibility to cefixime. *Antimicrob Agents Chemother* 2002;46:3744–3749.
9. Ochiai S, Sekiguchi S, Hayashi A, Shimadzu M, Ishiko H, Matsushima-Nishiwaki R, Kozawa O, Yasuda M, Deguchi T. Decreased affinity of mosaic-structure recombinant penicillin-binding peotein2 for oral cephalosporins in *Neisseria gonorrhoeae*. *J Antimicrob Chemother* 2007;60:54–60.
10. Whiley DM, Limnios EA, Ray S, Sloots TP, Tapsall JW. Further questions regarding the role of mosaic *penA* sequences in conferring reduced susceptibility to ceftriaxone in *Neisseria gonorrhoeae*. *Antimicrob Agents Chemother* 2007, 51:802–803.

# Australian Paediatric Surveillance Unit annual report, 2006

Yvonne Zurynski, Elizabeth J Elliott

## Background

The Australian Paediatric Surveillance Unit (APSU) conducts national active surveillance of rare diseases of childhood, including infectious and vaccine preventable diseases, genetic disorders, childhood injuries and mental health conditions.[1] The study of communicable and vaccine preventable diseases is supported in part by the Australian Government Department of Health and Ageing through its communicable diseases program. This report is a summary of surveillance results for communicable and/or vaccine preventable diseases studied through the APSU in 2006.

In 2006, nine communicable or vaccine preventable conditions were studied:

- acute flaccid paralysis (AFP);
- congenital cytomegalovirus infection;
- congenital rubella infection;
- perinatal exposure to HIV and HIV infection;
- neonatal herpes simplex virus infection;
- hepatitis C virus infection;
- non-tuberculous mycobacterium infection (surveillance to end in 2007);
- neonatal group B streptococcus infection;
- varicella: neonatal, congenital and severe complications of varicella infection requiring hospitalisation.

## Methods

APSU study protocols are developed with collaborating investigators and/or institutions and the objectives and chief investigators for each study are listed in Table 1. Detailed protocols including case definitions for each disease under surveillance are available at www.apsu.org.au

The APSU aims to provide epidemiological information that is representative of the Australian population and maximal case ascertainment is a high priority. Despite a representative mailing list (92% of all paediatricians in active clinical practice in Australia participate in monthly surveillance) and high response rates (97% for 2006), complete case ascertainment is unlikely. This is particularly relevant in remote communities where children have limited access to paediatricians. However, for most conditions studied by the APSU no national data are available to estimate completeness of ascertainment. APSU encourages the use of complementary data sources where available and reporting by a range of specialists to maximise cases identified. Reported rates for conditions ascertained through the APSU therefore represent a minimum estimate of the incidence of these conditions in the relevant Australian populations.

## Results

All data provided in this report are accurate as at 30 July 2007. It is possible that some notifications may be reclassified or the outcomes may change as additional clinical data are received. In 2006, 1,248 clinicians participated in the monthly surveillance of 16 uncommon childhood conditions, including the nine communicable diseases listed above. The report card return rate for 2006 was 97%. Enhanced data detailing diagnosis, clinical management and short-term outcome were available for more than 85% for all cases notified. Table 2 shows the number of cases reported in 2006 and for the whole study period and the reported rate per 100,000 population.

APSU data contribute significantly to the national surveillance effort, providing valuable information for clinicians, policymakers and the community.[1,2] The APSU is often the only source of national data that includes clinical and/or laboratory details, and data on both inpatients and outpatients.[1,2] The key findings for infectious diseases under surveillance by the APSU in 2006 are summarised in Table 1.

## Studies due to finish in 2007

Surveillance for non-tuberculous mycobacterial infection finished in 2007. Adequate data have been collected in order to address the specific aims for this surveillance as determined by the investigators group leading this study, and a journal article is in preparation.

## Table 1. Results summary

| Condition and principal investigator | Objectives | Key findings |
|---|---|---|
| **Acute flaccid paralysis (AFP)**<br>Prof. Heath Kelly, Victorian Infectious Diseases Reference Laboratory | To determine the notification rate of AFP in children aged < 15 years<br>To determine whether AFP is caused by poliovirus infection and if so, whether it is a wild, vaccine, or vaccine-derived strain of poliovirus<br>To determine other causes and the clinical picture of AFP in Australia | In 2006, Australia reached the WHO AFP surveillance target of one case /$10^5$ children aged <15 years per annum.<br>The primary diagnoses for AFP remain Guillain-Barre syndrome and transverse myelitis.<br>Only approximately 20% of cases had adequate faecal specimen collection in 2006 – well below the 80% WHO target.<br>An outbreak of approximately 300 cases of wild poliovirus recorded in Indonesia in 2005 and the recent importation into Australia of polio by an adult from Pakistan, highlights the need for continued surveillance to keep Australia polio free.[3] |
| **Congenital cytomegalovirus (cCMV) infection**<br>Prof. William Rawlinson, Virology Division, Department of Microbiology, Prince of Wales Hospital, Sydney | To determine the incidence of congenital and suspected congenital CMV infection<br>To determine the presenting features and clinical spectrum of disease due to congenital CMV<br>To determine the genotypes of CMV which cause congenital disease<br>To determine current therapy for congenital CMV infection<br>To determine the epidemiology of congenital CMV prior to trials of vaccines and antivirals | cCMV continues to be the most common infectious cause of malformations in Australia.<br>cCMV infection was not associated with maternal illness in approximately one third of cases, and should be considered regardless of maternal history.<br>cCMV remains under-diagnosed. Although most cases are diagnosed by urine culture; use of PCR for urinary screening for CMV may increase diagnostic yield.<br>Universal neonatal hearing screening programs may also help identify new cases. |
| **Congenital rubella (with defects)**<br>A/Prof. Cheryl Jones, The Children's Hospital at Westmead & Discipline of Paediatrics & Child Health, University of Sydney | To document the incidence of congenital rubella infection<br>To determine the vaccination status of mothers of infected infants<br>To monitor the effectiveness of the current vaccination program | There were no cases of congenital rubella reported in 2006.<br>As the risk of congenital rubella remains, particularly among immigrant women born in countries with poorly developed vaccination programs, such women should have serological testing for rubella after arrival in Australia, and vaccination when appropriate.<br>Travel to rubella endemic counties in the first trimester by women with no prior rubella immunity poses a risk to the foetus of congenital rubella. |
| **Perinatal exposure to HIV and HIV infection**<br>Ms Ann McDonald, National Centre in HIV Epidemiology and Clinical Research | To identify new cases of perinatal exposure to HIV, paediatric HIV infection, and AIDS<br>To describe the pattern of perinatal exposure to HIV in Australia<br>To monitor the perinatal HIV infection transmission rate and use of interventions for reducing the risk of mother-to-child transmission<br>To describe the natural history of paediatric HIV infection | Data from this study informed the section on perinatal HIV testing in the HIV Testing Policy 2006.<br>In 2006, 14 cases of perinatal exposure were reported.<br>11 mothers were diagnosed prenatally; nine of these made use of interventions, and no child in this group has acquired HIV infection to date (7 HIV status negative, 2 indeterminate).<br>For the two mothers who did not use intervention one child acquired HIV infection<br>Three mothers were diagnosed postnatally – two of these were migrants from sub-Saharan Africa.<br>Antenatal diagnosis of the mother's HIV infection and use of interventions is required to minimise the risk of mother-to-child HIV transmission.[4] |
| **Neonatal herpes simplex virus infection (HSV)**<br>A/Prof. Cheryl Jones, Herpes Virus Research Unit, The Children's Hospital at Westmead & Discipline of Paediatrics & Child Health, University of Sydney | To determine the incidence of neonatal HSV infection in Australia, its mortality and morbidity<br>To determine its mode of presentation e.g. localised, disseminated or complicated by encephalitis or pneumonitis and mode of transmission<br>To determine whether there is a delay between presentation, diagnosis and initiation of treatment | Over a half of neonatal HSV infections in Australia are caused by HSV type 1, in contrast to the USA where HSV type 2 predominates.<br>Typical herpetic lesions of the skin, eye or mouth were not evident in half of infants identified with neonatal HSV infection, which makes early diagnosis difficult.<br>Disseminated HSV infection in the newborn may be associated with the early onset of pneumonitis in infants (in whom the chest X-ray may be normal). This is highly lethal unless antiretroviral therapy is initiated. |

## Table 1. Results summary, *continued*

| Condition and principal investigator | Objectives | Key findings |
|---|---|---|
| **Hepatitis C virus infection (HCV)**<br>A/Prof Cheryl Jones, The Children's Hospital at Westmead & Discipline of Paediatrics & Child Health, University of Sydney | To determine the reported incidence of newly diagnosed HCV infection in Australian children<br>To describe the clinical presentation, investigation and management of newly diagnosed HCV infection in Australian children<br>To document the presence of known risk factors for HCV infection in an Australian paediatric population<br>To determine the prevalence of co-infection with hepatitis B virus and/or HIV in Australian children with newly diagnosed HCV infection | Perinatal transmission is the main source of HCV infection in Australian children.<br>In the APSU study infants at risk were born to mothers who used IV drugs, had invasive procedures overseas or had tattoos.<br>Most HCV-infected children are clinically asymptomatic with mildly elevated liver function test at diagnosis, however, HCV induced chronic liver disease and liver failure have been reported among children.[6]<br>Given that 1%–2% of Australian women of childbearing age are infected with HCV, the reported rates of infection are lower than predicted. This may be due to the lack of a consistent approach to identifying children with HCV infection.[6] |
| **Non-tuberculous mycobacterium infection (NTMI)**<br>Dr Pamela Palasanthiran, Paediatric Infectious Diseases Specialist, Department of Immunology and Infectious Diseases, Sydney Children's Hospital Randwick, NSW | To estimate the incidence of newly diagnosed NTM infection in children seen by child health specialists in Australia<br>To describe the epidemiology and spectrum of disease and document known risk factors<br>To describe diagnostic investigations used in Australia; frequency of use of skin testing and the clinical utility of the test, including differential skin testing<br>To describe the management of NTM in Australia and the response to treatment | This infection most often presents as lymphadenitis predominantly in immunocompetent children.<br>*Mycobacterium avium intracellulare* and *Mycobacterium fortuitum* are the most common organisms isolated in Australian children.<br>Surgery is the most commonly offered therapy and in NTMI lymphadenitis complete excision is associated with a lower risk of relapse.<br>There is marked heterogeneity in the antimicrobials and course prescribed.<br>Despite therapy, relapse occurs in about 20% of cases.[7] |
| **Neonatal and infant Streptococcus agalactiae (group B streptococcus – GBS) sepsis**<br>Prof Lyn Gilbert, Centre for Infectious Diseases and Microbiology, Institute for Clinical Pathology and Medical Research, Westmead Hospital, Westmead, NSW | To determine the current incidence of early and late onset neonatal GBS infection<br>To determine the incidence of maternal and infant risk factors<br>To determine the proportion of early onset GBS infection in infants of women who have been given intrapartum antibiotic prophylaxis<br>To determine the short-term mortality and morbidity of early and late onset GBS infection<br>To determine the distribution of GBS genotypes between isolates | Over half (57%) of the reported cases have been early onset at less than eight days of age.<br>The number of notifications received so far is consistent with other available data.<br>Reported rates of GBS infection are higher in Queensland than in any other state.<br>Meningitis is associated with late-onset cases (children aged 9 days or more).<br>Group B streptococcus isolates have been collected for approximately 75% of cases and these will be genotyped. |
| **Severe complications of varicella infection**<br>Prof Robert Booy, National Centre for Immunisation Research and Surveillance, The Children's Hospital at Westmead, NSW | To describe the incidence in children aged one month to 15 years<br>To describe the demographics of affected children<br>To describe the vaccination status<br>To describe the management of the disease<br>To describe The genotypes of varicella zoster viruses associated with severe complications of varicella in Australia[8] | **Preliminary results – surveillance period of seven months only**<br>Fourteen children were hospitalised with complications of varicella; average age = 5; range 8 months to 12 years.<br>The average stay in hospital was seven days; range 2–14 days.<br>Complications included bacteraemia, osteomyelitis, cellulitis, pneumonia, hepatitis, encephalitis and ataxia.<br>The main source of contact were siblings, other family members or friends at school or daycare.<br>All but one affected child were unvaccinated. |
| **Congenital and neonatal varicella**<br>As above | To describe the epidemiology of neonatal and congenital varicella and to compare results to a previous APSU study conducted in 1997. | One case of congenital varicella was reported in New South Wales.<br>Fourteen cases of neonatal varicella were reported.<br>Further analysis of these data is currently being completed by the investigators. |

**Table 2. Confirmed cases identified for 2006 and for the total study period**

| Condition | Date study commenced | Questionnaire response (%) for total study period | Number of confirmed cases for 2006 | Reported Rate for 2006 (per $10^5$) | Number of confirmed cases for total study period | Reported rate for total study period (per $10^5$ per annum) |
|---|---|---|---|---|---|---|
| Acute flaccid paralysis | March 1995 | 90 | 44* | 1.1† | 412* | 0.87† |
| Congenital cytomegalovirus | Jan 1999 | 69 | 18 | 6.8‡ | 75 | 3.70‡ |
| Congenital rubella (with defects) | May 1993 | 96 | NIL | NIL | 50 | 0.09† |
| Perinatal exposure to HIV | May 1993 | 90 | 14 | 5.2‡ | 282 | 7.90‡ |
| Neonatal herpes simplex virus infection | Jan 1997 | 95 | 11 | 4.1‡ | 88 | 3.52‡ |
| Hepatitis C virus infection | Jan 2003 | 85 | 9 | 0.22† | 41 | 0.26† |
| Non-tuberculous mycobacteria | July 2004 | 80 | 33§ | 0.81† | 77§ | 0.64† |
| Neonatal B group streptococcus Infection | July 2005 | 86 | 55 | 20.7‡ | 92 | 23.20‡ |
| Congenital varicella | May 2006 | 100 | 1 | \|\| | 1 | \|\| |
| Neonatal varicella | May 2006 | 91 | 8 | \|\| | 8 | \|\| |
| Severe complications of varicella | May 2006 | 90 | 14 | \|\| | 14 | \|\| |

* All reported cases that have been classified by the Polio Expert Committee were 'non-polio acute flaccid paralysis' according to World Health Organization criteria.

† Based on population of children aged ≤ 15 years as estimated by the Australian Bureau of Statistics.[9]

‡ Based on number of births as estimated by the Australian Bureau of Statistics.[9]

§ Includes confirmed and probable cases.

|| Surveillance for varicella commenced in May 2006. Due to the short surveillance period of only seven months, a rate is not reported for 2006.

## New surveillance studies

### Acute rheumatic fever

The acute rheumatic fever (ARF) surveillance is a joint project between the Menzies School of Public Health, The National Heart Foundation of Australia and the APSU. Surveillance commenced in September 2007. The burden of ARF has been recognised among Indigenous children and control programs and data collections in the top end of Australia have been invaluable. However, we know little about the incidence of ARF in the rest of Australia although the Australian Bureau of Statistics estimates that approximately 30% of Australia's Indigenous population lives in New South Wales.[10] The incidence of ARF in the non-Indigenous population is unknown, and this study may provide preliminary information on other high risk groups such as refugees. In order to improve surveillance coverage in rural and remote regions the APSU will recruit additional key clinicians from these areas. This is an important capacity building step for the surveillance mechanism.

### Intussusception

Surveillance for intussusception commenced in July 2007. Intussusception has been recognised as a potential complication of rotavirus vaccination and the APSU study will provide information on the diagnosis and clinical management of intussusception and any temporal association between rotavirus vaccination and intussusception.

## Acknowledgements

The APSU is funded by the Australian Government Department of Health and Ageing; Faculty of Medicine, the University of Sydney; the Division of Paediatrics and Child Health, Royal Australasian College of Physicians; National Health and Medical

Research Council Enabling Grant no:402784; and National Health and Medical Research Council Practitioner Fellowship no:457084 (Elizabeth Elliott).

We acknowledge the important continued contribution of all paediatricians and child health professionals who participate in surveillance studies conducted by the APSU.

## Author details

Yvonne Zurynski[1,2]
Elizabeth J Elliott[1,2,3]

1. Australian Paediatric Surveillance Unit, The Children's Hospital at Westmead, Westmead, New South Wales
2. Discipline of Paediatrics and Child Health, Faculty of Medicine, The University of Sydney, New South Wales
3. The Children's Hospital at Westmead, Westmead, New South Wales

Corresponding author: Dr Yvonne Zurynski, Assistant Director, Australian Paediatric Surveillance Unit, The Children's Hospital at Westmead, Locked Bag 4001, WESTMEAD NSW 2145.Telephone: +61 2 9845 1202/3005. Facsimile: +61 2 9845 3082. Email: yvonnez@chw.edu.au

## References

1. Zurynski YA, Peadon E, Bower C, Elliott EJ. Impacts of national surveillance for uncommon conditions in childhood. *J Paediatr Child Health* 2007;43:724–731.
2. Grenier D, Elliott EJ, Zurynski Y, Rodrigues Perera R, Preece M, Lynn R, et al. Beyond counting numbers: Public health impacts of national paediatric surveillance units. *Arch Dis Child* 2007;92:527–533.
3. Thorley BR, Brussen KA, Elliott EJ, Kelly HA. Vigilance is required for Australia to remain polio free. *Med J Aust* 2006;184:474–475.
4. McDonald A, Zurynski Y, Nadew K, Elliott E, Kaldor J, Ziegler J. Prevention of mother-to-child HIV transmission in Australia, 1982–2005. *J Paediatr Child Health 2007*;43:A11.
5. Elliott EJ, Jones CA, Mews C, Hardikar A, Kesson AM, Polis S, et al. Reported risk factors for hepatitis C virus Infection in Australian children: Results of National Surveillance. *J Paediatr Child Health* 2005;41 Suppl:S3.
6. Hardikar W, Elliott EJ, Jones CA. The silent infection: should we be testing for perinatal hepatitis C and, if so, how? *Med J Aust* 2006;184:54–55.
7. Blyth CC, Palasanthiran P, Best E, Jones C, Daley AJ, Burgner D, et al. Non-tuberculous mycobacterial infection: results from a national surveillance study. *J Paediatr Child Health* 2006;42:A11.
8. Peadon E, Burgner D, Nissen M, Buttery J, Zurynski Y, Elliott E, et al. Case for varicella surveillance in Australia. *J Paediatr Child Health* 2006;42:663–664.
9. Australia Bureau of Statistics. Australian Demographic Statistics. Catalogue No. 3101.0, March Quarter 2007.
10. Australian Bureau of Statistics. National Aboriginal and Torres-Strait Islander Health Survey 2007, Catalogue No: 4715.0.55.006.

Articles

# INFLUENZA VIRUSES WITH REDUCED SENSITIVITY TO THE NEURAMINIDASE INHIBITOR DRUGS IN UNTREATED YOUNG CHILDREN

Aeron C Hurt, Ian G Barr

## Abstract

The neuraminidase inhibitors are a class of antiviral drugs used for both the prophylaxis and treatment of influenza infections. Clinical trials of these inhibitors detected a low level of resistant viruses from treated individuals, although a higher frequency was detected in children (5%–6%) compared to adults (1%–4%). In addition, there have been some previous reports of NA inhibitor resistant viruses being isolated from untreated individuals. Here we report on the NA inhibitor sensitivity of over 1,000 influenza isolates collected through the World Health Organization (WHO) global influenza surveillance program. Of the total number of viruses analysed, only 2 (0.2%) strains (an A(H1N1) strain and an influenza B strain) were considered to have a significant reduction in sensitivity to at least one of the neuraminidase inhibitor drugs. Interestingly, both of these strains were isolated from untreated patients in the youngest age cohort (less than 2 years). Although the influenza B strain is unlikely to be clinically resistant, the A(H1N1) virus contained the same His274Tyr neuraminidase mutation that has been observed in resistant mutants following oseltamivir treatment. Given these results it may be important to enhance neuraminidase inhibitor susceptibility testing of viruses from patients in the less than two years cohort. *Commun Dis Intell* 2008;32:57–62.

Keywords: drug resistance, influenza, neuraminidase inhibitors

## Introduction

Influenza causes significant morbidity and mortality worldwide each year.[1] While the disease is generally self-limiting in healthy adults, deaths occur in infants and the elderly, usually due to life threatening complications associated with secondary bacterial infections such as pneumonia.[2] Annual vaccination is the primary option for the prevention of influenza, but efficacy relies on a good match between the strains currently circulating and those in the vaccine, and is estimated to be 60%–90% in children and healthy adults, but only 50%–60% in the elderly.[3] In Australia, influenza vaccine uptake within the elderly population (aged ≥ 65 years) is high, with states such as Victoria having an estimated 74.5% coverage in 2002.[4] In comparison, influenza vaccination of Australian children aged six months to five years is extremely low. Influenza vaccination of this age group is neither recommended nor funded by the Australian Government.

While influenza vaccination continues to be the most cost effective means of influenza prevention in the community, antiviral drugs provide an additional control measure for either the treatment or short-term prevention of influenza. Two classes of influenza antivirals exist – the adamantanes, or M2 ion channel inhibitors, and the neuraminidase (NA) inhibitors. The adamantanes, including amantadine and rimantadine, were first shown to be inhibitory for influenza A viruses in 1964,[5] and since this time have been widely used in many countries around the world, although amantadine is rarely used for influenza treatment in Australia and rimantadine is not licensed. Resistance to this class of drugs has however been identified with a high frequency, particularly in recent years where over 90% of A(H3N2) strains from North America and China were found to be resistant.[6] Adamantane resistant viruses have demonstrated no reduction in viral fitness compared to wild type viruses, and may in fact have some selective advantage given that adamantane resistance in A(H3N2) viruses is greater than 50% in Australia, even though very little of the drug is used.[7,8]

As a result of the high level of adamantane resistant viruses currently circulating, more emphasis has been placed on the role of the newer class of influenza antiviral drugs, the NA inhibitors (NAIs), in the management of influenza. This class of influenza drugs has two currently licensed products, zanamivir and oseltamivir, which are available in many countries, including Australia.[9] These inhibitors act by binding to the NA and preventing the release of newly formed virions from the host cell, disrupting further replication of the virus. Clinical studies and post marketing surveillance have shown that the frequency of NAI is significantly lower than that observed for the adamantanes.[9] Clinical

studies identified 1%–4% resistance in oseltamivir-treated adults,[10] while higher levels of resistance (5%–6%) were observed in oseltamivir treated children.[11] However more recent studies have identified levels of resistance as high as 16%–18% in oseltamivir-treated children.[12,13] Only one incident of significant resistance has been reported following zanamivir treatment and this was concerning an immunocompromised patient,[14] although this low incidence of resistance to zanamivir may be a result of the low usage of this drug compared to oseltamivir. In addition to the occurrence of NAI resistance following oseltamivir treatment, resistant strains have also been isolated from untreated individuals,[15] including one strain isolated from an 8-month old infant in Australia.[16] As NAI resistance has been more common in oseltamivir-treated children than in adults, together with our previous finding of a naturally occurring NAI resistant virus in Australia isolated from an infant, we decided to investigate further whether there was any variation in the levels of viral NAI resistance from influenza cases in different age cohorts. Given the low levels of NAI usage, it was assumed that all or nearly all of the samples that were tested in the study came from untreated patients. To achieve this, over 1,000 influenza viruses isolated between 2001 and 2006 were tested for their susceptibility to the NAI drugs and the data analysed based on the age of the patient.

## Materials and methods

### Viruses

All influenza viruses tested in this study were submitted to the WHO Collaborating Centre for Reference and Research on Influenza as part of the WHO global influenza surveillance program. Only specimens with patient age data available were included in the study (approximately 70% of the specimens or isolates received at the Centre). A total of 1,097 influenza viruses from Australasia (Australia 468, and New Zealand 150), South East Asia (Thailand 209, Philippines 45, Singapore 108, Vietnam 1, Cambodia 1, and Indonesia 14), South Pacific (New Caledonia 19, Solomon Islands 4, and Fiji 1), Eastern Asia (Republic of Korea 16, and Taiwan 45), and South Africa (16) were tested for their susceptibility to the NAIs. The number of viruses isolated from patients from different age groups and from different regions is shown in Table 1. The viruses tested were made up of influenza A(H1N1) (288 isolates), A(H1N2) (5 isolates), A(H3N2) (535 isolates) and influenza B strains (270 isolates). All viruses were isolated and passaged in Madin-Darby canine kidney (MDCK) cells [American Type Culture Collection (CCL-34)] maintained in DMEM Coons Basal Medium containing sodium bicarbonate (3%) with the addition of 2 mM glutamine, 1% non-essential amino acids, 0.05% $NaHCO_3$, 0.02M HEPES, 4% penicillin and streptomycin, 2 µg/ml amphotericin B and 4 µg/ml trypsin (all media were obtained from CSL Limited, Australia) and tested within a maximum of five passages from isolation.

### NA inhibitors

Zanamivir was used directly from the blister packaging of Relenza (5 mg zanamivir and 20 mg lactose) (GlaxoSmithKline) as distributed through pharmacies. Oseltamivir carboxylate (GS 4071), the active form of the ethyl ester prodrug oseltamivir phosphate, was kindly provided by Dr James Smith, F Hoffmann-La Roche Ltd, Basal, Switzerland. Each of the drugs was dissolved in assay buffer and stored for up to three months as a stock solution at 4°C prior to use.

### NA inhibition assay

A fluorescence-based NAI assay was used to determine the sensitivity of viruses to the NAI compounds. The assay was based on the release of the fluorescent product 4-methylumbelliferone from the substrate 2-(4-methylumbelliferyl)-a-D-N-acetylneuraminic acid (MUNANA) as a measure of NA activity.[17] Methods followed those described previously.[18] The data were plotted as the percentage of fluorescence activity inhibited against the log NA inhibitor concentration. A logistic curve fit program (kindly provided by Dr Trevor Rae, Roche Products, Welwyn Garden City) was used to produce a curve of best fit and calculate an $IC_{50}$ value for each virus. Known susceptible viruses and known resistant viruses were used as controls in each assay.

### RT-PCR and sequencing

RNA extraction was performed using the RNEasy kit (QIAGEN) and RT-PCR was performed using the SuperScript III One-Step RT-PCR System with Platinum Taq DNA Polymerase (Invitrogen, Australia) according to the manufacturer's protocol. Sequencing was performed using a Big Dye III kit (Perkin Elmer) and an ABI 310 genetic analyser (Institute of Medical and Veterinary Science, Adelaide). Nucleotide sequences were analysed using DNASTAR V.5 (Lasergene, USA).

## Results

One thousand and ninety-eight influenza viruses isolated between 2001 and 2006 were analysed for their susceptibility to the NAIs zanamivir and oseltamivir carboxylate. The data from this study demonstrated small differences in NA inhibitor susceptibility between the N1, N2 and B neuraminidases to both antiviral agents zanamivir and oseltamivir carboxylate (Table 2), similar to that

**Table 1. Number of viruses isolated from patients from different age groups and regions**

| Region | Age group of patient | | | |
|---|---|---|---|---|
| | <2 | 2–17 | 18–50 | >50 |
| Australasia | 270 (69%) | 155 (39%) | 129 (59%) | 64 (71%) |
| South East Asia | 108 (27%) | 185 (47%) | 64 (29%) | 21(23%) |
| South Pacific | 5 (1%) | 12 (3%) | 6 (3%) | 1 (1%) |
| East Asia | 6 (2%) | 40 (10%) | 14 (6%) | 2 (2%) |
| South Africa | 4 (1%) | 5 (1%) | 5 (2%) | 2 (2%) |
| Total | 393 | 397 | 218 | 90 |

reported previously.[18] $IC_{50}$ values (concentration of antiviral required to inhibit NA activity by 50%) for each virus were compared based on the age of the patient, to determine both the mean NAI susceptibility and the number of viruses within that cohort that had an $IC_{50}$ value that differed by 10-fold or greater from the mean $IC_{50}$ value (termed an 'outlier'). Of the total number of viruses analysed, only 2 (0.2%) strains were considered to be outliers to at least one of the NAIs (Table 3). Interestingly, both of these outliers were from patients in the youngest age cohort (less than 2 years) (Table 2). However, when the $IC_{50}$ data for the outlying strains were removed from the overall analysis (Table 2), the mean $IC_{50}$ values were very similar between all age cohorts for all subtypes and against both NAIs.

Of the two strains with raised $IC_{50}$ values, there was no predominance of one NA subtype, with one outlier being detected in each of the N1 and B NA subtypes. The outlying virus with the highest $IC_{50}$ was an A(H1N1) strain designated as A/Victoria/124/2005. This virus was isolated from a 15-month-old infant who had presented to a general practitioner with cough and fever on 7 June 2005. As part of the Victorian GP sentinel influenza surveillance program,[19] a combined nose and throat swab was taken from the patient and sent in viral transport medium to the Victorian Infectious Diseases Reference Laboratory for respiratory virus identification. Following influenza A detection by RT-PCR, the specimen was forwarded to the WHO Collaborating Centre for Reference and Research on Influenza for further analysis. After a single passage in MDCK cells the viral isolate was tested for NAI susceptibility and this revealed an $IC_{50}$ value for oseltamivir carboxylate that was approximately 900-fold higher than the mean oseltamivir carboxylate $IC_{50}$ value for other N1 viruses. The virus however was found to be fully sensitive to zanamivir ($IC_{50}$ = 0.4 nM). Sequence analysis of the NA gene from the virus revealed a His274Tyr mutation. Interestingly, the patient from whom the specimen was taken had not been treated with either NAI. It could not be determined if the child had contracted influenza from a drug treated individual.

The other outlier, B/Perth/211/2001, had significantly less resistance than was observed for A/Victoria/124/2005. B/Perth/211/2001, which we have reported previously,[16] demonstrated a 7-fold increase in $IC_{50}$ to zanamivir and an 18-fold increase to oseltamivir carboxylate. This virus was isolated from an 8-month-old infant from Western Australia who had also not been treated with either zanamivir or oseltamivir. Initial sequence analysis of the virus did not reveal any NA mutations. However on further analysis the isolate was found to contain a mixed viral population, from which a proportion contained a Asp197Glu amino acid mutation in the NA gene.[16]

## Discussion

The NAIs were the first class of drugs to be specifically designed for the treatment or prevention of influenza. However since their release into the market in 1999 there has been limited use of these drugs in most countries except Japan and the United States of America. In Japan, up to 18% of oseltamivir treated patients have been shown to shed resistant influenza viruses but, unlike the spread of adamantane resistant influenza viruses into populations where little drug is being used, there is little evidence of the subsequent spread of NAI resistant strains to other individuals. The results from this study found that only 0.2% of the influenza strains tested were found to have a significant reduction in sensitivity to at least one of the NAIs. While it is not possible to be definitive, it is highly likely based on the patients' records (both patients were confirmed not to have received either NAI drugs) and the low usage of NAI drugs in Australia, that these resistant strains did not arise as a result of drug selective pressure.

Although the $IC_{50}$ values of the two strains in this study were found to be significantly higher than the other viruses tested, one of these, B/Perth/211/2001, would probably not be considered clinically significant, as the $IC_{50}$ levels of this virus are exceeded following administration of the normal dosage of either drug.[20,21] However, the A/Victoria/124/2005 strain had an $IC_{50}$ value (585 nM for oseltamivir carboxylate) which was higher than the reported

## Table 2. NA inhibitor susceptibility of viruses taken from patients of different age cohorts

| Age | N1 | | | | N2* | | | | B | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | n = | Mean $IC_{50}$ ± 1 S.D. (nM)† Zanamivir | Mean $IC_{50}$ ± 1 S.D. (nM)† Oseltamivir carboxylate | Number of outliers‡ | n = | Mean $IC_{50}$ ± 1 S.D. (nM)† Zanamivir | Mean $IC_{50}$ ± 1 S.D. (nM)† Oseltamivir carboxylate | Number of outliers‡ | n = | Mean $IC_{50}$ ± 1 S.D. (nM)† Zanamivir | Mean $IC_{50}$ ± 1 S.D. (nM)† Oseltamivir carboxylate | Number of outliers‡ |
| <2 | 60 | 0.37 ± 0.19 | 0.64 ± 0.47 | 1 | 240 | 1.08 ± 1.07 | 0.35 ± 0.27 | 0 | 93 | 2.05 ± 1.08 | 13.24 ± 11.19 | 1 |
| 2–17 | 133 | 0.36 ± 0.27 | 0.69 ± 0.53 | 0 | 152 | 1.09 ± 0.66 | 0.31 ± 0.20 | 0 | 112 | 1.77 ± 1.26 | 15.49 ± 12.05 | 0 |
| 18–50 | 77 | 0.34 ± 0.30 | 0.64 ± 0.83 | 0 | 97 | 0.90 ± 0.29 | 0.29 ± 0.24 | 0 | 44 | 1.72 ± 1.42 | 8.02 ± 7.55 | 0 |
| >50 | 18 | 0.36 ± 0.27 | 0.68 ± 0.52 | 0 | 51 | 1.02 ± 0.80 | 0.32 ± 0.20 | 0 | 21 | 2.12 ± 1.63 | 10.08 ± 9.77 | 0 |
| Total | 288 | 0.36 ± 0.26 | 0.67 ± 0.62 | 1 | 540 | 1.02 ± 0.65 | 0.32 ± 0.24 | 0 | 270 | 1.88 ± 1.26 | 13.06 ± 11.23 | 1 |

* IC50 values of the five A(H1N2) viruses were not significantly different from the values of the A(H3N2) isolates.

† Mean IC50 value calculation does not include IC50 values of outliers.

‡ Outlier defined as a virus with an IC50 at least 10–fold greater than the mean of other viruses with the same NA subtype.

## Table 3. NA inhibitor susceptibility of outlier influenza isolates with $IC_{50}$ values at least 10-fold greater than the mean

| Designation | Subtype | Age of patient | Zanamivir | | Oseltamivir carboxylate | |
|---|---|---|---|---|---|---|
| | | | $IC_{50}$ (nM) | Fold difference* | $IC_{50}$ (nM) | Fold difference* |
| A/Victoria/124/2005 | H1N1 | 12 months | 0.4 ± 0.2 | – | 585.8 ± 61.2 | 874-fold |
| B/Perth/211/2001 | B | 8 months | 13.8 ± 1.7 | 7-fold | 233.9 ± 31.8 | 18-fold |

* Fold-difference calculated by comparing the IC50 of the isolate with the mean IC50 of viruses with the same NA subtype.

oseltamivir carboxylate steady-state plasma Cmin of 138 ng/ml (approximately 485 nM) achieved with the normal 75 mg twice daily oseltamivir dosing.[21] This suggests that oseltamivir carboxylate treatment of the patient with this strain may not be effective. The amino acid mutation His274Tyr observed in the NA gene of A/Victoria/124/2005 has been the most commonly NA mutation detected in NAI resistant N1 viruses, which includes both the commonly circulating human A(H1N1) viruses as well as highly pathogenic A(H5N1) influenza viruses.[9] To date there have been three reported cases of oseltamivir resistant A(H5N1) viruses that have been isolated from infected Vietnamese patients undergoing oseltamivir treatment.[22,23] In all cases the resistance was due to the His274Tyr mutation in the NA gene, the same mutation that was detected in the A/Victoria/124/2005 strain in this study. It should be remembered however that the resistance mutation in the strain from Victoria appears to have occurred spontaneously in an untreated individual, unlike the A(H5N1) viruses where resistance was likely to have been generated as a result of drug selective pressure. While A(H1N1) or A(H5N1) viruses with a His274Tyr NA mutation are resistant to oseltamivir, these viruses remain fully sensitive to the other NAI, zanamivir (the zanamivir $IC_{50}$ of A/Victoria/124/2005 was 0.4nM). As a result, zanamivir may be a better option for the treatment of patients who shed these viruses, although this drug is only licensed for use in patients aged five years or older.

Two previous studies have investigated the impact that a His274Tyr NA mutation has had on the infectivity and transmissibility of viruses in ferrets. While one study found that mutant viruses had significantly compromised fitness[24] (as is the case with many other NAI resistant mutants[9]), a second study found that transmissibility was possible, although a higher dose of the virus was necessary for infection compared to the wild type virus.[25] If the data from this second ferret study are indicative of the fitness of this virus in humans, it is therefore possible that infants may be able to facilitate the replication and transmission of NAI resistant strains better than adults due to the high titres and prolonged duration of influenza virus shed by individuals in the younger age group.[26,27] While further work is necessary to understand the risk that the two strains (with high $IC_{50}$) identified in this study may pose if they become widely circulating in the human population, it is of note that all of these isolates were from children aged 18 months or less. Given these results and the previous reports showing the significantly higher incidence of drug resistance in oseltamivir treated children compared to adults, it essential in the future to enhance NAI susceptibility testing in both treated and untreated individuals within this young age group.

## Acknowledgments

The authors would like to thank Heath Kelly, Joy Turner and Chris Birch of the Victorian Infectious Diseases Reference Laboratory, Victoria and Peter McMinn of the Princess Margaret Hospital for Children, Western Australia, for submitting the viruses A/Victoria/124/2005 and B/Perth/211/2001 and for gathering epidemiological information related to these viruses. In addition, the authors would like to thank all of the other laboratories that have submitted samples used in this study to the WHO Collaborating Centre. The Melbourne WHO Collaborating Centre for Reference and Research on Influenza is supported by the Australian Government Department of Health and Ageing.

## Author details

Aeron C Hurt[1,2]
Ian G Barr[1,2]

1. WHO Collaborating Centre for Reference and Research on Influenza, Parkville, Victoria
2. Monash University, School of Applied Sciences, Churchill, Victoria

Corresponding author: Mr Aeron Hurt, WHO Collaborating Centre for Reference and Research on Influenza, 45 Poplar Road, PARKVILLE VIC 3052. Telephone: +61 3 9389 1392. Facsimile: +61 3 9389 1881. Email: aeron.hurt@influenza-centre.org

## References

1. Simonsen L. The global impact of influenza on morbidity and mortality. *Vaccine* 1999;17:S3–S10.
2. Simonsen L, Clarke MJ, Williamson GD, Stroup DF, Arden NH, Schonberger LB. The impact of influenza epidemics on mortality: introducing a severity index. *Am J Pub Hlth* 1997;87:1944–1950.
3. Nichol KL. The efficacy, effectiveness and cost-effectiveness of inactivated influenza virus vaccines. *Vaccine* 2003;21:1769–1775.
4. Andrews RM, Skull SA, Byrnes GB, Campbell DA, Turner JL, McIntyre PB, et al. Influenza and pneumococcal vaccine coverage among a random sample of hospitalised persons aged 65 years or more, Victoria. *Commun Dis Intell* 2005;29:283–288.
5. Davies WL, Grunert RR, Haff RF, McGahen JW, Neumayer EM, Paulshock M, et al. Antiviral activity of 1-adamtanamine (amantadine). *Science* 1964;144:862–863.
6. Bright RA, Shay DK, Shu B, Cox NJ, Klimov AI. Adamantane resistance among influenza A viruses isolated early during the 2005–2006 influenza season in the United States. *JAMA* 2006;295:891–894.
7. Barr IG, Hurt AC, Iannello P, Tomasov C, Deed N, Komadina N. Increased adamantane resistance in influenza A(H3) viruses in Australia and neighbouring countries in 2005. *Antiviral Res* 2007;73:112–117.
8. Simonsen L, Viboud C, Grenfell BT, Dushoff J, Jennings L, Smit M, et al. The genesis and spread of reassortment human influenza A/H3N2 viruses conferring adamantane resistance. *Mol Biol Evol* 2007;24:1811–1820.

9. Hurt AC, Ho HT, Barr I. Resistance to anti-influenza drugs: adamantanes and neuraminidase inhibitors. *Expert Rev Anti Infect Ther* 2006;4:795–805.
10. Gubareva LV, Kaiser L, Matrosovich MN, Soo-Hoo Y, Hayden FG. Selection of influenza virus mutants in experimentally infected volunteers treated with oseltamivir. *J Infect Dis* 2001;183:523–531.
11. Whitley RJ, Hayden FG, Reisinger KS, Young N, Dutkowski R, Ipe D, et al. Oral oseltamivir treatment of influenza in children. *Pediatr Infect Dis J* 2001;20:127–133.
12. Kiso M, Mitamura K, Sakai-Tagawa Y, Shiraishi K, Kawakami C, Kimura K, et al. Resistant influenza A viruses in children treated with oseltamivir: descriptive study. *Lancet* 2004;364:759–765.
13. Ward P, Small I, Smith J, Suter P, Dutkowski R. Oseltamivir (Tamiflu(R)) and its potential for use in the event of an influenza pandemic. *J Antimicrob Chemother* 2005;55 Suppl 1:i5–i21.
14. Gubareva LV, Matrosovich MN, Brenner MK, Bethell RC, Webster RG. Evidence for zanamivir resistance in an immunocompromised child infected with influenza B virus. *J Infect Dis* 1998;178:1257–1262.
15. Monto AS, McKimm-Breschkin JL, Macken C, Hampson AW, Hay A, Klimov A, et al. Detection of influenza viruses resistant to neuraminidase inhibitors in global surveillance during the first 3 years of their use. *Antimicrob Agents Chemother* 2006;50:2395–2402.
16. Hurt AC, Iannello P, Jachno K, Komadina N, Hampson AW, Barr IG, et al. Neuraminidase inhibitor-resistant and -sensitive influenza B viruses isolated from an untreated human patient. *Antimicrob Agents Chemother* 2006;50:1872–1874.
17. Potier M, Mameli L, Belisle M, Dallaire L, Melancon SB. Fluorometric assay of neuraminidase with a sodium (4-methylumbelliferyl-alpha-D-N-acetylneuraminate) substrate. *Anal Biochem* 1979;94:287–296.
18. Hurt AC, Barr IG, Hartel G, Hampson AW. Susceptibility of human influenza viruses from Australasia and South East Asia to the neuraminidase inhibitors zanamivir and oseltamivir. *Antiviral Res* 2004;62:37–45.
19. Turner JL, Fielding JE, Clothier HJ, Kelly HA. Influenza surveillance in Victoria, 2005. *Commun Dis Intell* 2006;30:137–143.
20. Peng AW, Milleri S, Stein DS. Direct measurement of the anti-influenza agent zanamivir in the respiratory tract following inhalation. *Antimicrob Agents Chemother* 2000;44:1974–1976.
21. Roche Pharmaceuticals. Tamiflu (Oseltamivir carboxylate) Capsules and for Oral Suspension. *Package Insert* 2006. Available from: www.tamiflu.com
22. Le QM, Kiso M, Someya K, Sakai YT, Nguyen TH, Nguyen KH, et al. Avian flu: isolation of drug-resistant H5N1 virus. *Nature* 2005;437:1108.
23. de Jong MD, Tran TT, Truong HK, Vo MH, Smith GJ, Nguyen VC, et al. Oseltamivir resistance during treatment of influenza A (H5N1) infection. *N Engl J Med* 2005;353:2667–2672.
24. Ives JAL, Carr JA, Mendel DB, Tai CY, Lambkin R, Kelly L, et al. The H274Y mutation in the influenza A/H1N1 neuraminidase active site following oseltamivir phosphate treatment leave virus severely compromised both *in vitro* and *in vivo*. *Antiviral Research* 2002;55:307–317.
25. Herlocher ML, Truscon R, Elias S, Yen HL, Roberts NA, Ohmit SE, et al. Influenza viruses resistant to the antiviral drug oseltamivir: transmission studies in ferrets. *J Infect Dis* 2004;190:1627–1630.
26. Frank AL, Taber LH, Wells CR, Wells JM, Glezen WP, Paredes A. Patterns of shedding of myxoviruses and paramyxoviruses in children. *J Infect Dis* 1981;144:433–441.
27. Hall CB, Douglas RGJ, Geiman JM, Meagher MP. Viral shedding patterns of children with influenza B infection. *J Infect Dis* 1979;140:610–613.

# Higher than expected seasonal influenza activity in Victoria, 2007

Emma R Miller, James E Fielding, Kristina A Grant, Ian G Barr, Georgina Papadakis, Heath A Kelly

## Abstract

In 2007, the Victorian influenza season exceeded normal seasonal activity thresholds. The average rate of influenza-like illness (ILI) reported by general practitioners (GPs) participating in sentinel surveillance was 9.0 cases per 1,000 consultations, peaking at 22 cases per 1,000 consultations in mid-August. The average ILI rate reported by the Melbourne Medical Locum Service (MMLS) was 11.5 per 1,000 consultations over the season. The MMLS ILI rate peaked at 30 per 1,000 consultations at the same time as peak rates were reported by GPs, with a secondary peak observed three weeks later (22 cases per 1,000 consultations). Influenza cases notified to the Victorian Department of Human Services peaked in mid-August with a secondary peak of influenza A in early September. Of the influenza positive swabs collected by GPs and among those collected throughout the state, 92% were type A and 8% were type B. The most common strains identified in Victoria in the 2007 influenza season were A/Brisbane/10/2007-like followed by A/Solomon Islands/3/2006-like. While neither virus strain was specifically included in the 2007 Australian influenza vaccine, reasonable cross protection was afforded by the strains in the vaccine. *Commun Dis Intell* 2008;32:63–70.

Keywords: surveillance, epidemiology, influenza

## Introduction

A sentinel general practice (GP) program for the surveillance of influenza like illness (ILI) has been conducted in Victoria since 1993. Laboratory testing for cases meeting selection criteria was introduced to the GP sentinel system in 1998.[1] The Victorian Infectious Diseases Reference Laboratory (VIDRL) also monitors diagnoses of ILI made by GPs in the Melbourne Medical Locum Service (MMLS). Under the *Health (Infectious Disease) Regulations 2001,*[2] the Department of Human Services (DHS) coordinates the surveillance of laboratory notifications of influenza. These three elements (sentinel GP surveillance, MMLS monitoring and DHS surveillance) constitute the Victorian Influenza Surveillance System.

Supplementing the Victorian Influenza Surveillance System, the World Health Organization (WHO) Collaborating Centre for Reference and Research on Influenza (also based in Melbourne) undertakes strain typing of influenza isolates and influenza specimens identified through the GP sentinel surveillance programs as well as those forwarded by two Melbourne hospital-based laboratories.

The Victorian influenza surveillance system aims to:

- monitor the occurrence of laboratory-confirmed influenza in Victoria;
- describe the epidemiology of influenza in Victoria – identifying the onset, duration and magnitude of each influenza season; and
- characterise the strains of influenza in the community to assist in monitoring the impact of the current vaccine and the formulation of vaccines for the subsequent season.

In this paper we summarise findings from the Victorian Influenza Surveillance System in 2007.

## Methods

### General practice sentinel surveillance

Participating GPs were asked to report weekly on the total number of consultations they had for the week, and the age, sex and vaccination status of any patients presenting with ILI. In line with accepted case criteria, ILI was defined as history of fever, cough and fatigue/malaise.[3] Nose and throat swabs were offered to patients presenting within three days of the onset of their symptoms. Once formal consent was obtained from these patients, GPs collected data on their age, sex, symptoms (fever, cough, fatigue, myalgia, other) and vaccination status. GPs were also asked to provide an indication of their confidence in their clinical diagnosis of influenza ('almost certain,' 'probable' or 'less likely'). Registered as approved activities by the Royal Australian College of General Practitioners and the Australian College of Rural and Remote Medicine, GPs were offered Continuing Professional Development for their participation in the program.

Swabs were transported to VIDRL by courier where they were tested using an in-house respiratory multiplex polymerase chain reaction (PCR) test identifying influenza, adenovirus, picornavirus (enterovirus and rhinovirus), respiratory syncytial virus and parainfluenza viruses.

A development of a previous assay,[4] the current test uses type specific primers that identify influenza B and all influenza A sub-types.

Data for the 22-week period 30 April to 30 September 2007 were analysed using the set of threshold values previously established for Victorian influenza seasons.[5] Rates of ILI cases per 1,000 consultations for baseline activity, normal seasonality and higher than expected activity were defined as below 2.5, between 2.5 and <15, and between 15 and <35 respectively. According to these thresholds, 'epidemic influenza activity' is defined by rates exceeding 35 cases per 1,000 consultations.

The recommendation of the *Framework for an Australian Influenza Pandemic Plan* (1999)[6] was for sentinel surveillance to achieve recruitment coverage of approximately one practice per 200,000 in metropolitan areas and one practice per 100,000 population in rural areas. These recommendations, however, do not take into account the number of GPs participating in each practice or the fraction of time each GP spends consulting. We have previously suggested that a relatively small number of practices with better response rates is able to provide data on seasonal influenza activity that are as useful as those provided by a larger number of practices with poorer participation.[7] We demonstrated that the number of GP consultations per population in a given region better measured geographic representation than the number of practices per population, and we proposed that a small number of 'dedicated' participating sites seeing around 2,000 consultations per 100,000 population over the influenza season should be used as the recruiting target.

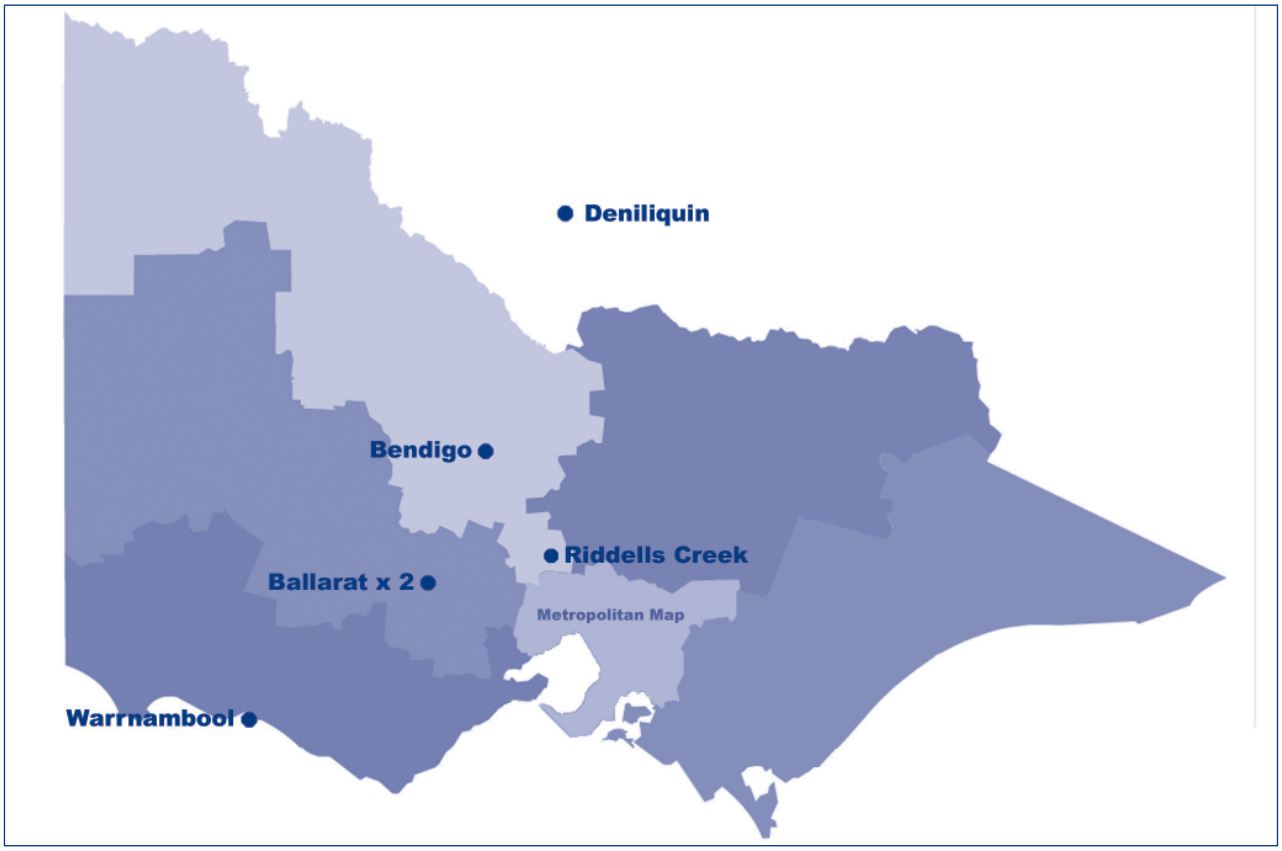
In 2007 there were 50 GPs participating in 15 metropolitan practices and 15 GPs participating in six rural practices. The geographical locations of recruited practices are presented in Figures 1a and 1b.

### Melbourne Medical Locum Service

The largest medical locum service in Australia, the Melbourne Medical Locum Service operates a 24-hour service throughout metropolitan Melbourne each day. Weekly searches of the clinical database maintained by the MMLS, using the search terms 'influenza' or 'flu,' provides data on the number of influenza-related diagnoses made by MMLS GPs. These data and the total number of consultations made by the MMLS are available from the password protected MMLS website.

**Figure 1a. Distribution of sentinel surveillance sites in metropolitan Victoria 2007**

**Figure 1b. Distribution of sentinel surveillance sites in rural Victoria 2007**




### Notified laboratory-confirmed influenza

Under the *Health (Infectious Diseases) Regulations 2001,*[2] medical practitioners and pathology services are required to notify confirmed influenza cases to the DHS within five days of the positive test. Data on cases notified during the surveillance period were identified in the DHS Notifiable Infectious Disease Surveillance database and extracted for analysis on 22 October 2007.

### Data collation and reporting

As well as requesting laboratory tests, GPs reported ILI cases to VIDRL by facsimile, each week. Data on influenza related diagnoses were extracted from the MMLS database each week. Weekly information was reported to the DHS Communicable Disease Control Unit and to the Australian Government Department of Health and Ageing. Every two weeks, structured reports on the previous fortnight were distributed to all participating GPs, state and Australian health departments and other interested health professionals and health agencies, and were also made available on the VIDRL web site (at http://www.vidrl.org.au). Summary reports of (laboratory-confirmed) influenza notifications were updated daily and posted on the DHS Communicable Disease Control Unit web site (http://www.health.vic.gov.au/ideas/surveillance/daily.htm).

## Results

### ILI surveillance

Across the 22 weeks of surveillance, an average of 95% (61/65) of GPs returned tally sheets to VIDRL each week (range 86% to 98%). GPs reported having conducted 115,646 consultations and identified 1,045 ILI cases during the season – an overall rate of 9.0 per 1,000 consultations. From a baseline rate averaging around 3.0 cases per 1,000 consultations in the first weeks of surveillance, the rate peaked in mid-August (week beginning 20 August) at 21.9 cases per 1,000 consultations, before declining to 7.1 ILI cases per 1,000 consultations at the end of the surveillance period – over two times the baseline rate. There appeared to be a differential metro–rural pattern of disease in 2007 (Figure 2). The rural peak (25.6 ILI cases per 1,000 consultations) occurred a week earlier than the metropolitan peak, and declined less steeply before a secondary peak was reported at the end of the surveillance period, or the last week in September (16.4 ILI cases per 1,000 consultations). In 2007, overall ILI rates reported by GPs did not return to baseline until mid-October (data not shown).

Among the total ILI cases reported by GPs, 55% (572/1,045) were female and 45% (472/1,045) were male. The median age of ILI cases was 32 years

**Figure 2. Weekly influenza-like illness rates reported, Victoria, 2007, by rural and metropolitan practices**

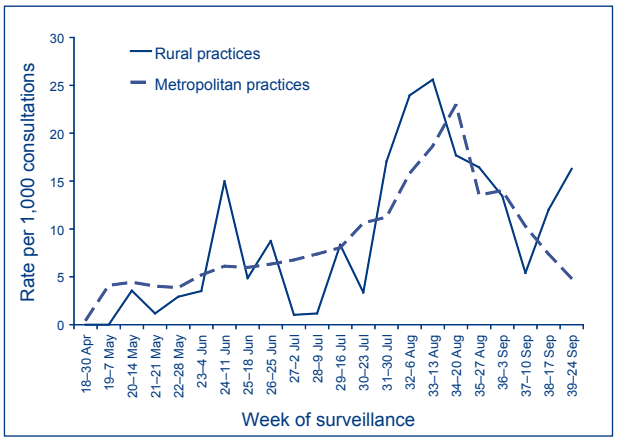
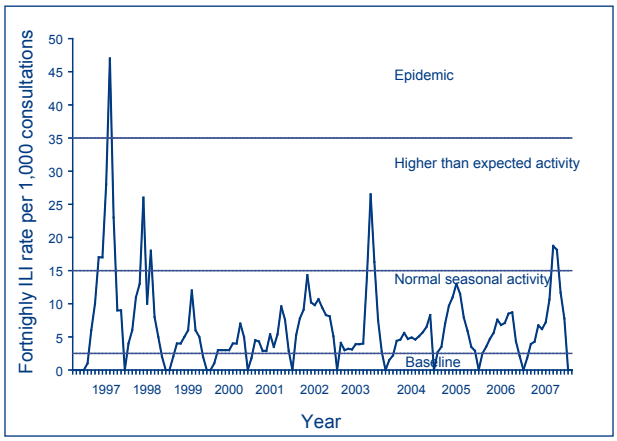



**Figure 3. Fortnightly GP sentinel surveillance influenza-like illness rates, Victoria, seasons 1997 to 2007**




(range one to 95 years) and 82% (854/1,045) were reported as being unvaccinated for the season. Among the cases aged 65 years or older, 16% (11/69) were unvaccinated.

Among consultations conducted by the MMLS during the 2007 season, 362 patients were diagnosed with 'flu' or 'influenza.' The average ILI rate in 2007 reported by the MMLS was 11.5 cases per 1,000 consultations. ILI rates followed a similar pattern to that reported by rural GPs with a peak over two weeks in mid-August (30.0 ILI cases per 1,000 consultations) and a secondary peak in mid-September (21.4 ILI cases per 1,000 consultations). By the final week of the surveillance period the ILI rate per 1,000 consultations had declined to 8.4, but did not return to baseline until mid-October (data not shown).

Using the previously described thresholds for GP sentinel surveillance in Victoria, ILI rates in 2007 exceeded those of the previous three seasons and, for the first time since 2003, were in the range of 'higher than expected activity' (Figure 3). The peak was not as high as earlier seasons in which higher than expected activity was reported (i.e. 1997, 1998 and 2003).

### Laboratory surveillance

Participating GPs submitted a total of 403 patient swabs to VIDRL, with a median of five swabs submitted per GP (range one to 24 swabs). Overall, 62% (248/403) of the total swabs tested positive to any of the respiratory viruses forming part of the multiplex PCR. As can be seen in Table 1, 67% (165/248) of those testing positive for respiratory viruses were positive for influenza A and 10% (24/248) were influenza B positive (including two swabs positive for both influenza A and B). Over 80% of influenza diagnoses were made between weeks 29 to 36 (16 July to 9 September). Approximately 17% (43/165) of the swabs tested positive for picornavirus, the diagnoses of which were distributed relatively equally throughout the surveillance period. The overall positive predictive value (PPV) for all clinical diagnoses of influenza made by all GPs was 47% (the median for individual GPs was 33%). PPV rose with the certainty of the diagnosis, with those reporting they were 'almost certain' of their clinical diagnosis achieving an overall positive predictive value of 64% (individual GP median 69%). PPV for the years 2002 to 2007 are presented in Table 2. Although not apparent in the 2003 season, also a year of above expected activity, PPVs can ordinarily be expected to increase with increasing prevalence of influenza in the population.[8]

Among those testing positive for influenza A or B, 45% (83/185) of the specimens were provided by female patients and 55% (102/185) by male patients. The median age of these patients was 27 years (range 0 to 92 years) and 70% (128/183) reported being unvaccinated for the 2007 season, 11% (21/183) reported being vaccinated and 19% (34/183) had unknown vaccination status.

VIDRL provided approximately 55% of all notifications to DHS during the surveillance period, with 12 other Victorian laboratories providing notifications for approximately 51% of cases; the sum of the percentages is greater than 100% because some cases were tested and notified by multiple laboratories. During the surveillance period, a total of 1,343 laboratory-confirmed cases of influenza were notified to the DHS, representing a 256% increase on the numbers notified during the 2006 surveillance period (377 cases). Approximately 15% of this total included the laboratory-confirmed cases identified through GP sentinel surveillance, and around 8% were identified during outbreak investigations. Around 54% (723/1,343) of the notifications were in

**Table 1. Respiratory viruses detected from general practice sentinel surveillance influenza-like illness patient swabs, 2007**

| Respiratory virus | n detected | % detected (total swabs) | % detected (PCR positive swabs) |
|---|---|---|---|
| Influenza A | 165 | 41 | 67 |
| Influenza B | 24 | 6 | 10 |
| Picornavirus | 43 | 11 | 17 |
| Adenovirus | 4 | 1 | 2 |
| Parainfluenza virus | 4 | 1 | 2 |
| Respiratory syncytial virus | 8 | 2 | 3 |
| Total | 248 | 62 | 100 |

**Table 2. Positive predictive value of clinical diagnoses of influenza, 2002 to 2007, by general practitioner certainty of diagnosis**

| Year of surveillance | General practice certainty of diagnosis – number laboratory confirmed (PPV) | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Almost certain | | Probable | | Less likely | | Not stated | | Total | |
| | n | PPV% | n | PPV% | n | PPV% | n | PPV% | n | PPV% |
| 2002 | 25 | 45 | 94 | 35 | 23 | 24 | 25 | 45 | 167 | 38 |
| 2003 | 87 | 45 | 73 | 29 | 4 | 9 | 20 | 38 | 184 | 34 |
| 2004 | 12 | 26 | 23 | 16 | 6 | 13 | 2 | 9 | 43 | 16 |
| 2005 | 74 | 61 | 90 | 41 | 8 | 15 | 10 | 43 | 182 | 43 |
| 2006 | 48 | 51 | 56 | 27 | 11 | 21 | 11 | 41 | 126 | 33 |
| 2007 | 85 | 64 | 91 | 44 | 5 | 13 | 8 | 38 | 189 | 47 |

females and 46% (611/1,343) were in males (sex was not reported in nine cases) and the median age was 31 years (range 0 to 97 years).

The majority of the DHS notifications—nearly 77%—resulted from clinical presentations not forming part of the surveillance system. Of the total cases, 86% (1156/1343) were influenza A, 14% (186/1,343) were influenza B and one case was notified with both influenza A and B. As can be seen in Figure 4, the number of notified influenza A cases increased in line with the MMLS and GP surveillance ILI rates, with a peak in mid-August (weeks 33 and 34). A secondary peak of influenza A occurred in early September (week 36), one week prior to the secondary peak in ILI cases diagnoses reported by the MMLS. Notifications for influenza B, which made up 16% of all influenza notifications in 2007, had a later peak in mid-September (weeks 37 and 38), which was preceded by a smaller peak in mid-August (week 34). A further peak in DHS notifications of laboratory-confirmed cases of influenza B occurred after the end of the surveillance period, in late October to early December (data not shown).

Figure 5 presents laboratory-confirmed influenza A and B are stratified according to source of identification. Broadly reminiscent of the bimodal presentation of DHS notifications, influenza A cases identified by the GP surveillance program peaked a week earlier than the DHS influenza A cases and plateaued two weeks ahead of the secondary DHS influenza A peak. The number of influenza B cases identified by GPs was too small to identify trends over time.

**Figure 4. Notified cases of laboratory-confirmed influenza and general practitioner sentinel surveillance and Melbourne Medical Locum Service influenza-like illness rates, Victoria, 30 April to 27 September 2007, by week**

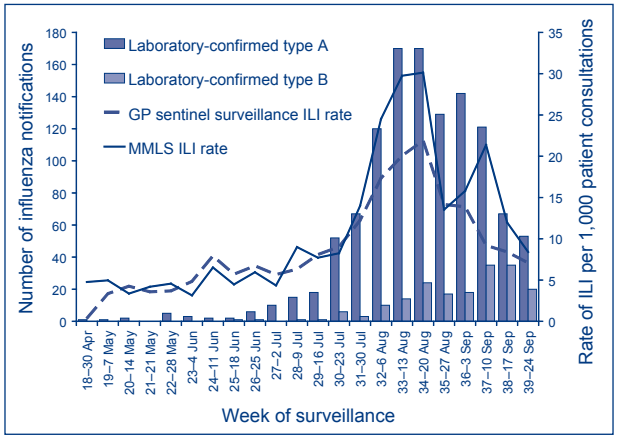
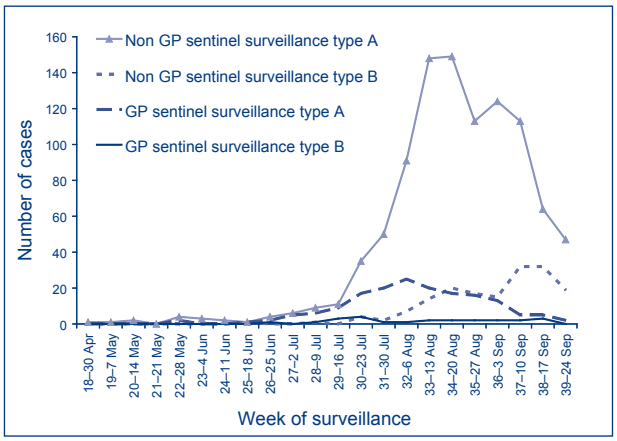
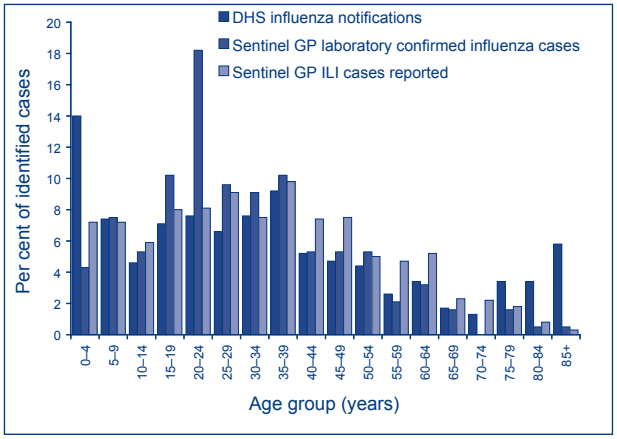


**Figure 5. Laboratory-confirmed influenza, Victoria, 30 April to 27 September 2007, by type and notification source**




The relative age distributions of cases according to notification source, DHS and GP sentinel surveillance (ILI cases and laboratory-confirmed cases) are presented in Figure 6. There were differences in the age distributions from the two surveillance sources. Around 14% (188/1,343) of the DHS notifications were in cases aged under five years, compared to 4% (8/187) of GP laboratory-confirmed cases and 7% (65/902) of ILI cases reported by GPs. Those aged 75 years or older made up 12% (168/1,343) of DHS notifications but only 3% of GP laboratory-confirmed cases (5/187) and GP reported ILI cases (26/902). GP laboratory-confirmed cases were most prevalent in those aged 20 to 24 years, who made up 18% (35/187) of cases from this source. In contrast, 8% of both DHS notifications (102/1,343) and GP ILI cases (73/902) were in the 20 to 24 year age group. Two cases among those notified during the surveillance period were reported to have died as a result of type A influenza virus infection: an 84-year-old female and a 91-year-old male. A further five cases aged five years and between 68 and 90 years were reported to have died with a type A influenza virus infection, but death was attributed to other causes.

**Figure 6. Laboratory-confirmed influenza and influenza-like illness cases, Victoria, 30 April to 27 September 2007, by proportion of age group and notification source**




A total of 105 isolates and 224 specimens collected in Victoria were referred to the WHO Collaborating Centre for Reference and Research on Influenza. Of these, 187 specimens (57%) were collected through GP sentinel surveillance. At time of writing, data were available from 170 of the influenza positive specimens sent (149 were influenza A, 21 influenza B). Only 24% (40/170) of specimens (mostly collected through the sentinel surveillance) yielded recoverable isolates. Of the 34 influenza A isolates, 56% (19/34) were A/Solomon Islands/3/2006-like (H1) and 29% (10/34) were A/Wisconsin/67/2005-like (H3) and (5/34) 15% were A/Brisbane/10/2007-like (H3). Three of the six influenza B samples were B/Malaysia/2506/2004-like, two were B/Shanghai/361/2002-like and one was B/Florida/4/2006-like.

Among the 329 specimens and isolates collected throughout Victoria during 2007 and referred to the WHO Centre, 50% (165/329) were recoverable of which 90% (148/165) were type A and 10% (17/165) were type B. Thirty-seven per cent (55/148) of the type A isolates were further characterised as H1N1 strains comprised of: A/Solomon Islands/3/2006-like (96%, including nine low reactors); and A/New Caledonia/20/99-like (4%). Approximately 63% (93/148) of the remaining influenza A isolates were H3N2 strains comprised of: A/Brisbane/10/2007-like (58%, including nine low reactors); and A/Wisconsin/67/2005-like [42%, most of which (30/39) were low reactors]. The 17 influenza type B isolates were designated as B/Florida/4/2006-like, 41% (7/17) and B/Shanghai/361/2002-like (low reactor), 35% (6/17) – giving a total of 76% B/Yamagata/16/88-like lineage virus. The remaining 24% (4/17) were B/Malaysia/2506/2004-like (low reactor), being of the B/Victoria/2/87-like lineage.

The Australian influenza vaccine for 2007 contained A/New Caledonia/20/99, A/Wisconsin/67/2005 and B/Malaysia/2506/67/2004 strains.[9]

## Outbreak investigations

The Communicable Disease Control Unit (DHS) investigated 24 respiratory outbreaks during the reporting period, of which 16 were confirmed as due to type A influenza virus, one was due to type B influenza virus, three were due to respiratory syncytial virus and a cause was unable to be established for four outbreaks. With the exceptions of a defence facility and rehabilitation centre, all notified influenza outbreaks occurred in aged care settings. Outbreaks occurred in aged care facilities, which accommodated between 24 and 107 residents, with

attack rates among residents ranging from 9% to 46%. These outbreaks were investigated and appropriate control measures implemented in accordance with the *Guidelines for the Control of Respiratory Disease Outbreaks in Aged Care Facilities in Victoria*.

## Discussion

Across Australia, more influenza cases were notified in 2007 relative to the past few years. The 2007 year-to-date national laboratory-confirmed notifications to the end of October were increased by close to 240% on the previous year and were 3.4 times the five-year mean number of notifications.[10] Consistent with the national influenza season, Victoria experienced a 270% increase in notifications this year compared with the same period in 2006 and ILI rates from GP sentinel surveillance were in the range of 'higher than expected activity.' While higher than at any time since 2003, the ILI rate was below that experienced in 2003 and considerably lower than during the influenza epidemic of 1997. The importance of historical threshold data in evaluating the relative severity of seasons is clearly demonstrated by these figures. While notification numbers were increased in Victoria in line with the national increase, threshold analysis indicated that the 2007 season was no worse than the 2003 season.

Regardless of the source of notification, the Victorian influenza season was characterised by a bimodal frequency distribution over time; reaching peak activity in mid-August in 2007. A secondary peak in early September was observed in notification data, in mid-September in MMLS data and in late September in the rural component of GP sentinel surveillance.

As has been noted in previous years, population age-structure varies according to reporting source. The DHS notifications contained a large proportion of notifications of laboratory-confirmed influenza in children below five years of age, as well as a relatively large proportion of notifications in elderly patients. This is largely because these notifications are predominantly made from hospitals, with these age groups most at risk of hospitalisation. In contrast, those aged between 20 and 24 years were the largest group with laboratory-confirmed influenza presenting with ILI to sentinel surveillance GPs. The age structure for non-laboratory-confirmed ILI cases reported by sentinel surveillance GPs, however, was more symmetrically distributed. The increased proportion of positive swabs from young adults, which also featured in previous years of GP sentinel surveillance, may be due to several factors. For instance, workplace or university requirements for sick certificates may increase the number of patients in this age group presenting to the GP within the first three days of symptoms appearing (one of the defined case criteria for swabbing). Although this was not reported by participating GPs, there may be greater compliance with the swabbing procedure in this age group.

The influenza typing and strain data available for Victoria suggest that the pattern of approximately 90% type A and 10% type B was distributed relatively evenly over the season. The Australian influenza vaccine for 2007 contained A/New Caledonia/20/99-like, A/Wisconsin/67/2005-like and B/Malaysia/2506/67/2004-like strains.[9] The most commonly isolated strains of influenza A were A/Solomon Islands/3/2006-like A(H1) and A/Brisbane/10/2007-like A(H3) strains, while B/Florida/4/2006 and B/Shanghai/361/2002–like strains were the most commonly isolated influenza B strains in Victoria. Both the influenza A(H1) and A(H3) strains most commonly circulating in Victoria in 2007 represented drift variants from the virus contained in the 2007 vaccines. While these viruses can be distinguished antigenically using specific ferret sera or by molecular sequencing of the haemagglutinin gene, a reasonable level of cross reactivity between vaccine and circulating influenza A strains would be expected for the influenza A viruses.

For the influenza B viruses, the most commonly circulating viruses were of the B/Yamagata/16/88-lineage (B/Florida/4/2006 and B/Shanghai/361/2002-like viruses) while the minority were similar to the 2007 vaccine strain, B/Malaysia/2506/2004. While there is some measurable cross reactivity generated with one B-lineage against the other, higher levels of immunity would be expected against viruses of the same lineage. As it is currently only possible to have one influenza B-lineage in the influenza vaccine, it is not possible to have a perfect match while both the influenza B-lineages are co-circulating.

The GP sentinel program continues to enhance Victoria's overall influenza surveillance effort by contributing additional information not easily obtained through passive surveillance systems. In 2007, the GP surveillance period was extended in order to accommodate the piloting of GP surveillance for varicella-zoster virus (chickenpox and shingles), which does not tend to follow a marked seasonal pattern in the manner of influenza.[11] While the increased demands on GPs resulted in the withdrawal of some GPs from both surveillance programs (65 GPs participated in 2007, compared to 74 in 2006), the average GP response rate of 95% was considerable higher than previous years. For instance in 2005, average response rate was 85% and it was as low as 68% in 2006. As we have previously canvassed,[7] high participation from a smaller number of participating practices may provide more representative surveillance data than larger numbers of practices with poorer response rates.

## Acknowledgements

We gratefully acknowledge the participation of general practitioners and their practice staff in the sentinel surveillance program; their support is critical to its success.

As critical is the ongoing support and participation of the MMLS, and our particular thanks go to MMLS Directors, Ms Josie Adams and Ms Bronwyn Hawking.

We also thank others involved in the operation of the sentinel surveillance program, including the private pathology providers who facilitate transport of respiratory specimens from rural and regional general practices. Surveillance of laboratory-confirmed influenza is made possible through notifying laboratories (particularly the Viral Identification Laboratory at VIDRL) and medical practitioners throughout Victoria.

We also thank staff in the Communicable Disease Control Unit at the Department of Human Services for data entry and follow up of notifications data.

The Melbourne WHO Collaborating Centre for Reference and Research on Influenza is supported by the Australian Government Department of Health and Ageing.

## Author details

Dr Emma R Miller[1]
Mr James E Fielding[2]
Ms Kristina A Grant[1]
Dr Ian G Barr[3]
Ms Georgina Papadakis[1]
Associate Professor Heath A Kelly[1]

1. Victorian Infectious Diseases Reference Laboratory, Melbourne, Victoria
2. Department of Human Services, Melbourne, Victoria
3. WHO Collaborating Centre for Influenza Reference and Research

Corresponding author: A/Prof Heath Kelly, Director, Epidemiology, Victorian Infectious Diseases Reference Laboratory, Locked Bag 815, CARLTON SOUTH VIC 3035. Telephone: +61 3 9342 2608. Facsimile: +61 3 0342 2665. Email: heath.kelly@mh.org.au

## References

1 Kelly H, Murphy A, Leong W, Leydon J, Tresise P, Gerrard M, et al. Laboratory-supported influenza surveillance in Victorian sentinel general practices. *Commun Dis Intell* 2000;24:379–383.

2 Rural Infection Control Practice Group. *Health (Infectious Disease) Regulations*. Melbourne: RICPRAC, Department of Human Services; 2001. (Policy no. 7.1).

3 Thursky K, Cordova SP, Smith D, Kelly H. Working towards a simple case definition for influenza surveillance. *J Clin Virol* 2003;27:170–179.

4 Druce JD, Tran T, Kelly H, Kaye M, Chibo D, Kostecki R, et al. Laboratory diagnosis and surveillance of human respiratory viruses by PCR in Victoria, Australia, 2002–2003. *J Med Virol* 2005;75: 122–129.

5 Watts CG, Andrews RM, Druce JD, Kelly HA. Establishing thresholds for influenza surveillance in Victoria. *Aust N Z J Public Health* 2003;27:409–412.

6 The Influenza Pandemic Planning Committee Communicable Diseases Network Australia and New Zealand. *A Framework for an Australian Influenza Pandemic Plan*. Canberra: Commonwealth Department of Health and Aged Care; 1999. (Version 1).

7 Clothier H, Turner J, Hampson A, Kelly H. Geographic representativeness for sentinel influenza surveillance: implications for routine surveillance and pandemic preparedness. *Aust N Z J Public Health* 2006;30:337–341.

8 Kelly H, Birch C. The causes and diagnosis of influenza-like illness. *Aust Fam Physician* 2004;33:305–309.

9 Therapeutic Goods Administration. AIVC recommendations for the composition of influenza vaccine for 2007. Canberra: Australian Government Department of Health and Ageing, TGA. [updated October 2006]. Available from: http://www.tga.gov.au/committee/aivc2007.htm Accessed November 2007.

10 Australian Government Department of Health and Ageing. Australian influenza report: week ending 13 October 2007. Canberra: Surveillance Policy and System Section; 2007.

# Enhanced surveillance for serious complications of influenza in children: role of the Australian Paediatric Surveillance Unit

Yvonne A Zurynski, David Lester-Smith, Marino S Festa, Alison M Kesson, Robert Booy, Elizabeth J Elliott

## Abstract

Influenza contributes significantly to disease burden among children aged less than five years. Existing influenza surveillance systems do not provide detailed data on clinical presentation, management, vaccination status, risk factors and complications in hospitalised children, or link such data with laboratory results. Following a number of child deaths due to influenza in 2007, the Australian Government Department of Health and Ageing approached the Australian Paediatric Surveillance Unit (APSU) to examine the feasibility of enhancing APSU surveillance to identify children hospitalised with severe complications of influenza. Active, national, weekly surveillance was conducted during September 2007 with reporting by 1,256 Australian paediatricians working in hospitals and outpatient settings. The weekly report card return rate was 93%; detailed clinical data were provided on 88% of all notified cases and 15 children met the case criteria for severe complications of influenza. Admission to hospital occurred within 48 hours of onset of symptoms in over half of the children, of whom 13 had influenza A and two had influenza B, confirmed mostly by polymerase chain reaction on nasopharyngeal aspirate. Serious complications included pneumonia, presumed viral (67%), secondary bacterial infection, shock, cardiomyopathy, myocarditis and hypoglycaemia. No child aged six months or older had been vaccinated against influenza, including three children with underlying chronic conditions. No eligible child received an antiviral agent for influenza. Length of hospital stay ranged from 2 to 34 days; four children were admitted to a Paediatric Intensive Care Unit and one was ventilated. This study demonstrates the feasibility of using the established APSU mechanism for enhanced emergency surveillance during disease outbreaks, emergence or importation. *Commun Dis Intell* 2008;32:71–76.

Keywords: influenza, surveillance, child, diagnosis, immunisation

## Introduction

Influenza is a common childhood disease with a wide spectrum of severity from minor respiratory symptoms to severe respiratory illness and life-threatening multi-system complications.[1–9] Significant morbidity and mortality of influenza has been reported in Australian children, with an estimated hospitalisation rate of 82 per 100,000 and death rate of 0.2 per 100,000 children aged less than five years.[10] Of 22 children admitted with complications of influenza to one paediatric intensive care unit (PICU) over a short period in 2003, three died and none had been immunised.[7] Compared with 2006, during the 2007 influenza season there were increased numbers of hospital admissions,[11] including a number of child deaths, attributed to influenza and its complications. In response to this, the Australian Paediatric Surveillance Unit (APSU, www.apsu.org.au) was approached in late August 2007 by the Office for Health Protection, Australian Government Department of Health and Ageing to conduct enhanced surveillance for children aged less than five years, hospitalised with serious complications of influenza. Surveillance commenced 10 days later on 1 September and continued until the end of the month.

Currently, influenza surveillance systems in Australia are based on laboratory reporting and sentinel surveillance for influenza-like illness with reporting by general practitioners and hospital emergency departments.[11,12] They provide the number of confirmed cases, trends over time and geographic distribution, however, limited timely information is available on clinical presentation and risk factors, investigations and hospital management, complications, treatment, and outcomes. A surveillance system able to rapidly provide detailed clinical and laboratory data on children with serious complications of influenza would add value to existing systems. It would enable us to identify clusters of severe disease, diagnostic and management practice, and raise awareness amongst clinicians. Surveillance would inform us of sub-groups at most risk of serious complications and these data would be useful in the development of future immunisation policy and guidelines for diagnosis and treatment of influenza in children.

APSU conducts active, national surveillance for incident cases of rare conditions or rare complications of common conditions in children.[13] Paediatricians and other child health specialists respond to a monthly report card that lists up to 16 different

conditions. The report card, sent by email (68%) or post, is expected to be returned whether a case has been seen or not. Currently, 96% of the monthly report cards sent to 1,256 clinicians (representing approximately 92% of paediatricians practicing in Australia and listed on the Royal Australasian College of Physicians list of Fellows) are returned.[13] Clinicians reporting cases are sent a two-page questionnaire (adapted for each condition) requesting information on the demographics, management and short-term outcomes for the child.

Our aim was to determine whether APSU can provide timely enhanced data during outbreaks, emergence or importation.

## Methods

A modified version of the routine APSU surveillance mechanism was used for surveillance of serious complications of influenza. A separate report card, case definition, study protocol and questionnaire was developed and sent by post to all 1,256 clinicians participating in routine APSU surveillance. Each week during the month of September 2007 a separate influenza report card was sent by post or email, reminding clinicians to report newly diagnosed cases. The questionnaire was included as an attachment to the email report card or as a paper copy with the paper card to clinicians who prefer this method. Clinicians reporting a case were encouraged to complete and return the questionnaire directly via facsimile, email or post. In addition, study materials including the case definition, study protocol and questionnaire were made available on the APSU website www.apsu.org.au. To raise awareness of this surveillance study, an item was placed in the weekly email newsletter of the RACP Division of Paediatrics and Child Health. Urgent ethics clearance was obtained from the Human Research Ethics Committee at the Children's Hospital at Westmead.

### Case definition

Clinicians were asked to report any child aged less than five years admitted to hospital with laboratory-confirmed influenza and with any of the complications listed in Table 1.

The questionnaire included items about demographics, presenting symptoms, method of diagnosis, investigations, complications, length of stay in hospital and/or PICU, immunisation status, risk factors such as pre-existing chronic conditions; and other factors of public health interest such as recent overseas travel, close contact with farm animals, and contact with other people infected with influenza.

## Results

### Surveillance results

In September 2007, a total of 5,073 report cards were sent either by email (65%) or surface mail (35%) to practising paediatricians and 4,745 (93%) cards were returned, confirming a high participation rate. There were 4,710 cards received indicating 'nothing to report' and 35 cards reporting a total of

**Table 1. Case definition criteria**

**Any child aged less than five years and admitted to hospital with laboratory confirmed influenza and any of the following complications:**

- pneumonia (x-ray confirmed)
- requirement for ventilation
- encephalitis/encephalopathy with or without seizures
- myocarditis; pericarditis; cardiomyopathy
- rhabdomyolysis
- purpura fulminans
- disseminated coagulopathy
- transverse myelitis
- polyneuritis
- Guillain-Barré syndrome
- shock (requiring >40 ml/kg fluid resuscitation)
- acute renal failure
- Reye's syndrome
- laboratory proven secondary bacterial infection; bacteraemia, septicaemia, bacterial pneumonia
- death

**Exclusion:** simple febrile seizures

58 potential cases of influenza. Questionnaires were completed for 51 of 58 (88%) notifications, allowing for case classification. Of the 51 cases, 15 were confirmed influenza cases, reported from five of the seven Australian states and territories (Table 2). There were 36 reporting errors including four cases that met all case definition criteria but were aged five years or more; 23 cases that did not meet the clinical case definition, most commonly because they had no serious complications; and eight cases were diagnosed outside of the study period, before September 2007 (Table 2).

### Demographics and diagnosis

Among the 15 confirmed cases hospitalised with complications of influenza, all were born in Australia, three (20%) identified as Aboriginal or Torres Strait Islander and most (78%) were male. At diagnosis, children ranged in age from 4 days to 3.7 years (median 1.5 years): over half (60%) were aged under two years and three (20%) were aged less than six months. Nasopharyngeal aspirate was the most common sampling method (n=13); a throat swab was taken in one child and the sampling method is not known in the other case. Thirteen children had influenza A and two had influenza B. Influenza was confirmed by polymerase chain reaction in 11 and immunofluorescence in four cases.

### Presenting features and complications

The most common symptoms at onset of illness were fever, cough, dyspnoea, and headache. Over half (53%) the children had a rapid clinical deterioration with admission to hospital within 48 hours of the onset of symptoms. Four (27%) children were admitted to a PICU and one child required ventilation (Table 3). The majority of children were seriously ill as indicated by the range of complications (Table 3). The most common complication was x-ray confirmed pneumonia, presumed viral in 10 (67%) children; secondary bacterial pneumonia was identified in only one case (Table 3). Other complications included shock, myocarditis, cardiomyopathy, need for ventilation and hypoglycaemia.

### Table 3. Symptoms and signs at onset of illness and complications at presentation to hospital

| Symptoms and complications | Confirmed cases (n=15) |
|---|---|
| Admitted to Intensive Care Unit | 4 |
| **Symptoms and signs at onset of illness*** | |
| Cough | 12 |
| Fever | 10 |
| Dyspnea | 8 |
| Sore throat | 3 |
| Vomiting | 2 |
| Headache | 5 |
| Stridor | 2 |
| Other | 3 |
| **Complications*** | |
| Pneumonia (presumed viral) | 10 |
| Ventilation | 1 |
| Shock | 1 |
| Secondary infection | |
| • blood culture +ve for *Moraxella catarrhali* | 3 |
| • norovirus and *Clostridium difficile* toxin A isolated from stool | |
| Hypoglycaemia | 2 |
| Cardiomyopathy | 1 |
| Dehydration | 1 |

* Some children had more than one symptom or sign and more than one complication.

### Table 2. Number of reports and case classification, by state or territory

| State | Total notifications | Errors | Questionnaire not returned | Confirmed cases |
|---|---|---|---|---|
| New South Wales | 9 | 7* | 0 | 2 |
| Northern Territory | – | – | – | – |
| Queensland | 13 | 9 | 2 | 2 |
| South Australia | 11 | 2 | 3 | 6 |
| Tasmania | – | – | – | – |
| Victoria | 12 | 6 | 2 | 4 |
| Western Australia | 13 | 12 | 0 | 1 |
| Total | 58 | 36 | 7 | 15 |

* One of these notifications was a duplicate report.

**Known risk factors, contacts, vaccination status and treatment**

Among the 15 confirmed cases, one was a neonate and four had pre-existing chronic conditions, including one child with Down's syndrome, laryngomalacia and hypothyroidism; one 12-week-old infant who was clinically malnourished; one child with congenital heart disease; and one child with asthma.

A contact with laboratory confirmed influenza (a sibling) was identified for only one child. No child had travelled overseas in the 10 days prior to onset of symptoms and no child had close recent contact with pigs, poultry or birds.

None of the 10 children aged six months or older had been vaccinated against influenza, including three children with pre-existing chronic conditions. Only four children had been vaccinated against pneumococcus. None of nine children aged over 12 months and eligible for treatment with the anti-influenza drug Tamiflu™ (oseltamivir phosphate) received the drug.

**Outcomes**

Admission to hospital occurred within 48 hours of onset of symptoms in over half of children. Length of hospital stay ranged from 2 to 30 days (median 6.5 days). Length of stay in PICU ranged from 3 to 20 days (3 cases). At the time of reporting, 13 of the 15 children hospitalised with complicated influenza had been discharged with no ongoing medical problems. There were no deaths reported. One child remained in PICU for 20 days after admission and one child remained in hospital 34 days after admission and final outcome data are not available for these two children.

## Discussion

We have demonstrated that the APSU surveillance system can be modified and mobilised quickly, in the event of an epidemic, to enable enhanced surveillance for diseases of public health interest, such as severe complications of influenza. Within 10 days of being commissioned by the Department of Health and Ageing to undertake this project, we had consulted experts in the field and formed an investigators group; developed a case definition, study protocol, reporting instructions, report card and questionnaire; obtained ethics approval; publicised the study among reporting paediatricians; and begun surveillance.

Our data demonstrate that the 1,256 clinicians who report each month to the APSU are willing to participate in emergency surveillance, for example during an epidemic. This is confirmed by the 93% response rate to the weekly report card and provision of clinical data for 88% of notified cases. Furthermore, the study provided an educational opportunity. At the outset of the study APSU provided all paediatricians with important information about the method of diagnosis, investigation, management and known risk factors for influenza and highlighted the importance of asking about vaccination status, pre-existing conditions and recent overseas travel in children presenting with flu-like symptoms. The data collected were timely, detailed, and nationally representative and clinical and laboratory data could be linked immediately and related to outcomes. Such data are currently not available from any other source. Potentially, such enhanced APSU surveillance could also be used to collect data on new, emerging, or introduced diseases and to inform public health response for disease management and prevention.

Our data highlight a number of important issues for clinicians. Young children who are subsequently hospitalised with complications of influenza present with typical 'flu-like' symptoms but deteriorate quickly (over a half were admitted within 48 hours of onset of symptoms); presumed viral pneumonia is the most common complication; and prognosis is usually excellent. None of the children in our study had been diagnosed before presenting to hospital. None of the children eligible for treatment with oseltamivir had been treated, despite evidence that treatment shortens the duration of illness.[14] Trials of point-of-care testing for influenza in the primary health care setting and in emergency departments, coupled with early treatment using oseltamivir are needed to determine whether such measures will reduce the number of children hospitalised due to influenza and the number of serious complications in children. Such trials may also inform the development of Australian clinical practice guidelines for diagnosis and treatment of children presenting with influenza-like illness.

None of the children in our study, who were eligible for vaccination, had been vaccinated against influenza, including four with pre-existing chronic conditions. Because influenza contributes to a significant disease burden among children, the USA Advisory Committee on Immunization Practices now recommends vaccination for all children aged six months to five years.[15] In 2003, Milne, et al. called for further discussion regarding inclusion of similar recommendations on the Australian Immunisation Schedule.[7] The 2007 *Australian Immunisation Handbook* includes a recommendation for the vaccination of children aged six months or older who are immunocompromised or who have chronic conditions that may predispose them to severe complications of influenza and states that 'influ-

enza vaccine should be administered to any person who wishes to reduce the likelihood of becoming ill with influenza'.[16] However, influenza vaccination for healthy children is not currently funded under the National Immunisation Schedule. Enhanced surveillance data and detailed review of hospital records of children admitted to hospital with laboratory-confirmed influenza will be important to inform changes to immunisation recommendations and to monitor the effectiveness of such changes. We are currently undertaking an audit of all (122) cases of influenza admitted to the Children's Hospital at Westmead in 2007.

Although the number of children identified in this short (one month) surveillance period at the end of the influenza season is small, our data show that children are hospitalised with a range of serious, often multiple, complications of influenza. We cannot compare the reporting rate in our study with data provided by laboratory and sentinel surveillance systems currently operating in Australia, as these existing systems do not provide information about severity of illness and specific complications by age group. The highest burden of influenza admissions is known to be in children aged under two years[10] and this is reflected in our study, in which 60% were aged under two years. However, four additional cases reported among older children but excluded from our analysis illustrate that children aged over five years may also suffer severe multisystem complications. Of the three males and one female (aged 7.5, 7.9, 9.4 and 13.7 years), three were admitted to PICU and two required ventilation (for 5 days and 11 days). Three had influenza A and one had influenza B. Their complications included pneumonia, encephalopathy, disseminated coagulopathy, seizure, shock, Reye's syndrome and acute necrotising encephalopathy. One of these children had cystic fibrosis; the other three were previously healthy. None had been vaccinated against influenza and two were treated with oseltamivir. These data suggest that future surveillance studies should include children up to the age of 15 years.

With the support of the Department of Health and Ageing we are currently piloting a new Paediatric Active Enhanced Disease Surveillance (PAEDS) system in four major paediatric hospitals in four states (Princess Margaret Hospital for Children in Perth, Women's and Children's Hospital in Adelaide, Royal Children's Hospital in Melbourne and the Children's Hospital at Westmead in Sydney). This hospital based surveillance system involves active case identification by specialist nurses and is modelled on the IMPACT system developed in Canada.[17] The strength of PAEDS lies in its ability to facilitate collection of timely, detailed clinical data in addition to biological specimens. PAEDS, is a collaboration between the APSU and the National Centre for Immunisation Research and Surveillance for Vaccine Preventable Diseases. If sucessfully rolled out to all major paediatric centres around Australia, PAEDS could offer an additional complementary source of ascertaining hospitalised cases of severe complications of influenza.

## Conclusions and recommendations

We have demonstrated the potential for using the APSU for emergency surveillance of uncommon conditions. This surveillance system has the advantage of being well established, cheap and enabling linkage of and timely access to epidemiological, clinical and virological data. With an extended period of surveillance (June to September) APSU and/or PAEDS could in future provide prospective enhanced data on hospitalised cases of severe seasonal influenza in Australian children aged under 15 years.

## Acknowledgements

We acknowledge all clinicians contributing data to the Australian Paediatric Surveillance Unit, including the Australian and New Zealand Paediatric Intensive Care Units. The APSU is a Unit of the Division of Paediatrics and Child Health, Royal Australasian College of Physicians and is funded by the Australian Government Department of Health and Ageing, the Faculty of Medicine at the University of Sydney, and a National Health and Medical Research Council (NHMRC) Enabling Grant (402784). Elizabeth Elliott is supported by an NHMRC Practitioner Fellowship (457084).

## Author details

Dr Yvonne A Zurynski[1,2]
Dr David Lester-Smith[1,2]
Dr Marino S Festa[3]
Associate Professor Alison M Kesson[2,4]
Professor Robert Booy[5]
Professor Elizabeth J Elliott[1,2]

1. Australian Paediatric Surveillance Unit, The Children's Hospital at Westmead, New South Wales
2. Discipline of Paediatrics and Child Health, The University of Sydney, New South Wales
3. Paediatric Intensive Care Unit, The Children's Hospital at Westmead, New South Wales
4. Department of Infectious Diseases and Microbiology, The Children's Hospital at Westmead, New South Wales
5. National Centre for Immunisation Research and Surveillance, The Children's Hospital at Westmead, New South Wales

Corresponding author: Dr Yvonne Zurynski, Assistant Director, Australian Paediatric Surveillance Unit, The Children's Hospital at Westmead, Locked Bag 4001, WESTMEAD NSW 2145.Telephone: +61 2 9845 1202 / 3005. Facsimile: +61 2 9845 3082. Email: yvonnez@chw.edu.au

## References


1. Bhat N, Wright JG, Broder KR, Murray EL, Greenberg ME, Glover MJ, et al. Influenza-associated deaths among children in the United States, 2003–2004. *N Engl J Med* 2005;353):2559–2567.
2. Centers for Disease Control and Prevention. Update: influenza activity—United States and worldwide, May 20-September 15, 2007. *Morb Mortal Wkly Rep* 2007;56:1001–1004.
3. Grose C. The puzzling picture of acute necrotizing encephalopathy after influenza A and B virus infection in young children. *Pediatr Infect Dis J* 2004;23:253–254.
4. Heikkinen T, Silvennoinen H, Peltola V, Ziegler T, Vainionpaa R, Vuorinen T, et al. Burden of influenza in children in the community. *J Infect Dis* 2004;190:1369–1373.
5. Kappagoda C, Isaacs D, Mellis C, Peat J, De Silva L, O'Connell A. Critical influenza virus infection. *J Paediatr Child Health* 2000;36:318–321.
6. Maricich SM, Neul JL, Lotze TE, Cazacu AC, Uyeki TM, Demmler GJ, et al. Neurologic complications associated with influenza A in children during the 2003–2004 influenza season in Houston, Texas. *Pediatrics* 2004;114:e626–e633.
7. Milne BG, Williams S, May ML, Kesson AM, Gillis J, Burgess MA. Influenza A associated morbidity and mortality in a Paediatric Intensive Care Unit. *Commun Dis Intell* 2004;28:504–509.
8. Newland JG, Laurich VM, Rosenquist AW, Heydon K, Licht DJ, Keren R, et al. Neurologic complications in children hospitalized with influenza: characteristics, incidence, and risk factors. *J Pediatr* 2007;150:306–310.
9. Smitherman HF, Caviness AC, Macias CG. Retrospective review of serious bacterial infections in infants who are 0 to 36 months of age and have influenza A infection. *Pediatrics* 2005;115:710–718.
10. Brotherton J, Wang H, Schaffer A, Quinn H, Menzies R, Hull B, et al. Vaccine preventable diseases and vaccination coverage in Australia, 2003 to 2005. *Commun Dis Intell* 2007; 31:Suppl–152.
11. Australian Government Department of Health and Ageing. Australian Influenza Report. Report No. 13 week ending 13 October 2007. Available from: http://www.health.gov.au/internet/wcms/publishing.nsf/Content/6EE650550BD9C27DCA25736B001219D0/$File/ozflu-no13-2007.pdf
12. O'Brien K, Barr IG, Annual report of the National Influenza Surveillance Scheme, 2006. *Commun Dis Intell* 2007;31:167–179.
13. Zurynski YA, Peadon E, Bower C, Elliott EJ. Impacts of national surveillance for uncommon conditions in childhood. *J Paediatr Child Health* 2007;43:724-731.
14. Matheson NJ, Harnden AR, Perera R, Sheikh A, Symmonds-Abrahams M, Neuraminidase inhibitors for preventing and treating influenza in children. *Cochrane Database of Systematic Reviews* 2007;(1):CD002744.
15. Fiore AE, Shay DK, Haber P, Iskander JK, Uyeki TM, Mootrey G, et al. Prevention and control of influenza. Recommendations of the Advisory Committee on Immunization Practices, 2007. *Morb Mortal Recomm Rep* 2007;56(RR–6):1–54.
16. National Health and Medical Research Council. *The Australian Immunisation Handbook*, 8th edition. Canberra: Australian Government Publishing Service, 2007; Chapter 3.9 Influenza.
17. Scheifele DW, Halperin SA, CPS/Health Canada IMP, Immunization Monitoring Program, Active: a model of active surveillance of vaccine safety. *Sem Pediatr Infect Dis* 2003;14:213–219.

# Absence of a *Chlamydia trachomatis* variant, harbouring a deletion in the cryptic plasmid, in clients of a sexually transmissible infection clinic and antenatal patients in Melbourne

Matthew P Stevens, Sarah E Tan, Leonie Horvath, Christopher K Fairley, Suzanne M Garland, Sepehr N Tabrizi

## Abstract

A *Chlamydia trachomatis* (CT) variant, harbouring a 377 bp deletion in the cryptic plasmid, recently identified in Europe, has caused false-negative reporting of CT infections by various assays. This report aimed at identifying whether this variant is present among clients of a sexual health clinic, or antenatal screening patients in Melbourne. Two hundred CT-positive specimens (by BDProbeTec™ ET assay) from Melbourne Sexual Health Centre (August 2005–November 2006) were tested by COBAS *Taq*Man 48 PCR assay. Discrepancies were tested by an in-house real-time (Re-Ti) polymerase chain reaction (PCR) assay, amplifying a 274-bp region of the *omp*1 gene. Additionally, 1,071 consecutive specimens from antenatal screening patients at the Royal Women's Hospital (December 2006–April 2007) were tested by COBAS *Taq*Man 48 and *omp*1 Re-Ti PCR. The CT variant was not detected among the 200 CT-positive specimens (95% confidence interval 0–2.3%). Three tested CT-negative by COBAS *Taq*Man 48, *omp*1 Re-Ti PCR and CT mutant-specific PCR, suggesting sample degradation or differential assay sensitivity. Of the 1,071 antenatal screening specimens, 56 tested CT-positive and 1,015 CT-negative by COBAS *Taq*Man 48. All of the CT-negatives tested negative by *omp*1 Re-Ti PCR (95% confidence interval 0–0.5%), with 51 of 56 CT-positives testing positive. These findings show there were no CT variants among attendees of a Melbourne sexual health clinic, nor among antenatal screening patients. It is likely that the variant strain has not yet entered circulation in these populations. However, given the current upsurge in urogenital CT-infections, continued surveillance is necessary to ensure timely detection of this variant, should it be introduced into the population. *Commun Dis Intell* 2008;32:77–81.

Keywords: *Chlamydia trachomatis*, variant, cryptic plasmid, real-time polymerase chain reaction

## Introduction

*Chlamydia trachomatis* (CT) infections are the most frequently reported bacterial sexually transmitted infection in the developed world, with the urogenital tract being the most common site of CT infection.[1,2] However, the majority of CT infections are asymptomatic (approximately 80% in women, and 50% in men) and thereby often remain undiagnosed.[3] Left untreated, chlamydial infections can lead to complications such as epididymitis in men, and pelvic inflammatory disease with long-term concomitant ectopic pregnancy or tubal infertility in women.

The CT bacterium contains approximately 7 to 10 copies of a 7.5-kb cryptic plasmid, which has an unknown function.[4,5] Given the cryptic plasmid is highly conserved in sequence, present in multiple copies and within all serovars, it has been the target for sensitive CT detection with several assays. However, in late 2006, a new CT variant was identified in Sweden, containing a 377-base pair deletion in the cryptic plasmid.[6] This finding came after the observation of an unexpected decrease in CT infections of up to 25% between November 2005 and August 2006 in Halland County, southwest Sweden.[6] To date, the CT variant has been detected throughout Sweden, with single cases reported in Denmark, France and Ireland, and two in Norway.[7–10] None have been detected in other parts of Europe, including the Netherlands and the United Kingdom.[11,12]

This finding has important ramifications in the diagnoses of CT infections, given that several commercially available CT assays target the cryptic plasmid, including the COBAS Amplicor and COBAS *Taq*Man 48 assays (Roche Diagnostics) and Abbott CT/NG test (Abbott Laboratories).[13,14] Assays targeting this deleted region of the cryptic plasmid would result in the false-negative reporting of specimens harbouring the CT variant. According to a recent Microbiology Quality Assurance Program report by the Royal College of Pathologists of Australia, approximately 65% of Australian laboratories are using CT assays potentially susceptible to false-negative CT diagnosis should this CT variant enter circulation.[15] Alternative assays which target areas of the cryptic plasmid outside this deletion region, such as the BDProbeTec™ ET assay (Becton Dickinson); or those targeting the *omp*1 gene, such as the ARTUS CT kit (Qiagen); or rRNA, such as the Aptima

Combo 2 assay (Gen-Probe), can effectively detect this new CT variant. The failure to detect CT infections due to this variant provides a selection advantage over the 'wild-type' CT strains assuming it is at no biological disadvantage. The CT variant would go undetected, the infection left untreated and allowed to persist and potentially spread more widely. Therefore, vigilance to monitor the presence of such variant strains in a given population is of considerable importance. A knowledge of which CT strains are in general circulation, particularly new variants, will improve our understanding of their epidemiology and may improve strategies for diagnosis and disease control.

In this study, we describe a comparative study testing two cohorts from the Melbourne population with the *omp*1 Re-Ti genomic assay and the *Taq*Man 48 and BDProbeTec™ ET assays. We detected no CT variants among the current population, as determined by this alternative target assay for CT detection.

## Methods

### Specimens

Specimens included in this study consisted of CT-positive specimens from the Melbourne Sexual Health Centre (MSHC) (collected from August 2005 to November 2006). These specimens were collected from both male (88.5%) and female (11.5%) clientele. In addition, consecutive antenatal screening clinical specimens routinely tested at the Royal Women's Hospital (RWH) Department of Microbiology (collected from December 2006 to April 2007) were included. All specimens comprised of first pass urine (FPU). Specimens from the MSHC (n = 200) all previously tested CT-positive by BDProbeTec™ ET assay. Consecutive specimens tested at the RWH (n = 1,071) by COBAS *Taq*Man 48 assay comprised: CT-negative (n = 1,015), or CT-positive (n = 56).

DNA extracts from two CT strains harbouring the deletion mutation were kindly supplied by Dr P Nilsson from the Uppsala University Hospital, Uppsala, Sweden.

The Alfred Hospital ethics committee approved the use of anonymous specimens (from MSHC clients) for research purposes using an 'opt out' consent procedure. Analysis of antenatal screening patient specimens was undertaken with approval covered by public health legislation. All specimens were tested anonymously.

*BDProbeTec ET and COBAS TaqMan 48 assay*

Two hundred CT-positive FPU specimens, as detected by the BDProbeTec™ ET assay, were tested using the COBAS *Taq*Man 48 PCR Analyzer according to the manufacturer's instructions. Both assays target the CT cryptic plasmid, though at different locations. Given there are up to 10 copies of the cryptic plasmid per bacterium, these assays were designed for their high sensitivity.

*CT-consensus real-time PCR assay – Re-Ti PCR assay*

All 1,071 consecutive antenatal screening specimens from RWH were tested by CT Re-Ti PCR assay. CT-specific forward and reverse primers and hydrolysis '*Taq*Man' probe were manually designed using alignment of the *omp*1 nucleotide sequences of CT reference strains obtained from GenBank (accession numbers in parentheses): A/Sa1/OT (M58938), B/Alpha-95 (U80075), B-Jali-20 (M33636), Ba/Apache-2 (AF063194), C/TW3/OT (M17343), D/B120 (X62918), D/B185 (X62919), D/IC-Cal8 (X62920), E/Bour-1990 (X52557), F/IC-Cal3 (X52080), G/UW57/Cx (AF063199), H/Wash (X16007), I/UW-12 (AF063200), J/UW36/Cx (AF063202), K/UW31/Cx (AF063204), L1/440-Bu (M36533), L2/434-Bu (M14738), and L3/404-Bu (X55700). The primers (CT-F: 5´-CATGARTGGCAAGCAAGTTTA-3´ and CT-R: 5´-GCAATACCGCAAGATTTTCTAG-3´) were directed to amplify a 274 bp region spanning VD-IV and the hydrolysis probe (5´- HEX-TGTTCACTCCYTACATTGGAGT-BHQ1-3´) targeting a consensus region upstream of VDIV. Underlined nucleotides represent locked nucleic acid bases.

The polymerase chain reaction (PCR) was prepared in a LightCycler® 480 Multiwell Plate 96 containing 1 µM of primer (CT-F and CT-R), 0.2 µM of probe and 1x LightCycler® 480 Probes Master (Roche Diagnostics) per well. The DNA template (7 µl) was added to the appropriate well resulting in a final reaction volume of 20 µl. The PCR was performed under the following parameters: initial enzyme activation at 95°C for 10 minutes; followed by 45 cycles of 95°C for 10 seconds, 60°C for 20 seconds, and 65°C for 30 seconds. Following the amplification phase, a cooling step was performed at 40°C for 10 seconds (ramp rate of 1.5°C per second). Acquisition of the fluorescence signal was performed using the Mono Hydrolysis Probe setting (483–523 nm) following the 65°C extension phase of each cycle.

*CT mutant-specific real-time PCR assay*

Specimens testing CT-negative by COBAS *Taq*Man 48 and CT-positive by either BDProbeTec™ ET assay or *omp*1 Re-Ti PCR were further tested by a CT mutant-specific real-time PCR assay, previously published.[14] Minor changes to the method included the use of 1.0 µM of each primer and 0.2 µM of each probe. In addition, a melting curve step was incorporated.

*Statistical analysis*

The 95% confidence intervals (CI) of proportions were calculated using the modified Wald method.[16]

## Results

Of the 200 CT-positive specimens identified at the MSHC, as determined by BDProbeTec, three tested CT-negative using the COBAS *Taq*Man 48 assay (Table 1), and were thus potential CT variants. These three specimens subsequently tested CT-negative by both *omp*1 Re-Ti PCR and CT mutant-specific PCR assays. These three discrepancies could be due to sample degradation or differential sensitivity between BDProbeTec and other assays. The fact that none of the three CT-positives (BD ProbeTec)/CT-negative (COBAS *Taq*Man 48) were positive by *omp*1 Re-Ti PCR, confirms that the CT variant was not present among this cohort (95% CI 0–2.3%).

Of the 1,071 clinical specimens derived from consecutive routine antenatal screening at the RWH, 94.8% (1,015/1,071) tested CT-negative by COBAS *Taq*Man 48. Among these 1,015 CT-negative specimens, none tested CT-positive by the Re-Ti PCR assay, confirming the CT variant was not present in this cohort (95% CI 0–0.5%). Conversely, among the 56 CT-positive specimens, five tested CT-negative using the Re-Ti PCR assay (Table 2). The overall concordance between the COBAS *Taq*Man 48 and Re-Ti PCR results was 99.5% (kappa = 0.951), indicating a near perfect agreement.

Two CT strains harbouring the deletion mutation were tested by *omp*1 Re-Ti PCR and CT mutant-specific PCR to confirm that the *omp*1 Re-Ti PCR could detect the CT variant. The *omp*1 Re-Ti PCR was able to detect both variant CT strains, as well as CT-positive specimens, such as from the antenatal screening cohort (Figure). However, the CT

**Table 1. Comparative detection of *Chlamydia trachomatis*-positive infections among Melbourne Sexual Health Centre clientele, by BDProbeTec ET assay and COBAS *Taq*Man 48 assay**

| BDProbeTec ET* | COBAS *Taq*Man 48† | | Total |
|---|---|---|---|
| | Positive | Negative | |
| Positive | 197 | 3 | 200 |
| Negative | 0 | 0 | 0 |
| Total | 197 | 3 | 200 |

* BD ProbeTec ET assay targets a region of the cryptic plasmid outside of where the mutation occurs.

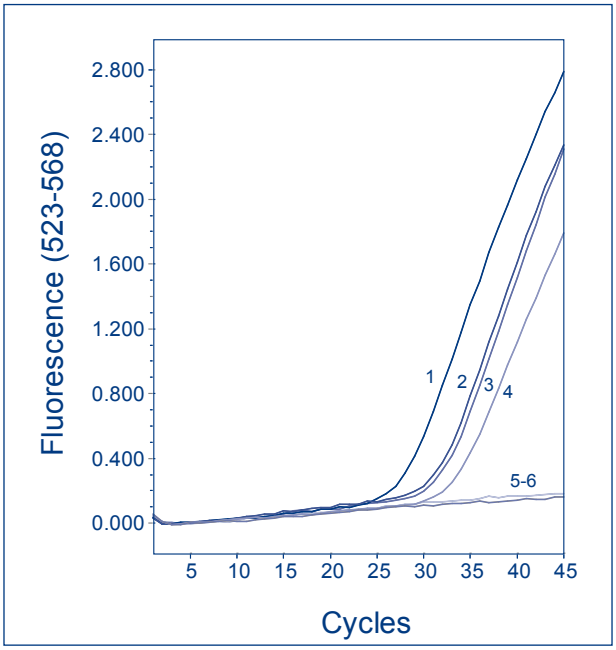
† COBAS TaqMan 48 assay targets the region of the cryptic plasmid where the mutation occurs.

**Table 2. Comparative detection of *Chlamydia trachomatis* infections among a Royal Women's Hospital antenatal screening cohort by polymerase chain reaction targeting the cryptic plasmid or *omp*1 gene**

| *Chlamydia trachomatis* real-time PCR* | COBAS *Taq*Man 48 assay | | Total |
|---|---|---|---|
| | Positive | Negative | |
| Positive | 51 | 0 | 51 |
| Negative | 5 | 1,015 | 1,020 |
| Total | 56 | 1,015 | 1,071 |

* *Chlamydia trachomatis* real-time polymerase chain reaction (PCR) assay targets the genomic *omp*1 gene.

**Figure. *Omp*1 real-time polymerase chain reaction of *Chlamydia trachomatis* variants and 'wild-type' strains**




Confirmation of the *omp*1 real-time PCR assay's capability of detecting the CT variant. Two CT strains, harbouring the deletion mutation, tested positive by *omp*1 Re-Ti PCR (2 and 3), as did 'wild-type' CT-positive specimens from the antenatal screening cohort (1 and 4). Conversely, two CT-negative specimens are shown (5 and 6), in which no amplification curves are present.

**Table 3. Comparison of assay sensitivity (using CP values) in the detection of the *Chlamydia trachomatis* variant**

| *Chlamydia trachomatis* mutants strain and dilution | | CP values | | CP difference |
|---|---|---|---|---|
| | | Mutant-specific PCR* | *omp*1 Re-Ti PCR | |
| 1 | 1/10 | 26.41 | 30.56 | 4.15 |
| | 1/100 | 30.60 | 33.84 | 3.24 |
| | 1/1000 | 37.61 | 39.45 | 1.84 |
| 2 | 1/10 | 29.02 | 31.20 | 2.18 |
| | 1/100 | 33.39 | 36.70 | 3.31 |
| | 1/1000 | 36.91 | 38.52 | 1.61 |

* The *Chlamydia trachomatis* mutant-specific polymerase chain reaction (PCR) targets the region of the cryptic plasmid whereby the mutation occurs.

CP Crossing point.

mutant-specific assay was more sensitive in detecting CT positivity than the *omp*1 Re-Ti PCR, as seen through the later crossing point (average: 2.7 cycles difference) (Table 3).

## Discussion

This report describes the absence of CT variants among two study cohorts in the Melbourne population using a novel real-time PCR assay targeting the CT *omp*1 gene encoding the major outer membrane protein. This Re-Ti assay was utilised for the detection of CT infections, including both 'wild-type' and variant strains. The assay was shown to successfully detect CT DNA among 91.1% of antenatal screening specimens previously identified as CT-positive by COBAS *Taq*Man 48. The lower sensitivity of the current assay was not unexpected, given that the COBAS *Taq*Man 48 assay is based on amplification of a region within the multi-copy cryptic plasmid whilst the Re-Ti assay targets the single copy *omp*1 gene. Therefore, we would anticipate a tenfold difference in sensitivity as was indeed demonstrated through comparison of the crossing point values of the Re-Ti *omp*1 assay and Roche mutant-specific assay. In addition, the five discrepancies (negative by Re-Ti *omp*1 positive by *Taq*Man 48) were demonstrated as containing CT DNA in low copy numbers, with *Taq*Man 48 CP values of >40.

Given the lack of detection of the CT variant among the current study cohorts it was imperative to verify that the *omp*1 PCR assay was indeed capable of detecting the CT variant. DNA extracts from two separate CT strains harbouring the deletion mutation, confirmed by DNA sequencing,[6,14] were readily detected by the Re-Ti *omp*1 PCR, providing confidence that we were seeing true negative findings.

It is important to highlight two potential limitations of this study. The first being that collection of the MSHC specimens preceded initial reports of CT variant detection in Sweden. The second is that the antenatal screening cohort could be considered one of lower risk and thus less likely to have the CT variant in circulation.

This was the first description of a CT consensus real-time PCR assay utilising the recently released LC 480 platform. From these results, it is probable that the CT variant strains have not yet entered circulation in Australia. The continued use of CT assays targeting the cryptic plasmid is appropriate in Australia and there is no immediate requirement for performing supplementary assays for CT variants by routine diagnostic laboratories. However, given the current upsurge in urogenital CT infections worldwide, continued surveillance is necessary to ensure timely detection of this variant strain.

## Acknowledgements

We would like to thank Anna-Maria Costa, Shujun Chen and Nicole Gardiner for the routine CT testing of consecutive antenatal screening specimens at the Royal Women's Hospital, Department of Microbiology.

## Author details

Dr Matthew P Stevens, Research Officer[1]
Ms Sarah E Tan, Research Assistant[1]
Ms Leonie Horvath, Medical Scientist[2,3]
Professor Christopher K Fairley, Director[2,4]
Professor Suzanne M Garland, Director[1,5]
Associate Professor Sepehr N Tabrizi, Senior Research Scientist[1,4]

1. Department of Microbiology and Infectious Diseases, The Royal Women's Hospital, Carlton, Victoria
2. Melbourne Sexual Health Centre, Victoria
3. Microbiological Diagnostic Unit, University of Melbourne, Victoria
4. School of Population Health, University of Melbourne, Victoria
5. Department of Obstetrics and Gynaecology, University of Melbourne, Victoria

Corresponding author: Dr Matthew P Stevens, Research Officer, Department of Microbiology, The Royal Women's Hospital, 132 Grattan Street, CARLTON VIC 3053. Telephone: +61 3 9344 3108. Facsimile: +61 3 9344 2713. Email: matthew.stevens@mcri.edu.au

## References

1. Cates WJ, Wassenheit JN. Genital chlamydial infections: epidemiology and reproductive sequelae. *Am J Obstet Gynecol* 1991;164:1771–1781.
2. Quinn TC, Gaydos C, Shepherd M, Bobo L, Hook III W, Viscidi R, et al. Epidemiologic and microbiologic correlates of *Chlamydia trachomatis* infection in sexual partnerships. *JAMA* 1996;276:1737–1742.
3. Gaydos CA, Theodore M, Dalesio N, Wood BJ, Quinn TC. Comparison of three nucleic acid amplification tests for detection of *Chlamydia trachomatis* in urine specimens. *J Clin Microbiol* 2004;42:3041–3045.
4. Lovett M, Kuo KK, Holmes K, Falkow S. Plasmids of the genus *Chlamydia*. In: Nelson J, Grassi C, editors. *Current Chemotherapy and Infectious Diseases*. Vol. 2. Washington, DC: American Society for Microbiology; 1980. p. 1250–1252.
5. Palmer L, Falkow S. A common plasmid of *Chlamydia trachomatis*. *Plasmid* 1986;16:52–62.
6. Ripa T, Nilsson P. A variant of *Chlamydia trachomatis* with deletion in cryptic plasmid: implications for use of PCR diagnostic tests. *Euro Surveill* 2006;11 Available from: http://www.eurosurveillance.org/ew/2006/061109.asp#2 Accessed on 5 October 2007.
7. Herrmann B. A new genetic variant of *Chlamydia trachomatis*. *Sex Transm Infect* 2007;83:253–254.
8. de Barbeyrac B, Raherison S, Cado S, Normandin F, Clerc M, Clairet V, et al. French situation concerning the Swedish *Chlamydia trachomatis* variant. *Euro Surveill* 2007;12. Available from: http://www.eurosurveillance.org/em/v12n10/1210–226.asp Accessed on 24 January 2008.
9. Lynagh Y, Walsh A, Crowley B. First report of the new variant strain of *Chlamydia trachomatis* in Ireland. Epi-Insight 2007;8. Available from: http://www.ndsc.ie/hpsc/EPI-Insight/Volume82007/File,2424,en.pdf Accessed on 24 January 2008.
10. Moghaddam, A, Reinton N. Identification of the Swedish *Chlamydia trachomatis* variant among patients attending a STI clinic in Oslo, Norway. *Euro Surveill* 2007;12 Available from: http://www.eurosurveillance.org/ew/2007/070301.asp#3 Accessed 5 October 2007.
11. de Vries HJ, Catsburg A, van der Helm JJ, Beukelaar EC, Morre SA, Fennema JS, et al. No indication of Swedish *Chlamydia trachomatis* variant among STI clinic visitors in Amsterdam. *Euro Surveil*. 2007;12. Available from: http://www.eurosurveillance.org/ew/2007/070208.asp#3 Accessed on 5 October 2007.
12. Sillis M, Skidmore S, Mallinson H, Todd T, Coupland L, Oliver P, et al. No evidence of the *Chlamydia trachomatis* variant in the UK. *Sex Transm Infect* 2007;83:488–489.
13. Soderblom T, Blaxhult A, Fredlund H, Herrmann B. Impact of a genetic variant of *Chlamydia trachomatis* on national detection rates in Sweden. *Euro Surveill* 2006;11. Available from: http://www.eurosurveillance.org/ew/2006/061207.asp#1 Accessed 5 October 2007.
14. Ripa T, Nilsson PA. A *Chlamydia trachomatis* strain with a 377-bp deletion in the cryptic plasmid causing false-negative nucleic acid amplification tests. *Sex Transm Dis* 2007;34:255–256.
15. Microbiology Quality Assurance Program. RCPAQAP NSW, Australia: RCPA QAP Pty Limited:. Available from: http://www.rcpaqap.com.au/micro/index.html [updated on 11 December 2007] Accessed 23 January 2008.
16. Agresti A, Coull BA. Approximate is better than 'Exact' for interval estimation of binomial proportions. *Am Stat* 1998;52:119–126.

# Short reports

# Rotavirus surveillance in Australia

Kate A Ward, Peter B McIntyre, Carl D Kirkwood, Paul W Roche, Mark J Ferson, Paul G Van Buynder, April R Roberts-Witteveen, Alison M Kesson, Vicki L Krause, Jeremy M McAnulty

## Background

### Rotavirus

Globally, rotavirus is the most common cause of severe gastroenteritis in early childhood[1] infecting almost all children by five years of age. Rotavirus is estimated to be responsible for more than 600,000 deaths per year in these children with 85% occurring in low-income countries.[2]

In Australia, an estimated 10,000 hospitalisations and one death occur annually in children under 5 years, mostly in children aged under two years.[3] Among those under one year, hospitalisation rates are five times greater in Indigenous Australians compared with non-Indigenous children.[4] In addition to hospital admissions, 22,000 emergency department visits attributable to rotavirus are estimated to occur annually.[3] Direct medical costs in Australia are estimated at over $30 million.[3]

Rotaviruses are primarily spread by the faecal-oral route. Symptoms include diarrhoea, vomiting and fever. In severe cases acidosis, electrolyte abnormalities, severe dehydration and death can occur. Infection can be asymptomatic, especially in infants aged under three months. Diagnosis is usually confirmed by detection of rotavirus in stool samples.[5]

Rotaviruses are RNA viruses. They are classified into serogroups (A–G) with most human infections caused by Group A. They can be further subdivided into G and P serotypes by the VP7 and VP4 outer proteins of the virus (e.g. G1-4, G9, P[4], P[8]).[5]

In Australia, from 1 July 2005 to 30 June 2006, national rotavirus serotype surveillance data indicated that serotype G1 was the most dominant serotype representing 40% of all strains. Serotype G1 continues to be the most frequently reported serotype worldwide and has been the most common Australian serotype, for all but two years since 1999. From July 2001 to June 2003, serotype G1 was replaced by G9 as the most dominant serotype. Substantial geographical variation in prevalent serotypes has also been identified within Australia.[6] This geographical and temporal variation in strains along with the diversity of strains capable of causing severe disease in children in Australia has potential implications for vaccine effectiveness should the prevalent strains diverge from those primarily targeted by the available vaccines.

Two vaccines have been recently developed. Rotarix® (GlaxoSmithKline) is a live attenuated G1P[8] human rotavirus, which provides cross protection against most other serotypes when administered in two doses, one to two months apart.[7] Rotateq® (Merck) is a live pentavalent human-bovine (WC3 strain) reassortant rotavirus vaccine providing protection against five human serotypes G1, G2, G3, G4 and P[8] when administered in three doses four to ten weeks apart.[8]

In 2006, Rotateq was licensed for use in the United States of America (USA) and Rotarix was licensed for use in the United Kingdom. Both vaccines have been shown to be highly efficacious against severe rotavirus disease with 85% efficacy for Rotarix,[7] and 98% efficacy for Rotateq.[8]

Rotarix and Rotateq were licensed in Australia in 2006. Both vaccines have been available on the private market since June 2006 and Rotarix was publicly funded for babies in the Northern Territory from August 2006. Rotavirus vaccination has been included in the National Immunisation Program, commencing from 1 July 2007, with all infants born after 1 May 2007 eligible for vaccination. Victoria, South Australia and Queensland have included Rotateq in their vaccination program while the remaining jurisdictions have elected to use the Rotarix vaccine.

### Surveillance

According to published US Centers for Disease Control and Prevention (CDC) guidelines,[9] the public health importance of a disease can be measured using the following parameters:

- frequency of disease;
- severity of disease;
- inequities associated with disease;
- costs;
- preventability (or amenability to public health intervention); and
- public interest.

Prior to establishing a surveillance system for any condition, it is crucial to identify specific aims and ensure that any system is adequately sensitive and specific to achieve these aims.

The aims of surveillance vary depending on the health condition of interest, and may include to:

- control the spread of disease (with public health follow up for each case);
- estimate the burden of disease;
- monitor trends in the burden over time;
- assess the effectiveness of interventions (e.g. vaccines);
- monitor changes in disease characteristics over time (e.g. change in serotypes, strains);
- enhance understanding of the epidemiology and clinical course of the disease;
- provide a basis for epidemiologic research; and
- inform policy makers.

Following the identification of surveillance system aims, further consideration needs to be given to the specific analyses that will be conducted, the importance of timeliness and the potential public health actions in response to surveillance data. Finally, the resources (including funding and personnel) that would be required to operate the system must be allocated and key stakeholders consulted prior to its introduction.

## Rotavirus surveillance

### Aims

Based on the above criteria, the following is a list of possible aims of rotavirus surveillance in Australia with a brief discussion surrounding each aim.

*Control the spread of disease*

Laboratories and doctors are likely to identify only a small proportion of all rotavirus cases (Figure). Public health follow-up on individual cases for rotavirus will therefore not be a feasible or appropriate method to control the spread of rotavirus.

*Estimate the burden of a disease, monitor trends in the burden of disease over time and provide a basis for epidemiologic research*

Given that many rotavirus cases do not make contact with a health provider, the burden of rotavirus infection must be estimated.

Laboratory counts of patients who test positive for rotavirus depend on the number of tests done that is influenced by a number of factors including physician practice and access to laboratory facilities. While access to laboratory testing may remain stable within regions, other more unpredictable changes such as physician practices following the introduction of the vaccination and epidemics of gastroenteritis infection due to norovirus, may influence testing requests (Table). Thus interpreting trends over time in positive laboratory tests for rotavirus requires caution, especially at the national level.

Assuming that moderate to severe cases of rotavirus make contact with the hospital system, monitoring rotavirus coded hospitalisation data and mortality from all gastroenteritis-related codes over time could broadly detect changes in the burden of severe rotavirus.

Case surveillance could direct future scientific studies with clearly defined hypotheses and study population e.g. an increase in incidence in certain groups or geographic areas may warrant further research. With the introduction of the vaccine and expected decline in cases, outbreaks will provide an increasingly valuable opportunity to assess vaccine effectiveness.

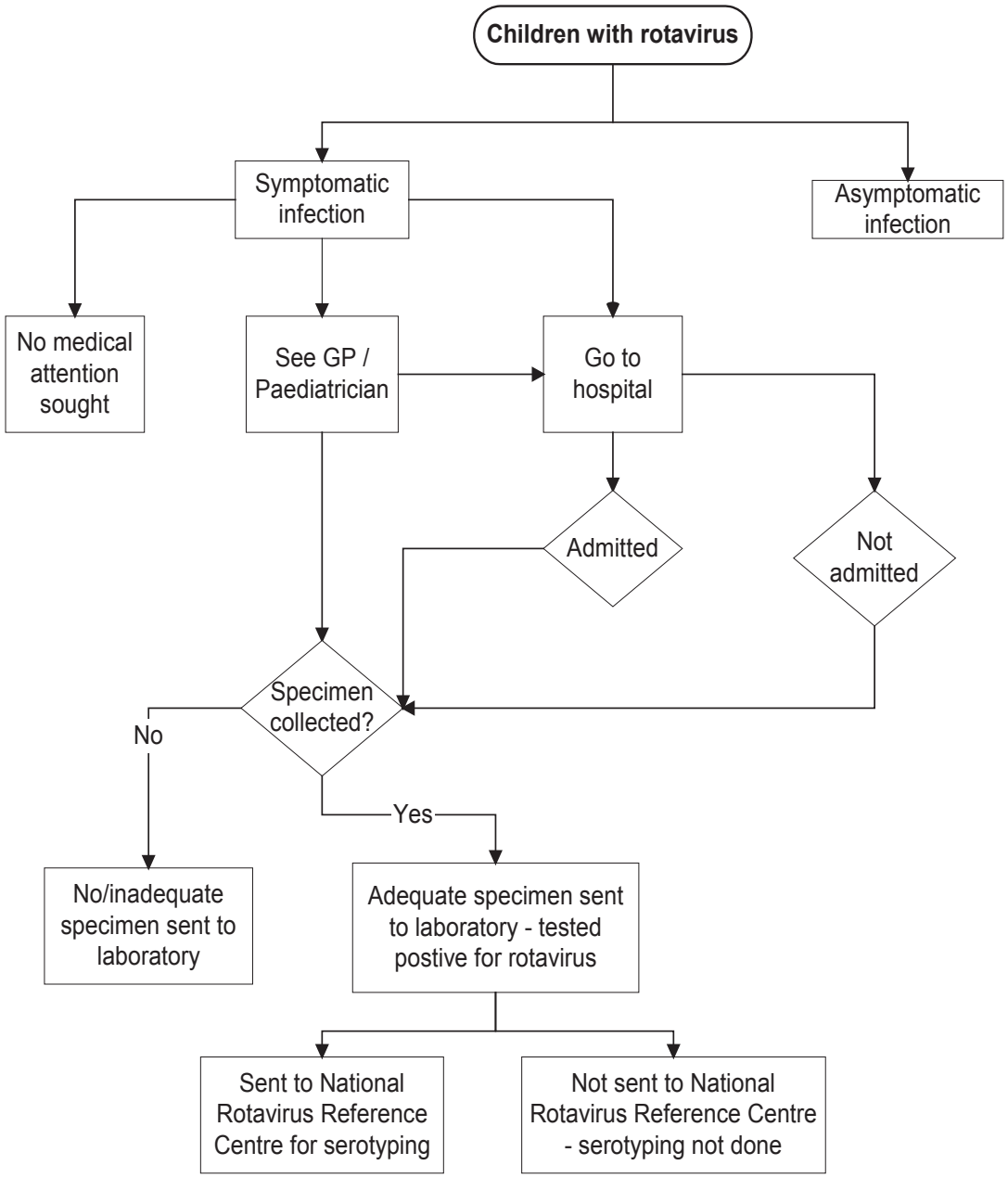
*Assess the effectiveness of vaccines*

The two rotavirus vaccines have undergone rigorous safety and efficacy trials each involving approximately 70,000 children.[7,8] While public funding is not contingent on national effectiveness data, evaluating the impact of any rotavirus vaccination program is worthwhile to ensure maximum impact of the vaccination. The absence of a stable national baseline of rotavirus notification patterns and rates prior to vaccine introduction and the fact that laboratory data reflect only a proportion of the true incidence of rotavirus disease makes it difficult to assess the impact of vaccination at the national level. The opportunity cost of investing public health resources into individual case follow-up to determine vaccine status must be considered at the jurisdictional level. Specific scientific studies to better assess vaccine efficacy in defined populations may be appropriate and feasible under some circumstances, especially in areas of high incidence such as the Northern Territory.[10] Australia may have the unique opportunity to compare the two vaccines, given that individual jurisdictions have chosen different vaccines.

*Monitor changes in serotypes over time*

The National Rotavirus Reference Centre has documented temporal and geographical variations in strain type in isolates sent from participating laboratories throughout Australia since 1999. Given the

**Figure. Diagram of children infected with rotavirus captured by laboratory surveillance**




introduction of a vaccine in subgroups of the population, understanding the impact of vaccination on circulating serotypes is important. The prevalent strains of rotavirus have been shown to vary substantially in the absence of vaccine pressure so interpretation of changes following vaccine introduction is complex and likely to require a long time period. To maximise the usefulness of such data, the National Rotavirus Reference Centre must receive specimens from a sufficiently large and representative sample across the country.

*Enhance understanding of the epidemiology of the disease*

Laboratory and hospitalisation data following vaccine introduction will add to the available descriptive epidemiological data from the pre-vaccine period. Analysis of additional risk factors including indigenous status would require follow-up of individual cases.

**Table. Potential sources of data for rotavirus surveillance**

| | Advantages | Limitations |
|---|---|---|
| Mortality data | Mortality due to any gastroenteritis-related codes could be evaluated<br>Captures most severe cases<br>Additional resources required for data analysis and reporting | Mortality extremely low in Australia therefore not likely to be very informative source of data<br>Biased sample given that only the most severe cases will die<br>Delays in accessing data (approximately 1 month)<br>Potential coding/ cause of death assignment errors |
| Inpatient statistics data | Has been shown in Australia to provide a good source of information on moderate to severe disease over time (of primary importance for vaccination)<br>Specific in the inpatient data collection code for rotavirus already collected<br>Captures sex, age, aboriginality and length of time in hospital<br>Can be used to help assess the economic burden of rotavirus<br>Additional resources required for data analysis and reporting | Limited to rotavirus cases associated with moderate to severe disease<br>Potentially underestimate cases if rotavirus ICD code not assigned[4]<br>Hospital practices/admission rates may change over time<br>Variations in admission practices between jurisdictions complicates national summary data<br>Delays in accessing data (available within six months) |
| Laboratory data – sentinel (e.g. LabVISE) or all laboratories | Timely<br>Accurate | Only represent a proportion of all rotavirus cases (moderate to severe disease)<br>May be variations in testing practice over time (especially considering the introduction of a vaccine)<br>Can be influenced by other factors e.g. epidemic season of norovirus can result in increased in all virus testing<br>Additional resources required for data entry, reporting and analysis |
| Emergency department (ED) surveillance | Timely<br>Resource intensive to set up but minimal additional resources required once established as reports can be automated | Low specificity: only captures data on ED presentations that are allocated a provisional diagnosis of gastrointestinal disease. Laboratory results not usually available in ED setting<br>Data not captured nationally |
| Sentinel GPs | More representative sample than hospital data<br>Vaccine information available | Resource intensive to maintain |
| Institutional outbreak reports | Identifies outbreak, which enables public health intervention<br>No additional resources required as data already collected by OzFoodNet | Incomplete reporting as reliant on institution notifying public health unit |

## Current rotavirus surveillance systems in similar countries

### United States of America

Rotavirus is not a notifiable disease in the USA. The CDC recommendations for national surveillance systems for rotavirus infections include national hospital discharge databases for rotavirus diagnoses and laboratory reports from sentinel laboratories. A system of sentinel laboratories has also been established by the CDC to monitor the prevalence of rotavirus serotypes over time. Special studies (e.g. case control and retrospective cohort studies) are planned to assess vaccine effectiveness at state and local levels.[11]

### United Kingdom

Rotavirus is not a notifiable disease in England or Wales. Surveillance is based on routine laboratory reports to monitor secular trends.[12] Ad hoc disease burden studies have estimated the proportion of acute gastroenteritis that is attributable to rotavirus using data from laboratories, general practitioners, hospital admissions and deaths.[13]

### Canada

Rotavirus is not a notifiable disease in Canada. Surveillance is based on laboratory data reported through the National Enteric Surveillance Program.

These data represent a subset of infections and are meant to monitor trends rather than provide an estimate of incidence. Ad hoc cohort studies have been conducted to estimate the burden of rotavirus in Canada. Prospective hospital based studies will be used to assess vaccine effectiveness in the future.[14]

## Current rotavirus surveillance systems in Australia

In Western Australia (since 2006), Queensland (since 2005) and the Northern Territory (since 1994), all laboratory-confirmed cases of rotavirus infection are notifiable conditions, which are reported to the health department under public health legislation. On the basis of notifications to health departments in 2006, there were 605 cases (rate = 311/100,000) in the Northern Territory and 2,508 cases (rate = 64/100,000) reported in Queensland. Laboratory surveys in New South Wales suggest that just over 1,000 (rate = 18/100,000) stool samples tested positive for rotavirus in 2006.

The National Rotavirus Reference Centre in Melbourne currently collects specimens from nine collaborating centres in New South Wales, the Northern Territory, South Australia, Victoria and Western Australia in order to characterise rotavirus strains causing annual epidemics of severe diarrhoea in young children. Participating laboratories predominantly test specimens from hospitalised children.

Potential data sources in Australia include death registry data (although this is limited by small numbers), inpatient statistics for all gastroenteritis and episodes coded as due to rotavirus, emergency department presentations and institutional outbreak reports. Hospital and mortality data are delayed and are currently only analysed by researchers on an ad hoc basis.

Laboratory diagnoses and sentinel general practice presentations are two potential data sources for rotavirus surveillance but such data are not currently collected nationally. The advantages and limitations of these data sources are discussed in the Table.

### Monitoring adverse events

Extensive safety data exists for both rotavirus vaccines[7,8] and post licensure studies are underway in the USA. While intussusception was identified as an adverse event of the older rotavirus vaccine licensed in 1998 in the USA[15] (prompting its later withdrawal), no association with intussusception has been found for the two new rotavirus vaccines. The existing reporting through the Adverse Drug Reactions Advisory Committee can be used to monitor any adverse events associated with rotavirus vaccines but has substantial limitations, as it is a passive reporting system. In 2007, to coincide with the introduction of rotavirus vaccines into Australia, the Australian Paediatric Surveillance Unit commenced a national study to monitor intussusception incidence in children aged less than 24 months.

## Summary

Rotavirus is a cause of significant morbidity in children aged under five years in Australia. Clinical trials have shown that two available vaccines are highly efficacious in the prevention of severe diarrhoea and hospitalisation due to rotavirus. A national rotavirus vaccination program was initiated in Australia in July 2007.

Apart from immunisation, current tools are unlikely to be effective in controlling the spread of rotavirus in the wider community. Routine surveillance has limited value in assessing vaccine effectiveness.

The available evidence indicates that surveillance is likely to be useful for (and therefore aim to):

1. Detect changes in the rotavirus serotypes to track whether prevalent strains match the available vaccine. This requires the National Rotavirus Surveillance Program to test a representative sample of rotavirus cases across Australia.
2. Monitor trends in the burden of rotavirus over time using data on positive laboratory tests stratified by jurisdiction and hospitalisations from all gastroenteritis-related codes and those coded as due to rotavirus. Mortality data are likely to be of limited value but are important to evaluate over a longer time period.
3. Provide a basis for further epidemiologic research.

## Recommendations

1. States and territories should consider mandating laboratory reporting of patients with rotavirus infection under public health legislation to broadly monitor the burden due to rotavirus and facilitate collection of representative specimens for the National Rotavirus Reference Centre.
2. States and territories should ensure representative samples are submitted to the National Rotavirus Reference Centre for serotyping.
3. The National Rotavirus Reference Centre will continue to distribute annual reports to laboratories for circulation to stakeholders.
4. The Surveillance Branch of the Australian Government Department of Health and Ageing and the National Centre for Immunisation Research and Surveillance should regularly monitor mor-

tality, hospitalisation data already collected and additional laboratory data, for changes in the burden of rotavirus over time.

5. Special studies that address specific research questions such as vaccine efficacy in certain groups may be undertaken by jurisdictions with the interest and capacity to do so, especially for groups at special risk such as Indigenous children in high incidence areas.

## Author details

Ms Kate Ward, Trainee Public Health Officer[1]
Professor Peter B. McIntyre, Director[2,3]
Dr Carl D Kirkwood, Senior Research Fellow[4]
Dr Paul W Roche, Epidemiologist[5]
Professor Mark J Ferson[6,7]
Associate Professor Paul G Van Buynder, Director[8]
Mrs April R Roberts-Witteveen[5,9]
Associate Professor Alison M Kesson, Head of Infectious Diseases and Microbiology[10]
Associate Professor Vicki L Krause, Director[11]
Dr Jeremy M McAnulty, Director[1]

1. Communicable Diseases Branch, NSW Health, North Sydney, New South Wales
2. National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, Westmead, New South Wales
3. Discipline of Paediatrics and Child Health and School of Public Health, University of Sydney, New South Wales
4. Enteric Virus Research Group, Murdoch Childrens Research Institute, Parkville, Victoria
5. Surveillance Policy and Systems Section, Australian Government Department of Health and Ageing, Canberra, Australian Capital Territory
6. Director, Public Health Unit, South Eastern Sydney Illawarra Health, Randwick, New South Wales
7. Conjoint Professor, School of Public Health & Community Medicine, University of NSW, Kensington, New South Wales
8. Communicable Disease Control, WA Department of Health, Subiaco, Western Australia
9. Masters of Applied Epidemiology Scholar, Australian National University, Acton, Australian Capital Territory
10. The Children's Hospital at Westmead and Discipline of Paediatrics and Child Health, The University of Sydney, New South Wales
11. Centre for Disease Control, Department of Health and Community Services, Casuarina, Northern Territory

Corresponding author: Ms Kate Ward, Communicable Diseases Branch, NSW Health, Locked Mail Bag 961, North Sydney NSW 2059. Telephone: +61 2 9391 9279. Facsimile: +61 2 9391 9189. Email: kward@doh.health.nsw.gov.au

## References

1. Parashar UD, Bresee JS, Gentsch JR, Glass RI. Rotavirus. *Emerg Infect Dis* 1998;4:561–570.
2. Parashar UD, Gibson CJ, Bresse JS, Glass RI. Rotavirus and severe childhood diarrhea. *Emerg Infect Dis* 2006;12:304–306.
3. Galati JC, Harsley S, Richmond P, Carlin JB. The burden of rotavirus-related illness among young children on the Australian health care system. *Aust N Z J Public Health* 2006;30:416–421.
4. Newall AT, MacIntyre R, Wang H, Hull B, Macartney K. Burden of severe rotavirus disease in Australia. *Paediatr Child Health* 2006;42:521–527.
5. Glass RI, Parashar UD, Bresee JS, Turcios R, Fischer TK, Widdowson MA, et al. Rotavirus vaccines: current prospects and future challenges. *Lancet* 2006;368:323–332.
6. Kirkwood CD, Cannan D, Bogdanovic-Sakran N, Bishop RF, Barnes GL, National Rotavirus Surveillance Group. National Rotavirus Surveillance Program annual report, 2005–06. *Commun Dis Intell* 2006;30:434–438.
7. Ruiz-Palacios GM, Perez-Schael I, Velazquez FR, Abate H, Breuer T, Clemens SC. Safety and efficacy of an attenuated vaccine against severe rotavirus gastroenteritis. *N Engl J Med* 2006;354:11–22.
8. Vesikari T, Matson DO, Dennehy P, Van Damme P, Santosham M, Rodriguez Z; et al. Safety and efficacy of a pentavalent human-bovine (WC3) reassortant rotavirus vaccine. *N Engl J Med* 2006;354:23–33.
9. Centers for Disease Control and Prevention. Updated Guidelines for evaluating public health surveillance systems. *MMWR Recomm Rep* 2001;50 (RR13):1–35 Available from: http://iier.isciii.es/mmwr/preview/mmwrhtml/rr5013a1.htm
10. Schultz R. Rotavirus gastroenteritis in the Northern Territory, 1995–2004. *Med J Aust* 2006;2;185:354–356.
11. Centers for Disease Control and Prevention. Prevention of rotavirus gastroenteritis among infants and children. Recommendations of the Advisory Committee on Immunization Practices (ACIP) *MMWR Recomm Rep* 2006;55(RR12):1–13. Available from: http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5512a1.htm
12. Health Prevention Agency. Rotavirus surveillance data from England and Wales. Available from: http://www.hpa.org.uk/infections/topics_az/rotavirus/data.htm
13. Harris JP, Jit M, Cooper D, Edmunds WJ. Evaluating rotavirus vaccination in England and Wales. Part I. Estimating the burden of disease. *Vaccine* 2007;25:3962–3970.
14. Landry L. Rotavirus disease in Canada. *7th Canadian Immunization Conference;* 3–6 December 2006. Winnipeg, Manitoba, Canada. Available from: http://www.phac-aspc.gc.ca/cnic-ccni/2006/pres/_pdf-mon-lun-dec04/PM/B-Hall-B-Rotavirus/Landry_Rotavirus-disease-in-Canada_Imm-Conference-2006.pdf
15. Centers for Disease Control and Prevention. Suspension of rotavirus vaccine after reports of intussusception—United States, 1999 [published erratum appears in *MMWR Morb Mortal Wkly Rep* 2004;53:879]. *MMWR Morb Mortal Wkly Rep* 2004;53:786–789.

# A norovirus outbreak associated with consumption of NSW oysters: implications for quality assurance systems

Clare Huppatz, Sally A Munnoch, Tory Worgan, Tony D Merritt, Craig Dalton, Paul M Kelly, David N Durrheim

## Abstract

Norovirus is a common cause of gastroenteritis outbreaks associated with raw shellfish consumption. In Australia there have been several reports of norovirus outbreaks associated with oysters despite the application of regulatory measures recommended by Food Standards Australia New Zealand. This study describes an outbreak of norovirus gastroenteritis following the consumption of New South Wales oysters. In September 2007, OzFoodNet conducted a cohort study of a gastroenteritis outbreak amongst people that had dined at a Port Macquarie restaurant. Illness was strongly associated with oyster consumption, with all cases having eaten oysters from the same lease (RR undefined, $p<0.0001$). Norovirus was detected in a faecal specimen. Although no pathogen was identified during the environmental investigation, the source oyster lease had been closed just prior to harvesting due to sewage contamination. Australian quality assurance programs do not routinely test oysters for viral contamination that pose a risk to human health. It is recommended that the feasibility of testing oysters for norovirus, particularly after known faecal contamination of oyster leases, be assessed. *Commun Dis Intell* 2008;32:88–91.

Keywords: disease outbreak, environmental investigation, foodborne disease, norovirus

## Introduction

In Australia, there have been several published outbreaks of norovirus, which have implicated oysters.[1–5] Between 2001–2006 in Australia, there were 13 outbreaks of gastroenteritis due to oysters reported to OzFoodNet; half of these outbreaks were due to norovirus (OzFoodNet, unpublished data, 2007). However, norovirus is a reasonably common gastroenteritis-causing pathogen in the community and disease can be mild. It is likely that there is significant under-reporting of norovirus gastroenteritis related to oyster consumption.

This report describes the epidemiological investigation of a norovirus outbreak following consumption of a meal at a northern New South Wales restaurant. The ability of current quality assurance control measures to protect the public from viral infections is questioned.

## Methods

On 24 September 2007, Hunter New England OzFoodNet was informed of 19 cases of gastrointestinal illness amongst 54 individuals who had attended a golfing event in Port Macquarie in mid-September. Hypothesis generating interviews established that the point source of the outbreak was a restaurant dinner function on 11 September. This was the only meal shared by all cases and no attendees had been unwell at the function.

A retrospective cohort investigation of attendees of the function on 11 September was undertaken to identify the source of the outbreak. A questionnaire was administered by telephone between 25 and 28 September. Information was gathered regarding the onset and nature of any gastrointestinal illness, food and drink exposures, and contact with ill people.

A case was defined as a person who attended the restaurant for dinner and subsequently developed diarrhoea plus one or more additional symptoms of gastroenteritis (nausea, vomiting, abdominal pain, fever or lethargy).

Data were entered into a Microsoft Access database and then imported into STATA™ (version 8.0, Stata Corporation, College Station, Texas, USA) for analysis using Fisher's exact test for exploring rates by exposure status.

To investigate the environmental cause of the outbreak, the local council inspected the restaurant and the NSW Food Authority commenced a trace-back investigation of the oysters. This involved a site visit to the oyster wholesaler premises and to the oyster grower.

Faecal specimens were examined for parasitic, bacterial and viral pathogens by microscopy and culture, and enzyme immunoassay (EIA) and polymerase chain reaction for norovirus. Oysters obtained during the environmental investigation were tested for bacterial and viral pathogens.

## Results

### Cohort study

Interviews were undertaken with 53/54 people in the cohort, with one person being uncontactable. Nineteen cases were identified, with a median incubation period of 35 hours (range 10–59 hours) and the median duration of illness was 2.5 days (range 1–8 days). Two cases visited a doctor (11%), and no cases were hospitalised. All cases reported diarrhoea, and 18/19 (95%) experienced watery diarrhoea and one (5%) reported possible blood in the stool. Other symptoms reported included: abdominal pain (95%), lethargy (79%), nausea (79%), vomiting (58%), fever (58%) and headache (63%).

The dinner consisted of a choice of two entrée dishes, a buffet style main meal and a selection of desserts. The entrée choices were a plate of chicken skewers, served with risotto or a plate of six prawns and six natural oysters in the half-shell, served with cocktail sauce (served on the side) and lettuce garnish.

Illness was strongly associated with oyster consumption, with all cases having eaten oysters (RR undefined, $p<0.0001$) (Table). Other food items that were associated with illness included: prawns (RR=3.7, 95% CI 1.0–14.1), lettuce garnish (RR=2.8, 95%CI 1.4–5.5), cocktail sauce (RR=2.3, 95%CI 1.1–4.7), cucumber and tomato salad (RR=2.3, 95%CI 1.2–4.5) and pavlova (RR= 2.3, 95%CI 1.2–4.3). Stratified analysis could not be performed, as all cases consumed oysters.

### Environmental investigation

The restaurant inspection found the facility to be compliant with the Australia New Zealand Food Standards Code[6] with no breaches in the handling or storage of food items.

The trace-back investigation identified that the estuary from which the oysters were harvested had been closed for three months prior to opening on 4 September, the day on which the implicated batch of oysters supplied to the restaurant was harvested. The estuary had been closed on 4 June due to rainfall, with continued closure due to a faecal spill from a sewage treatment plant near the oyster lease in the estuary on 20 August. The implicated oysters underwent post-harvest depuration for 36 hours, a process during which oysters are held in tanks of disinfected sea-water, allowing the oysters to feed in clean water prior to sale.[7] Quality assurance sampling performed post-depuration reported *Escherichia coli* <0.5 CFU/g, which is the limit of detection for the method used.

**Table. Attack rates and relative risk for foods eaten at the restaurant dinner in Port Macquarie, 11 September 2007**

| Food | Ate | | | Did not eat | | | Relative risk | 95% CI |
|---|---|---|---|---|---|---|---|---|
| | Ill n=19 | Total | Attack rate (%) | Ill | Total | Attack rate (%) | | |
| **Entrée** | | | | | | | | |
| Oysters | 19 | 34 | 55.9 | 0 | 19 | 0.0 | undefined | |
| Prawns | 17 | 37 | 45.9 | 2 | 16 | 12.5 | 3.7 | 1.0 – 14.1 |
| Lettuce garnish | 10 | 15 | 66.7 | 9 | 38 | 23.7 | 2.8 | 1.4–5.5 |
| Cocktail sauce | 11 | 20 | 55.0 | 8 | 33 | 24.2 | 2.3 | 1.1–4.7 |
| Chicken skewers | 1 | 16 | 6.3 | 18 | 37 | 48.6 | 0.1 | 0.0–0.9 |
| **Main course** | | | | | | | | |
| Leg ham | 17 | 40 | 42.5 | 2 | 13 | 15.4 | 2.8 | 0.7–10.4 |
| Lamb | 15 | 42 | 35.7 | 4 | 11 | 36.3 | 1.0 | 0.4–2.4 |
| Beef | 11 | 41 | 26.8 | 8 | 12 | 66.7 | 0.4 | 0.2–0.8 |
| Chicken | 12 | 37 | 32.4 | 7 | 16 | 43.8 | 0.7 | 0.4–1.5 |
| Cucumber and tomato salad | 5 | 7 | 71.4 | 14 | 46 | 30.4 | 2.3 | 1.2–4.5 |
| **Dessert** | | | | | | | | |
| Pavlova | 6 | 9 | 66.7 | 13 | 44 | 29.5 | 2.3 | 1.2–4.3 |
| Toffee pudding | 0 | 6 | 0.0 | 19 | 47 | 40.4 | 0.0 | – |
| Apple strudel | 3 | 6 | 50.0 | 16 | 47 | 34.0 | 1.4 | 0.6–3.6 |
| Fruit salad | 3 | 8 | 37.5 | 16 | 45 | 35.6 | 1.1 | 0.4–2.8 |
| Cream | 11 | 25 | 44.0 | 8 | 28 | 28.6 | 1.5 | 0.7–3.2 |

The environmental investigation revealed that there were no sick food handlers at the time of the function, either at the restaurant, wholesaler or oyster farm.

### Microbiology

Faecal specimens were collected from only one case. Of two collected specimens, one was tested by EIA and found to be positive for norovirus.

Fresh oysters obtained during the environmental investigation were negative for pathogens, including norovirus, but were from a different batch to that consumed on the night of the function.

## Discussion

This outbreak of norovirus gastroenteritis was associated with oyster consumption. While consumption of prawns, lettuce and cocktail sauce were associated with illness, their association was likely to represent confounding, as the oysters were served on a plate with these food items. All of those cases that ate prawns (n=17), lettuce (n=10) or cocktail sauce (n=11), had also consumed oysters (n=19). Other foods (cucumber and tomato salad and pavlova) accounted for fewer cases of illness (5/19 and 6/19 respectively).

This study highlights the importance of quality assurance processes within the oyster industry. In New South Wales there is a mandatory, industry-funded Shellfish Program, which is administered by the NSW Food Authority.[8] The NSW Shellfish Program incorporates the principles and objectives of the Australian Shellfish Quality Assurance Program.[9] Quality assurance programs monitor *E. coli* and faecal coliforms as indicator organisms for contamination of shellfish.[9]

Reviews of foodborne viral illness have suggested that bacteriological testing of shellfish provides an unreliable indication of viral contamination.[4,10] Several studies have detected norovirus in oysters which have met bacteriologic standards required for human consumption.[11,12] In addition, shellfish have the ability to accumulate viruses in their digestive tracts to levels that are much higher than those of the surrounding water.[10] One study found that Eastern oysters accumulate a viral surrogate, F+ coliphage, to densities that were on average 19 times greater than the levels in the surrounding estuarine water.[13]

Although depuration effectively decreases bacterial levels, including *E. coli*,[7,10] it is generally accepted that this process is inadequate for viral decontamination.[10,14] It has been demonstrated experimentally that radio-labelled virus particles can persist in oysters for 64 hours after depuration.[15]

While norovirus usually causes a mild illness, its presence may indicate increased risk of other viruses, including hepatitis A.[16] Some have suggested that warning labels accompany oysters at points of sale to warn of the potential risks.[14,17] An alternative could be to test specifically for norovirus and other viral pathogens, however, the cost effectiveness and feasibility of this in Australia would require evaluation.

Several countries in the Asia-Pacific region are currently evaluating or instituting norovirus testing of oysters. In 2007, the Agri-Food and Veterinary Authority (AVA) of Singapore introduced the mandatory testing of imported oysters.[18] The AVA requires all consignments of frozen oysters to be tested by the exporting country and certified as being free from norovirus.[18] In New Zealand, the Environmental Science and Research (ESR) laboratory is working with the New Zealand Food Safety Authority to improve methods for norovirus testing of shellfish.[19] The goal of the ESR oyster research program is to establish protocols and quality systems suitable for regulatory use in New Zealand.[19]

## Conclusion

Epidemiological evidence from this cohort study showed a significant association between oysters and gastroenteritis. Although environmental evidence was lacking, a potential mechanism for viral contamination leading to accumulation of virus within the oysters was the reported sewage spill in the vicinity of the oyster lease less than two weeks prior to harvesting. Current quality assurance systems may be inadequate to guarantee public safety from viral contamination of oysters. The feasibility and cost effectiveness of enterovirus testing of oysters, particularly after high risk events such as faecal spills, should be further evaluated.

## Acknowledgements

We would like to acknowledge the NSW Food Authority who were involved in this outbreak investigation. We are also grateful to OzFoodNet staff, Australian Government Department of Health and Ageing, Australian Capital Territory. The Masters of Applied Epidemiology Program (CH & PK) is funded by the Australian Government Department of Health and Ageing. Paul Kelly is also supported by the National Health and Medical Research Council. This paper was prepared with infrastructure support from Hunter Medical Research Institute.

## Author details

Clare Huppatz[1,2]
Sally Munnoch[1,3]
Tory Worgan[1,3]
Tony Merritt[1,3]
Craig Dalton[1]
Paul M Kelly[2]
David N Durrheim[1]

1. Hunter New England Population Health, NSW Health, Locked Bag 10, Wallsend, New South Wales
2. National Centre for Epidemiology and Population Health, College of Medicine and Health Sciences, Australian National University, Canberra, Australian Capital Territory
3. OzFoodNet, Australian Government Department of Health and Ageing, Hunter New England Office, Wallsend, New South Wales

Corresponding author: Dr Clare Huppatz, Master of Applied Epidemiology Scholar, Hunter New England Population Health, NSW Health, Locked Bag 10, WALLSEND NSW 2287. Telephone: +61 2 4924 6353. Facsimile: +61 2 4924 6212. Email: Clare.Huppatz@hnehealth.nsw.gov.au

## References

1. Webby RJ, Carville KS, Kirk MD, Greening G, Ratcliff RM, Crerar SK, et al. Internationally distributed frozen oyster meat causing multiple outbreaks of norovirus infection in Australia. *Clin Infect Dis* 2007;44:1026–1031.
2. Stafford R, Strain D, Heymer M, Smith C, Trent M, Beard J. An outbreak of Norwalk virus gastroenteritis following consumption of oysters. *Commun Dis Intell* 1997;21:317–320.
3. Symes SJ, Gunesekere IC, Marshall JA, Wright PJ. Norovirus mixed infection in an oyster-associated outbreak: an opportunity for recombination. *Arch Virol* 2007;152:1075–1086.
4. Fleet GH, Heiskanen P, Reid I, Buckle KA. Foodborne viral illness—status in Australia. *Int J Food Microbiol* 2000;59:127–136.
5. Murphy AM, Grohmann GS, Christopher PJ, Lopez WA, Davey GR, Millsom RH. An Australia-wide outbreak of gastroenteritis from oysters caused by Norwalk virus. *Med J Aust* 1979;2:329–333.
6. Food Standards Australia New Zealand. Australia New Zealand Food Standards Code. Available from: http://www.foodstandards.gov.au/thecode/. Accessed on 7 January 2008.
7. Son NT, Fleet GH. Behavior of pathogenic bacteria in the oyster, *Crassostrea commercialis*, during depuration, re-laying, and storage. *Appl Environ Microbiol* 1980;40:994–1002.
8. NSW Government. NSW Oyster Industry: Sustainable Aquaculture Strategy. Available from: http://www.dpi.nsw.gov.au/fisheries/aquaculture/publications/oysters/industry/nsw-oyster-industry-sustainable-aquaculture-strategy Accessed on 21 November 2007.
9. SafeFood New South Wales. New South Wales Shellfish Program Operations Manual. Available from: http://www.foodauthority.nsw.gov.au/industry/pdf/Manual-Shellfish-Program.pdf Accessed 21 November 2007.
10. Richards GP. Enteric virus contamination of foods through industrial practices: a primer on intervention strategies. *J Ind Microbiol Biotechnol* 2001;27:117–125.
11. Nishida T, Kimura H, Saitoh M, Shinohara M, Kato M, Fukuda S, et al. Detection, quantitation, and phylogenetic analysis of noroviruses in Japanese oysters. *Appl Environ Microbiol* 2003;69:5782–5786.
12. Dore WJ, Henshilwood K, Lees DN. Evaluation of F-specific RNA bacteriophage as a candidate human enteric virus indicator for bivalve molluscan shellfish. *Appl Environ Microbiol* 2000;66:1280–1285.
13. Burkhardt W, 3rd, Calci KR. Selective accumulation may account for shellfish-associated viral illness. *Appl Environ Microbiol* 2000;66:1375–1378.
14. Potasman I, Paz A, Odeh M. Infectious outbreaks associated with bivalve shellfish consumption: a worldwide perspective. *Clin Infect Dis* 2002;35:921–928.
15. Hay B, Scotti P. Evidence for intracellular absorption of virus by the Pacific oyster, *Crassostrea gigas*. *N Z J Mar Freshwater Res* 1986;20:655–659.
16. Kingsley DH, Meade GK, Richards GP. Detection of both hepatitis A virus and Norwalk-like virus in imported clams associated with food-borne illness. *Appl Environ Microbiol* 2002;68:3914–3918.
17. Dalton C. Commentary. An outbreak of Norwalk virus gastroenteritis following consumption of oysters. *Commun Dis Intell* 1997;21:321–322.
18. Agri-food & Veterinary Authority of Singapore. Annual Report 2006/07. Going the Extra Mile. Available from: http://www.ava.gov.sg/NR/rdonlyres/0676D1EB-C401-4038-9D8D-84A01B52DD27/18527/AVA07_CorporateReview.pdf Accessed 9 January 2008.
19. Institute of Environmental Science and Research. Norovirus Research Project Goal and Activities. Available from: http://www.esr.cri.nz/competencies/foodsafety/Norovirus+Research+Projects.htm Accessed 22 November 2007.

# THE PUBLIC HEALTH VALUE OF EMERGENCY DEPARTMENT SYNDROMIC SURVEILLANCE FOLLOWING A NATURAL DISASTER

Kirsty Hope, Tony Merritt, Keith Eastwood, Kelly Main, David N Durrheim, David Muscatello, Kerry Todd, Wei Zheng

## Abstract

During a recent natural disaster public health staff required timely and comprehensive surveillance of priority health conditions, including injury, mental health disorders and selected infectious diseases, to inform response and recovery activities. Although traditional surveillance is of value in such settings it is constrained by a focus on notifiable conditions and delays in reporting. The application of an electronic emergency department syndromic surveillance system proved valuable and timely in informing public health activities following a natural disaster in New South Wales. *Commun Dis Intell* 2008;32:92–94.

Keywords: syndromic surveillance, natural disaster, disease surveillance

## Introduction

The coastal region of Newcastle, north of Sydney in New South Wales, Australia, experienced severe storms over a two day period from 7 June 2007. Wind gusts exceeding 120 km/hour and rainfall of 209 mm in one day[1,2] fell in Newcastle and the inland Hunter region resulting in extensive damage, power failures and flooding to houses, businesses, schools, aged care facilities, hospitals and local infrastructure, severely affecting roads, power, water supplies and sewage systems. Over 200,000 homes were without power for up to seven days, resulting in loss of heating and considerable food spoilage. Seven evacuation centres were established for displaced residents. The storm also resulted in the grounding of a coal ship, the Pasha Bulker, storing over 700 tonnes of fuel oil,[2] on a Newcastle beach.

Hunter New England Population Health (HNEPH) contributed to the acute disaster response and was the lead health agency during the recovery phase. While maintaining core public health services, additional responsibilities included: environmental health field assessments, oversight of water quality, enhanced surveillance for health events, and maintaining communication systems by providing daily situation reports to relevant agencies, and preparing information for the public and media.

Natural disasters of a similar type and magnitude internationally have underlined the importance of timely surveillance systems for early identification of infectious disease outbreaks. In developed countries gastrointestinal and respiratory illnesses following flooding events are common.[3,4] Early identification allows appropriate resource allocation and preventative measures to be implemented to prevent further illness and to minimise the impact on the hospital/medical system and the community in general. Early identification is also vital in ensuring timely and accurate messages to the community.

Traditional surveillance focuses on notifiable infectious diseases with significant time delays while awaiting laboratory confirmation. Thus, new information sources were required to obtain a timely and comprehensive picture of priority health related events including infectious disease clusters, injury and mental health issues. Emergency Department (ED) data were regarded as the most reliable and timely source for this health intelligence.

## Method

The NSW Department of Health had previously established syndromic surveillance using a database of data routinely entered into computerised ED patient management information systems of 30 EDs in New South Wales; but this database did not cover the Hunter storm-affected region at the time of the storm. Following intense advocacy in the immediate period following the storm event, the network was extended to include 11 EDs in the disaster area from 16 June. From this time, standard reports from the New South Wales ED syndromic surveillance system were available, listing presentations for 37 syndromes based on ED provisional diagnosis. These syndromes included gastrointestinal, influenza, pneumonia, other/unspecified respiratory infections, all injury and mental health presentations. The reports were updated four times per day using data extracted from the ED surveillance database six times per day.

The New South Wales ED surveillance system standard reports are described elsewhere.[5] Briefly, statistical control charts are used to automatically detect increases in syndrome activity, using Poisson z-scores of observed versus expected day-of-week

and weekly counts, and a modified cumulative sum (cusum) method for counts. Expected syndrome incidence is determined using a baseline of the previous 51 weeks.[5]

Prior to these reports being available, ED data were manually extracted from the computerised patient management information system of all EDs in storm-affected areas, and stored in an Excel spreadsheet. The data extracted provided information on all gastroenteritis using ICD-10 codes and a summary of all visits that were identified as related to the storm event. Triage staff were requested to flag all storm-related presentations using a specific incident flag available in the standard computerised patient management software. In addition, further storm-related presentations were identified retrospectively using keyword searches in the nursing assessment field.

## Results

Prior to 16 June, the initial manual extraction of data required a hospital staff member to set aside their normal tasks and extract the information separately for each hospital each day. Due to the time required to extract data, we were obliged to limit syndromic surveillance to only one syndrome, gastroenteritis. Once the electronic ED-based syndromic surveillance system was implemented it provided reports on 37 syndromes. These reports were updated four times daily and were also run retrospectively for the period immediately following the storm to ensure that no post-storm presentation increases had been missed.

No gastroenteritis outbreaks were identified during the recovery phase. The New South Wales syndromic surveillance system detected increases in presentations of respiratory syndromes. However, these increases were within seasonally expected levels given the onset of winter when compared with ED data for the previous five years.

In the first two days following the storm event, 60% of the storm-related ED visits were due to hypothermia and the majority of the remainder resulted from traumatic injuries, including fractures and lacerations. The incident flag was poorly utilised by triage staff and the bulk of storm-related visits were identified retrospectively.

## Discussion

The initial manual extraction of ED data was time consuming and this limited the range of data that could be realistically obtained. The actual prevalence of hypothermia was not realised until after retrospective coding had occurred. In addition, as coding was conducted using keywords in the triage nursing assessment text, it is possible that additional presentations related to the storm were missed.

Once implemented the electronic ED-based syndromic surveillance system provided easily accessible and timely reports on a wide range of indicator syndromes. A hypothermia syndrome, based on ICD-10 codes can be made available through the electronic ED-based system for future incidents. If required a syndrome can also be made available for incident related presentations using key word searches in the nursing assessment field, negating the need for staff to manually flag presentations.

ED syndromic data proved a valuable source of information for public health staff and complemented existing surveillance. The data informed response planning, while also reassuring the public that measures taken were successfully mitigating the threat of infectious disease outbreaks.

The use of ED data during this natural disaster has shown that the timeliness and coverage of ED data captured through the New South Wales syndromic surveillance system makes it a valuable surveillance tool for the response and recovery phases following natural emergencies.

## Acknowledgement

We thank Hunter New England Health Information Technology staff and NSW Health Centre for Epidemiology and Research for implementing the ED surveillance system.

## Author details

Kirsty Hope[1,2]
Tony Merritt[1]
Keith Eastwood[1]
Kelly Main[1]
David N Durrheim[1,2]
David Muscatello[3]
Kerry Todd[1]
Wei Zheng[3]

1. Hunter New England Population Health, Wallsend, New South Wales
2. Newcastle Institute of Public Health, Newcastle, New South Wales
3. New South Wales Health Department

Corresponding author: Ms Kirsty Hope, Epidemiologist, Newcastle Institute of Public Health, Locked Bag 10, WALLSEND, NSW 2287. Telephone: +61 2 4926 4604 Email: kirsty.hope@hnehealth.nsw.gov.au

## References

1. Australian Government Bureau of Meteorology. Newcastle, New South Wales June 2007 daily weather observations. Available from: www.bom.gov.au/climate/dwo/200706/html/IDCJDW2097.200706.shtml. Accessed August 2007.
2. Guy Carpenter and Company Ltd. Australian East Coast Storm 2007: Impact of east coast lows. Available from http://gcportal.guycarp.com/portal/extranet/popup/insights/reportsPDF/2007/Australian%20East%20Coast%20Storm%20Report%202007.pdf?vid=4 Accessed 3 January 2008.

3. Watson J, Gayer M, Connolly M. Epidemics after Natural Disasters. *EID* 2007;13:1-5.
4. Centers for Disease Control and Prevention. After a Hurricane: Key facts about infectious disease. Available from: www.bt.cdc.gov/disasters/hurricanes/infectiousdisease.asp Accessed August 2007.
5. Muscatello D, Churches T, Kaldor J, Zheng W, Chiu C, Correll P, et al. An automated, broad-based, near real-time public health surveillance system using presentations to hospital Emergency Departments in New South Wales, Australia. *BMC Pub Hlth* 2005;5:141-152.

# School-based vaccinations delivered by general practice in rural north Queensland: an evaluation of a new human papilloma virus vaccination program

Carole Reeve, Stephanie De La Rue, Dennis Pashen, Margaret Culpan, Tracy Cheffins

## Abstract

A local general practice was contracted to provide the school-based immunisation program over two years in Mount Isa, Queensland. The schedule was for female Year 10, 11 and 12 students to receive three doses of human papilloma virus (HPV) vaccination (Gardasil). This was provided as part of the broader immunisation program that involved providing Year 8 students with two doses of hepatitis B vaccination and one dose of varicella-zoster, and Year 10 students with one dose of diphtheria-tetanus-pertussis (DTPa). Data were collected on the number of consent forms returned and how many declined vaccination, how many students were vaccinated and those requiring catch-up vaccinations, as well as the total number completing the full course of immunisations. Adverse events were also recorded. The total cohort of girls eligible for HPV vaccination was 304 (consented to vaccination – 275 (90%), declined vaccination – 13 (4%), coverage for first HPV dose – 89%, coverage for second HPV dose – 88%, coverage for third HPV dose – 79%). When compared with other adolescent vaccinations given concurrently as part of the broader vaccination program, HPV coverage was higher. There were only three significant adverse events. Three girls fainted at the time of immunisation but recovered immediately. The HPV immunisation had a good uptake and was well tolerated. Integrating school immunisation provision with general practice provides continuity with preschool immunisations and provides a convenient location for parents to bring children who have missed out on immunisations or would like to discuss the immunisation program further. *Commun Dis Intell* 2008;32:94–98.

Keywords: human papilloma virus, vaccination, school-based immunisation, consent, immunisation coverage

## Introduction

Worldwide, cervical cancer is the second most common cancer in women. Human papilloma virus (HPV) DNA is found in virtually all invasive cervical cancers.[1] There are over 100 HPV subtypes and at least 15 (HPV) types are human carcinogens that play a role in the pathogenesis of cervical and other cancers.[2]

Although the incidence of cervical cancer in Australia is low, the incidence of psychological and physical morbidity due to precancerous lesions is high.[3] Every year in Australia, pap screening detects about 100,000 abnormal smears and about 15,000 women undergo treatment for high-grade lesions.[4,5] Therefore the prophylactic prevention of HPV-related dysplasia and cancer would save lives, and reduce the need for colposcopy and other procedures.

The quadrivalent vaccine Gardasil (Merck) was developed in Australia and was approved in August 2006 for use in young females aged 9–25 years. If administered prior to sexual activity, Gardasil provides 90%–100% protection against persistent infection and cervical/genital disease due to HPV types 16 and 18 which cause 70% of cervical cancer cases and 50% of high grade cervical abnormalities, and HPV types 6 and 11,[6,7] which are associated with 90% of cases of genital warts and approximately 10% of low grade cervical abnormalities.[3]

Though Gardasil is very effective in providing protection against HPV infection it is not a treatment and consequently has no effect in women with infection prior to vaccination. However, clinical trials have found that many women are only infected with one type of the virus so sexually active women are still likely to benefit from vaccination.[7]

In November 2006 the Australian Government announced that a national school-based HPV immunisation program would be funded from April 2007. The program has two phases delivered in schools (Box):

- a two year catch-up for girls aged 13–18 years provided in schools;
- from 2009, an on-going program for girls aged 12–13 years.

A further catch-up program for women up to and including 26 years will be funded for two years from 2007 and is to be delivered through general practice.

This paper presents data regarding the implementation of the HPV vaccination program in Mount Isa, a rural north Queensland city with two high schools and an Indigenous population of 19% based on census figures.[8]

## Method

In Mount Isa, the school-based vaccination program was tendered out to a local general practice. The program was carried out in the two local high schools. The practice manager, administration staff and practice nurse organised the program and nurse immunisers were employed on a casual basis to provide the immunisations in the schools.

Consent forms were delivered to the schools by the immunisation team and sent home to parents with the students for signing. The immunisation team then collected them from the school office.

An immunisation day was allocated for each age group and the team vaccinated all students with signed consent forms. Follow-up days were allocated to provide vaccinations for students who were absent on the initial day of immunisation. In addition, students who missed out on immunisation at school could be brought in by their parents to the general practice providing the school-based program.

Data were collected on the number of consent forms returned, the number declined, the number of students actually vaccinated and the number requiring catch-up vaccination. Also recorded were minor and significant adverse events.

These data were entered into the Queensland Health MS Excel datasheet provided as part of the contract. The rates of consent form return and immunisation coverage were calculated automatically using the total cohort of students as the denominator. The data presented in this paper are from the first six months of the program, July–December 2007.

## Results

### Human papilloma virus consent

The total cohort of girls eligible for HPV vaccination was 304, of which:

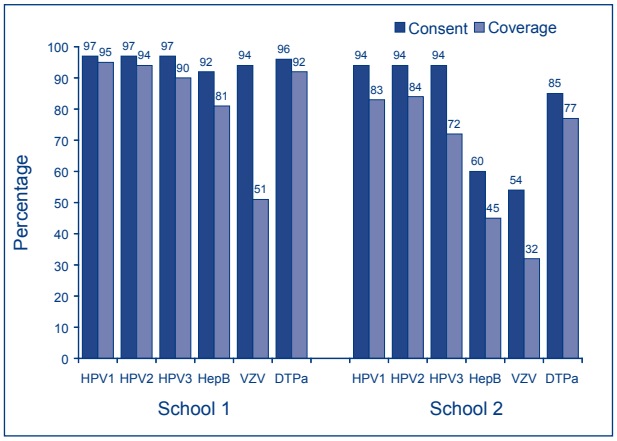
- 285 (94%) returned consent forms;
- 275 (90%) consented to vaccination; and
- 13 (4.3%) declined vaccination.

**Table. Schedule for the Queensland school-based vaccination program**

| HPV |
|---|
| **2007** |
| Three doses of HPV vaccination for females in Years 10, 11 and 12 with one to two months between 1st and 2nd doses and four months for the 3rd (abbreviated catch-up schedule) |
| **2008** |
| Three doses of HPV vaccination for females in Years 8, 9 and 10 with one to two months between 1st and 2nd doses and six months for the 3rd |
| **2009** |
| HPV will form part of the ongoing school based vaccination program delivered to female Year 8 students in conjunction with hepatitis B and varicella |
| **Hepatitis B vaccination** |
| Two doses for Year 8 students not previously vaccinated, with an interval of four to six months. |
| **Varicella-zoster** |
| One dose for Year 8 students not previously infected or vaccinated, given at the same time as hepatitis B. |
| **Diphtheria-tetanus-pertussis (DTPa)** |
| One dose for Year 10 students with no previous booster who have had a previous primary course. |

The overall rate of consent form return for HPV was 97% in School One and 94% in School Two. Consent rates of greater than 80% were achieved for HPV, hepatitis B and varicella-zoster in School One, while in School Two only the HPV vaccination achieved consent rates above 85% (Figure 1).

**Figure 1. HPV consent form return rates and actual vaccination coverage compared with the other vaccinations given as part of the same vaccination program HPV coverage**




HPV Human papilloma virus.
HepB Hepatitis B vaccine
VZV Varicella zoster vaccine
DTPa Diphtheria-tetanus-pertussis vaccine

One parent declined to consent to the HPV vaccination in School One.

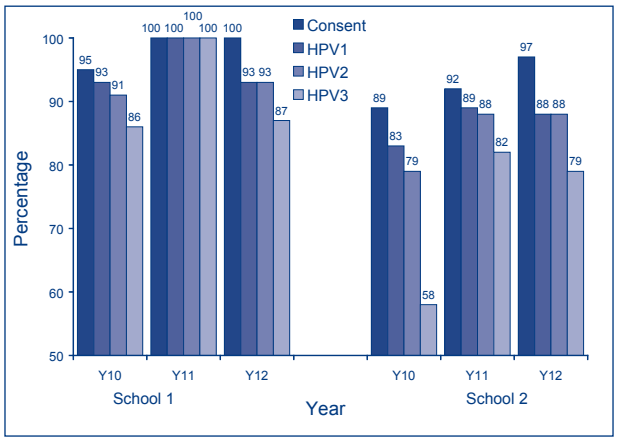
In School Two, 12 parents declined HPV vaccination for their daughters.

A combined coverage rate of 82% was achieved for both schools for the first HPV dose. After the catch-up program had been administered this rose to 89%.

Coverage rate for the second dose was 76% and was particularly low among Year 10 students in School Two, a large number of whom were absent on the day of immunisation. Coverage for the second HPV dose was 88% after parents brought their adolescents into the practice for catch-up doses. Coverage for the third dose was 72%, and 79% after the catch-up program (Figure 2).

Fifty-three adolescents had their catch-up doses through the general practice.

**Figure 2. Consent and coverage rates for human papilloma virus vaccination doses administered as part of the Mount Isa school-based vaccination program**




HPV Human papilloma virus.

### Adverse events

There were only three significant adverse events. Three girls fainted at the time of immunisation but recovered immediately. Quite a few girls complained of nausea and feeling dizzy but did not require any medical intervention.

### Queensland immunisation coverage

The school based immunisation program commenced simultaneously across Queensland in June 2007 and was divided into Northern, Central and Southern regions.

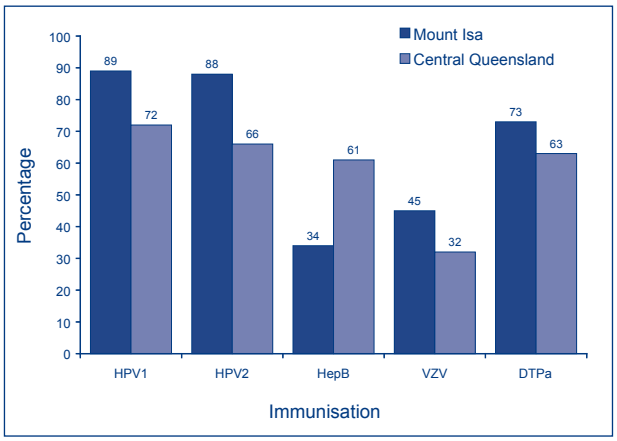
Vaccination coverage data from the Central region was provided by written communication from Queensland Health for comparative purposes. This program was delivered at the same time and using the same schedule as the Mount Isa program and involved a cohort of 39,000 students in 228 schools. Central Queensland HPV coverage rates for doses one and two were 72% and 66% respectively. Overall immunisation rates achieved in Mount Isa were higher than those in Central Queensland, although hepatitis B was lower (Figure 3).

## Discussion

HPV vaccination is a new program and there are little data available on HPV immunisation coverage in Australia. In this study two comparisons were made.

The overall rate of consent form return and coverage for HPV was higher than for the other vaccinations administered as part of the broader immunisation program. Coverage decreased with subsequent

**Figure 3. Comparison of immunisation coverage between Mount Isa and Central Queensland**




| | |
|---|---|
| HPV | Human papilloma virus. |
| HepB | Hepatitis B vaccine |
| VZV | Varicella zoster vaccine |
| DTPa | Diphtheria-tetanus-pertussis vaccine |

doses of HPV. This has also been noted with school hepatitis B programs.[9] When compared with other adolescent vaccinations given in Mount Isa, HPV coverage was the highest.

Overall North Queensland data are currently unavailable, but comparisons can be made with data from Central Queensland programs that were conducted mostly through local councils rather than general practice. Overall immunisation rates achieved in Mount Isa were higher than those in Central Queensland, although hepatitis B was lower. This is thought to be due to the fact that many children had already been immunised against hepatitis B in childhood.

High overall coverage in Mount Isa was likely to be due to the relatively small number of adolescents in the cohort. The prominent and convenient location of the practice also made it easy for parents to bring their adolescents in for catch-up doses.

Two surveys prior to the introduction of HPV predicted consent rates of 77% and 54% in spite of the fact that few participants linked HPV with cervical cancer.[10,11] Coverage rates achieved for HPV in this study were higher than these predictions.

Importantly, significant adverse events from HPV vaccination experienced during this study were limited to vasovagal events. Anecdotally, it was noted that students complained that each subsequent dose was more painful that the first with increased stinging occurring immediately at the site of injection but resolving over several minutes.

Getting signed consent forms back from parents in a timely manner was the most challenging aspect of the program. In 2008, the HPV immunisation program will cover girls in Years 8–10 and from 2009 HPV will become part of the Year 8 schedule. This study found that the Years 8–10 students had lower consent and coverage rates, which could impact coverage levels in the future.

The other difficulty encountered was adolescents being absent from school. This was addressed, in part, by allowing parents to bring students into the practice themselves to ensure that the immunisations were administered.

Of the 53 adolescents who had their catch-up doses through the general practice, most had signed consent forms but were absent from school on the immunisation day. However, eight doses were given opportunistically to adolescents when they presented at the practice for other reasons and the parents signed their consent forms.

Australia has had immunisation programs since the 1920s. However, it wasn't until the polio campaigns of the 1950s that these programs were delivered in schools. Since then, there has been a move towards immunisation in general practice as part of the National Immunisation Strategy.[12] With more immunisations being given in childhood and the potential loss of lifetime immunity provided by the disease, boosters are now being required during adolescence and schools provide a suitable environment for their administration.

Based on this experience, we believe general practice to be an ideal provider of school based immunisation programs. Utilising a combined approach of mass school based vaccination programs and catch-up and opportunistic doses given in the general practice, very good coverage rates can be achieved.

This approach also provides good continuity with existing preschool immunisation, and HPV immunisation of women who have left school.

## Acknowledgements

We would like to acknowledge the school-based immunisation team lead by Sheila Simpkins; Nicola Moorby, Bubs Davis, Kevin Gallagher and Ken Gilberd. The assistance of the Tropical Population Health Unit is gratefully acknowledged.

Financial support for writing this article was provided by a Primary Health Care Research, Evaluation and Development Fellowship.

## Author details

Dr Carole Reeve[1]
Dr Stephanie De La Rue[1]
Assoc Prof Dennis Pashen[1]
Dr Margaret Culpan[2]
Dr Tracy Cheffins[3]

1. Mount Isa Centre for Rural and Remote Health, Mount Isa, Queensland
2. Outback Family Medicine, Mount Isa, Queensland
3. School of Medicine Rural Health Research Unit, James Cook University, Townsville, Queensland

Corresponding author: Dr Stephanie De La Rue, Mount Isa Centre for Rural and Remote Health, PO Box 2572, MOUNT ISA QLD 4825. Telephone: +61 7 4744 7913. Facsimile: +61 7 4749 5130. Email: Stephanie.delarue@jcu.edu.au

## References

1. Parkin DM, Bray F, Ferlay J, Pisani P. Estimating the world cancer burden: Globocan 2000. *Int J Cancer* 2001;94:153–156.
2. Gillison ML, Shah KV. Role of mucosal human papillomavirus in nongenital cancers. *J Natl Cancer Inst Monogr* 2003;9:57–65.
3. May J. HPV vaccination – a paradigm shift in public health. *Aust Fam Physician* 2007;36:10.
4. Wain, GV. Cervical cancer prevention: the saga goes on, but so much has changed! *Med J Aust* 2006;185:476–477.
5. National Health and Medical Research Council. Screening to prevent cervical cancer: guidelines for the management of asymptomatic women with screen detected abnormalities. Canberra: National Health and Medical Research Council; 2005.
6. Villa LL, Costa RL, Petta CA, Andrade RP, Ault KA, et al. Prophylactic quadrivalent human papillomavirus (types 6, 11, 16, and 18) L1 virus-like particle vaccine in young women: a randomised double-blind placebo-controlled multicentre phase II efficacy trial. *Lancet Oncology* 2005;6:271–278.
7. Siddiqui A, Perry C. Human papillomavirus quadrivalent (types 6, 11, 16, 18) recombinant vaccine (Gardasil). *Drugs* 2006;66:1263–1271.
8. Australian Burea of Statistics. Census of Population and Housing, Indigenous Profile, Table 101. Canberra: Australian Burea of Statistics,1996.
9. Dilraj A, Strait-Jones J, Nagao M, CuiK, Terrell-Perica S, Effler PV. A statewide hepatitis B vaccination program for school children in Hawaii; vaccination series completion and participation rates over consecutive school years. *Public Health Rep* 2003;118:127–133.
10. Marshall H, Ryan P, Roberton D, Baghurst P. A cross-sectional survey to assess community attitudes to introduction of human papillomavirus vaccine. *Aust N Z J Public Health* 2007;31:235–242.
11. Hausdorf K, Newman B, Whiteman D, Aitken J, Frazer I. HPV vaccination what do Queensland parents think? *Aust N Z J Public Health* 2007;31:288–289.
12. National Immunistion Strategy 1993. National Health and Medical Research Council, Commonwealth of Australia 1993.

Quarterly reports

# OzFoodNet quarterly report, 1 October to 31 December 2007

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 outbreaks of gastrointestinal illness and clusters of disease potentially related to food, occurring in Australia from 1 October to 31 December 2007.

Data were received from OzFoodNet representatives in all Australian states and territories and a sentinel site in the Hunter/New England region of New South Wales. The data in this report are provisional and subject to change as the results of outbreak investigations can take months to finalise.

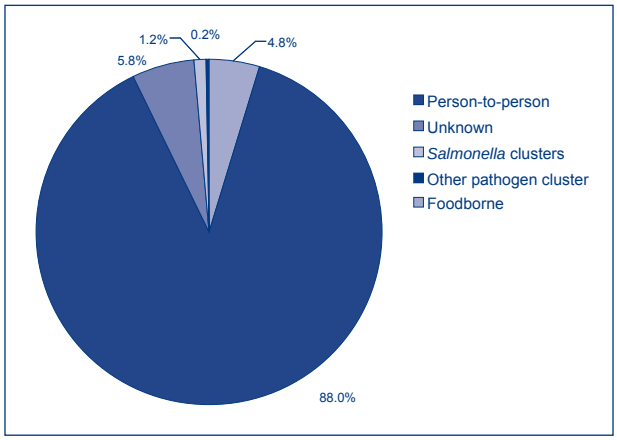
During the fourth quarter of 2007, OzFoodNet sites reported 565 outbreaks of enteric illness, including those transmitted by contaminated food. Outbreaks of gastroenteritis are often not reported to health agencies or the reports are delayed, meaning that these figures under-represent the true burden of enteric illness. In total, these outbreaks affected 9,891 people, of which 256 were hospitalised and 29 people died. The majority (88%, n=497) of outbreaks resulted from infections due to person-to-person transmission (Figure).

## Foodborne disease outbreaks

There were 27 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as the primary mode of transmission (Table). These outbreaks affected 314 people and resulted in 31 people being admitted to hospital. There were no deaths reported. This compares with 34 outbreaks for the fourth quarter of 2006 and 36 outbreaks in the previous quarter of 2007.

*Salmonella* was responsible for 13 outbreaks during this quarter, with *Salmonella* Typhimurium being the most common serotype. Various phage types of *S.* Typhimurium were reported as the cause of these outbreaks including *S.* Typhimurium 44 in four outbreaks, *S.* Typhimurium U307 and *S.* Typhimurium 29 each in two outbreaks, and *S.* Typhimurium 135a

**Figure. Mode of transmission for outbreaks of gastrointestinal illness reported by OzFoodNet sites, 1 October to 31 December 2007**




and *S.* Typhimurium 9 each in one outbreak. The other *Salmonella* serotypes causing outbreaks were *S.* Kiambu, *S.* Saintpaul, and *S.* Tennessee.

Norovirus was associated with six foodborne outbreaks during this quarter. *Campylobacter* was reported in a single outbreak. There were four toxin-related outbreaks during the quarter including two ciguatera fish poisoning outbreaks, and single outbreaks of *Bacillus cereus* intoxication and *Clostridium perfringens* intoxication. The remaining three outbreaks were caused by unknown aetiological agents.

Seven outbreaks reported in this quarter were associated with food prepared at restaurants, six from food prepared at private residences and four with food prepared by commercial caterers. Food prepared by takeaway outlets, institutions and aged care facilities were each associated with two outbreaks. Two outbreaks resulted from contaminated primary produce—Spanish mackerel and coral trout. Single outbreaks were associated with food prepared for a camp and by a bakery.

To investigate these outbreaks, sites conducted one cohort study, and collected case series data in 19 investigations. There were seven outbreaks where no individual patient data were collected.

## Table. Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 October to 31 December 2007

| State | Month of outbreak | Setting prepared | Aetiological agent | Number affected | Evidence | Responsible vehicles |
|---|---|---|---|---|---|---|
| NSW | October | Private residence | Unknown | 7 | D | Suspected watermelon |
| | | Bakery | *S.* Typhimurium 44 | 27 | D | Uncooked cheese/cream cake |
| | November | Takeaway | *Campylobacter* | 2 | D | Meat kebab |
| | | Private residence | *S.* Typhimurium 9 | 11 | D | Multiple foods |
| | | Restaurant | Unknown | 3 | D | Unknown |
| | | Private residence | *S.* Typhimurium 29 | 8 | D | Suspected beef patties/ home made icecream |
| | | Private residence | *S.* Typhimurium 29 | 3 | D | Eggnog |
| Qld | October | Aged care | *S.* Kiambu | 2 | D | Unknown |
| | November | Takeaway | *Bacillus cereus* | 3 | M | Fried rice/honey chicken |
| | | Primary produce | Ciguatera fish poisoning | 2 | D | Coral trout |
| | | Restaurant | *S.* Typhimurium U307 | 3 | D | Unknown |
| | | Institution | *S.* Typhimurium U307 | 6 | D | Unknown |
| | December | Primary produce | Ciguatera fish poisoning | 2 | D | Spanish mackerel |
| | | Restaurant | Norovirus | 34 | D | Unknown |
| | | Private residence | Norovirus | 5 | D | Salad |
| Tas | October | Restaurant | Unknown | 12 | D | Unknown |
| | | Restaurant | *S.* Typhimurium 135a | 2 | D | Unknown |
| Vic | October | Commercial caterer | Norovirus | 18 | A | Fruit salad |
| | | Restaurant | *S.* Typhimurium 44 | 16 | M | Chicken foccacia/raw egg aioli |
| | | Aged care | *S.* Saintpaul | 3 | D | Unknown |
| | | Commercial caterer | Norovirus | 34 | D | Unknown |
| | | Commercial caterer | Norovirus | 53 | D | Unknown |
| | November | Private residence | *S.* Typhimurium 44 | 13 | D | Unknown |
| | | Institution | *Clostridium perfringens* | 7 | D | Unknown |
| | December | Restaurant | *S.* Typhimurium 44 | 13 | M | Eggs used in an undercooked food (risottini) and cross contamination |
| WA | November | Camp | *S.* Tennessee | 12 | D | Unknown |
| | December | Commercial caterer | Norovirus | 13 | D | Unknown |

* No foodborne outbreaks were reported in the Australian Capital Territory, South Australia or the Northern Territory during the quarter.

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.

Investigators obtained analytical epidemiological evidence in one outbreak and microbiological evidence in three outbreaks. For the remaining 23 outbreaks, investigators obtained descriptive evidence implicating the food vehicle or suggesting foodborne transmission.

The following jurisdictional summaries describe key outbreaks that occurred in this quarter.

### New South Wales

New South Wales reported seven outbreaks of foodborne illness during this quarter. An outbreak of *Salmonella* Typhimurium 9 affected 11 of 13 people attending a private residence. Illness was associated with a shared meal of multiple foods that included raw eggs as ingredients.

*S.* Typhimurium 29 was identified as the aetiologic agent in two outbreaks. One outbreak affected three people who drank an eggnog milkshake that included raw egg as an ingredient. The second outbreak affected eight family members attending a home-catered party. The food source for the party was not identified but the meal included beef patties, eggs and a home-made icecream.

New South Wales also investigated a geographic cluster (27 cases) of *S.* Typhimurium 44. Interviews of some cases (11/17) showed that they had eaten either a cheese or cream cake from one bakery. The New South Wales Food Authority noted that raw egg was an ingredient used in the preparation of the base for these cakes. New South Wales also reported an outbreak of two cases of campylobacteriosis associated with meat kebabs and prepared by a takeaway outlet.

**Queensland**

Queensland reported eight outbreaks during this quarter. *Salmonella* Kiambu caused illness in two residents of a Queensland aged care facility. Two outbreaks of *Salmonella* Typhimurium U307 caused three cases of illness among patrons of the same restaurant and six cases of illness among residents of a mental health residential facility. Investigators were unable to identify the food or environmental cause for these three *Salmonella* outbreaks.

Queensland reported two ciguatera fish poisoning outbreaks during the quarter. Coral trout purchased from a fish market in Gladstone caused two cases of this toxin-related illness. The other outbreak of two cases was from consumption of a Spanish mackerel caught by a recreational fisherman in a channel between Upolu, Vlasoff and Michaelmas Cays (Great Barrier Reef).

Queensland investigated an outbreak of three cases of *Bacillus cereus* intoxication from a meal of fried rice and honey chicken. Both food and vomitus specimens were positive for *Bacillus cereus* toxin gene.

Norovirus caused two outbreaks of gastrointestinal illness; both were likely due to food handlers working while they were infectious. These outbreaks of norovirus involved illness among 34 patrons who had dined at a restaurant and five people eating at a private residence.

**Tasmania**

Tasmania reported two outbreaks during this quarter. Two cases of *Salmonella* Typhimurium 135a were notified from the same household in north-west Tasmania. A restaurant was suspected as being the source of infection. No other salmonellosis cases, associated with this business, were identified nor any links identified between these cases and recent *S.* Typhimurium 135a clusters.[1]

Tasmania investigated foodborne illness among 12 patrons who had eaten the same meal at a restaurant. Cases onset of symptoms began 30–40 hours after meals were consumed and included abdominal pains, vomiting and diarrhoea. A source for the outbreak was not identified.

**Victoria**

Victoria reported eight outbreaks of foodborne illness during this quarter. Two cases became sick within one day of each other in an outbreak caused by *Salmonella* Saintpaul in an aged care facility. Both cases were residents of the facility but did not have contact with each other. A third case was a staff member who had an onset of illness at the same time as the residents – this staff member was negative for *Salmonella*. A source for this outbreak was unable to be identified despite an extensive investigation.

There were three outbreaks caused by *Salmonella* Typhimurium 44 investigated in Victoria during the quarter. *S.* Typhimurium 44 affected 16 patrons of a restaurant. All cases ate the chicken foccacia that included a raw egg aioli. A sample from the outside of the blender used to make the aioli was positive for *S.* Typhimurium 44.

A second outbreak caused by *S.* Typhimurium 44 was reported in 13 cases (patrons and staff) of a restaurant. Food (risottini and stuffed mushrooms) and a sample from the outside of the blender that was used to make a raw egg aioli were positive for *S.* Typhimurium 44. This outbreak was suspected to have been caused by the use of raw eggs in an undercooked food, and cross contamination of other food within the restaurant.

Victoria investigated a third outbreak of *S.* Typhimurium 44 where several home prepared foods were served at a private residence. Thirteen cases of illness were identified including two cases who provided faecal specimens that were positive for *S.* Typhimurium 44. The cause of this outbreak was unable to be identified.

Three outbreaks were suspected to have been caused by food handlers working while they were infectious with norovirus. The first outbreak involved a commercial caterer who provided a lunch that included various meats, vegetables and desserts. A food handler was symptomatic with vomiting and diarrhoea a few days prior to preparing food for the function. An investigation showed a statistically significant association between consumption of fruit salad and illness (RR 4.5; 95%CI 1.2–16.7). The

second norovirus outbreak was confirmed in four separate groups who had a catered lunch provided by the same commercial caterer over a two day period. One food handler was ill with abdominal pain and nausea on the day prior to the food being provided to customers. Three further staff became ill with vomiting and diarrhoea at the same time the customers became ill three days later. One of the positive specimens was from a food handler. The third of these norovirus outbreaks saw two separate groups of illness on successive weekends. The first group involved 53 guests who became ill. It is likely that a child who had vomited in the dining room was the source of illness for the guests and some of the staff. Two staff became ill after this function, and their illness was consistent with the guests. The following weekend there were two separate groups who dined at the same function centre and illness was reported in guests of both groups. It is suspected that an infectious food handler contaminated multiple foods during preparation and was the source of illness for the people who dined on this second weekend.

**Western Australia**

Western Australia reported two outbreaks of foodborne illness during this quarter. An outbreak of gastroenteritis caused by norovirus occurred among people who ate at a Western Australia function centre on two evenings. The cause of illness in the 13 cases was suspected to be foodborne as two food handers prepared food while ill with symptoms consistent with norovirus infection.

An outbreak of *S.* Tennessee infection that occurred at a railway construction camp in the north of Western Australia was suspected to be either food– or water-borne. *S.* Tennessee has been detected previously in drinking water from this region and drinking water at the camp had a history of contamination. A treatment system was fitted and the drinking water sampled was subsequently free of microbial contamination. In addition, a food handler who had prepared salads for lunches had *S.* Tennessee infection. Lunches were prepared in the morning and stored at ambient temperature until consumption. Ambient temperatures at the time of the outbreak were greater than 40°C.

## Comments

During the quarter, Western Australia also investigated a travel-acquired outbreak of *Cyclospora cayetanensis,* which is rarely reported as a cause of illness in Australia. It involved a cluster of eight cases from a family of 13 members, including two cases who provided faecal specimens that were positive for *C. cayetanensis*. The family had recently completed a five day cruise on a ship that departed and returned to Singapore. Symptoms reported by cases included diarrhoea, nausea and abdominal discomfort. A source for the infection was not identified.

*C. cayetanensis* infection is usually via a food– or water-borne contamination typically associated with imported produce or with international travel.[2] The mode of transmission is faecal–oral or ingestion through ingestion of contaminated food or water. Person to person exposure is unlikely as oocysts typically become infectious after maturing days to weeks after excretion. Outbreaks have not been associated with cooked or frozen food.[3]

*C. cayetanensis* gastroenteritis causes watery diarrhoea, anorexia, fatigue and weight loss. In Australia, the infection should be considered as a potential cause of traveller's diarrhoea in people returning from overseas.[2,4] However, a seemingly isolated case of *C. cayetanensis* without a travel history may be outbreak related and the need for further action discussed with public health officials.[4]

Eggs and egg-containing dishes were identified as the most common outbreak food vehicle in 2006 and responsible for 14% (16/115) of foodborne outbreaks.[3] This continued in 2007 and the fourth quarter with 19% (5/27) of foodborne outbreaks associated with uncooked or lightly cooked foods that had raw eggs as an ingredient. All jurisdictions, except for the Northern Territory, have reported egg-related *Salmonella* outbreaks due to various strains of *Salmonella* Typhimurium in 2006 and 2007.[3] An assortment of food vehicles were identified in these outbreaks, including dessert, salad dressing, sauce, milkshake and under/lightly cooked egg.

The reasons for the increase in outbreaks linked with eggs are unclear, but it has stimulated national discussions about means to prevent disease from potentially contaminated eggs. In August 2007, Federal, State and Territory governments met with industry and stakeholders at the National Egg Food Safety Summit to discuss how to tackle the problem of egg-associated illness.

Continued liaison between food safety agencies and the egg industry are paramount given egg-associated outbreaks of salmonellosis. There would be value in the development of nationally consistent guidelines on the use of eggs for the entire food service industry, specifically targeting restaurants and bakeries that have been the source of many of these outbreaks. In addition, the general public require continuing education on the safe handling and use of eggs in the home.

## Acknowledgements

OzFoodNet thanks the investigators in the public health units and state and territory departments of health, as well as public health laboratories and local government environmental health officers who provided data used in this report. We would also like to thank laboratories conducting serotyping and phage typing of *Salmonella* for their ongoing work during this quarter.

The OzFoodNet Working Group is *(in alphabetical order)*: Mary Barker (WA), Robert Bell (Qld), Charlie Blumer (ACT), Roy Byun (NSW), Adam Craig (NSW), Craig Dalton (Hunter New England), Gerard Fitzsimmons (DoHA), Kathleen Fullerton (DoHA) Robyn Gibbs (WA), Joy Gregory (Vic), Gillian Hall (NCEPH), Michelle Harlock (NT), Geoff Hogg (MDU), Martyn Kirk (DoHA), Karin Lalor (Vic), Michelle McPherson (SA), Tony Merritt (Hunter New England), Sally Munnoch (Hunter New England), Jennie Musto (NSW), Lillian Mwanri (SA), Rhonda Owen (DoHA), Chris Oxenford (ACT), Raj Patil (DAFF), Nevada Pingault (WA), Jane Raupach (SA), Mark Salter (FSANZ), Cameron Sault (Tas), Russell Stafford (Qld), Nicola Stephens (Tas), Hassan Vally (NCEPH), Tory Worgan (Hunter New England).

## Author details

Correspondence: Mr Gerard Fitzsimmons, Epidemiologist, OzFoodNet, Office of Health Protection, Australian Government Department of Health and Ageing, GPO Box 9848, MDP 14, CANBERRA ACT 2601. Telephone: +61 2 6289 2748. Facsimile:+61 2 6289 2500. Email: gerard.fitzsimmons@health.gov.au

## References

1. Stephens N, Sault C, Firestone SM, Lightfoot D, Bell C. Large outbreaks of *Salmonella* Typhimurium phage type 135 infections associated with the consumption of products containing raw egg in Tasmania. *Commun Dis Intell* 2007;31:118–124.
2. Herwaldt B. *Cyclospora cayetanensis*: A Review, Focusing on the outbreaks of cyclosporiasis in the 1990s. *Clin Infect Dis* 2000;31:1040–1057.
3. OzFoodNet Working Group. Monitoring the incidence and causes of diseases potentially transmitted by food in Australia: Annual report of the OzFoodNet network, 2006. *Commun Dis Intell* 2007;31:345–365.
4. Pingé-Suttor V, Douglas C, Wettstein A. *Cyclospora* infection masquerading as coeliac disease. *Med J Aust* 2004;180:295–296.

# Communicable diseases surveillance

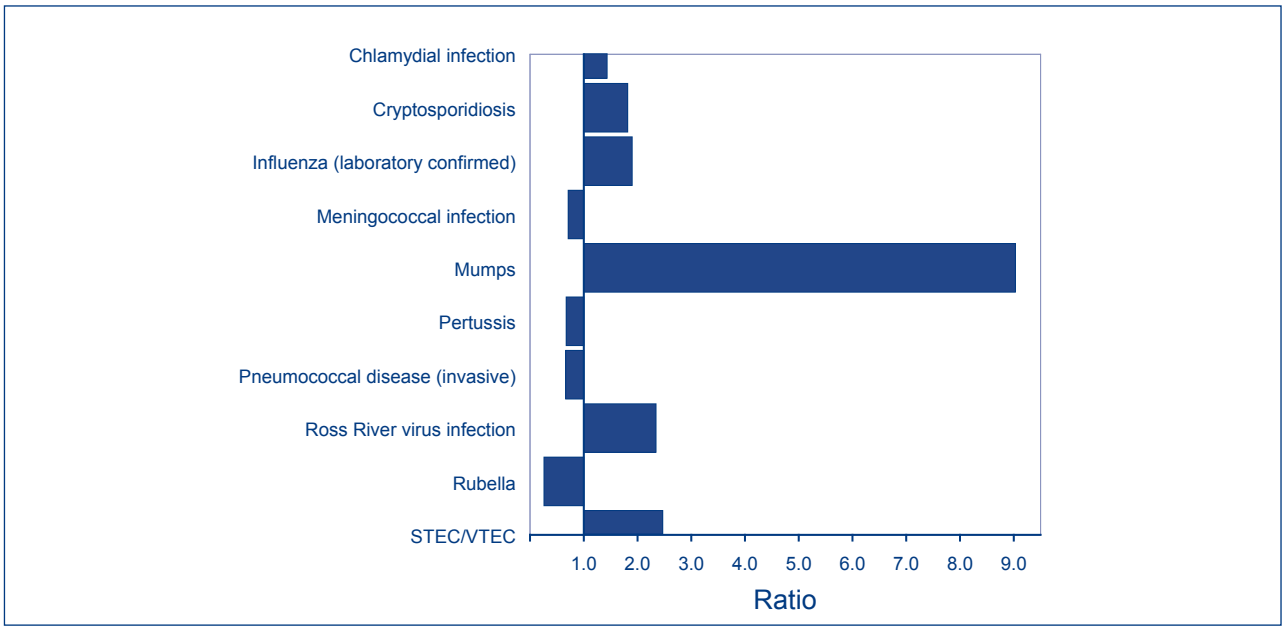
## Highlights for 4th quarter, 2007

*Communicable diseases 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 and 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' while data from the LabVISE scheme are referred to as 'laboratory reports'.*

Figure 1 shows the changes in selected disease notifications to the National Notifiable Diseases Surveillance System (NNDSS) with an onset in the fourth quarter (October to December) 2007, in comparison with the five-year mean for the same period. Notifications were above the five-year mean for chlamydial infections, cryptosporidiosis, influenza (laboratory confirmed), mumps, Ross River virus and Shiga toxin-producing/verotoxin-producing *Escherichia coli* (STEC/VTEC). Notifications were below the five-year mean for meningococcal infection, pertussis, invasive pneumococcal disease and rubella.

**Figure 1. Selected* diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 October to 31 December 2007 with historical data***




* Selected diseases are chosen each quarter according to current activity. Five year averages and the ratios of notifications in the reporting period in the five year mean should be interpreted with caution. Changes in surveillance practice, diagnostic techniques and reporting, may contribute to increases or decreases in the total notifications received over a five year period. Ratios are to be taken as a crude measure of current disease activity and may reflect changes in reporting rather than changes in disease activity. See Table 1 for all diseases.

† Ratio of current quarter total to mean of corresponding quarter for the previous five years.

## Gastrointestinal diseases

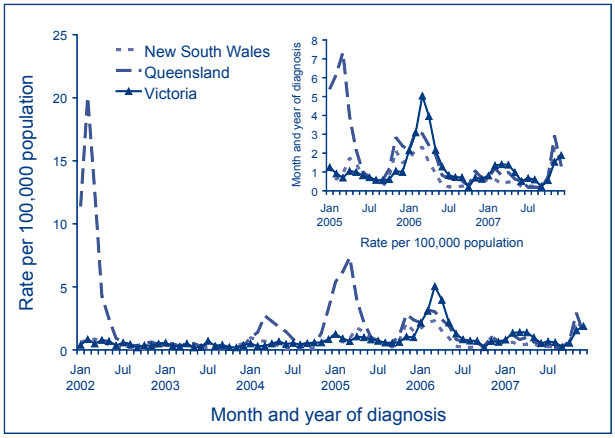
### Cryptosporidiosis

There were 805 notifications of cryptosporidiosis between 1 October and 31 December 2007, which was 80% higher than the five-year mean for previous corresponding quarters. All jurisdictions reported cases during the fourth quarter, with the majority from New South Wales (300), Queensland (207) and Victoria (207) (Figure 2).

The total for the quarter represented a substantive increase from the previous quarter and the same quarter for 2006. Notifications of cryptosporidiosis peak in the summer months, with increases in notifications commencing in the fourth quarter and peaking in the first quarter of the following year.

New South Wales reported 37% (300) of the total number of cases reported nationally. Over a third of the New South Wales cases notified were in the age range of 1 to 4 years. A range of possible risk factors reported in a New South Wales media release included: contact with farm animals, consumption of untreated water and swimming; however there were no significant common source outbreaks identified.[1]

**Figure 2. Notification rates of cryptosporidiosis, New South Wales, Queensland and Victoria, 2002 to 2007**




### Haemolytic uraemic syndrome

Haemolytic uraemic syndrome (HUS) is a rare condition characterised by progressive renal failure associated with microangiopathic haemolytic anaemia (red blood cell destruction), and thrombocytopaenia (platelet reduction and bleeding into the skin). HUS can occur following a variety of associated diseases, diarrhoeal and non-diarrhoeal. Diarrhoeal associated disease is the most common cause of HUS, specifically, infections associated with *Shigella dysenteriae* type 1 and, most commonly, STEC/VTEC.[2]

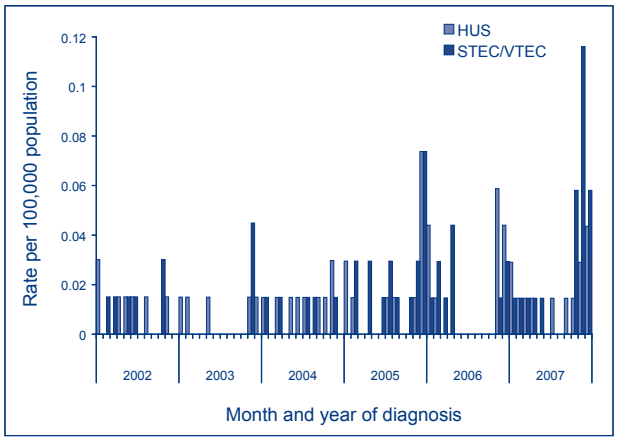
Reporting of a confirmed case of HUS on the NNDSS requires clinical evidence based on acute microangiopathic anaemia on a peripheral blood smear and either acute renal impairment or thrombocytopaenia.[3]

Approximately 10% of persons with an infection of STEC/VTEC will go on to develop this condition.[4,5] Where STEC/VTEC is isolated in the context of HUS, they are notified as both STEC/VTEC and HUS in NNDSS.

There were seven cases of HUS notified in the fourth quarter of 2007. This was 16% higher then the five-year mean for the corresponding period. Six of the cases notified were in New South Wales, however no common associations or exposures were identified between the cases.

Of these six HUS cases notified in New South Wales, one case was co-notified with a STEC infection. The number of STEC/VTEC cases notified during this period was 41, New South Wales notified 39% (16) of these cases, and nationally this was 2.5 times the five-year mean for the corresponding period. Figure 3 shows the notification rates of HUS and STEC/VTEC in New South Wales between 2002 and 2007.

**Figure 3. Notification rates of haemolytic uraemic syndrome and Shiga toxin-producing/verotoxin-producing *Escherichia coli*, New South Wales, 2002 to 2007**



## Quarantinable diseases

### Cholera

Cholera is one of eight human diseases that are currently subject to quarantine controls in Australia. The notifiable serogroups for cholera are toxigenic *Vibrio cholerae* O1 and O139.[3] Although there are over 200 *V. cholerae* serogroups, non-O1 and non-O139 groups rarely elaborate cholera enterotoxin.[4]

One case of cholera was notified in Queensland during the fourth quarter of 2007. The case was a 46-year-old female who acquired the infection whilst travelling through India as part of a tour group. The infecting organism was identified as toxigenic *Vibrio cholerae* O1 Ogawa. Investigations undertaken by Queensland Health noted that other members of the tour group also became ill during the tour, however no additional cases were discovered in Australia.

This case represented one of three cholera notifications that were reported in Australia in 2007. The average number of cases over the last five years was 3.4 cases per year.

## Vaccine preventable diseases

### Mumps

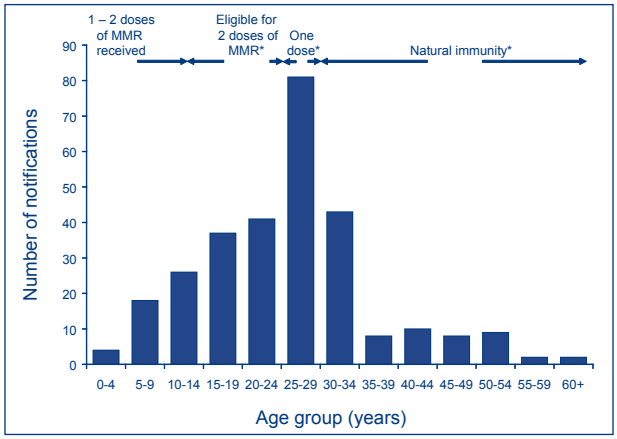
During the fourth quarter of 2007, 290 cases of mumps were reported. Over half of the cases notified to NNDSS (155) were from New South Wales. Western Australia reported 86 cases (30%) and the Northern Territory reported 32 cases (11%). In comparison to the five-year mean for the corresponding period (32.2), the fourth quarter of 2007 was nine times higher and exceeded the 95th percentile of the five-year mean by 233 cases.

In Western Australia, 86 cases of mumps were notified during the quarter. The majority of these cases (76, 88%) were from the Kimberley region, and almost all were Indigenous persons (71, 93%); and aged between 10 and 29 years (54, 71%). Some of the Indigenous cases had epidemiological links to cases in a Northern Territory outbreak. Of all cases notified in the Kimberley region of Western Australia, 38 (50%) were fully vaccinated, 10 (13%) were partially vaccinated, 9 (12%) were not vaccinated and information was unknown, not applicable or missing for 22% of cases.

Ten of the cases reported in the Northern Territory occurred in students at a boarding school. Following public health investigation, it was noted that these cases were likely to have received early immunisation with the measles-mumps-rubella (MMR) vaccine at 9–10 months of age. This was consistent with historical recommendations in the Northern Territory, which no longer apply.

The current National Immunisation Program Schedule recommends two doses of MMR at 12 months and at four years, unless there is a contraindication. The efficacy following immunisation at less than 12 months may be reduced when compared to those who are immunised at 12 months, due to the natural persistence of maternal antibodies in the child. *The Australian Immunisation Handbook* recommends that when MMR is given under 12 months of age, that the dose be repeated at or after 12 months.[6,7] Figure 4 highlights the number of notifications associated with each age group, highlighting mumps vaccine eligibility based on historical vaccination policies.[7]

**Figure 4. Notifications of mumps and mumps vaccine eligibility, Australia, 1 October to 31 December 2007, by age group**




* Mumps monovalent vaccine introduced in 1980 for children over 12 months. Mumps monovalent vaccine replaced by measles-mumps-rubella (MMR) in 1988. MMR second dose recommendation for the 10–16 year age group from 1993 and at four years from 1998.[7]

## Other bacterial infections

### Meningococcal infections

There were 79 notifications of meningococcal infection reported in the fourth quarter of 2007, 22% more than the corresponding period in 2006. Serogroup data were available on 72 (92%) of the notified cases in the quarter. Sixty-two (79%) were serogroup B, 3 (4%) were serogroup C, 2 (3%) were serogroup W135, 3 (4%) were serogroup Y, and in 8 (10%) the serogroup was either not typed or no data were provided.

Those notified were aged from one month to 79 years; nine cases (11%) were aged less then 12 months, 18 cases (23%) were aged 1–years, and there were 19 (24%) cases aged 15–20 years.

There were three deaths associated with meningococcal infection reported from New South Wales (2) and Queensland (1). One case was in a 15-year-old female with serogroup Y infection, another case in a 6-month-old male with serogroup B infection and one case in an 8-month-old male with serogroup C infection. The case with serogroup C infection was too young to be vaccinated under the current immunisation schedule.

The current National Immunisation Program Schedule recommends one dose of the meningococcal C vaccine at 12 months of age.[6]

## Acknowledgements

Thanks go to staff of the Surveillance Policy and Systems Section of the Australian Government Department of Health and Ageing and all our state and territory data managers.

Special thanks to Carolien Giele, Mark Bartlett, Frances Birrell, Shelley Deeks and Peter McIntyre for their contributions.

## References

1. NSW Health. *Increase in cryptosporidiosis cases*. NSW Health Media Release; 11 December 2007. Available from: http://www.health.nsw.gov.au/news/2007/20071211_01.html. Accessed on 11 January 2008.
2. Warrell DA, Cox TM, Firth JD, Benz EJ, editors. *Oxford Textbook of Medicine*. 4th edn. Oxford: Oxford University Press; 2003.
3. Communicable Diseases Network Australia. *Interim Surveillance Case Definitions for the Australian National Notifiable Diseases Surveillance System*. 2004.
4. Heymann DL, ed. *Control of Communicable Diseases Manual*. 18th edn. Washington: American Public Health Association; 2004.
5. Bartlett M, McAnulty J, Rutherford A, Gupta L, Karalis T, Maywood P, et al. Haemolytic uraemic syndrome: A cluster of cases in early 1999. *NSW Public Health Bulletin* 1999;10:9.
6. National Health and Medical Research Council. *The Australian Immunisation Handbook*. 8th edn. Canberra: Commonwealth of Australia; 2003. p.183. Available from: http://www9.health.gov.au/immhandbook/pdf/handbook.pdf Accessed on 11 January 2008
7. Aratchige P, McIntyre P, Quinn H. Recent increases in mumps in Australia—the "forgotten" age group in the 1998 Measles Control Campaign. Unpublished work. National Centre for Immunisation Research and Surveillance of Vaccine-Preventable Diseases; 2007.

# Tables

## National Notifiable Diseases Surveillance System

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 35,690 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 October and 31 December 2007 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

### Table 1. Reporting of notifiable diseases by jurisdiction

| Disease | Data received from: |
|---|---|
| **Bloodborne diseases** | |
| Hepatitis (NEC) | |
| Hepatitis B (incident) | All jurisdictions |
| Hepatitis B (unspecified) | All jurisdictions |
| Hepatitis C (incident) | All jurisdictions except Qld |
| Hepatitis C (unspecified) | All jurisdictions |
| Hepatitis D | All jurisdictions |
| **Gastrointestinal diseases** | |
| Botulism | All jurisdictions |
| Campylobacteriosis | All jurisdictions except NSW |
| Cryptosporidiosis | All jurisdictions |
| Haemolytic uraemic syndrome | All jurisdictions |
| Hepatitis A | All jurisdictions |
| Hepatitis E | All jurisdictions |
| Listeriosis | All jurisdictions |
| Salmonellosis | All jurisdictions |
| Shigellosis | All jurisdictions |
| STEC, VTEC | All jurisdictions |
| Typhoid | All jurisdictions |
| **Quarantinable diseases** | |
| Cholera | All jurisdictions |
| Highly pathogenic avian influenza | All jurisdictions |
| Plague | All jurisdictions |
| Rabies | All jurisdictions |
| Severe acute respiratory syndrome | All jurisdictions |
| Smallpox | All jurisdictions |
| Viral haemorrhagic fever | All jurisdictions |
| Yellow fever | All jurisdictions |
| **Sexually transmissible infections** | |
| Chlamydial infection | All jurisdictions |
| Donovanosis | All jurisdictions |
| Gonococcal infection | All jurisdictions |
| Syphilis (all) | All jurisdictions |
| Syphilis <2 years duration | All jurisdictions |
| Syphilis >2 years or unspecified duration | All jurisdictions |
| Syphilis - congenital | All jurisdictions |

| Disease | Data received from: |
|---|---|
| **Vaccine preventable diseases** | |
| Diphtheria | All jurisdictions |
| *Haemophilus influenzae* type b | All jurisdictions |
| Influenza - laboratory confirmed* | All jurisdictions |
| Measles | All jurisdictions |
| Mumps | All jurisdictions |
| Pertussis | All jurisdictions |
| Pneumococcal disease (invasive) | All jurisdictions |
| Poliomyelitis | All jurisdictions |
| Rubella | All jurisdictions |
| Rubella - congenital | All jurisdictions |
| Tetanus | All jurisdictions |
| Varicella zoster (chickenpox) | All jurisdictions except NSW |
| Varicella zoster (shingles) | All jurisdictions except NSW |
| Varicella zoster (unspecified) | All jurisdictions except NSW |
| **Vectorborne diseases** | |
| Arbovirus infection (NEC) | All jurisdictions |
| Barmah Forest virus infection | All jurisdictions |
| Dengue virus infection | All jurisdictions |
| Japanese encephalitis virus infection | All jurisdictions |
| Kunjin virus infection | All jurisdictions |
| Malaria | All jurisdictions |
| Murray Valley encephalitis virus infection | All jurisdictions |
| Ross River virus infection | All jurisdictions |
| **Zoonoses** | |
| Anthrax | All jurisdictions |
| Australian bat lyssavirus | All jurisdictions |
| Brucellosis | All jurisdictions |
| Leptospirosis | All jurisdictions |
| Lyssaviruses (unspecified) | All jurisdictions |
| Ornithosis | All jurisdictions |
| Q fever | All jurisdictions |
| Tularaemia | All jurisdictions |
| **Other bacterial infections** | |
| Legionellosis | All jurisdictions |
| Leprosy | All jurisdictions |
| Meningococcal infection - invasive | All jurisdictions |
| Tuberculosis | All jurisdictions |

* Laboratory confirmed influenza is not notifiable in South Australia but reports are forwarded to NNDSS.

NEC Not elsewhere classified

## Table 2. Notifications of diseases received by state and territory health authorities in the period 1 October to 31 December 2007, by date of onset*

| Disease | State or territory | | | | | | | | Total 4th quarter 2007† | Total 3rd quarter 2007 | Total 4th quarter 2006 | Last 5 years mean 4th quarter | Year to date 2007 | Last 5 years YTD mean | Ratio‡ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | | | | | | | |
| **Bloodborne diseases** | | | | | | | | | | | | | | | |
| Hepatitis (NEC) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.0 | 0.0 |
| Hepatitis B (incident) | 2 | 15 | 1 | 9 | 4 | 4 | 25 | 11 | 71 | 62 | 79 | 68.2 | 290 | 313.8 | 1.0 |
| Hepatitis B (unspecified) | 13 | 691 | 57 | 227 | 110 | 19 | 465 | 147 | 1,729 | 2,221 | 1,629 | 1,515.8 | 7,550 | 6,173.0 | 1.1 |
| Hepatitis C (incident) | 4 | 4 | 0 | NN | 6 | 4 | 33 | 19 | 70 | 109 | 95 | 105.4 | 353 | 447.0 | 0.7 |
| Hepatitis C (unspecified) | 32 | 1,369 | 45 | 659 | 129 | 73 | 680 | 350 | 3,337 | 3,596 | 3,039 | 3,207.2 | 13,132 | 13,217.0 | 1.0 |
| Hepatitis D | 0 | 1 | 0 | 3 | 0 | 0 | 3 | 0 | 7 | 10 | 5 | 4.8 | 34 | 27.8 | 1.5 |
| **Gastrointestinal diseases** | | | | | | | | | | | | | | | |
| Botulism | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0.2 | 1 | 1.3 | 0.0 |
| Campylobacteriosis§ | 133 | NN | 45 | 1,447 | 554 | 151 | 1,796 | 569 | 4,695 | 3,840 | 4,453 | 4,471.2 | 17,671 | 15,526.4 | 1.1 |
| Cryptosporidiosis | 7 | 300 | 18 | 207 | 5 | 11 | 207 | 50 | 805 | 275 | 444 | 443.2 | 2,877 | 2,521.2 | 1.8 |
| Haemolytic uraemic syndrome | 0 | 6 | 0 | 0 | 0 | 0 | 1 | 0 | 7 | 2 | 8 | 6.0 | 20 | 15.4 | 1.2 |
| Hepatitis A | 0 | 14 | 0 | 6 | 1 | 1 | 8 | 4 | 34 | 43 | 54 | 76.0 | 164 | 349.6 | 0.4 |
| Hepatitis E | 0 | 2 | 0 | 1 | 0 | 0 | 0 | 0 | 3 | 2 | 6 | 3.4 | 18 | 21.2 | 0.9 |
| Listeriosis | 0 | 6 | 0 | 2 | 4 | 0 | 3 | 1 | 16 | 10 | 14 | 15.8 | 50 | 62.6 | 1.0 |
| Salmonellosis | 22 | 531 | 131 | 511 | 194 | 39 | 517 | 211 | 2,156 | 1,554 | 2,108 | 1,999.6 | 9,685 | 7,885.4 | 1.1 |
| Shigellosis | 0 | 14 | 50 | 26 | 31 | 1 | 14 | 22 | 158 | 172 | 117 | 124.4 | 615 | 548.8 | 1.3 |
| STEC, VTEC‖ | 0 | 16 | 1 | 12 | 10 | 0 | 2 | 0 | 41 | 13 | 20 | 16.6 | 112 | 63.4 | 2.5 |
| Typhoid | 0 | 3 | 1 | 3 | 0 | 0 | 6 | 2 | 15 | 20 | 21 | 14.8 | 92 | 65.0 | 1.0 |
| **Quarantinable diseases** | | | | | | | | | | | | | | | |
| Cholera | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 3 | 0.8 | 3 | 3.4 | 1.3 |
| Highly pathogenic avian influenza | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.0 | 0.0 |
| Plague | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.0 | 0.0 |
| Rabies | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.0 | 0.0 |
| Severe acute respiratory syndrome | 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 |
| 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 |

## Table 2. Notifications of diseases received by state and territory health authorities in the period 1 October to 31 December 2007, by date of onset,* *continued*

| Disease | State or territory | | | | | | | | Total 4th quarter 2007† | Total 3rd quarter 2007 | Total 4th quarter 2006 | Last 5 years mean 4th quarter | Year to date 2007 | Last 5 years YTD mean | Ratio‡ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | | | | | | | |
| **Sexually transmissible infections** | | | | | | | | | | | | | | | |
| Chlamydial infection¶ | 196 | 3,063 | 483 | 3,293 | 787 | 304 | 2,777 | 1,906 | 12,809 | 12,376 | 11,615 | 8,922.0 | 51,458 | 35,946.4 | 1.4 |
| Donovanosis | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 3.0 | 3 | 12.4 | 0.0 |
| Gonococcal infection | 17 | 333 | 329 | 345 | 95 | 8 | 228 | 430 | 1,785 | 1,708 | 1,870 | 1,772.2 | 7,619 | 7,418.2 | 1.0 |
| Syphilis (all) | 9 | 344 | 58 | 97 | 10 | 7 | 191 | 46 | 762 | 843 | 730 | 570.2 | 3,127 | 2,257.6 | 1.3 |
| Syphilis <2 years duration | 6 | 88 | 17 | 53 | 0 | 0 | 77 | 23 | 264 | 339 | 267 | 196.0 | 1,274 | 696.3 | 1.3 |
| Syphilis >2 years or unspecified duration | 3 | 256 | 41 | 44 | 10 | 7 | 114 | 23 | 498 | 504 | 463 | 452.6 | 1,853 | 1,839.8 | 1.1 |
| Syphilis - congenital | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 1 | 1 | 2.4 | 9 | 14.4 | 0.8 |
| **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 | 3 | 2 | 0 | 0 | 0 | 1 | 0 | 6 | 5 | 7 | 4.0 | 17 | 20.8 | 1.5 |
| Influenza - laboratory confirmed | 17 | 128 | 8 | 366 | NDP | 12 | 130 | 94 | 755 | 9,070 | 421 | 407.4 | 10,745 | 3,421.6 | 1.9 |
| Measles | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 4 | 10 | 9.8 | 11 | 61.0 | 0.0 |
| Mumps | 3 | 155 | 32 | 6 | 3 | 0 | 5 | 86 | 290 | 151 | 44 | 32.0 | 584 | 152.6 | 9.0 |
| Pertussis | 19 | 708 | 5 | 387 | 88 | 3 | 227 | 33 | 1,470 | 1,610 | 1,305 | 2,145.4 | 5,472 | 8,326.8 | 0.7 |
| Pneumococcal disease - invasive | 7 | 99 | 13 | 50 | 22 | 9 | 69 | 20 | 289 | 612 | 277 | 435.0 | 1,494 | 2,051.8 | 0.7 |
| Poliomyelitis | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0.0 | 1 | 0.0 | 0.0 |
| Rubella | 0 | 1 | 0 | 2 | 0 | 0 | 1 | 1 | 5 | 6 | 12 | 19.2 | 35 | 85.8 | 0.3 |
| Rubella - congenital | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.2 | 1 | 1.4 | 0.0 |
| Tetanus | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 2 | 2 | 1.0 | 3 | 3.6 | 1.0 |
| Varicella zoster (chickenpox) | NDP | NN | 67 | 110 | 189 | 3 | NN | 117 | 486 | 595 | 719 | NA | 1,837 | NA | NA |
| Varicella zoster (shingles) | NDP | NN | 21 | 99 | 99 | 32 | NN | 106 | 357 | 385 | 415 | NA | 1,671 | NA | NA |
| Varicella zoster (unspecified) | NDP | NN | 0 | 839 | 163 | 12 | NN | 164 | 1,178 | 1,134 | 918 | NA | 4,309 | NA | NA |
| **Vectorborne diseases** | | | | | | | | | | | | | | | |
| Arbovirus infection (NEC) | 0 | 0 | 0 | 3 | 0 | 0 | 0 | 0 | 3 | 3 | 4 | 8.0 | 23 | 50.6 | 0.4 |
| Barmah Forest virus infection | 1 | 107 | 12 | 213 | 11 | 0 | 7 | 24 | 375 | 315 | 367 | 249.8 | 1,699 | 1,365.8 | 1.5 |
| Dengue virus infection | 2 | 16 | 4 | 18 | 6 | 2 | 7 | 19 | 74 | 71 | 37 | 70.6 | 324 | 358.4 | 1.0 |
| Japanese encephalitis virus infection | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.4 | 0.0 |
| Kunjin virus infection | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.6 | 0 | 6.8 | 0.0 |
| Malaria | 3 | 23 | 12 | 52 | 6 | 4 | 20 | 23 | 143 | 118 | 149 | 137.0 | 580 | 641.2 | 1.0 |
| Murray Valley encephalitis virus infection | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 1.2 | 0.0 |
| Ross River virus infection | 3 | 238 | 62 | 505 | 59 | 2 | 29 | 220 | 1,118 | 647 | 544 | 475.8 | 4,177 | 3,512.4 | 2.3 |

## Table 2. Notifications of diseases received by state and territory health authorities in the period 1 October to 31 December 2007, by date of onset,* *continued*

| Disease | State or territory | | | | | | | | Total 4th quarter 2007† | Total 3rd quarter 2007 | Total 4th quarter 2006 | Last 5 years mean 4th quarter | Year to date 2007 | 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 | 1 | 0.2 | 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 | 7 | 0 | 0 | 0 | 0 | 7 | 11 | 13 | 12.6 | 40 | 37.8 | 0.6 |
| Leptospirosis | 0 | 0 | 0 | 7 | 0 | 0 | 7 | 2 | 16 | 10 | 20 | 24.0 | 106 | 148.0 | 0.7 |
| Lyssavirus (unspecified) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.0 | 0.0 |
| Ornithosis | 0 | 9 | 0 | 1 | 0 | 0 | 18 | 2 | 30 | 14 | 42 | 43.2 | 100 | 197.4 | 0.7 |
| Q fever | 0 | 58 | 0 | 35 | 3 | 0 | 6 | 2 | 104 | 115 | 100 | 124.8 | 458 | 514.6 | 0.8 |
| Tularaemia | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.0 | 0.0 |
| **Other bacterial infections** | | | | | | | | | | | | | | | |
| Legionellosis | 0 | 28 | 1 | 13 | 5 | 1 | 11 | 36 | 95 | 50 | 96 | 88.0 | 311 | 328.6 | 1.1 |
| Leprosy | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 2 | 0 | 1 | 0.6 | 12 | 6.8 | 3.3 |
| Meningococcal infection - invasive** | 1 | 31 | 0 | 23 | 3 | 1 | 13 | 7 | 79 | 126 | 65 | 111.2 | 311 | 472.2 | 0.7 |
| Tuberculosis | 5 | 78 | 19 | 52 | 21 | 3 | 103 | 23 | 304 | 282 | 331 | 318.4 | 1,096 | 1,120.8 | 1.0 |
| Total | 496 | 8,398 | 1,477 | 9,637 | 2,618 | 706 | 7,611 | 4,747 | 35,690 | 42,195 | 32,212 | 28,061.8 | 150,301 | 115,780.1 | 1.3 |

* Date of onset = the true onset. If this is not available, the 'date of onset' is equivalent to the earliest of two dates: (i) specimen date of collection, or (ii) the date of notification to the public health unit. Hepatitis B and C unspecified and tuberculosis were analysed by the date of notification.

† 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.

‡ Ratio = ratio of current quarter total to the mean of last five years for the same quarter. Note: Ratios for syphilis <2 years; syphilis >2 years or unspecified duration based on four years data

§ Not reported for New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.

|| Infections with Shiga toxin (verotoxin) producing *Escherichia coli* (STEC/VTEC).

¶ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia which reports only genital tract specimens, Northern Territory which excludes ocular specimens, and Western Australia which excludes ocular and perinatal infections.

** Only invasive meningococcal disease is nationally notifiable. However, New South Wales, the Australian Capital Territory and South Australia also report conjunctival cases.

NN Not notifiable.

NEC Not elsewhere classified.

NDP No data provided.

## Table 3. Notification rates of diseases, 1 October to 31 December 2007, by state or territory. (Annualised rate per 100,000 population)

| Disease* | State or territory | | | | | | | | Aust |
|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | |
| **Bloodborne diseases** | | | | | | | | | |
| Hepatitis (NEC) | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Hepatitis B (incident) | 2.4 | 0.9 | 1.9 | 0.9 | 1.0 | 3.2 | 1.9 | 2.1 | 1.4 |
| Hepatitis B (unspecified) | 15.3 | 40.1 | 106.1 | 21.9 | 27.8 | 15.4 | 35.7 | 27.9 | 32.9 |
| Hepatitis C (incident) | 4.7 | 0.2 | 0.0 | NN | 1.3 | 3.2 | 2.5 | 3.6 | 1.3 |
| Hepatitis C (unspecified) | 37.7 | 79.5 | 83.7 | 63.0 | 34.8 | 59.2 | 52.3 | 66.5 | 63.5 |
| Hepatitis D | 0.0 | 0.1 | 0.0 | 0.3 | 0.0 | 0.0 | 0.2 | 0.0 | 0.1 |
| **Gastrointestinal diseases** | | | | | | | | | |
| Botulism | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Campylobacteriosis† | 156.5 | NN | 83.7 | 138.4 | 139.9 | 122.4 | 138.0 | 108.1 | 89.4 |
| Cryptosporidiosis | 8.2 | 17.4 | 33.5 | 19.8 | 1.3 | 8.9 | 15.9 | 9.5 | 15.3 |
| Haemolytic uraemic syndrome | 0.0 | 0.3 | 0.0 | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 | 0.1 |
| Hepatitis A | 0.0 | 0.8 | 0.0 | 0.6 | 0.3 | 0.8 | 0.6 | 0.8 | 0.6 |
| Hepatitis E | 0.0 | 0.1 | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.1 |
| Listeriosis | 0.0 | 0.3 | 0.0 | 0.2 | 1.0 | 0.0 | 0.2 | 0.2 | 0.3 |
| Salmonellosis | 25.9 | 30.9 | 243.7 | 48.9 | 49.0 | 31.6 | 39.7 | 40.1 | 41.0 |
| Shigellosis | 0.0 | 0.8 | 93.0 | 2.5 | 7.8 | 0.8 | 1.1 | 4.2 | 3.0 |
| STEC, VTEC‡ | 0.0 | 0.9 | 1.9 | 1.1 | 2.5 | 0.0 | 0.2 | 0.0 | 0.8 |
| Typhoid | 0.0 | 0.2 | 1.9 | 0.3 | 0.0 | 0.0 | 0.5 | 0.4 | 0.3 |
| **Quarantinable diseases** | | | | | | | | | |
| Cholera | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Highly pathogenic avian influenza | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Plague | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Rabies | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Severe acute respiratory syndrome | 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 |
| 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 infections** | | | | | | | | | |
| Chlamydial infection§ | 230.7 | 177.8 | 898.7 | 315.2 | 198.7 | 246.5 | 213.2 | 362.1 | 243.7 |
| Donovanosis | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Gonococcal infection | 20.0 | 19.3 | 612.2 | 33.0 | 24.2 | 6.5 | 17.5 | 82.2 | 34.0 |
| Syphilis (all) | 10.5 | 20.0 | 107.9 | 9.3 | 2.5 | 5.7 | 14.7 | 8.7 | 14.5 |
| Syphilis <2 years duration | 7.1 | 5.1 | 31.6 | 5.1 | 0.0 | 0.0 | 5.9 | 4.4 | 5.0 |
| Syphilis >2 years or unspecified duration | 3.5 | 14.9 | 76.3 | 4.2 | 2.5 | 5.7 | 8.8 | 4.4 | 9.5 |
| Syphilis - congenital | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| **Vaccine preventable diseases** | | | | | | | | | |
| Diphtheria | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| *Haemophilus influenzae* type b | 0.0 | 0.2 | 3.7 | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 | 0.1 |
| Influenza - laboratory confirmed | 20.0 | 7.4 | 14.9 | 35.0 | NDP | 9.7 | 10.0 | 17.9 | 14.4 |
| Measles | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Mumps | 3.5 | 9.0 | 59.5 | 0.6 | 0.8 | 0.0 | 0.4 | 16.1 | 5.5 |
| Pertussis | 22.4 | 41.2 | 9.3 | 37.0 | 22.2 | 2.4 | 17.4 | 6.6 | 28.0 |

## Table 3. Notification rates of diseases, 1 October to 31 December 2007, by state or territory. (Annualised rate per 100,000 population), *continued*

| Disease* | State or territory | | | | | | | | Aust |
|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | |
| **Vaccine preventable diseases, continued** | | | | | | | | | |
| Pneumococcal disease (invasive) | 8.2 | 5.7 | 24.2 | 4.8 | 5.6 | 7.3 | 5.3 | 3.8 | 5.5 |
| Poliomyelitis | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Rubella | 0.0 | 0.1 | 0.0 | 0.2 | 0.0 | 0.0 | 0.1 | 0.2 | 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 | 0.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Varicella zoster (chickenpox) | NDP | NN | 124.7 | 10.5 | 47.7 | 2.4 | NN | 22.2 | 9.3 |
| Varicella zoster (shingles) | NDP | NN | 39.1 | 9.5 | 25.0 | 25.9 | NN | 20.1 | 6.8 |
| Varicella zoster (unspecified) | NDP | NN | 0.0 | 80.2 | 41.1 | 9.7 | NN | 31.2 | 22.4 |
| **Vectorborne diseases** | | | | | | | | | |
| Arbovirus infection (NEC) | 0.0 | 0.0 | 0.0 | 0.3 | 0.0 | 0.0 | 0.0 | 0.0 | 0.1 |
| Barmah Forest virus infection | 1.2 | 6.2 | 22.3 | 20.5 | 2.8 | 0.0 | 0.5 | 4.6 | 7.1 |
| Dengue virus infection | 2.4 | 0.9 | 7.4 | 1.7 | 1.5 | 1.6 | 0.5 | 3.6 | 1.4 |
| Japanese encephalitis virus | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Kunjin virus infection | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Malaria | 3.5 | 1.3 | 22.3 | 5.0 | 1.5 | 3.2 | 1.5 | 4.4 | 2.7 |
| Murray Valley encephalitis virus infection | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Ross River virus infection | 3.5 | 13.8 | 115.4 | 48.3 | 14.9 | 1.6 | 2.2 | 41.8 | 21.3 |
| **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 | 0.7 | 0.0 | 0.0 | 0.0 | 0.0 | 0.1 |
| Leptospirosis | 0.0 | 0.0 | 0.0 | 0.7 | 0.0 | 0.0 | 0.5 | 0.4 | 0.3 |
| Lyssavirus (unspecified) | 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 | 0.1 | 0.0 | 0.0 | 1.4 | 0.4 | 0.6 |
| Q fever | 0.0 | 3.5 | 0.0 | 3.3 | 0.8 | 0.0 | 0.5 | 0.4 | 2.0 |
| Tularaemia | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| **Other bacterial infections** | | | | | | | | | |
| Legionellosis | 0.0 | 1.6 | 1.9 | 1.2 | 1.3 | 0.8 | 0.8 | 6.8 | 1.8 |
| Leprosy | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 | 0.0 |
| Meningococcal infection - invasive‖ | 1.2 | 1.8 | 0.0 | 2.2 | 0.8 | 0.8 | 1.0 | 1.3 | 1.5 |
| Tuberculosis | 5.9 | 4.5 | 37.2 | 5.0 | 5.3 | 2.4 | 7.9 | 4.4 | 5.8 |

* Rates are subject to retrospective revision.

† Not reported for New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.

‡ Infections with Shiga toxin (verotoxin) producing *Escherichia coli* (STEC/VTEC).

§ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia which reports only genital tract specimens, Northern Territory which excludes ocular specimens, and Western Australia which excludes ocular and perinatal infections.

‖ Only invasive meningococcal disease is nationally notifiable. However, New South Wales, the Australian Capital Territory and South Australia also report conjunctival cases.

NN Not notifiable.

NEC Not elsewhere classified.

NDP No data provided.

## Laboratory Serology and Virology Reporting Scheme

There were 6,058 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 October to 31 December 2007 (Tables 4 and 5).

### Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 October to 31 December 2007, and total reports for the year†

| | State or territory | | | | | | | | This period 2007 | This period 2006 | Year to date 2007 | Year to date 2006 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | | | | |
| Measles, mumps, rubella | | | | | | | | | | | | |
| Measles virus | – | – | – | – | 1 | – | – | – | 1 | 3 | 19 | 57 |
| Mumps virus | – | 3 | 1 | 2 | 3 | – | 2 | – | 12 | 2 | 53 | 27 |
| Hepatitis viruses | | | | | | | | | | | | |
| Hepatitis A virus | – | 2 | – | 6 | 1 | – | – | – | 10 | 7 | 41 | 31 |
| Hepatitis D virus | – | – | – | – | 2 | – | – | – | 2 | 2 | 22 | 7 |
| Arboviruses | | | | | | | | | | | | |
| Barmah Forest virus | – | 8 | – | 79 | 10 | – | – | – | 97 | 23 | 506 | 288 |
| Flavivirus (unspecified) | – | – | – | 15 | – | – | – | 1 | 16 | 4 | 97 | 47 |
| Ross River virus | 1 | 5 | 7 | 156 | 27 | – | 1 | 6 | 203 | 41 | 1,090 | 1,062 |
| Adenoviruses | | | | | | | | | | | | |
| Adenovirus not typed/ pending | – | 77 | – | 44 | 156 | 1 | 25 | – | 309 | 144 | 1,199 | 636 |
| Herpes viruses | | | | | | | | | | | | |
| Cytomegalovirus | – | 22 | 1 | 111 | 100 | 3 | 14 | – | 251 | 137 | 1,196 | 872 |
| Epstein–Barr virus | – | 9 | 18 | 268 | 109 | 3 | 11 | 75 | 493 | 261 | 2,458 | 1,445 |
| Varicella–zoster virus | 1 | 108 | 4 | 413 | 121 | 4 | 33 | – | 684 | 142 | 2,809 | 1,039 |
| Other DNA viruses | | | | | | | | | | | | |
| Parvovirus | – | 2 | – | 91 | 6 | – | 22 | – | 121 | 37 | 410 | 186 |
| Poxvirus group not typed | – | – | – | – | – | – | 1 | – | 1 | 1 | 3 | 3 |
| Picornavirus family | | | | | | | | | | | | |
| Enterovirus not typed/ pending | – | 21 | – | 11 | 17 | 1 | 1 | – | 51 | 9 | 173 | 103 |
| Picornavirus not typed | – | – | – | – | – | 2 | – | – | 2 | – | 9 | 2 |
| Rhinovirus (all types) | – | 68 | – | – | 10 | – | – | – | 78 | 65 | 323 | 207 |
| Ortho/paramyxoviruses | | | | | | | | | | | | |
| Influenza A virus | – | 2 | 1 | 25 | 22 | – | 16 | – | 66 | 39 | 2,293 | 340 |
| Influenza B virus | – | 3 | – | 3 | 12 | – | 16 | – | 34 | 6 | 165 | 176 |
| Parainfluenza virus type 1 | – | 6 | – | 1 | 3 | – | 1 | – | 11 | – | 50 | 74 |
| Parainfluenza virus type 2 | – | – | – | 2 | – | – | – | – | 2 | 3 | 61 | 15 |
| Parainfluenza virus type 3 | – | 28 | – | 46 | 45 | – | 9 | – | 134 | 118 | 507 | 233 |
| Respiratory syncytial virus | 2 | 35 | 1 | 82 | 26 | 11 | 10 | – | 176 | 62 | 2,239 | 1,816 |
| Other RNA viruses | | | | | | | | | | | | |
| HTLV–1 | – | – | – | – | 2 | – | – | – | 2 | 2 | 14 | 6 |
| Norwalk agent | – | 23 | – | 1 | – | 1 | 346 | – | 371 | 440 | 1,138 | 1,550 |
| Rotavirus | – | 57 | – | – | 150 | 3 | 25 | – | 253 | 428 | 617 | 1,318 |

## Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 October to 31 December 2007, and total reports for the year,† *continued*

| | State or territory | | | | | | | | This period 2007 | This period 2006 | Year to date 2007 | Year to date 2006 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | | | | |
| Other | | | | | | | | | | | | |
| *Chlamydia pneumoniae* | 1 | – | – | – | – | – | 2 | – | 3 | – | 4 | 1 |
| *Chlamydia psittaci* | – | – | – | 1 | – | 1 | 25 | – | 27 | 21 | 66 | 64 |
| *Chlamydia trachomatis* – not typed | 3 | 133 | – | 1,025 | 328 | 14 | 6 | 1 | 1,512 | 471 | 7,837 | 3,875 |
| *Chlamydia* species | – | 1 | – | – | – | – | – | – | 1 | – | 3 | 2 |
| *Coxiella burnetii* (Q fever) | 1 | 33 | – | 28 | 9 | – | 19 | 3 | 93 | 11 | 276 | 106 |
| *Mycoplasma pneumoniae* | – | 5 | 2 | 70 | 25 | 6 | 47 | 11 | 166 | 134 | 1,155 | 1,035 |
| *Rickettsia* – spotted fever group | – | 3 | – | 1 | 2 | 1 | 7 | – | 14 | 2 | 112 | 87 |
| *Streptococcus* group A | – | 7 | 35 | 197 | – | 1 | 21 | – | 261 | 48 | 1,087 | 377 |
| *Bordetella pertussis* | – | 7 | – | 97 | 32 | 2 | 13 | – | 152 | 90 | 818 | 1,313 |
| *Brucella* species | – | – | – | 3 | – | – | – | – | 3 | – | 10 | 5 |
| *Legionella longbeachae* | – | – | – | – | – | – | 2 | – | 2 | 6 | 8 | 21 |
| *Legionella pneumophila* | – | 1 | – | – | – | – | 1 | – | 2 | 3 | 30 | 28 |
| *Cryptococcus* species | – | 1 | – | 1 | 1 | – | – | – | 3 | 2 | 45 | 19 |
| *Leptospira* species | – | – | – | 8 | 1 | – | – | – | 9 | 2 | 61 | 18 |
| *Treponema pallidum* | – | 25 | 9 | 211 | 143 | – | 1 | 1 | 412 | 99 | 2,196 | 785 |
| *Entamoeba histolytica* | – | – | – | 1 | – | – | 1 | – | 2 | – | 8 | 1 |
| *Toxoplasma gondii* | – | 1 | – | 2 | 1 | 1 | 3 | – | 8 | 3 | 29 | 39 |
| *Echinococcus granulosus* | – | – | – | – | 4 | 1 | 3 | – | 8 | – | 24 | 3 |
| Total | 9 | 696 | 79 | 3,001 | 1,369 | 56 | 684 | 98 | 6,058 | 2,868 | 31,261 | 19,319 |

* State or territory of postcode, if reported, otherwise state or territory of reporting laboratory.

† Data presented are for reports with reports dates in the current period.

– No data received this period.

## Table 5. Virology and serology reports by laboratories for the reporting period 1 October to 31 December 2007*

| State or territory | Laboratory | October 2007 | November 2007 | December 2007 | Total this period |
|---|---|---|---|---|---|
| Australian Capital Territory | The Canberra Hospital | – | – | – | – |
| New South Wales | Institute of Clinical Pathology and Medical Research, Westmead | 49 | 3 | 90 | 142 |
| | New Children's Hospital, Westmead | 118 | 73 | 50 | 241 |
| | Repatriation General Hospital, Concord | – | – | – | – |
| | Royal Prince Alfred Hospital, Camperdown | 15 | 22 | 11 | 48 |
| | South West Area Pathology Service, Liverpool | 57 | 9 | 31 | 97 |
| Queensland | Queensland Medical Laboratory, West End | 1,594 | 1,341 | 351 | 3,286 |
| | Townsville General Hospital | – | – | – | – |
| South Australia | Institute of Medical and Veterinary Science, Adelaide | 746 | 73 | 574 | 1,363 |
| Tasmania | Northern Tasmanian Pathology Service, Launceston | 25 | 15 | 14 | 54 |
| | Royal Hobart Hospital, Hobart | – | – | – | – |
| Victoria | Australian Rickettsial Reference Laboratory | 38 | 38 | 14 | 90 |
| | Monash Medical Centre, Melbourne | 17 | 28 | 25 | 70 |
| | Royal Children's Hospital, Melbourne | 20 | 13 | 12 | 45 |
| | Victorian Infectious Diseases Reference Laboratory, Fairfield | 398 | 60 | 49 | 507 |
| Western Australia | PathWest Virology, Perth | – | – | – | – |
| | Princess Margaret Hospital, Perth | – | – | – | – |
| | Western Diagnostic Pathology | 85 | – | 30 | 115 |
| Total | | 3,162 | 1,675 | 1,251 | 6,058 |

* The complete list of laboratories reporting for the 12 months, January to December 2007, will appear in every report regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.

– No data received this period.

# Additional reports

## Australian Sentinel Practice Research Network

*The Australian Sentinel Practices Research Network (ASPREN) is a national surveillance system that is owned and operated by the Royal Australian College of General Practitioners and directed through the Discipline of General Practice at the University of Adelaide.*

*The network consists of general practitioners who report presentations on a number of defined medical conditions each week. ASPREN was established in 1991 to provide a rapid monitoring scheme for infectious diseases that can alert public health officials of epidemics in their early stages as well as play a role in the evaluation of public health campaigns and research of conditions commonly seen in general practice. The aim of ASPREN is to also provide an indicator of the burden of disease in the primary health care setting and to detect trends in consultation rates.*

*The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published. In 2007, four conditions are being monitored all of which are related to communicable diseases. They include influenza like illness (ILI), gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, published in Commun Dis Intell 2008;32:134–135.*

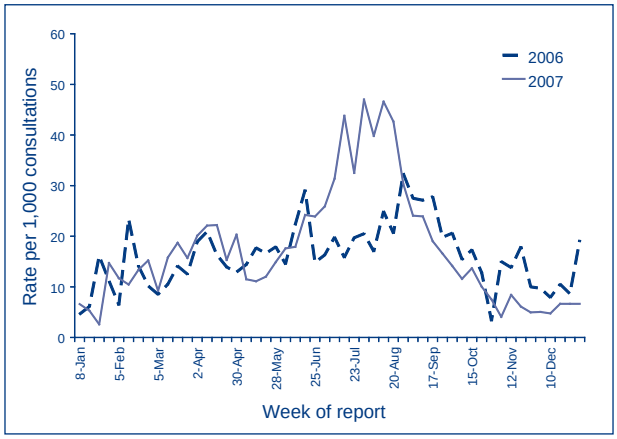
*Data on influenza-like illness, gastronenteritis, chickenpox and shingles from 1 January to 31 December 2007 compared with 2006, are shown as the rate per 1,000 consultations in Figures 1, 2, 3 and 4, respectively.*

**Reporting period 1 October to 31 December 2007**

Sentinel practices contributing to ASPREN were located in all jurisdictions other than the Northern Territory. A total of 92 general practitioners contributed data to ASPREN in the fourth quarter of 2007. Each week an average of 72 general practitioners provided information to ASPREN at an average of 7,231 (range 3,008 to 8,197) consultations per week.

In the fourth quarter of 2007, influenza-like illness (ILI) rates began to decrease from early November. From November to end of December, ILI rates were lower (4 to 8 cases per 1,000 consultations) compared with 8 to 18 cases per 1,000 consultations for the same period in 2006 (Figure 1).

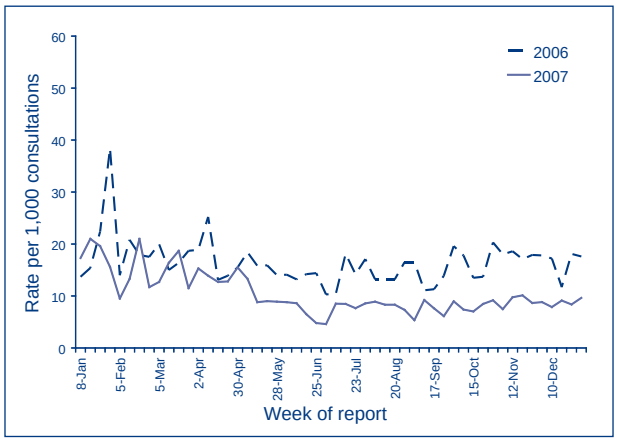
**Figure 1. Consultation rates for influenza-like illness, ASPREN, 2006 to 31 December 2007, by week of report**




Reports of gastroenteritis from 1 October to 31 December 2007 were lower compared with the same period in 2006 (Figure 2). During this reporting period, consultation rates for gastroenteritis remained constant (between 7 to 10 cases per 1,000 consultations).

Reports of varicella infections were reported at a lower rate for the fourth quarter of 2007 compared with the same period in 2006, but there was no recognisable seasonal pattern. From 1 October to 31 December 2007, rates for chickenpox fluctuated between 0 to 1.3 case per 1,000 consultations (Figure 3).

**Figure 2. Consultation rates for gastroenteritis, ASPREN, 2006 to 31 December 2007, by week of report**



In the fourth quarter of 2007, rates for shingles fluctuated between less than 1 to 1.4 cases per 1,000 consultations (Figure 4).

**Figure 3. Consultation rates for chickenpox, ASPREN, 2006 to 31 December 2007, by week of report**

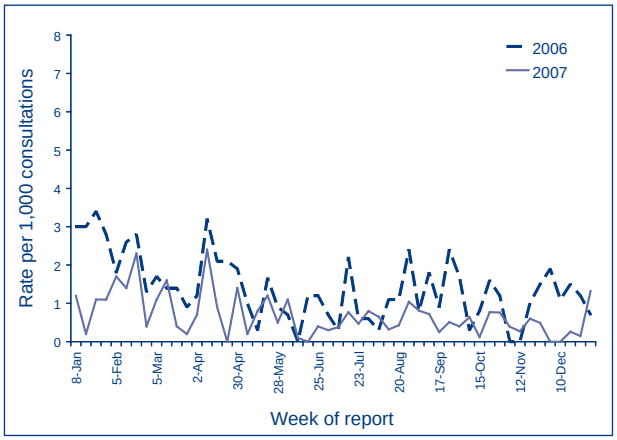
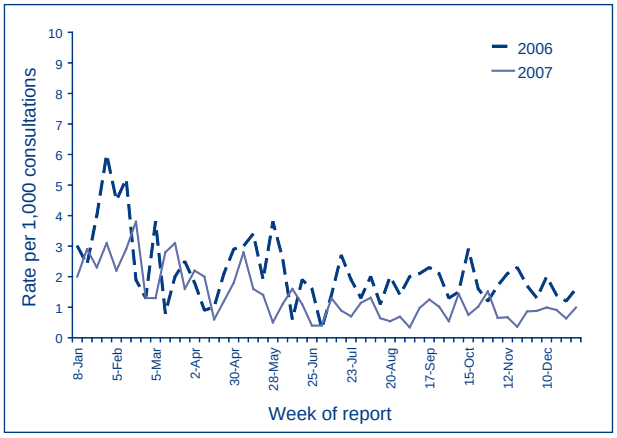



**Figure 4. Consultation rates for shingles, ASPREN, 2006 to 31 December 2007, by week of report**




## 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% 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 *2008;32:134.*

### Reporting period 1 July to 30 September 2007

The AGSP laboratories received a total of 651 gonococcal isolates of which 636 remained viable for susceptibility testing. This was about 25% less than the 869 gonococci reported for the same period in 2006. About 30% of this total was from New South Wales, 20% from Victoria, 17% from Queensland, 13% from each of Western Australia and the Northern Territory and 5% from South Australia. There were nine isolates from Tasmania and three from the Australian Capital Territory.

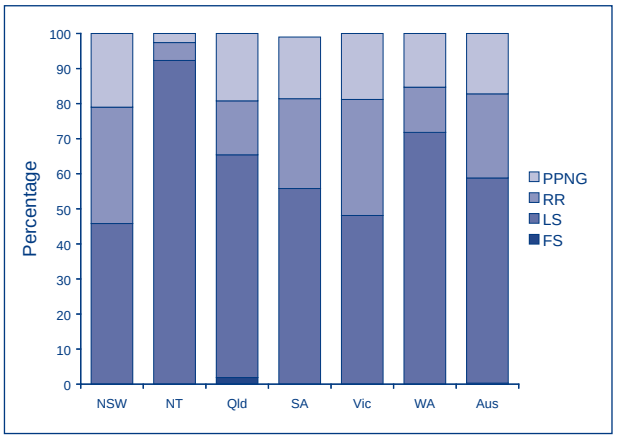
### Penicillins

Two hundred and sixty-two (41.1%) of the 636 isolates examined were penicillin resistant by one or more mechanisms. One hundred and nine (17.1%) were penicillinase producing *Neisseria gonorrhoeae* (PPNG) and 153 (24%) resistant by chromosomal mechanisms, (CMRP). The proportion of all strains resistant to the penicillins by any mechanism ranged from 7.5% in the Northern Territory to 54% in New South Wales and 52% in Victoria. High rates of penicillin resistance were also found in South Australia (44%), Queensland (34.6%) and in Western Australia (28.2%). All nine gonococci tested in Tasmania, but none of the three in the Australian Capital Territory, were penicillin resistant.

Figure 5 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 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 New South Wales most of the penicillin resistance was with CMRP (63, 33.2%) with 40 PPNG (21%) and a similar distribution was also present in Victoria where 25 PPNG represented 18.8% of isolates tested, but 44 CMRP was 33% of isolates tested. In Queensland CMRP represented 15.4% of isolates tested, while PPNG were 19.2%, in South Australia PPNG were 17.6% and CMRP 26.5% and in Western Australia PPNG 15.3% and CMRP 12.9%. PPNG were also present in Tasmania and Northern Territory (3 and 2 isolates respectively), but there were no PPNG in the Australian Capital Territory. CMRP were present in Tasmania (6 isolates) and the Northern Territory (4). All the penicillin resistant strains in the Northern Territory were from Darwin.

**Figure 5. Categorisation of gonococci isolated in Australia, 1 July to 30 September 2007, 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.*

## Ceftriaxone

Four isolates with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected, one each in New South Wales and South Australia and two in Queensland. It is emphasised that no treatment failures have been documented locally when a 250 mg IM dose of ceftriaxone has been used.

## Spectinomycin

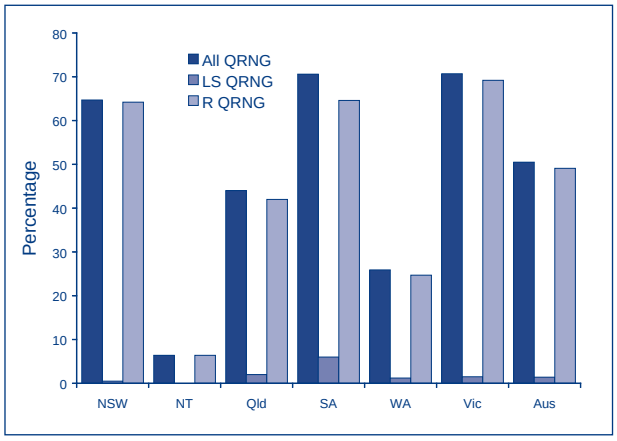
All isolates susceptible to this injectable agent.

## Quinolone antibiotics

Nationally, the 321 quinolone resistant *N. gonorrhoeae* (QRNG) detected in this quarter represented 50.5% of all isolates tested. In the third quarter of 2006, the 325 QRNG represented 38% of all isolates while in 2005 there were 35.5% QRNG and in 2004 QRNG were 24% of all gonococci tested. The majority of QRNG (272 of 321, 98.6%) had higher-level resistance to ciprofloxacin of 1 mg/L or more. 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.

QRNG were detected in all states and territories with the exception of the Australian Capital Territory (Figure 6). The highest proportion of QRNG was found in Victoria where 94 QRNG represented 70.7% of isolates tested and South Australia where there were 24 QRNG (70.6% of isolates). In New South Wales there were 123 QRNG (64.7%), in Queensland 44 (42.3%) and in Western Australia 22 (25.9 %) with five QRNG detected the Northern Territory and nine (of 9 tested) in Tasmania.

**Figure 6. The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 July to 30 September 2007, 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 (129) and proportion (20.3%) of high level tetracycline resistance (TRNG) detected was higher than that recorded in this quarter in 2006 (102, 11.9%). TRNG were found in all states and territories except for Tasmania and the Australian Capital Territory and represented between 3.8% (Northern Territory) and 36.5% (Western Australia) of all isolates tested.

### Reference

1. Management of sexually transmitted diseases. World Health Organization 1997; Document WHO/GPA/TEM94.1 Rev.1 p 37.

## 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 *2005;29:91–92.*

*HIV and AIDS diagnoses and deaths following AIDS reported for 1 April to 30 June 2007, as reported to 30 September 2007, and reported for 1 July to 30 September 2007, as reported to 31 December 2007 are included in this issue of* Communicable Diseases Intelligence *(Tables 1, 2, 3 and 4).*

**Table 1. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 April to 30 June 2007, by sex and state or territory of diagnosis**

| | Sex | State or territory | | | | | | | | Totals for Australia | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | This period 2007 | This period 2006 | YTD 2007 | YTD 2006 |
| HIV diagnoses | Female | 0 | 13 | 0 | 6 | 2 | 0 | 5 | 2 | 28 | 30 | 64 | 69 |
| | Male | 0 | 101 | 2 | 40 | 9 | 1 | 68 | 8 | 229 | 195 | 488 | 416 |
| | Not reported | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | Total* | 0 | 116 | 2 | 46 | 11 | 1 | 73 | 10 | 259 | 225 | 554 | 485 |
| AIDS diagnoses | Female | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 6 | 1 | 10 |
| | Male | 0 | 5 | 0 | 5 | 0 | 0 | 9 | 1 | 20 | 39 | 44 | 85 |
| | Total* | 0 | 5 | 0 | 5 | 0 | 0 | 10 | 1 | 21 | 45 | 46 | 96 |
| AIDS deaths | Female | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 2 | 1 | 2 | 4 |
| | Male | 0 | 0 | 0 | 3 | 0 | 0 | 1 | 0 | 4 | 17 | 15 | 33 |
| | Total* | 0 | 0 | 0 | 3 | 0 | 0 | 2 | 1 | 6 | 19 | 17 | 39 |

* Totals include people whose sex was reported as transgender.

## Table 2. Cumulative diagnoses of HIV infection, AIDS, and deaths following AIDS since the introduction of HIV antibody testing to 30 June 2007, and reported by 30 September 2007, by sex and state or territory

| | Sex | State or territory | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Australia |
| HIV diagnoses | Female | 32 | 898 | 23 | 285 | 104 | 12 | 379 | 212 | 1,945 |
| | Male | 260 | 13,661 | 135 | 2,814 | 964 | 110 | 5,380 | 1,239 | 24,563 |
| | Not reported | 0 | 230 | 0 | 0 | 0 | 0 | 22 | 0 | 252 |
| | Total* | 292 | 14,818 | 158 | 3,108 | 1,069 | 122 | 5,803 | 1,458 | 26,828 |
| AIDS diagnoses | Female | 10 | 251 | 4 | 72 | 32 | 4 | 111 | 41 | 525 |
| | Male | 92 | 5,432 | 45 | 1,043 | 409 | 53 | 2,015 | 428 | 9,517 |
| | Total* | 102 | 5,701 | 49 | 1,117 | 442 | 57 | 2,139 | 471 | 10,078 |
| AIDS deaths | Female | 7 | 136 | 1 | 42 | 20 | 2 | 62 | 27 | 297 |
| | Male | 73 | 3,586 | 28 | 672 | 280 | 33 | 1,416 | 295 | 6,383 |
| | Total* | 80 | 3,733 | 29 | 716 | 300 | 35 | 1,487 | 323 | 6,703 |

* Totals include people whose sex was reported as transgender.

## Table 3. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 July to 30 September 2007, by sex and state or territory of diagnosis

| | Sex | State or territory | | | | | | | | Totals for Australia | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | This period 2007 | This period 2006 | YTD 2007 | YTD 2006 |
| HIV diagnoses | Female | 0 | 17 | 0 | 5 | 4 | 0 | 10 | 3 | 39 | 31 | 105 | 100 |
| | Male | 2 | 80 | 0 | 36 | 7 | 1 | 69 | 18 | 213 | 203 | 707 | 619 |
| | Not reported | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 4 | 0 |
| | Total* | 2 | 101 | 0 | 41 | 11 | 1 | 79 | 21 | 256 | 236 | 818 | 721 |
| AIDS diagnoses | Female | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 2 | 6 | 4 | 16 |
| | Male | 0 | 3 | 0 | 3 | 0 | 2 | 10 | 2 | 20 | 49 | 71 | 134 |
| | Total* | 0 | 4 | 0 | 3 | 0 | 2 | 11 | 2 | 22 | 57 | 76 | 153 |
| AIDS deaths | Female | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 5 | 4 |
| | Male | 0 | 3 | 0 | 0 | 0 | 0 | 1 | 1 | 5 | 24 | 23 | 57 |
| | Total* | 0 | 3 | 0 | 0 | 0 | 0 | 1 | 2 | 6 | 24 | 28 | 63 |

* Totals include people whose sex was reported as transgender.

## Table 4. Cumulative diagnoses of HIV infection, AIDS, and deaths following AIDS since the introduction of HIV antibody testing to 30 September 2007, and reported by 31 December 2007, by sex and state or territory

| | Sex | State or territory | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Australia |
| HIV diagnoses | Female | 32 | 914 | 23 | 291 | 108 | 12 | 389 | 217 | 1,986 |
| | Male | 262 | 13,740 | 135 | 2,851 | 972 | 111 | 5,449 | 1,262 | 24,782 |
| | Not reported | 0 | 232 | 0 | 0 | 0 | 0 | 22 | 0 | 254 |
| | Total* | 294 | 14,917 | 158 | 3,151 | 1,081 | 123 | 5,882 | 1,486 | 27,092 |
| AIDS diagnoses | Female | 10 | 252 | 4 | 73 | 32 | 4 | 113 | 41 | 529 |
| | Male | 92 | 5,438 | 45 | 1,046 | 409 | 55 | 2,035 | 434 | 9,554 |
| | Total* | 102 | 5,708 | 49 | 1,121 | 442 | 59 | 2,161 | 477 | 10,119 |
| AIDS deaths | Female | 7 | 136 | 1 | 42 | 20 | 2 | 64 | 28 | 300 |
| | Male | 73 | 3,589 | 29 | 673 | 280 | 33 | 1,418 | 297 | 6,392 |
| | Total* | 80 | 3,736 | 30 | 717 | 300 | 35 | 1,491 | 326 | 6,715 |

* Totals include people whose sex was reported as transgender.

## Childhood immunisation coverage

*Tables 5, 6 and 7 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 July and 30 September 2006, at 24 months of age for the cohort born between 1 July and 30 September 2005, and at 6 years of age for the cohort born between 1 July and 30 September 2001 according to the National Immunisation Program.*

*For information about the Australian Childhood Immunisation Register see Surveillance systems reported in* CDI, *published in* Commun Dis Intell *2008;32:133–134 and for a full description of the methodology used by the Register see* 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 1435, Email: brynleyh@chw.edu.au.*

Immunisation coverage for children 'fully immunised' at 12 months of age for Australia increased marginally by 0.2 percentage points to 91.5% (Table 5). There were no important changes in coverage for any individual vaccines due at 12 months of age or by jurisdiction.

Immunisation coverage for children 'fully immunised' at 24 months of age for Australia increased by 0.5 percentage points to 93.0% and is now at its highest recorded level (Table 6). The greatest increase occurred in Western Australia where 'fully immunised' coverage increased by a significant 0.9 percentage points and coverage for individual

**Table 5. Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 July to 30 September 2006; assessment date 31 December 2007**

| Vaccine | State or territory | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Australia |
| Total number of children | 1,206 | 24,414 | 866 | 14,845 | 4,848 | 1,687 | 18,023 | 7,581 | 73,470 |
| Diphtheria, tetanus, pertussis (%) | 93.0 | 92.0 | 90.9 | 92.3 | 92.3 | 93.8 | 93.1 | 89.4 | 92.1 |
| Poliomyelitis (%) | 93.0 | 92.0 | 90.9 | 92.2 | 92.3 | 93.7 | 93.1 | 89.4 | 92.1 |
| *Haemophilus influenzae* type b (%) | 95.4 | 94.8 | 95.3 | 93.9 | 94.6 | 96.1 | 94.8 | 92.7 | 94.4 |
| Hepatitis B (%) | 95.4 | 94.8 | 95.4 | 93.8 | 94.5 | 96.0 | 94.8 | 92.9 | 94.4 |
| Fully immunised (%) | 92.8 | 91.7 | 90.7 | 91.4 | 91.6 | 93.5 | 92.2 | 88.8 | 91.5 |
| Change in fully immunised since last quarter (%) | -1.6 | +0.0 | +0.0 | +0.5 | +0.4 | +1.9 | +0.7 | -0.8 | +0.2 |

**Table 6. Percentage of children immunised at 2 years of age, preliminary results by disease and state or territory for the birth cohort 1 July to 30 September 2005; assessment date 31 December 2007**

| Vaccine | State or territory | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Australia |
| Total number of children | 1,106 | 23,440 | 945 | 14,679 | 4,610 | 1,571 | 16,716 | 6,982 | 70,049 |
| Diphtheria, tetanus, pertussis (%) | 95.5 | 95.3 | 95.8 | 94.6 | 95.2 | 96.9 | 95.9 | 94.5 | 95.3 |
| Poliomyelitis (%) | 95.4 | 95.2 | 95.8 | 94.5 | 95.1 | 96.9 | 95.9 | 94.5 | 95.2 |
| *Haemophilus influenzae* type b (%) | 95.7 | 95.4 | 94.8 | 93.6 | 94.2 | 96.8 | 95.1 | 94.4 | 94.8 |
| Measles, mumps, rubella (%) | 94.7 | 94.0 | 95.6 | 93.6 | 94.2 | 96.1 | 95.3 | 93.2 | 94.3 |
| Hepatitis B (%) | 96.2 | 96.0 | 97.1 | 95.5 | 95.7 | 97.3 | 96.6 | 95.3 | 96.0 |
| Fully immunised (%) | 93.9 | 92.9 | 94.1 | 92.1 | 92.9 | 95.7 | 94.1 | 91.4 | 93.0 |
| Change in fully immunised since last quarter (%) | -0.1 | +0.6 | +0.3 | +0.3 | +0.3 | +0.8 | +0.6 | +0.9 | +0.5 |

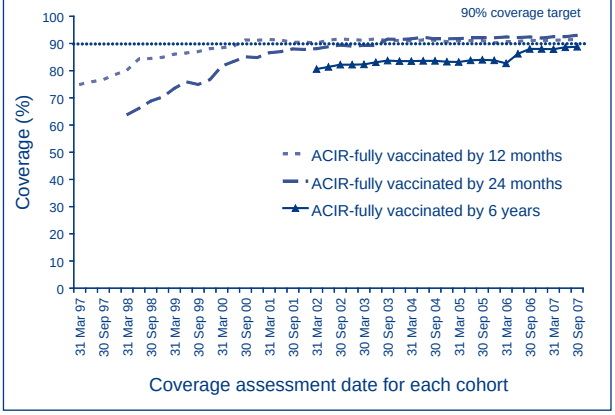
* The 12 months age data for this cohort was published in *Commun Dis Intell* 2007;32:148.

vaccines also increased in similar amounts, up to 1.2 percentage points for *Haemophilus influenzae* type b vaccine.

Immunisation coverage for children 'fully immunised' at six years of age for Australia increased a further 0.2 percentage points from the last quarter's 0.7 percentage points increase to reach 88.8%, its highest recorded level (Table 7). There were no important changes in coverage for any individual vaccines due at six years of age or by jurisdiction.

Figure 7 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 few years for all age groups. It should be noted that currently, coverage for the vaccines added to the NIP since 2003 (varicella at 18 months, meningococcal C conjugate at 12 months and pneumococcal conjugate at 2, 4, and 6 months) are not included in the 12 or 24 months coverage data respectively.

**Figure 7. Trends in vaccination coverage, Australia, 1997 to 30 September 2007, by age cohorts**




## 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.* Communicable Diseases Intelligence *NEPSS quarterly reports include only* Salmonella. *NEPSS receives reports of* Salmonella *isolates that have been serotyped and phage typed by the five* Salmonella *typing laboratories in Australia.* Salmonella *isolates are submitted to these laboratories for typing by primary diagnostic laboratories throughout Australia.*

*A case is defined as the isolation of a* Salmonella *from an Australian resident, either acquired locally or as a result of overseas travel, including isolates detected during immigrant and refugee screening. Second and subsequent identical isolates from an individual within six months are excluded, as are isolates from overseas visitors to Australia. The date of the case is the date the primary diagnostic laboratory isolated* Salmonella *from the clinical sample.*

*Quarterly reports include historical quarterly mean counts. These should be interpreted cautiously as they may be affected by outbreaks and by surveillance artefacts such as newly recognised and incompletely typed* Salmonella.

**Table 7. Percentage of children immunised at 6 years of age, preliminary results by disease and state or territory for the birth cohort 1 July to 30 September 2001; assessment date 31 December 2007**

| Vaccine | State or territory | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Australia |
| Total number of children | 1,061 | 22,548 | 877 | 14,350 | 4,721 | 1,511 | 16,472 | 6,878 | 68,418 |
| Diphtheria, tetanus, pertussis (%) | 89.6 | 89.8 | 88.8 | 88.2 | 88.0 | 89.0 | 91.7 | 86.0 | 89.4 |
| Poliomyelitis (%) | 90.1 | 89.8 | 88.9 | 88.3 | 88.2 | 88.9 | 91.9 | 86.3 | 89.5 |
| Measles, mumps, rubella (%) | 89.3 | 89.8 | 88.7 | 88.3 | 88.1 | 89.7 | 91.9 | 86.2 | 89.5 |
| Fully immunised (%) | 88.8 | 89.1 | 88.4 | 87.6 | 87.6 | 88.2 | 91.4 | 85.2 | 88.8 |
| Change in fully immunised since last quarter (%) | -0.3 | +0.9 | +1.1 | -0.9 | -0.2 | -2.1 | +0.3 | +0.5 | +0.2 |

* The 12 months age data for this cohort was published in *Commun Dis Intell* 2002;26:88

*NEPSS may be contacted at the Microbiological Diagnostic Unit, Public Health Laboratory, Department of Microbiology and Immunology, The University of Melbourne; by telephone: +61 3 8344 5701, facsimile: +61 3 8344 7833 or email joanp@unimelb.edu.au*

*Scientists, diagnostic and reference laboratories contribute data to NEPSS, which is supported by state and territory health departments and the Australian Government Department of Health and Ageing.*

*Reports to the National Enteric Pathogens Surveillance System of* Salmonella *infection for the period 1 October to 31 December 2007 are included in Tables 8 and 9. Data include cases reported and entered by 23 January 2008. 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 *2008;32:136.*

**Reporting period 1 October to 31 December 2007**

There were 1,815 reports to NEPSS of human *Salmonella* infection in the fourth quarter of 2007, approximately 40% more than in the third quarter of 2007. Although this count is fairly typical of the incidence of salmonellosis at this time of year, final inclusion of all data will probably see a count around 10% more than the recent historical average.

During the fourth quarter of 2007, the 25 most common *Salmonella* types in Australia accounted for 1,121 cases, 62% of all reported human *Salmonella* infections. Twenty-one of the 25 most common *Salmonella* infections in the fourth quarter of 2007 were also among those most commonly reported in the preceding quarter.

The most notable feature of the current data is a large outbreak of *S*. Typhimurium phage type 44, with cases reported predominantly from Victoria and New South Wales, but also South Australia, Queensland, the Northern Territory and Tasmania. Cases of *S*. Typhimurium (not phage typed), apparently reflecting one or more outbreaks in Western Australia during the third quarter of 2007, have declined considerably.

Other increases above the historical average for the period include *S*. Aberdeen (in the eastern states), *S*. Stanley (widespread, but typically acquired overseas), *S*. Typhimurium phage type 12 (widespread), *S*. Newport (particularly Victoria), and *S*. Singapore (Victoria, with cases in several other states).

**Acknowledgement:** We thank scientists, contributing laboratories, state and territory health departments, and the Australian Government Department of Health and Ageing for their contributions to NEPSS.

**Table 8. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 October to 31 December 2007, as reported to 23 January 2008**

| | State or territory | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Australia |
| Total all *Salmonella* for quarter | 25 | 455 | 97 | 458 | 88 | 38 | 478 | 176 | 1,815 |
| Total contributing *Salmonella* types | 17 | 117 | 43 | 95 | 39 | 13 | 115 | 46 | 218 |

## Table 9. Top 25 *Salmonella* types identified in Australia, 1 October to 31 December 2007, by state or territory

| National rank | *Salmonella* type | State or territory | | | | | | | | Total 4th quarter 2007 | Last 10 years mean 4th quarter | Year to date 2007 | Year to date 2006 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | | | | |
| 1 | *S.* Typhimurium PT 135 | 0 | 34 | 0 | 21 | 3 | 7 | 79 | 0 | 144 | 169 | 669 | 668 |
| 2 | *S.* Typhimurium PT 44 | 0 | 30 | 3 | 7 | 14 | 1 | 82 | 0 | 137 | 47 | 470 | 241 |
| 3 | *S.* Saintpaul | 0 | 10 | 9 | 61 | 1 | 0 | 3 | 7 | 91 | 95 | 371 | 569 |
| 4 | *S.* Birkenhead | 1 | 35 | 1 | 31 | 0 | 0 | 1 | 0 | 69 | 62 | 232 | 271 |
| 5 | *S.* Typhimurium PT 9 | 4 | 22 | 0 | 4 | 5 | 1 | 30 | 0 | 66 | 114 | 677 | 355 |
| 6 | *S.* Typhimurium (not phage typed) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 58 | 58 | 0 | 190 | 0 |
| 7 | *S.* Virchow PT 8 | 1 | 8 | 3 | 32 | 3 | 0 | 4 | 0 | 51 | 54 | 235 | 272 |
| 8 | *S.*Typhimurium PT 170 | 0 | 19 | 0 | 7 | 0 | 1 | 22 | 0 | 49 | 73 | 275 | 412 |
| 9 | *S.* Infantis | 1 | 23 | 5 | 0 | 2 | 1 | 11 | 5 | 48 | 30 | 193 | 176 |
| 10 | *S.* Aberdeen | 0 | 5 | 3 | 26 | 0 | 2 | 1 | 0 | 37 | 22 | 145 | 152 |
| 11 | *S.* Typhimurium PT 197 | 2 | 18 | 0 | 8 | 0 | 0 | 5 | 0 | 33 | 30 | 194 | 147 |
| 12 | *S.* Stanley | 0 | 11 | 0 | 5 | 2 | 2 | 7 | 6 | 33 | 17 | 134 | 104 |
| 13 | *S.* Chester | 1 | 10 | 3 | 12 | 0 | 0 | 2 | 3 | 31 | 38 | 158 | 158 |
| 14 | *S.* Muenchen | 1 | 8 | 4 | 14 | 0 | 1 | 1 | 1 | 30 | 29 | 137 | 156 |
| 15 | *S.* Typhimurium PT 12 | 2 | 9 | 0 | 5 | 3 | 0 | 7 | 3 | 29 | 18 | 108 | 117 |
| 16 | *S.* Enteritidis (not phage typed) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 28 | 28 | 0 | 48 | 0 |
| 17 | *S.* Waycross | 0 | 12 | 0 | 11 | 0 | 0 | 0 | 1 | 24 | 19 | 101 | 142 |
| 18 | *S.* Newport | 0 | 5 | 1 | 5 | 1 | 1 | 9 | 2 | 24 | 10 | 74 | 51 |
| 19 | *S.* Hvittingfoss | 0 | 1 | 0 | 20 | 0 | 0 | 1 | 1 | 23 | 20 | 115 | 137 |
| 20 | *S.* Typhimurium PT RDNC | 0 | 5 | 1 | 3 | 1 | 0 | 11 | 0 | 21 | 18 | 117 | 103 |
| 21 | *S.* Mississippi | 0 | 0 | 0 | 1 | 0 | 18 | 1 | 0 | 20 | 17 | 135 | 91 |
| 22 | *S.* Singapore | 0 | 4 | 0 | 3 | 3 | 0 | 10 | 0 | 20 | 13 | 75 | 54 |
| 23 | *S.* Typhimurium (PT pending) | 0 | 0 | 0 | 0 | 0 | 0 | 20 | 0 | 20 | 0 | 23 | 0 |
| 24 | *S.* Typhimurium untypable | 1 | 7 | 1 | 3 | 1 | 0 | 6 | 0 | 19 | 15 | 90 | 69 |
| 25 | *S.* Montevideo | 0 | 6 | 1 | 4 | 1 | 0 | 2 | 2 | 16 | 13 | 113 | 65 |

# OVERSEAS BRIEF

*The Overseas brief highlights disease outbreaks during the quarter that were of major public health significance worldwide or those that may have important implications for Australia.*

**Reporting period 1 October to 31 December 2007**

## Chikungunya

### Indonesia

An extensive outbreak of chikungunya was reported from the Indonesian islands of Java and Sumatra in late 2007. Between 11 and 24 November 2007, regional health authorities in Bogor, West Java reported 60 suspected cases of chikungunya infection. Numerous reports of chikungunya in Central and East Java occurred with outbreaks in these areas thought to have begun in early October 2007. Both *Aedes aegypti,* (the principal vector of chikungunya virus) and *Aedes albopictus* mosquitoes were thought to be transmitting the virus in some areas.[1] In a five day period between 10 and 15 December 2007, 997 suspected cases were reported by the Japara Regency Health Service in Central Java. In Sumatra, attacks rates of up to 90% were reported in some neighbourhoods in the worst affected areas of Wayadadi sub-district. Thousands of people were reportedly infected during December 2007 in the city of Bandar Lampang in South East Sumatra.[2] Outbreaks continued into 2008 in some areas.

Chikungunya occurred sporadically in Indonesia until 1985, but there were no reports of the disease between 1986 and 2000. Between 2001 and 2007, Indonesia reported several outbreaks, including 30 cases in central Jakarta (on the island of Java) in May 2007.[3] There are few reported laboratory confirmations of chikungunya cases in Indonesia and widespread under-reporting is likely.[4]

### Taiwan (ex Indonesia)

The Taiwan Centre for Disease Control reported two imported cases of chikungunya in Indonesia nationals, identified through fever screening at Taiwan's international airport on 28 and 30 December 2007.[5] Blood samples were taken from the cases after fever was identified at the airport and chikungunya was subsequently confirmed by polymerase chain reaction.[6]

## Cholera

An outbreak of cholera in the Vietnamese capital Hanoi began on 23 October 2007 and spread to 14 out of 64 provinces and cities across Vietnam.[7] National media reported nearly 2,000 people were hospitalised with acute diarrhoea in October and November 2007, with a total of 295 cases testing positive for cholera, including three foreign nationals from the Philippines, Belgium and Japan.[7,8] No deaths were reported and the epidemic was declared under control in December 2007 following extensive control campaigns by the Vietnamese Ministry of Health to improve food safety, disease surveillance, environmental hygiene and education.[8]

The outbreak originated in flood affected areas and health officials linked the initial source of illness to a popular fermented shrimp paste and other raw foods. Transmission is thought to then have occurred via contact with infected persons or contaminated water.[7]

Cholera was first reported in Vietnam in 1964 and is now endemic in 35 out of the 45 provinces.[9] The infection is found predominantly along the central coastal regions of the country and is positively correlated with rainfall and drinking water from public wells.[10] The last widespread cholera outbreak in Vietnam occurred in 2004.

## Dengue fever

### South East Asia

Across the South East Asia region in 2007, the number of cases of dengue fever reported has increased by 18% and fatal cases by 15% compared with 2006. The seasonal nature of dengue fever is well established in most countries with studies suggesting that peak dengue transmission occurs about six to eight weeks after peak rainfall.[11]

Thailand, Indonesia and Myanmar have all reported significantly increased numbers of dengue fever cases during 2007. Case fatality rates (CFRs) in urban areas are low: approximately 0.2% in Thailand and 1% in Indonesia and Myanmar where good health facilities are available, with much higher CFRs of up to 5% during localised outbreaks outside urban areas in both Indonesia and Myanmar.[11]

### Indonesia

Dengue fever is endemic across more than 300 regencies in Indonesia with the principle vector, *Aedes aegypti,* breeding extensively throughout all the country's islands. Dengue is endemic in predominantly urban areas with all serotypes (DEN 1 to 4) present in the majority of the large cities. The transmission season begins in November/December each year with the peak usually reached by February. Between January and November 2007 Indonesia reported 127,687 cases compared with 106,425 cases between January and September 2006.

Jakarta and West Java reported more than 25,000 cases each in 2007 (approximately 279 cases per 100,000 population in Jakarta and 63 cases per 100,000 population in West Java). East and Central Java each reported between 10,000 and 20,000 cases while Bali, East Kalimantan, Lampung, South Sumatra, Yogyarta, West Sumatra, North Sumatra, North Sulawesi, South Kalimantan and South Sulawesi reported between 1,000 and 5,000 cases with the remainder of the provinces reporting less than 1,000 cases each.[11]

### Thailand

Thailand reported 58,836 cases of dengue fever between January and November 2007, a 43% increase compared with 2006 when 41,116 cases were reported. Thailand reports cases of dengue fever annually from all its four regions: Northern, Central, North-eastern and Southern with peaks occurring in the months of June and July in recent years.[11]

### Myanmar

Between January and September 2007, Myanmar reported 9,578 cases of dengue fever compared to 8,674 cases during the same period of 2006. Similar to recent years, the peak number of cases in 2007 occurred in July, with the states/divisions of Mon, Yangon, Ayayarwaddy, Kayin, Yanintharyi, Rakhine, Mandalay, Bago (W), Bago (E), Magway and Sagaing reporting the most cases.[11]

## Ebola haemorrhagic fever

The World Health Organization (WHO) and the Ugandan Ministry of Health reported an outbreak of ebola haemorrhagic fever in the Bundibugyo District. Initial field investigations indicated that the outbreak began in September 2007. Confirmation of the outbreak was not obtained until 28 November 2007, after eight of the 20 samples collected by the national field team were sent to the national reference laboratories and to the United States Centers for Disease Control and Prevention (CDC) Special Pathogens Branch in Atlanta, tested positive for the infection.

A National Task Force was set up to co-ordinate the outbreak response and included the Ugandan Ministry of Health, the WHO and other experts from international NGOs including Médecins Sans Frontierès; African Field Epidemiology Network; International Federation of Red Cross and Red Crescent Societies and the CDC, Atlanta. The Global Outbreak and Alert Response Network along with other regional networks and technical institutions, supported international and operational co-ordination efforts. An active surveillance system for case detection and contact follow-up was established and isolation wards set up at hospitals in Kikyo and Bundibugyo. The Ugandan National Task Force reported 148 cases including 37 deaths (CFR 25%) by 3 January 2008.[12] The outbreak was officially declared over on 20 February 2008, 42 days after the last infected person was discharged from hospital on 8 January 2008.

There are currently four known species of Ebola virus, three of which are found in Africa (Zaire, Sudan and Ivory Coast species). Genetic sequencing conducted by the CDC in Atlanta of samples collected during this outbreak has confirmed that this Ebola virus is a new fifth species.[13,14]

## Influenza (avian)

The World Health Organization confirmed 19 cases of human H5N1 with dates of onset between 1 October and 31 December 2007[15] compared with 10 cases during the same period of 2006 (CFR 74%).[16] These WHO-confirmed cases were from six countries including the first ever confirmed cases in Burma and Pakistan (one case each). The remaining cases were reported from China (2 cases), Egypt (5 cases), Indonesia (9 cases) and Vietnam (1 case). The source of infection for nearly all cases was established as exposure to sick and dead poultry.[15]

There was no evidence of sustained human-to-human transmission of avian influenza during the reporting period, but a number of suspected clusters were investigated by the WHO and Ministries of Health in Burma, China and Pakistan. The son of one of the Chinese cases had initially been monitored as a suspected case. The case confirmed from Pakistan was a veterinarian who had worked on culling operations during H5N1 outbreaks in poultry. Up to 10 family members and contacts of this confirmed case had initially been suspected to have been infected with the virus, but none were confirmed and at least one was ruled out (by the US CDC) as ever having been infected. Similarly, four contacts of the Burmese cases had been symp-

tomatic, but none were confirmed as definite cases. If there was any human-to-human transmission amongst any of the confirmed or suspected cases it was limited and not sustained.

Indonesia reported the largest number of cases between 1 October and 31 December 2007 (9 cases, 7 of them fatal) and continues to report the highest number of cases of all countries since the beginning of the global outbreak in November 2003.[17]

## Plague (bubonic)

The Minister of Primary Healthcare in Uganda reported an outbreak of bubonic plague in the Nebbi district in the north-west of the country, with 100 cases between October and November 2007 (CFR 20%). The majority of fatal cases were women, who are at increased risk of exposure through sleeping on the floor (their indigenous Nebbi custom). There was no suggestion of any pneumonic plague cases in this outbreak.[18] Plague outbreaks are regularly reported from Uganda with the Nebbi district in the western region and the Arua district in the northern region considered to be endemic for the disease.[19]

## Poliomyelitis

### Global update

In 2007, the Global Polio Eradication Initiative (GPEI) intensified efforts to eradicate wild poliovirus type 1 (WPV1), because it is more highly transmissible and has higher attack rates of paralysis than WPV3.[20] This intensified effort has resulted in an 84% decrease in reported cases of WPV1 worldwide compared with 2006 (246 cases in 2007 compared with 1,463 cases in 2006) and limited transmission to small areas in the four endemic countries of Afghanistan, India, Nigeria and Pakistan.[20,21] The majority of this reduction in WPV1 case numbers has been from Nigeria.

Between 11 October and 4 January 2008, the GPEI reported 231 cases of wild poliovirus infection from the endemic countries of Afghanistan (1), India (177), Nigeria (50) and Pakistan (3) and 24 cases of wild poliovirus from the re-infected countries of Chad (11), the Democratic Republic of Congo (DRC) (7) and Niger (5).[22]

### India

In 2007 there were a total of 831 cases of wild poliovirus reported in India consisting of 76 WPV1, 752 WPV3 and three cases which were positive for both WPV1 and WPV3. This represents a 23% increase in the total number of polio cases in 2007 compared with 2006 but an 88% decrease in WPV1. The overall increase in cases is due to, not unexpectedly, outbreaks of WPV3 in Bihar. The impact of two large-scale supplementary immunisation campaigns using the monovalent oral polio vaccine type 3 won't start to impact these case numbers until mid-February 2008.[20]

### Afghanistan, Nigeria and Pakistan

In Nigeria, Pakistan and Afghanistan, (all of them considered polio-endemic) have all reported dramatic declines in total polio case numbers compared with 2006, with 90% fewer in Nigeria, 20% fewer in Pakistan and 45% fewer in Afghanistan.

### Chad

Chad (a re-infected country) reported 19 cases of polio in 2007, 11 of these between 1 October and 31 December 2007. Two of these cases were reported from the previously polio-free southern province of Tandjile. This area of southern Chad was not covered by the recent December supplementary immunisation campaign, increasing the risk of transmission and importation to the neighbouring countries of Sudan, Cameroon and the Central African Republic. The risk has since increased further with the movement of large numbers of Chadian refugees (estimated at over 30,000) into Cameroon following fighting between the government and opposition groups in the capital N'Djamena between 1 and 6 February 2008.[23]

### Democratic Republic of Congo

The DRC reported more cases of polio than any other re-infected country, with a total of 41 cases during 2007. Between 1 October and 31 December, the DRC reported six cases of wild poliovirus infection.[22]

### Nepal

Nepal reported four cases of WPV3 infection, all of which had onset of paralysis in December 2007. These were the first cases reported from Nepal since 2006. All of these cases were reported from areas bordering Bihar, India where an outbreak of WPV3 is continuing. Nepal remains at high risk of continued infection due to its proximity to the endemic areas of India and the movement of people across this border.[20,22]

## Rift Valley fever

An outbreak of Rift Valley fever (RVF) in the Sudan that was first reported in mid-October 2007 affected 15 localities in three states (White Nile, Sinnar and Gazeera) in the country. The Sudanese Federal Ministry of Animal Resources and Fisheries

reported confirmation of RVF in samples taken on 29 October 2007 from animals in the White Nile State.[24] A total of 698 human cases, including 222 deaths (CFR 32%), were reported between mid-October 2007 and 5 January 2008, the majority from Gazeera State. There have been no new cases reported since 5 January 2008, with active surveillance continuing in all affected states.[25]

RVF is endemic in the Sudan, but the last major outbreak was in 1976. The first outbreak of RVF outside sub-Saharan Africa occurred in Egypt in 1977–1978 (with 18,000 human cases including 598 deaths) and was thought to have begun with imported a case(s) from the Sudan. A later epizootic in Egypt in 1997 was also believed to have been imported from the Sudan. In September 2000, outbreaks of RVF were reported for the first time outside the continent of Africa, in Yemen and Saudi Arabia.[26,27]

## Tuberculosis

Approximately two million people in the Western Pacific Region become infected with tuberculosis (TB) annually with the potential for a rapid increase in the number of extensively drug-resistant TB (XDR-TB) cases if drug management is not optimal.[28] Access to appropriate treatment for multi-drug-resistant TB (MDR-TB) is not uniformly available increasing the risk of XDR-TB developing.

TB is one of the three leading infectious causes of mortality in Papua New Guinea (PNG), with a mortality rate of 42 deaths per 100,000 population and an annual incidence of 233 cases per 100,000 population. There is a high risk of TB infection spreading to northern Queensland because of Australia's close proximity to PNG and the Torres Strait Treaty arrangements that facilitate movement of people between PNG and the Torres Strait Islands. This risk is highlighted by 10 of the 30 MDR-TB strains isolated in Australia in the past two years originating from the Western Province of PNG.[29]

A recent study found that 15 out of 60 cases of tuberculosis amongst PNG nationals from the Western Province presenting to health clinics in Queensland between 2000 and 2006 were infected with MDR-TB strains, with evidence of primary transmission of the infection. These 15 cases were all infected with the *Mycobacterium tuberculosis* Beijing genotype, which is increasingly linked to MDR-TB and is highly transmissible.[29]

While introduction of the 'directly observed therapy short course program (DOTS)' prevents resistant strains developing, this optimal therapy is not consistently available or utilised in PNG. The magnitude of MDR-TB transmission in PNG is not known but its control is essential to stop it becoming an emerging issue in Queensland with long term social and economical sequelae.[29]

## References


1. ProMed-Mail [Online] [Surya]. 15 December 2007. Available from: www.promedmail.org
2. ProMed-Mail [Online] [The Jakarta Post]. 28 December 2007. Available from: www.promedmail.org
3. Global Public Health Intelligence Network GPHIN [Online], [The Jakarta Post]. 1 August 2007. Public Health Agency of Canada.
4. World Health Organization. Outbreak and spread of chikungunya. *Wkly Epidemiol Rec* 2007;82;409–416.
5. Infectious Disease Surveillance and Monitoring System for Animal and Human Health: summary of notable events/incidents of public health significance for the period 1–31 January 2008.
6. ProMed-Mail [Online]. 9 January 2008. Available from: www.promedmail.org
7. Reliefweb [Online] [Xinhua News Agency]. 14 November 2007. Available from: http://www.reliefweb.int/rw/dbc.nsf/doc100?OpenForm
8. Yahoo News [Online]. 16 January 2008. Available from: http://news.yahoo.com/
9. Gideon [Online]. Available from: http://www.gideononline.com/ Accessed 12 February 2008.
10. Kelly-Hope LA, Alonso WJ, Thiem VD, Anh DD, Canh do G, Lee H, et al. Geographic distribution and risk factors associated with enteric diseases in Vietnam. *Am J Trop Med Hyg* 2007;76:706–712.
11. World Health Organization. Regional Office of South-East Asia (SEARO) [Online]. Situation update of dengue in the SEA Region, 2007. Available from: http://www.searo.who.int/LinkFiles/Dengue_DengueSEAR-07.pdf
12. Government of Uganda. Ebola outbreak in Uganda: National Task Force situation report no. 18; 3 January 2008.
13. Centers for Disease Control and Prevention. Travel notices [Online]. Outbreak notice: Ebola outbreak in the district of Bundibugyo, Uganda. 6 December 2007. Available from: http://wwwn.cdc.gov/travel/contentEbolaUganda.aspx
14. World Health Organization [Online]. End of Ebola outbreak in Uganda. 20 February 2008. Available from: http://www.who.int/csr/don/2007_02_20b/en/index.html
15. World Health Organization. Disease Outbreak News [Online]. Avian influenza situation updates, 8 October 2007 to 11 January 2008. Available from: http://www.who.int/csr/don/en/
16. World Health Organization. Disease Outbreak News [Online]. Avian influenza situation updates, 16 October 2006 to 10 January 2007. Available from: http://www.who.int/csr/don/en/
17. World Health Organization. Disease Outbreak News [Online]. Avian influenza situation updates, 13 January 2004 to 11 January 2008. Available from: http://www.who.int/csr/don/en/
18. Global Public Health Intelligence Network GPHIN [Online], [Journal Chretien]. 4 December 2007. Public Health Agency of Canada.
19. Gideon [Online]. Available from: http://www.gideononline.com/ Accessed on 20 February 2008.

20. Global Polio Eradication Initiative [Online]. Monthly Situation Reports. January 2008. Available from: http://www.polioeradication.org/content/general/current_monthly_sitrep.asp

21. Conclusions and recommendations of the Advisory Committee on Poliomyelitis Eradication. Geneva, 27–28 November 2007. *Wkly Epidemiol Rec* 2008;83:25-35.

22. Global Polio Eradication Initiative [Online]. Wild Polio Weekly Updates 11 October 2007–4 January 2008. Available from: http://www.polioeradication.org/

23. United States Agency for International Development [Online]. Chad: Complex Emergency Fact Sheet #1 Fiscal year (FY) 2008. 8 February 2008. Available from: http://www.usaid.gov/our_work/humanitarian_assistance/disaster_assistance/countries/chad/template/fs_sr/fy2008/chad_ce_fs01_02-08-2008.pdf

24. World Health Organization [Online]. Rift Valley fever in Sudan, updates 1–4. 5 November–21 December 2007. Available from: http://www.who.int/csr/don/archive/disease/rift_valley_fever/en/

25. ProMed-Mail [Online]. (WHO) Rift Valley fever in Sudan – update. 22 January 2008. Available from: www.promedmail.org

26. World Health Organization [Online]. Rift Valley Fever fact sheet No. 207. [Revised September 2007]. Available from: http://www.who.int/mediacentre/factsheets/fs207/en/ Accessed on 21 February 2008.

27. Gideon [Online]. Available from: http://www.gideononline.com/ Accessed on 13 February 2008.

28. World Health Organization. Regional Office for the Western Pacific [Online} Press release. Drug-resistant tuberculosis threatens the Western Pacific Region. Manila: 22 March 2007. Available from: http://www.wpro.who.int/media_centre/press_releases/pr_20070322.htm

29. Gilpin CM, Simpson G, Vincent S, O'Brien TP, Knight TA, Globan M, et al. Evidence of primary transmission of multidrug-resistant tuberculosis in the Western Province of Papua New Guinea. *Med J Aust* 2008;188:148–152.

## Administrative

# COMMUNICABLE DISEASES INTELLIGENCE
# INSTRUCTIONS FOR AUTHORS

*Communicable Diseases Intelligence* (*CDI*) is published quarterly (March, June, September and December) by the Surveillance Branch, Office of Health Protection, Australian Government Department of Health and Ageing.

The aim of *Communicable Diseases Intelligence (CDI)* is to disseminate information on the epidemiology of communicable disease in Australia, including surveillance, prevention and control.

The objectives of *CDI* are to:

- report on surveillance of communicable diseases of relevance to Australia;
- publish other articles relevant to communicable disease epidemiology in Australia; and
- provide information on other activities relevant to the surveillance, prevention and control of communicable disease in Australia.

*CDI* invites contributions dealing with any aspect of communicable disease epidemiology, surveillance, prevention or control in Australia. Submissions can be in the form of original articles, short reports, or letters to the editor.

*CDI* will invite guest editorials and review articles on occasion and publish guidelines and position papers from the Communicable Diseases Network Australia (CDNA) and its expert sub-committees.

### Manuscripts for submission

Manuscripts submitted to *CDI* must be offered exclusively to the journal. All manuscripts should be accompanied by a covering letter that should include:

- a list of all authors;
- confirmation that the manuscript content (in part or in full) has not been submitted or published elsewhere; and
- whether the manuscript is being submitted as an article, short report, surveillance summary, outbreak report or case report.

In addition, manuscripts should include a title page that should contain the following information:

- title (e.g. Prof, Dr, Ms, Miss, Mrs, Mr), full name including middle initial, position held, and institution at the time the article was produced, of each author;

- name of corresponding author, including current postal address, telephone, facsimile and email; and
- word count of the main text and of the abstract.

On receipt of a manuscript, authors will be sent a brief acknowledgment. Accepted manuscripts are edited for style and clarity and final proofs are returned to the corresponding author for checking prior to printing.

## Authorship

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

## Types of manuscript

### Original articles

The text of articles must be structured to contain an abstract, introduction, methods, results, discussion, acknowledgments and references. Manuscripts submitted as articles must be 3,000 words or less and are peer-reviewed. Occasionally, reports of urgent public health importance may be published immediately, at the discretion of the Editor.

### Short reports

Short reports are not subject to peer review and should be of less than 2,000 words. Types of short reports include:

*Surveillance summaries*

A report of 1,000 words or less which briefly reports on changes in the local epidemiology of communicable disease, changes in surveillance systems, or new interventions, such as implementing vaccination in an at-risk group. Surveillance summaries should provide a brief description of the setting and a discussion of the significance of the events, changes or interventions.

*Outbreak reports*

Unstructured reports of communicable disease outbreaks of 500 to 1,000 words will be considered for publication based on their public health significance. Reports should include details of the investigation, including results of interventions and the significance of the outbreak for public health practice. More comprehensive reports on outbreaks should be submitted as articles.

*Case reports*

Brief unstructured reports of 500 to 1,000 words on unique cases of communicable disease will be considered based on their public health significance. Authors must note the instructions on the protection of patient's right to privacy (see Ethics committee approvals and patient's right to privacy below). Some discussion of the significance of the case for communicable disease control should be included.

### Letters to the Editor

The editors welcome comments on articles published in *CDI* in the form of letters to the Editor. Letters should normally be less than 500 words, include no more than a single chart and less than six references.

## Document preparation

Authors are asked to provide an electronic copy of the manuscripts. Microsoft Word for Windows 2003 or an earlier version is preferred. Alternatively files should be saved as Rich Text Format (rtf).

In addition:

- Arial font is preferred but if not available use Times New Roman.
- Abstracts should not exceed 250 words. Do not cite references in abstracts. Structured abstracts are not acceptable.
- Include up to 10 keywords.
- Avoid too many abbreviations.
- Do not use numbered paragraphs.
- Do not use page numbering.
- Do not use headers or footers.

Final manuscripts should not include any field codes such as automatic numbering for references. Electronic referencing software (e.g. Endnote) field codes should be embedded before submission of the final version.

## Tables

- Tables and table headings should be provided in the manuscript at the end of the text and should be referred to within the results section.
- Information in tables should not be duplicated in the text.
- Headings should be brief.
- Simplify the information as much as possible, keeping the number of columns to a minimum.

- Separate rows or columns are to be used for each information type (e.g. percentage and number should be in separate columns rather than having one in parentheses in the same column).
- If abbreviations are used these should be explained in a footnote.
- Footnotes should use the following symbols in sequence: * † ‡ § || ¶ ** †† ‡‡
- Do not use borders, or blank rows or blank columns for spacing.

## Figures and illustrations

Figures and illustrations, including headings, should be provided in the manuscript at the end of the text and should be referred to within the results section. In addition, they should also be provided as a separate file in accordance with the following requirements.

Examples of each of the following can be found in the on-line version of Instructions to authors at: http://www.health.gov.au/internet/wcms/publishing.nsf/Content/cda-pubs-cdi-auth_inst.htm

### Charts

- Use Microsoft Excel for Windows.
- Each figure should be created on a separate worksheet rather than as an object in the datasheet (use the 'as new sheet' option for chart location).
- The numerical data used to create each figure must be included on a separate worksheet.
- Worksheets should be appropriately titled to distinguish each graph.
- Do not include the graph heading on the Excel worksheet.

### Illustrations

- Black and white illustrations or flow charts can be included if required.
- Images should preferably be at least 300 dpi.
- Electronic copies of computer-generated illustrations should preferably be saved in a vector image program such as Adobe Illustrator but other similar graphic software is acceptable. Files should be saved in one of the following graphic formats (in preferential order): AI, TIFF, EPS, or GIF.
- Use a sans serif font for figures (e.g. arial). Symbols, lettering and numbering should be clear and large enough to be legible when reduced in size.

### Photographs

- Photographs may be submitted if required.
- Photos need to be at least 300 dpi.
- Electronic copies should be saved in Adobe Photoshop, or similar graphic software in one of the following graphic formats (in preferential order): PSD, TIFF, EPS or JPEG (JPG).

### Maps

- Electronic copies of black and white (outline) maps should be saved in Adobe Photoshop, or similar graphic software in one of the following graphic formats (in preferential order): PSD, TIFF, EPS, or GIF.
- Thermal maps created by mapping programs such as MapInfo or Arc GIS should be saved at 300 dpi and in one of the following graphic formats (in preferential order): TIFF, EPS, or JPEG (JPG). Shading of map areas should be distinguishable when printed in black and white.
- Use a sans serif font for text. Symbols, lettering and numbering should be clear and large enough to be legible when reduced in size.

## References

References should be identified consecutively in the text by the use of superscript numbers without brackets. Any punctuation should precede the reference indicators.

The accuracy of references is the responsibility of authors. Use the Vancouver reference style (see International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. *Ann Intern Med* 1997;1126:36–47 available from: http://www.nlm.nih.gov/bsd/uniform_requirements.html) and abbreviate journal names as in Medline (e.g. *Commun Dis Intell*). The Medline journal database is available from: http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=journals. Include the surnames and initials of all authors (or only the first six authors, et al, if there are more than six). Cite the first and last page numbers in full, and specify the type of reference (e.g. a letter, an editorial, an abstract, or supplement).

Cite personal communications and unpublished papers in the text, not in the reference list, with the exception of material that has been accepted for publication (in press). Obtain written permission from people cited, and include their title, position and affiliation.

## Ethics committee approvals and patients' rights to privacy

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

When informed consent has been obtained this should be included in the text.

Ethical approval and patient consent may also be required for case reports. Identifying details about patients should be omitted if they are not essential, but data should never be altered or falsified in an attempt to attain anonymity.

## Review process

Articles provisionally accepted for publication undergo a peer review process. Manuscripts are reviewed by two experts in the topic area. Authors may be asked to revise articles as a result of the review process before the final decision about publication is made by the Editor. Revised articles are to be returned with a covering letter addressing each comment made by each reviewer.

Occasionally, reports of urgent public health importance may be published immediately without peer review, at the discretion of the Editor. Articles may also be rejected without peer review.

Short reports are not subject to peer review.

## Copyright

All authors are asked to transfer copyright to the Commonwealth before publication. A copyright form will be sent to the corresponding author. All authors are required to sign the copyright release. The Commonwealth copyright will be rescinded if the article is not accepted for publication.

## Submission of manuscripts

Manuscripts should be provided electronically by email to: cdi.editor@health.gov.au

Requests for further information can be obtained either by telephone to (02) 6289 2717, by facsimile: (02) 6289 2600 or by email to the address above.

# SURVEILLANCE SYSTEMS REPORTED IN *CDI*, 2008

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

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

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

The major features of the surveillance schemes for which *CDI* publishes regular reports are described below.

Other surveillance schemes for which *CDI* publishes annual reports include tuberculosis notifications (*Commun Dis Intell* 2008;32:1–11), the Australian Mycobacterium Reference Laboratory Network (*Commun Dis Intell* 2008;32:12–17), invasive pneumococcal disease surveillance (*Commun Dis Intell* 2008;32:18–30), the National Arbovirus and Malaria Advisory Committee (*Commun Dis Intell* 2008;32:31–46), and the Australian Rotavirus Surveillance Program (*Commun Dis Intell* 2007;31:375–379).

## Australian Childhood Immunisation Register

Accurate information on the immunisation status of children is needed at the community level for program management and targeted immunisation efforts. A population-based immunisation register can provide this need. The Australian Childhood Immunisation Register (ACIR) commenced operation on 1 January 1996 and is now an important component of the *Immunise Australia Program*. It is administered and operated by Medicare Australia (formerly the Health Insurance Commission). The Register was established by transferring data on all children under the age of seven years enrolled with Medicare to the ACIR. This constitutes a nearly complete population register, as approximately 99% of children are registered with Medicare by 12 months of age. Children who are not enrolled in Medicare are added to the Register when a recognised immunisation provider supplies details of an eligible immunisation. Immunisations are generally notified to Medicare Australia either by electronic means, the Internet or by paper ACIR notification forms. Immunisations recorded on the Register must have been given in accordance with the guidelines for immunisation determined by the National Health and Medical Research Council.

From the data finally entered onto the ACIR, Medicare Australia provides regular quarterly coverage reports at the national and state level. Coverage for these reports is calculated using the cohort method described in *Commun Dis Intell* 1998;22:36–37. With this method, a cohort of children is defined by date of birth in three-month groups. This birth cohort has the immunisation status of its members assessed at the three key milestones of 12 months, 24 months and 6 years of age. Analysis of coverage is undertaken three months after the due date for completion of each milestone, so that time is available for processing notifications and the impact on coverage estimates of delayed notification to the ACIR is minimised. Only children enrolled with Medicare are included in order to minimise inaccuracies in coverage estimates due to duplicate records.

Medicare Australia coverage reports for the three milestones are published in *CDI* each quarter. Coverage estimates are provided for each state and

territory and Australia as a whole and for each individual vaccine assessed at each milestone. Changes in 'fully immunised' coverage from the previous quarter are also included in the tables.

A commentary on ACIR immunisation coverage estimates is included with the tables in each issue and graphs are used to provide trends in immunisation coverage.

## Australian Gonococcal Surveillance Programme

The Australian Gonococcal Surveillance Programme (AGSP) is a continuing program to monitor antimicrobial resistance in *Neisseria gonorrhoeae* and includes the reference laboratories in all states and territories. These laboratories report data on sensitivity to an agreed core group of antimicrobial agents on a quarterly basis and provide an expanded analysis as an annual report in *CDI* (*Commun Dis Intell* 2007;31:180–184). The antibiotics that are currently routinely surveyed are the penicillins, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens. One main purpose of the AGSP is to help define standard protocols for antibiotic treatment of gonococcal infection. When *in vitro* resistance to a recommended agent is demonstrated in 5% or more of isolates, it is usual to reconsider the inclusion of that agent in current treatment schedules. Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level resistance to the tetracyclines and intermittent surveys of azithromycin resistance are conducted. Comparability of data is achieved by means of a standardised system of MIC testing and a program-specific quality assurance process.

## Australian Meningococcal Surveillance Programme

The reference laboratories of the Australian Meningococcal Surveillance Programme report data of laboratory-confirmed cases confirmed either by culture or by non-culture 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 are reported annually and quarterly in *CDI*. Data 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, including phenotyping and antibiotic susceptibility data are published annually (*Commun Dis Intell* 2007;31:185–194).

## Australian Paediatric Surveillance Unit

The Australian Paediatric Surveillance Unit (APSU) is a national, active surveillance mechanism with monthly reporting by approximately 1,270 Australian paediatricians and other child health clinicians. APSU conducts surveillance for uncommon conditions of childhood, including genetic disorders, child mental health problems, rare injuries, and rare infections and vaccine preventable diseases. Communicable diseases currently under surveillance include: acute flaccid paralysis (to identify potential cases of poliovirus infection); congenital cytomegalovirus infection; congenital rubella; perinatal exposure to HIV, HIV infection and AIDS; neonatal herpes simplex virus infection; hepatitis C virus infection; group B *Streptococcus* sepsis; and neonatal, congenital and severe complications of varicella virus infection. Surveillance for intussusception and its causes (e.g. rotavirus infection) and for acute rheumatic fever (group A *Streptococcus* infection) commenced in 2007. APSU may occasionally be used for short-term rapid response surveillance e.g. surveillance for the severe complications of influenza in children <5 years of age in 2007.

The primary objectives of the APSU are to document the number of Australian children aged 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 2007, over 1,270 clinicians participated in the surveillance of 16 conditions through the APSU, with an overall monthly response rate of 96%. APSU is a unit of the Royal Australasian College of Physicians, and its activities are supported by the Department of Health and Ageing; the Faculty of Medicine, University of Sydney; and the National Health and Medical Research Council Enabling Grant 402784. For further information please contact the APSU Director, Professor Elizabeth Elliott on telephone: +61 2 9845 3005, facsimile +61 2 9845 3082 or email: apsu@chw.edu.au

## Australian Sentinel Practice Research Network

The Royal Australian College of General Practitioners and the Department of General Practice at the University of Adelaide operate the Australian Sentinel Practice Research Network (ASPREN). ASPREN is a national network of

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

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published. In 2008, four conditions are being monitored; all of which are related to communicable diseases. These include influenza like illness, gastroenteritis, chickenpox and shingles.

There are currently 96 general practitioners participating in the network from all jurisdictions other than the Northern Territory. Sixty-eight per cent of these are in metropolitan areas, 24% in rural and 7% in remote areas of Australia. Approximately 6,000 consultations are recorded each week..

Data for communicable diseases are published in *CDI* every quarter. Data are presented in graphic format as the rate of reporting per 1,000 consultations per week. The conditions are defined as follows.

**Influenza-like illness – record once only per patient**

Must have the following: fever, cough and fatigue.

**Gastroenteritis – record once only per patient**

Three or more loose stools, and/or two vomits in a 24 hour period excluding cases who have a known cause, for example bowel disease, alcohol, pregnancy.

**Chickenpox – record once only per patient**

An acute, generalised viral disease with a sudden onset of slight fever, mild constitutional symptoms and a skin eruption which is maculopapular for a few hours, vesicular for three to four days and leaves a granular scab.

**Shingles – record once only per patient**

Recurrence, recrudescence or re-activation of chickenpox infection. Vesicles with any erythematous base restricted to skin areas supplied by sensory nerves of a single or associated group of dorsal root ganglia. Lesions may appear in crops in irregular fashion along nerve pathways, are usually unilateral, deeper seated and more closely aggregated than those of chickenpox.

Note: Those conditions which show 'record once only per patient' are to have each occurrence of the condition only recorded on one occasion no matter how many patient contacts are made for this condition. If the condition occurs a second or subsequent time, it is to be recorded again. Conversely, for other conditions each attendance at which they are addressed in some way is to be recorded.

## HIV and AIDS surveillance

National surveillance for HIV and AIDS is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with state and territory health authorities, the Australian Government Department of Health and Ageing, the Australian Institute of Health and Welfare and other collaborating networks in surveillance for HIV/AIDS.

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

Currently, two tables presenting the number of new diagnoses of HIV infection, AIDS and deaths following AIDS are published in each issue of *CDI*. The tabulations are based on data available three months after the end of the reporting period, to allow for reporting delay and to incorporate newly available information.

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

## Laboratory Virology and Serology Reporting Scheme

The Laboratory Virology and Serology Reporting Scheme (LabVISE) began operating in 1977. The scheme currently comprises 17 laboratories from all states and the Australian Capital Territory. Contributors submit data fortnightly on the laboratory identification of viruses and other organisms. Each record includes mandatory data fields (laboratory, specimen collection date, a patient identifier code and organism), and optional fields (patient's

sex, date of birth or age, postcode of residence, specimen source, clinical diagnosis and the method of diagnosis). Reports are collated, analysed and published quarterly in *CDI*. Each report includes summary tables of total numbers of organisms identified by state or territory and numbers of reports by month and participating laboratory. Monthly updates of LabVISE data are also published on the *Communicable Diseases Australia* website.

LabVISE data should be interpreted with caution. The number and type of reports received is subject to a number of biases. These include the number of participating laboratories, which has varied over time. The locations of participating laboratories also create bias, as some jurisdictions are better represented than others. Also changes in diagnostic practices, particularly the introduction of new testing methodologies, may affect laboratory reports. The ability of laboratory tests to distinguish acute from chronic or past infection must also be considered in interpretation of the data. Although changes in incidence cannot be determined with precision from this data, general trends can be observed, for example with respect to seasonality and the age-sex distribution of patients. See review in *Commun Dis Intell* 2002;26:323–374).

## National Enteric Pathogens Surveillance System

Since 1980, the National Enteric Pathogens Surveillance Scheme (NEPSS) has collected, analysed and disseminated data on human enteric bacterial infections diagnosed in Australia. These pathogens include *Salmonella*, *Escherichia coli*, *Vibrio*, *Yersinia*, *Plesiomonas*, *Aeromonas* and *Campylobacter*.

*Communicable Diseases Intelligence* NEPSS quarterly reports include only *Salmonella*. NEPSS receives reports of *Salmonella* isolates submitted from primary diagnostic laboratories throughout Australia to any of the five serotyping laboratories, two of which (MDU and IMVS) also perform phage typing.

A case is defined as the isolation of a *Salmonella* from an Australian resident, either acquired locally or as a result of overseas travel, including isolates detected during immigrant and refugee screening. Second and subsequent identical isolates from an individual within six months are excluded, as are isolates from overseas visitors to Australia. The date of the case is the date the primary diagnostic laboratory isolated a *Salmonella* from the clinical sample.

NEPSS is operated by the Microbiological Diagnostic Unit — Public Health Laboratory, Department of Microbiology and Immunology, The University of Melbourne; and is overseen by a Steering Committee of state, territory and Commonwealth stakeholders. NEPSS may be contacted at the Microbiological Diagnostic Unit, by telephone +61 3 8344 5701, facsimile +61 3 8344 7833 or email joanp@unimelb.edu.au

Scientists, diagnostic and reference laboratories, clinicians and public health professionals generate and contribute data to NEPSS, which is supported by state and territory health departments and the Australian Government Department of Health and Ageing.

## OzFoodNet: enhanced foodborne disease surveillance

The Australian Government Department of Health and Ageing established the OzFoodNet network in 2000 to collaborate nationally in the investigation of foodborne disease. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease.

OzFoodNet reports quarterly on investigations of gastroenteritis outbreaks and clusters of disease potentially related to food. Annual reports have been produced and published in *CDI* (*Commun Dis Intell* 2007;31:345–365) since 2002. Data are reported from all Australian jurisdictions.

## National Influenza Surveillance Scheme

Influenza surveillance in Australia is based on several schemes collecting a range of data that can be used to measure influenza activity.

- Since 2001, laboratory-confirmed influenza has been a notifiable disease in all Australian states and territories (except South Australia) and reported in the National Notifiable Diseases Surveillance System (see above).

- In 2007, six sentinel general practitioner schemes contribute reports of influenza-like illness: the Australian Sentinel Practice Research Network, the Tropical Influenza Surveillance from the Northern Territory, the New South Wales Sentinel General Practice Scheme, the Victorian Sentinel General Practice Scheme, Queensland and Western Australian sentinel general practices.

- The Laboratory Virology and Serology Reporting Scheme laboratory reports of influenza diagnoses including virus type.

The results of each of the schemes are published together fortnightly throughout the influenza season (May to October) on the *Communicable Diseases Australia* Website as the Australian Influenza Report.

Annual reports on influenza in Australia are published in *CDI* each year (*Commun Dis Intell* 2007;31:167–179). These reports include the above data as well as absenteeism data from a major national employer, hospitalisation and mortality data and influenza typing data from the WHO Collaborating Centre for Influenza Reference and Research.

## National Notifiable Diseases Surveillance System

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

The system coordinates the national surveillance of more than 60 communicable diseases or disease groups endorsed by the CDNA. Under this scheme, notifications are made from doctors and laboratories to state or territory health authorities under the provisions of the public health legislation in their jurisdiction. Electronic, de-identified unit records of notifications are supplied to the Australian Government Department of Health and Ageing for collation, analysis and reporting in *CDI*.

Data provided for each notification include a unique record reference number, state or territory, disease code, date of onset, date of notification to the relevant health authority, sex, age, indigenous status and postcode of residence. Additional data include: infecting organism and subtype; the diagnosis method; full details of vaccination where appropriate; resident location; dates of onset, specimen collection, notification and date when notification was received by health authorities; outbreak reference number; how the case was found; whether the case was confirmed; and whether the case was imported from overseas.

Aggregated data are presented on the *Communicable Diseases Australia* Internet site and updated three times a week (www.health.gov.au/cda). Data are published in *CDI* every quarter and in an annual report. The reports include numbers of notifications for each disease by state or territory, and totals for Australia for the current period, the year to date, and for the corresponding period of the previous year. The national total for each disease is compared with the average number of notifications over the previous five years in the same period. A commentary on the notification data is included with the tables in each issue of *CDI* and graphs are used to illustrate important aspects of the data.

HIV infection and AIDS surveillance is conducted by the National Centre for HIV Epidemiology and Clinical Research and is reported in the HIV and AIDS surveillance reports (see below).

## Sentinel Chicken Surveillance Programme

The Sentinel Chicken Surveillance Programme is used to provide an early warning of increased flavivirus activity in Australia. The main viruses of concern are Murray Valley encephalitis (MVEV) and Kunjin viruses. MVEV causes the disease Murray Valley encephalitis (formerly known as Australian encephalitis), a potentially fatal disease in humans. Encephalitis is less frequent in cases of Kunjin virus infection and these encephalitis cases have a lower rate of severe sequelae.

These viruses are enzootic in parts of the north-east Kimberley region of Western Australia and the Top End of the Northern Territory but are epizootic in other areas of the Kimberley, Pilbara, Gascoyne Murchison and Mid-west regions of Western Australia, in north Queensland and in Central Australia. MVEV is also responsible for occasional epidemics of encephalitis in eastern Australia. Since 1974, a number of sentinel chicken flocks have been established in Australia to provide an early warning of increased MVEV activity. These programs are supported by individual state health departments. Each state has a contingency plan that will be implemented if one or more chickens in a flock seroconverts to MVEV.

Currently, flocks are maintained in the north of Western Australia, the Northern Territory, New South Wales and in Victoria. The flocks in Western Australia and the Northern Territory are tested all year round but those in New South Wales and Victoria are tested only in the summer months, during the main MVEV risk season. Results are posted on the National Arbovirus Surveillance Website by state representatives. A yearly summary is presented in *CDI* (*Commun Dis Intell* 2008;32:31–46).

## References

1. Last JM. A dictionary of epidemiology. New York: Oxford University Press, 1988.
2. Hall R. Notifiable diseases surveillance, 1917 to 1991. *Commun Dis Intell* 1993;226–236.