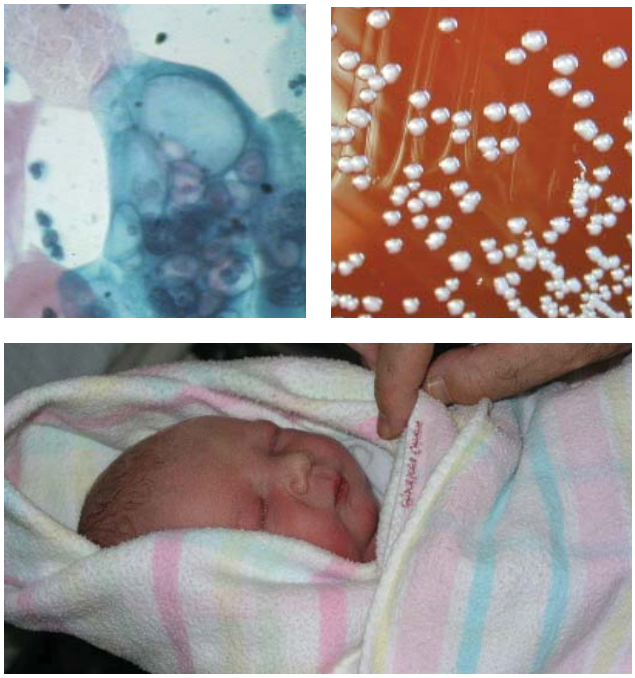
Australian Government
Department of Health and Ageing

# *Communicable Diseases Intelligence*



## Quarterly report

Volume 28
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# Communicable Diseases Intelligence

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

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Front cover: Surveillance and Epidemiology Section, Australian Government Department of Health and Ageing.

Clockwise from top left: Chlamydia. Image © Bristol Biomedical Image Archive, University of Bristol. *Burkolderia pseudomallei* grown on sheep blood agar for 48 hours. *Burkolderia pseudomallei* is the causative agent for melioidosis. Image sourced from the Centers for Disease Control and Prevention Public Health Image Library, courtesy of the Centers for Disease Control and Prevention, Atlanta, Georgia. A beautiful, healthy baby.

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# Contents

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# Invasive pneumococcal disease in Australia, 2003

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**Some minor amendments have been made to this article on 4 May 2005, post-printing. In particular, Tables 15 and 16 have been updated and the Map has been replaced.**

## Abstract

**There were 2,174 cases of invasive pneumococcal disease (IPD) noti ed to the National Noti able Diseases Surveillance System (NNDSS) in Australia in 2003; a rate of 10.9 per 100,000 population. The noti cation rate varied between states and territories and by geographical region with the highest rates in the north of the country. Invasive pneumococcal disease was reported most frequently in children aged less than two years (98.8 cases per 100,000 population). Enhanced surveillance for IPD in 2003 was carried out in all states and territories, providing additional data on 1,842 (85%) of all noti ed cases. Rates of IPD in Indigenous Australians were three times the rate in non-Indigenous Australians. There were 125 deaths attributed to IPD resulting in an overall case fatality rate of 6.8 per cent. Seventy-one percent of all pneumococcal isolates serotyped were serotypes in the seven-valent conjugate vaccine and 91 per cent were serotypes in the 23-valent polysaccharide pneumococcal vaccine. The clinical presentation and risk factors for IPD varied between Indigenous and non-Indigenous cases and non-vaccine serotypes occurred more frequently among Indigenous children and adults. Data from three years of surveillance indicate an early impact of the 7-valent vaccine in the target population. *Commun Dis Intell* 2004;28:441–454.**

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

## *Introduction*

Infection with *Streptococcus pneumoniae* is a leading cause worldwide of otitis media, pneumonia, bacteraemia, meningitis, responsible for significant morbidity and mortality in infants, the elderly and those with predisposing risk factors. Invasive pneumococcal disease (IPD) is the clinical condition in which *S. pneumoniae* infects a normally sterile site such as blood, cerebrospinal fluid or pleural fluid. IPD presents most commonly as pneumonia in adults and bacteraemia in children. The risk of disease is highest among people who are immunocompromised or have a chronic illness. IPD disease in Australia is generally a disease of the very young and the very old. The incidence of IPD in Indigenous Australians has been much higher than that of non-Indigenous Australians.

The escalating resistance of the pneumococcus to antibiotics has been an important factor for the development and introduction of new pneumococcal vaccines. In Australia the rate of penicillin resistant pneumococci increased from 1 per cent in 1984

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to 25 per cent in 1997.[1] Reduced susceptibilities to other antimicrobials has also emerged in recent years with the rate of reduced susceptibility to third generation cephalosporins in Australia reaching 13 per cent in 1997.[2] Multi-drug resistant pneumococci have been documented around the world and have been associated with outbreaks of meningitis in infants.[3] In 1999, 6.8 percent of invasive and 16.7 per cent of non-invasive pneumococcal isolates in Australia were multi-drug resistant.[4]

Ninety serotypes of *S. pneumoniae* identified by the polysaccharide composition of their capsule have been described. Immunity to pneumococcal infection is thought to be serotype specific. Vaccines containing pneumococcal polysaccharides from a varying number of serotypes have been available for many years, with a 23-valent polysaccharide vaccine (23vPPV) being used in Australia from 1986 (Table 1). Polysaccharide pneumococcal vaccines were shown to be poorly immunogenic in young children.[5] A vaccine in which polysaccharides from seven serotypes coupled to a protein carrier (mutated diphtheria toxoid) was developed to provide an effective pneumococcal vaccine for children and in a trial in the USA in infants aged 2 to 15 months of age demonstrated an efficacy of 93.9 per cent. [6] This conjugate vaccine (7vPCV) was licensed for use in Australia in January 2001 and a nationally funded vaccination program for children at high risk commenced in June 2001 (Table 1).

IPD was made a notifiable disease in all Australian states and territories in 2001 and surveillance data are reported to the National Notifiable Diseases Surveillance System (NNDSS). Additional enhanced surveillance data on IPD has also been collected since 2001 and annual reports have been published.[7,8] In this third report, an analysis of the influence of the 7-valent vaccine on IPD in vaccine eligible children has been performed with respect to overall rates of disease, disease caused by vaccine and non-vaccine serotypes and levels of antimicrobial resistance. Baseline data on IPD in all children and the elderly prior to the introduction of universal child and 65 year and older immunisation programs commencing in January 2005 are presented.

## *Methods and Materials*

### Case definition

A case of invasive pneumococcal disease was defined as the isolation from or the detection 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 NNDSS. Since this required changes to state and territory public health legislation, the data in 2001 was incomplete in some states and territories, but was complete for all jurisdictions from 2002.

NNDSS data in 2003 comprised core data, which is a set of data collected on all cases of all notifiable diseases as well as data specific for IPD. The list of the data fields collected the IPD enhanced data set is shown in Table 2.

### Clinical Presentation

Clinical presentations were coded as pneumonia, meningitis, bacteraemia, other or unknown. Pneumonia was defined as blood culture or nucleic

**Table 1. Recommendations for pneumococcal vaccination, Australia, 2003**

| 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 |
| Date implemented | 1998 | July 2001 |
| Target populations | All individuals aged 65 years and over<br>Individuals with asplenia<br>Immuncompromised patients<br>Aboriginal and Torres Strait Islander people aged 50 years and over<br>Immunocompetent individuals with chronic illness including chronic cardiac, renal or pulmonary disease, diabetes and alcohol-related problems | Tier 1: Indigenous children less than 5 years living in central Australia<br>Tier 2: Indigenous children aged less than 2 years particularly in rural and remote settings<br>Tier 3: Indigenous children under 2 years living in other settings<br>Non-Indigenous children less than 2 years living in Central Australia<br>Non-Indigenous children with conditions predisposing to pneumococcal infection |
| Data source | *NHMRC Immunisation Handbook* 7th edition, 2000 | ATAGI recommendations, 2001 |

**Table 2. Invasive pneumococcal disease surveillance data supplied by states and territories used in this report**

| Data type | Data fields |
|---|---|
| Demographic | Date of Birth |
| | Age |
| | Indigenous status: (Aboriginal, Torres Strait Islander, Aboriginal and Torres Strait Islander, Other, Unknown) |
| | Location (optional) |
| | Postcode |
| Risk factors | Premature birth (gestation less than 37 weeks) |
| | Congenital abnormality |
| | Anatomical or congenital asplenia |
| | Immunocompromised (e.g. HIV, lymphoma, transplant, multiple myeloma, nephrotic syndrome etc.) |
| | Chronic illness (e.g. cardiac disease, pulmonary disease, CSF leak, diabetes) |
| | Other risk factors (variable by State) including chronic suppurative otitis media, failure to thrive, previous IPD or pneumonia, excessive alcohol consumption, smoking or smoke exposure |
| Clinical data | Clinical presentation (pneumonia, meningitis, bacteraemia, other, unknown) |
| | Date of onset |
| | Death due to IPD |
| Microbiology data | Specimen collection date |
| | Date laboratory results issued (report date) |
| | Date notification received |
| | Specimen type (blood, CSF, pleural fluid, joint fluid, other sterile site) |
| | Specimen culture positive or *S. pneumoniae* detected by nucleic acid tests |
| | Antibiotic susceptibility (penicillin, cefotaxime/ceftriaxone) |
| | Pneumococcal serotype |
| Vaccination history | Source of vaccination history (validated, not validated, information not collected) |
| | Pneumococcal vaccination dates, number of doses and type of vaccine |
| | Vaccination status: fully vaccinated for age, partially vaccinated for age, not vaccinated, unknown |

acid test (NAT) positive for *S. pneumoniae* with clinical and/or radiological signs of pneumonia. Meningitis was defined as the detection of *S. pneumoniae* in the cerebrospinal fluid (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.

### Vaccination

The consensus definitions of vaccination status, vaccination confirmation and vaccine failure are shown in Table 3.

### Populations under surveillance

There were differences in populations under surveillance between jurisdictions in the collection of enhanced IPD data. The age groups on whom enhanced data was collected in 2003 are shown in Table 4.

NNDSS data for 2003 was analysed by date of disease onset while data in the enhanced data sets was analysed by date of notification.

### 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 June 30, 2003, was used as the denominator in rate calculations. Estimates of the Indigenous Australia population were based on projections from the 2001 census. The ABS calculated projections based on assump-

**Table 3. De nitions of vaccination status and vaccine failure used in this report**

| Category | Definition* |
|---|---|
| Fully vaccinated (child) | Those that have had the required doses for age of 7vPCV (or 23vPPV if age > 18 months) at least 2 weeks prior to infection. Children aged less than 7 months analysed on an individual basis |
| Fully vaccinated (adult) | Those that have had the required doses of 23vPPV at least 2 weeks prior to infection |
| Partially vaccinated (child) | Those that have received at least one dose, but not all the recommended doses of vaccine for age |
| Partially vaccinated (adult) | Those that have been vaccinated with 23vPPV but the time frame is outside the recommended schedule |
| Not vaccinated (child or adult) | Those that have never received a pneumococcal vaccine |
| Vaccination confirmation | Written confirmation through Australian Childhood Immunisation Register, State or Territory Immunisation register or health record |
| Vaccine failure | A fully vaccinated person (as per the above criteria) with disease due to a serotype found in the corresponding vaccine |

* According to the *Australian Immunisation Handbook*, 8th edition 2003.

**Table 4. Enhanced Invasive pneumococcal disease surveillance data collection by states and territories in 2003**

| Age group | Jurisdictions |
|---|---|
| Under 5 years | Australian Capital Territory (except vaccination status and risk factors) |
| | Queensland |
| | Victoria (except risk factor information) |
| | South Australia (except risk factor information) |
| | New South Wales (Cases in rural Public Health Units (PHU) and South Western Sydney, Hunter, Illawara, Greater Western Sydney, Wentworth and Southeastern Sydney PHUs) |
| Over 50 years | Victoria (Indigenous cases and vaccine failures only) |
| | South Australia (Vaccine failures only) |
| | New South Wales (Cases in Rural Public Health Units (PHU) and South Western Sydney, Hunter, Illawara, Greater Western Sydney PHUs) |
| All ages | North Queensland |
| | Northern Territory |
| | Western Australia |
| | Tasmania |

tions 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 proportions was calculated using the Chi-square test with Yates correction using Epi-Info 6.

## *Results*

### Notifications to NNDSS

There were 2,174 notifications of IPD to the NNDSS in 2003. The numbers of notification and the notification rate per 100,000 population are shown in Table 5.

The rates of IPD disease ranged between 7.7 and 12.4 per 100,000 except in the Northern Territory where the rate was 36.3 per 100,000. When the notification rates of IPD were examined by geographical distribution, variation within states was evident (Map).

The frequency of cases varied by season with 816 (38%) reported in winter months (June to August). The effect of season was more marked in cases aged five years or more than in younger children (Figure 1).

As previously noted, IPD in Australia is largely a disease of the very young and very old. The highest rates of disease were in children aged less than five years and adults aged more than 85 years (Figure 2). Among children aged less than five years, the highest rates were recorded in children aged one year (119 per 100,000 population). There were 488 cases in children aged less than two years of age (98.8 per 100,000) in 2003. In all age groups there were more male than female cases (overall male to female ratio 1.3:1).

**Table 5. Noti cations and noti cation rate per 100,000 population, Invasive pneumococcal disease, Australia, 2003***

| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Australia |
|---|---|---|---|---|---|---|---|---|---|
| Notifications | 40 | 784 | 72 | 466 | 176 | 43 | 443 | 150 | 2,174 |
| Rate per 100,000 population | 12.4 | 11.7 | 36.3 | 12.3 | 11.5 | 9.0 | 9.0 | 7.7 | 10.9 |

* By date of disease onset.

**Map. Noti cation rates of invasive pneumococcal disease, Australia, 2003 by statistical division of residence**

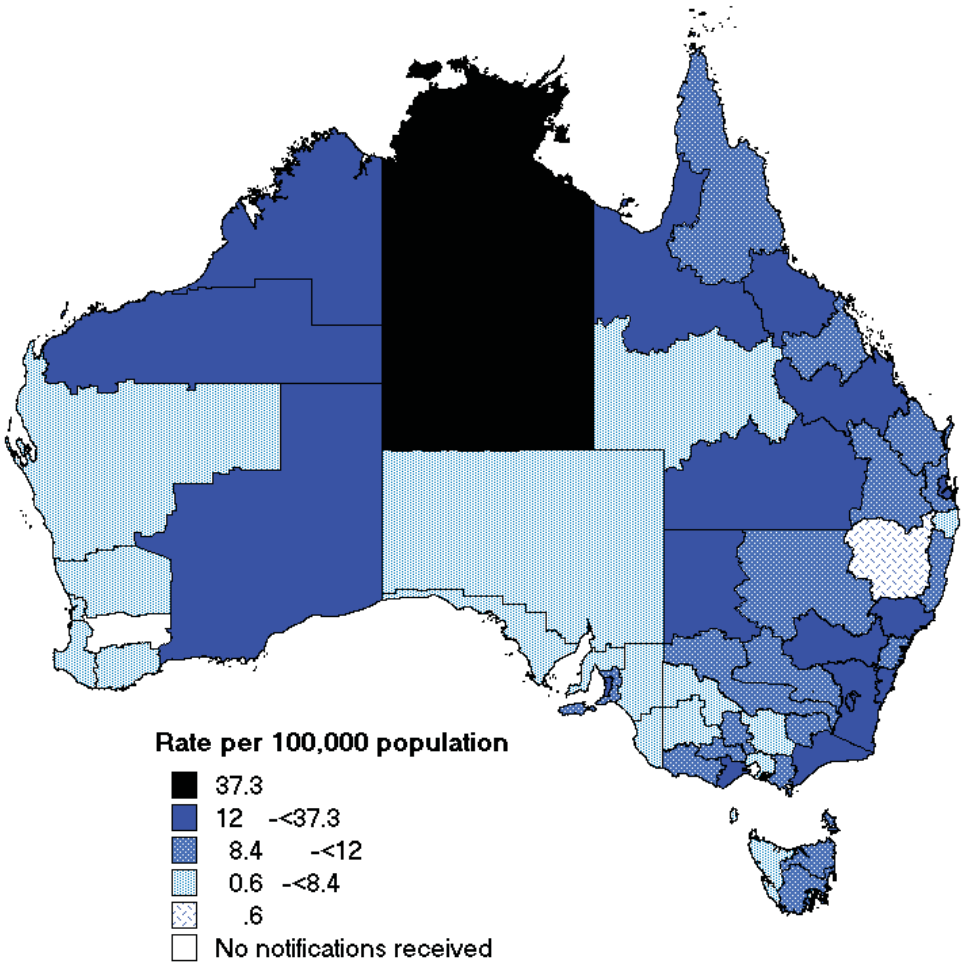



**Figure 1. Noti cations of invasive pneumococcal disease, by month of report and age group, Australia, 2003**

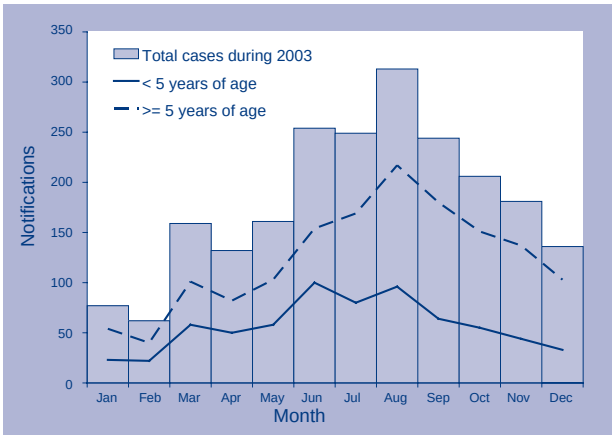
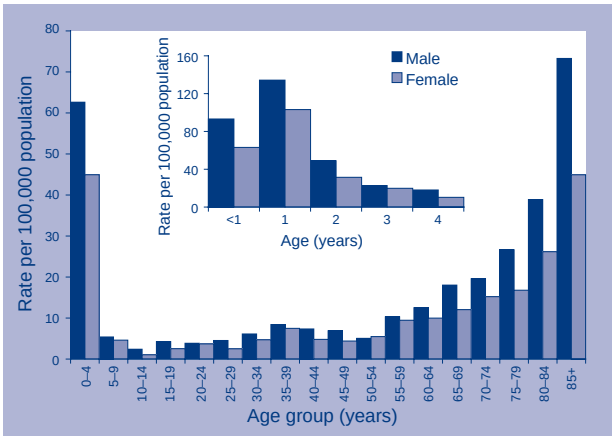



**Figure 2. Noti cation rates of invasive pneumococcal disease, by age and sex, Australia, 2003**



### Enhanced IPD surveillance data

Enhanced data were available for 1,842 cases or 85 per cent of notified cases—a similar proportion of cases to that reported on in the 2002 annual report.

*Demographics*

The demographic characteristics of cases on which enhanced data were collected are shown in Table 6.

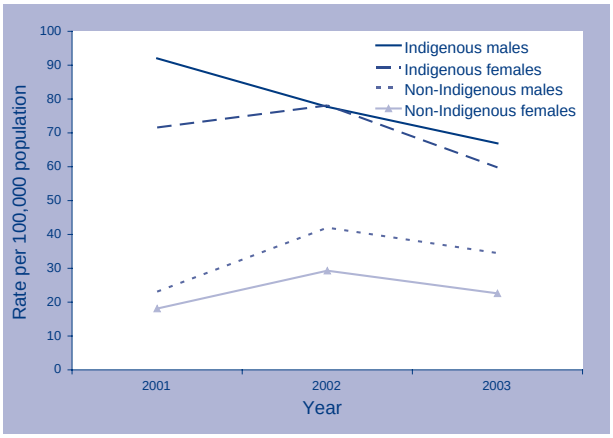
The enhanced data identified 149 cases of IPD among Indigenous people, which represented eight per cent of all cases, a similar proportion to that in 2002. This represented a national rate of 33.5 per 100,000 in Indigenous people compared with the national rate of 10.7 per 100,000. The rates of IPD in Indigenous people were highest in the Northern Territory (87.7 per 100,000) and Queensland (42.6 per 100,000). The national rate of IPD in Indigenous people is likely to be underestimated as incomplete reporting of Indigenous status continues to be a problem in some jurisdictions. Rates in Indigenous children under five years have fallen from 92 to 67 per 100,000 in males and 72 to 60 per 100,000 in females, between 2001 and 2003 respectively, but these represent small declines in total numbers (Figure 3).

*Clinical presentation*

The clinical presentation was reported in 69.8 per cent (1,286/1,842) of cases with enhanced surveillance information (Table 7).

Pneumonia was the most common clinical presentation (662 cases, 3.3 per 100,000 population) followed by bacteraemia (592 cases, 2.9 per 100,000 population) and meningitis (109 cases, 0.5 per 100,000 population). Other clinical presentations of IPD accounted for 45 cases. These clinical presentation rates were similar to those reported in 2002.

**Figure 3. Noti cation rates of invasive pneumococcal disease, Australia 2001 to 2003 in children under 5 years of age, by Indigenous status**




Clinical presentation varied by age with pneumonia being the most common presentation among cases over 65 years (72%) and bacteraemia the most common presentation among cases in children under five years (68%).

The proportion of IPD cases presenting as pneumonia was significantly higher in Indigenous children (37%) compared with non-Indigenous children (22%). There were no significant differences between Indigenous and non-Indigenous children in the proportions of other clinical presentations (Table 8).

**Table 6. Demographic pro le of Invasive pneumococcal disease cases reported by enhanced surveillance systems, by jurisdiction, Australia, 2003***

| Data | | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Total |
|---|---|---|---|---|---|---|---|---|---|---|
| Number | | 44 | 428 | 72 | 464 | 176 | 43 | 465 | 150 | 1,842 |
| Sex | Male:Female ratio | 1.2:1 | 1.3:1 | 2:1 | 1.3:1 | 1.4:1 | 1.9:1 | 1.4:1 | 1.3:1 | 1.3:1 |
| Age | <5 years | 11 | 192 | 20 | 163 | 73 | 4 | 131 | 43 | 637 |
| | 5 to 64 years | 20 | 69 | 49 | 200 | 50 | 27 | 181 | 68 | 664 |
| | ≥65 years | 13 | 167 | 3 | 101 | 53 | 12 | 153 | 39 | 541 |
| Indigenous status | Indigenous | 0 (0%) | 13 (3%) | 51 (71%) | 53 (11%) | 3 (2%) | 0 (0%) | 7 (2%) | 22 (15%) | 149 (8%) |
| | Non-indigenous | 19 (43%) | 403 (94%) | 21 (29%) | 305 (66%) | 163 (93%) | 39 (91%) | 288 (62%) | 128 (85%) | 1366 (74%) |
| | Unknown | 25 (57%) | 12 (3%) | 0 | 106 (23%) | 10 (5%) | 4 (9%) | 170 (36%) | 0 | 327 (18%) |

* See Table 4 for details of populations under enhanced surveillance in different jurisdictions in 2003.

**Table 7. Clinical presentations of Invasive pneumococcal disease, by jurisdiction, Australia, 2003**

| Clinical presentation* | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Total |
|---|---|---|---|---|---|---|---|---|---|
| Pneumonia (%) | 0 | 208 (48%) | 46 (64%) | 78 (17%) | 48 (27%) | 32 (74%) | 146 (31%) | 104 (69%) | 662 (36%) |
| Meningitis (%) | 1 (2%) | 27 (6%) | 6 (8%) | 19 (4%) | 8 (5%) | 5 (12%) | 32 (7%) | 11 (7%) | 109 (6%) |
| Bacteraemia (%) | 36 (82%) | 180 (42%) | 19 (26%) | 145 (31%) | 87 (49%) | 6 (14%) | 70 (15%) | 49 (32%) | 592 (32%) |
| Other (%) | 3 (7%) | 8 (2%) | 1 (1%) | 10 (2%) | 5 (3%) | 0 | 5 (1%) | 13 (9%) | 45 (2%) |
| Unknown (%) | 4 (9%) | 5 (1%) | 0 | 252 (54%) | 40 (23%) | 0 | 215 (46%) | 0 | 516 (28%) |

* Totals may exceed case total and percentages exceed 100 per cent since cases may have had more than one type of clinical presentation in some jurisdictions.

**Table 8. Clinical presentations of Invasive pneumococcal disease in Indigenous and non-Indigenous children aged less than 5 years, Australia, 2003**

| | Number of cases (%) | | |
|---|---|---|---|
| | Indigenous n=45 | Non-Indigenous n=516 | Significance of difference* |
| Pneumonia | 17 (37%) | 112 (22%) | p<0.05 |
| Meningitis | 8 (17%) | 54 (10%) | NS |
| Bacteraemia | 24 (53%) | 329 (64%) | NS |
| Other | 0 | 20 (4%) | – |

* Chi-square test with Yates correction.

NS = not significant.

IPD resulted in 125 deaths in Australia in 2003, a case fatality rate of 6.8 per cent (Table 9). The case fatality rate was significantly higher in cases aged more than 65 years (16.6%) compared with children aged less than five years (1.9%, p<0.001). The case fatality rate was not significantly different in Indigenous cases (4.7%) and non-Indigenous cases (6.9%). There were seven deaths in children aged less than two years of age.

### Risk factors for pneumococcal disease

The national surveillance working group defined risk factor categories for IPD. Other risk factors were recorded but varied between jurisdictions. More than one risk factor could be recorded for each case. Recognised risk factors for pneumococcal disease were reported in 640 cases. The most common of these was chronic illness, which included chronic respiratory, cardiac and renal disease. Immunocompromising conditions such as long-term immunosuppressant use were common among IPD cases.

The frequency of risk factors for IPD in Indigenous and non-Indigenous people are shown in Table 10. The rates of chronic illness were significantly higher in Indigenous children aged less than five years with IPD compared with non-Indigenous children in the same age group. Among cases aged five years or more, the proportion of immunocompromised cases was significantly higher among non-Indigenous cases than Indigenous cases.

### Pneumococcal serotypes causing disease in Australia

Pneumococcal serotypes were identified in 86 per cent (1,583/1,842) of the cases under enhanced surveillance in 2003. Of these, 71 per cent (1,129/1,583) of serotypes contained in the 7-valent conjugate pneumococcal vaccine and 91 per cent (1,444/1,583) were serotypes contained in the 23-valent polysaccharide pneumococcal vaccine (Table 11).

The frequency of vaccine serotypes in the conjugate and polysaccharide was further analysed in the target age groups for these vaccines and

**Table 9. Case fatality rates for Invasive pneumococcal disease, by jurisdiction, Australia, 2003**

| Data | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Total |
|---|---|---|---|---|---|---|---|---|---|
| Cases | 44 | 428 | 72 | 464 | 176 | 43 | 465 | 150 | 1,842 |
| Deaths | 4 | 63 | 4 | 8 | 9 | 6 | 22 | 9 | 125 |
| Case fatality rate (%) | 9% | 14.7% | 5.5% | 1.7% | 5.1% | 13.9% | 4.7% | 6% | 6.8% |
| Deaths in Aged < 5y /total cases aged <5y (%) | 1/11 9% | 4/192 (2.1%) | 2/20 (10%) | 1/163 (0.6%) | 1/73 (1.4%) | 0/4 (0%) | 2/131 (1.5%) | 1/43 (2.3%) | 12/ 637 (1.9%) |
| Deaths in Aged >65y /total cases aged >65y (%) | 2/13 15% | 54/167 (32.3%) | 0/3 (0%) | 5/101 (5%) | 6/53 (11.3%) | 3/12 (25%) | 15/153 (9.8%) | 5/39 (12.8%) | 90/541 (16.6%) |
| Deaths in Indigenous people/total Indigenous cases (%) | 0/0 (0%) | 2/13 (15.4%) | 2/51 (3.9%) | 2/53 (3.7%) | 0/3 (0%) | 0/0 (0%) | 1/7 (14.3%) | 0/22 (0%) | 7/149 (4.7%) |
| Death in non-Indigenous /total non-Indigenous + 'unknown' cases (%) | 4/44 (9%) | 61/415 (14.7%) | 2/21 (9.5%) | 6/411 (1.4%) | 9/173 (5.2%) | 6/43 (13.9%) | 21/458 (4.6%) | 9/128 (7%) | 118/1693 (6.9%) |

**Table 10. The frequency of risk factors for Invasive pneumococcal disease, by age group and Indigenous status, Australia, 2003**

| | Cases aged less than 5 years | | | Cases aged 5 years or over | | |
|---|---|---|---|---|---|---|
| | Indigenous (n=19) | Non Indigenous (n=93) | Significance of difference* | Indigenous (n=76) | Non-Indigenous (n=452) | Significance of difference* |
| Premature birth | 2 (11%) | 28 (30%) | NS | – | – | – |
| Congenital abnormality | 3 (16%) | 11 (12%) | NS | – | – | – |
| Asplenia | 0 | 1 (1.1%) | – | – | 7 (1.5%) | – |
| Immuno-compromised | 2 (11%) | 8 (8.6%) | NS | 7 (9%) | 98 (22%) | p<0.05 |
| Chronic illness | 9 (47%) | 15 (16%) | p<0.01 | 53 (70%) | 275(61%) | NS |

* Chi-square test with Yates correction.

NS = not significant.

**Table 11. Proportion of pneumococcal serotypes in cases of Invasive pneumococcal disease, covered by the 7-valent and 23-valent pneumococcal vaccines* by jurisdiction, Australia, 2003**

| Vaccine | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Total |
|---|---|---|---|---|---|---|---|---|---|
| 7v | 23/40 58% | 256/339 76% | 18/69 26% | 298/410 73% | 118/153 77% | 25/35 71% | 298/395 75% | 93/142 65% | 1,129/1,583 71% |
| 23v | 36/40 90% | 320/339 94% | 51/69 74% | 374/410 91% | 137/153 89% | 31/35 88% | 371/395 94% | 124/142 87% | 1,444/1,583 91% |

* As a proportion of serotyped isolates.

by Indigenous status (Table 12). The proportion of 7-valent conjugate vaccine serotypes was significantly lower in Indigenous children aged less than two years (34.6%) than in non-Indigenous children (88.3%, p<0.001). Similarly the proportion of 23-valent polysaccharide vaccine serotypes in Indigenous cases aged two years and above was significantly lower (69%) than in non-Indigenous cases (91%, p<0.001).

Trends in the numbers of 7-valent vaccine and non-7-valent vaccine serotypes in Indigenous and non-Indigenous cases aged less than two years between 2001 and 2003 are shown in Figure 4A and 4B.

The decline in IPD due to 7-valent serotypes in Indigenous children under two years was largely seen in the Northern Territory, Queensland and Western Australia (Table 13), the jurisdictions with the largest proportion of Indigenous people.

**Table 12. The proportion of pneumococcal serotypes isolated from cases of invasive pneumococcal disease, which were serotypes in the 7-valent and 23-valent pneumococcal vaccine, by age and Indigenous status, Australia, 2003**

| | Number (%) serotypes in pneumococcal vaccines | | | | | |
|---|---|---|---|---|---|---|
| | Cases aged less than 2 years with serotypes in 7-valent conjugate vaccine | | | Cases aged 2 years or more with serotypes in 23-valent vaccine | | |
| | Indigenous | Non-Indigenous | Significance of difference* | Indigenous | Non-Indigenous | Significance of difference* |
| ACT | – | 3/7 (42%) | – | – | 30/33 (90%) | – |
| NSW | 3/3 (100%) | 96/107 (89.7%) | NS | 5/6 (83%) | 203/224 (90.6%) | NS |
| NT | 0/11 (0%) | 4/4( 100%) | p<0.001 | 26/38 (68%) | 14/16 (87.5%) | NS |
| Qld | 1/7(14%) | 91/102 (89%) | p<0.001 | 32/44 (73%) | 235/257 (91.4%) | p<0.005 |
| SA | – | 41/44 (93.2%) | – | 0/1 (0%) | 95/108 (87.9%) | p=0.07 |
| Tas | – | 1/1 (100%) | – | – | 27/34 (79%) | – |
| Vic | 3/3 (100%) | 63/75 (85%) | NS | 2/4 (50%) | 294/313 (94%) | p<0.005 |
| WA | 2/2 (100%) | 19/21 (90%) | NS | 12/19 (63%) | 90/100 (90%) | p<0.01 |
| Australia | 9/26 (34.6%) | 318/361 (88.3%) | p<0.001 | 77/112 (69%) | 988/1,085 (91.3%) | p<0.001 |

* Differences tested by Chi square test with Yates correction.

NS = not significant.

**Figure 4A. Number of 7-valent vaccine and non-7-valent vaccine serotypes causing cases of invasive pneumococcal disease in Indigenous children aged less than 2 years, Australia 2001 to 2003**

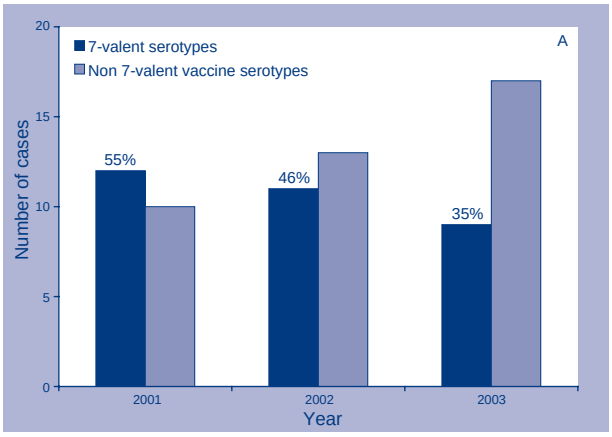
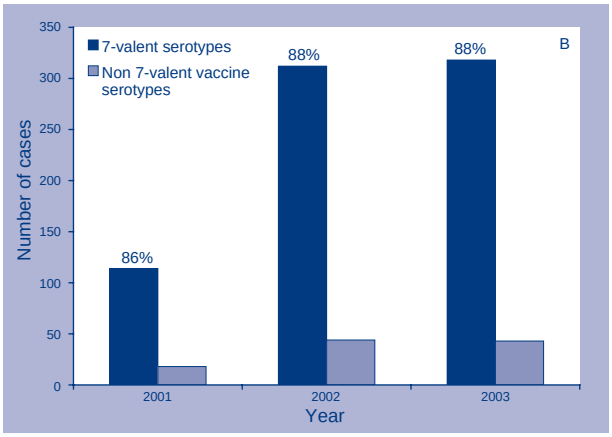



**Figure 4B. Number of 7-valent vaccine and non-7-valent vaccine serotypes causing cases of invasive pneumococcal disease in non-Indigenous children aged less than 2 years, Australia 2001 to 2003**



### Table 13. Pneumococcal serotypes in Indigenous children aged less than two years, 2001 to 2003, Western Australia, Queensland and Northern Territory

#### A. 7 valent vaccine serotypes

| | Northern Territory | Queensland | Western Australia | Total |
|---|---|---|---|---|
| 2001 | 8 | 9 | 2 | 19 |
| 2002 | 4 | 4 | 1 | 9 |
| 2003 | 0 | 1 | 2 | 3 |

#### B. non-7 valent vaccine serotypes

| | Northern Territory | Queensland | Western Australia | Total |
|---|---|---|---|---|
| 2001 | 4 | 0 | 6 | 10 |
| 2002 | 3 | 6 | 4 | 13 |
| 2003 | 11 | 8 | 0 | 19 |

## Vaccination status of Invasive pneumococcal disease cases

Data on pneumococcal vaccination were available for 58 per cent of the cases in 2003. Of the 1,082 cases with a vaccination history, the majority (758, 70%) was reported as unvaccinated. IPD was reported in 12 cases who had received vaccination with the 7-valent conjugate vaccine and in 161 cases who had received the 23-valent polysaccharide pneumococcal vaccine (Table 14).

Further investigations were made of the 12 cases of IPD presumptively vaccinated with 7-valent conjugate vaccine. Of the six cases in the Northern Territory, five were infected with pneumococcal serotypes not in the 7-valent vaccine and one case had no serotype information. Similarly four cases in Queensland were infected with non-vaccine serotypes and one case had no serotype information. There was no serotype data in the South Australian case. Therefore there was no evidence for any vaccine failure with the 7-valent conjugate pneumococcal vaccine in Australia in 2003 for those fully vaccinated. (Table 15).

The majority of the 161 cases of IPD in recipients of the 23-valent vaccine, occurred in those with predisposing risk factors for IPD. Thirty-six (22%) of the serotypes causing disease in these patients were not in 23-valent vaccine. In total 131 cases were presumptive 23-valent vaccine failures (Table 16).

## Antibiotic resistance in pneumococcal cases

The antibiotic susceptibilities of 1,193 isolates to penicillin were tested in six jurisdictions and 719 isolates to ceftriaxone were tested in five jurisdictions (Table 17).

### Table 14. Vaccination status of Invasive pneumococcal disease cases (all serotypes), by age group and jurisdiction, Australia, 2003.

#### A. Invasive pneumococcal disease cases aged less than 2 years

| Vaccination status | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Total |
|---|---|---|---|---|---|---|---|---|---|
| Fully vaccinated for age | 0 | 0 | 6 | 5 | 1 | 0 | 0 | 0 | 12 |
| Partially vaccinated for age | 0 | 0 | 5 | 3 | 50 | 0 | 0 | 1 | 59 |
| Not vaccinated | 0 | 131 | 5 | 14 | 0 | 0 | 79 | 21 | 250 |
| Unknown | 7 | 4 | 0 | 101 | 0 | 1 | 10 | 4 | 127 |

#### B. Invasive pneumococcal disease cases aged 2 years or more

| Vaccination status | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Total |
|---|---|---|---|---|---|---|---|---|---|
| Fully vaccinated for age | 0 | 53 | 16 | 21 | 20 | 4 | 44 | 3 | 161 |
| Partially vaccinated for age | 0 | 1 | 5 | 6 | 0 | 0 | 3 | 1 | 16 |
| Not vaccinated | 1 | 207 | 34 | 44 | 57 | 30 | 128 | 74 | 575 |
| Unknown | 36 | 32 | 1 | 270 | 46 | 7 | 197 | 46 | 635 |

**Table 15. Details of the cases of invasive pneumococcal disease that occurred in those fully vaccinated for age with 7-valent conjugate pneumococcal vaccines, by jurisdiction, Australia, 2003**

| | NSW | NT | Qld | SA | Tas | Vic | WA | Total |
|---|---|---|---|---|---|---|---|---|
| Number | 0 | 6 | 5 | 1 | 0 | 0 | 0 | 12 |
| Age range (years) | – | 0–1y | 0–1y | 0–1y | – | – | – | 0–1 |
| Indigenous | – | 5 | 4 | 0 | – | – | – | 9 (65%) |
| Risk factors present | – | 5/6 | 1/5 | – | – | – | – | 6 (43%) |
| 7-valent vaccination confirmed | – | 6/6 | 5/5 | 0/1 | – | – | – | 11 (78%) |
| Serotypes in 7-valent vaccine/ number with known serotype | – | 0/5 | 0/4 | 0/0 | – | – | – | 7/12 (58%) |
| Number of identified vaccine failures | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

**Table 16. Details of the cases of invasive pneumococcal disease that occurred in those fully vaccinated for age with 23-valent pneumococcal vaccines, by jurisdiction, Australia, 2003**

| | NSW | NT | Qld | SA | Tas | Vic | WA | Total |
|---|---|---|---|---|---|---|---|---|
| Number | 53 | 16 | 21 | 20 | 4 | 44 | 3 | 161 |
| Age range (years) | 51–91 | 21–57 | 2–80 | 44–90 | 21–88 | 46–90 | 12–48 | 2–91 |
| Indigenous | 1 | 16 | 15 | 1 | 0 | 0 | 2 | 35 |
| Risk factors present | 51 | 16 | 12 | 11 | 4 | 43 | 1 | 138 |
| 23-valent vaccination confirmed?* | 53 | 16 | 21 | 0 | 0 | 48 | 1 | 135 |
| Serotypes in 23-valent vaccine/ number with known serotype | 37/43 | 8/16 | 12/21 | 11/20 | 4/5 | 37/43 | 1/3 | 110/151 |
| Number of identified vaccine failures | 37 | 8 | 12 | 11 | 4 | 37 | 0 | 109 |

**Table 17. *S. pneumoniae* susceptibility to penicillin and ceftriaxone, New South Wales, Northern Territory, South Australia, Queensland, Tasmania and Western Australia, by jurisdiction**

| Antibiotic | Susceptibility | NSW | NT | Qld | SA | Tas | WA | Total |
|---|---|---|---|---|---|---|---|---|
| Penicillin | Resistant | 14 | 0 | 18 | 0 | 0 | 1 | 33 |
| | Intermediate | 29 | 7 | 45 | 7 | 1 | 20 | 109 |
| | Susceptible | 346 | 64 | 384 | 101 | 42 | 114 | 1,051 |
| | Total tested | 389 | 71 | 447 | 108 | 43 | 135 | 1,193 |
| | % reduced susceptibility | 11% | 9.8% | 14.1% | 6.4% | 2.3% | 15.5% | 11.9% |
| Ceftriaxone | Resistant | NT | 0 | 3 | 0 | 0 | 0 | 3 |
| | Intermediate | NT | 0 | 5 | 0 | 0 | 1 | 6 |
| | Susceptible | NT | 61 | 439 | 34 | 43 | 133 | 710 |
| | Total tested | NT | 61 | 447 | 34 | 43 | 134 | 719 |
| | % reduced susceptibility | – | 0% | 1.8% | 0% | 0% | 0.7% | 1.3% |

NT = not tested.

Trends in antibiotic susceptibility were examined in three years combined data from the Northern Territory, South Australia, Tasmania and Western Australia, 2001 to 2003 (Table 18). There were decreases in the number of non-susceptible isolates in the Northern Territory and South Australia, while the proportion of non-susceptible isolates remained almost constant in Tasmania and Western Australia.

There was a decline in penicillin non-susceptible strains in children under five years of age over the three years and a significantly lower proportion of isolates with reduced susceptibility to penicillin compared with isolates from older cases in 2003 (Table 18). In all three years, the rates of penicillin non-susceptible strains were higher in Non-Indigenous cases compared with Indigenous cases, but these differences did not reach statistical significance. There was a small decline in the proportion of penicillin resistant isolates that were in the 7-valent vaccine (92 % in 2001 to 84% in 2003) but the change in proportion was not statistically significant.

Susceptibility to ceftriaxone was less frequently measured. The overall proportion of non-susceptible strains fell from 5.1 per cent to 0.3 per cent over the three years. This decline was seen in non-Indigenous cases and in cases aged five years and older where the changes in proportion of ceftriaxone-non-susceptible strains between 2001 and 2003 were statistically significant. There was

**Table 18. Characteristics of patients with non-susceptible pneumococcal isolates, Northern Territory, South Australia, Tasmania and Western Australia combined, 2001 to 2003**

| **Penicillin** | **2001** | **2002** | **2003** |
|---|---|---|---|
| Total number of cases with reduced susceptibility | 55 | 41 | 36 |
| Total cases tested | 464 | 539 | 357 |
| Percent non-susceptible isolates | 11.8% | 7.6% | 10.1% |
| Proportion of cases under 5 years with reduced susceptibility | 15/182<br>8.2% | 13/191<br>6.8% | 4/108<br>3.7% |
| Proportion of cases 5 years and older with reduced susceptibility | 40/282<br>14.2% | 28/348<br>8.0% | 32/245<br>13.1%* |
| Proportion of Indigenous cases with reduced susceptibility | 7/67<br>10.4% | 6/ 82<br>7.3% | 6/69<br>8.7% |
| Proportion of non-Indigenous cases with reduced susceptibility | 48/362<br>13.3% | 35/457<br>7.6% | 30/284<br>10.6% |
| Proportion of cases with serotypes in 7-valent vaccine | 49/53<br>92% | 31/35<br>88% | 30/32<br>94% |
| Proportion of cases in 23-valent vaccine | 53/53<br>100% | 35/35<br>100% | 34/35<br>97% |
| Proportion of cases with reduced susceptibility vaccinated | 2/55<br>3.6% | 2/41<br>4.8% | 3/36<br>8% |
| **Ceftriaxone** | | | |
| Total number of cases with reduced susceptibility | 17 | 9 | 1 |
| Total cases tested | 332 | 372 | 272 |
| Percent non-susceptible isolates | 5.1% | 2.4% | 0.3%** |
| Proportion of cases under 5 years with reduced susceptibility | 7/140<br>5% | 4/191<br>2.1% | 0/108<br>0% |
| Proportion of cases 5 years and older with reduced susceptibility | 10/192<br>5.2% | 5/181<br>2.8% | 1/164†<br>0.6% |
| Proportion of Indigenous cases with reduced susceptibility | 3/43<br>7% | 2/82<br>2.4% | 0/69<br>0% |
| Proportion of non-Indigenous cases with reduced susceptibility | 14/289<br>4.8% | 7/290<br>2.4% | 1/203†<br>0.5% |
| Proportion of cases with serotypes in 7-valent vaccine | 17/17<br>100% | 8/8<br>100% | 0<br>0% |
| Proportion of cases in 23-valent vaccine | 17/17<br>100% | 8/8<br>100% | 1<br>100% |
| Proportion of all cases with reduced susceptibility vaccinated | 2/17? | 0/9? | 0/1? |

* Significant difference in proportions, between under 5 and ≥5 years in 2003 $p<0.05$.

† Significant change in proportions 2001 to 2003: ** $p<0.01$ † $p<0.05$.

only a single isolate of ceftriaxone non-susceptible pneumococci in 2003, which was a serogroup 1 from Western Australia in a non-Indigenous case (Table 18).

## *Discussion*

Surveillance data in 2003 suggests a moderate impact of the 7vPCV vaccine on the incidence of IPD since the introduction of the vaccine program in Indigenous children in mid-2001. Evidence for this includes a decrease in the notification rates in Indigenous children under five years, (from 92 to 67 per 100,000 in males and 72 to 60 per 100,000 in females) and a decrease in the rate of disease caused by vaccine serotypes in Indigenous children (from 55% to 34%). A more marked effect on pneumococcal disease will be seen in Australia when a government-funded universal childhood pneumococcal vaccination program commences in January 2005.[9] Continued surveillance to assess whether non 7-valent vaccine serotypes will increase is supported by a suggestion of increase in the northern jurisdiction's Indigenous less than 2 year old populations. Additionally, Whitney *et al* have observed a decreasing incidence of IPD in older adults possibly via herd immunity following universal childhood 7vPCV immunisation in the USA.[10] The impact of the upcoming Australian childhood 7vPCV universal program might also provide herd immunity on other age groups—and therefore enhanced surveillance of all ages is strongly supported.

In 2005, free 23vPPV vaccine will be made available to all Australians aged 65 years and over. Vaccine 'failure' in recipients of the polysaccharide vaccine was noted in this and the two previous IPD surveillance reports.[7,8] There is a need for more data on apparent vaccine failure in the vaccinated elderly to inform re-vaccination schedules. The effectiveness of the polysaccharide vaccine in preventing invasive disease has been estimated at 53 per cent, implying that 20,000 vaccinations are needed to prevent one infection.[11,12] A universal vaccination program for elderly Australians should provide the vaccine coverage required to reduce the incidence of invasive disease in the elderly, which has not declined in the last three years. The need to provide re-vaccination for the elderly at high risk of IPD requires continual evaluation as there is limited data on the precise timing and effectiveness of re-vaccination. The 8th edition of the Australian Immunisation Handbook recommends re-vaccination in non-Indigenous adults aged less than 65 years with risk factors at 65 years or 10 years after the first dose, whichever comes later. Indigenous adults 15 to 49 years with risk factors* should be re-vaccinated five years after the first dose and again at age 50 or 10 years after the first re-vaccination, whichever comes later. Indigenous adults without risk factors should be re-vaccinated five years after initial vaccination.[9]

Ascertainment of IPD cases is necessary for effective surveillance. In Victoria a capture-recapture study in 2004 found, in addition to their 465 notified cases under enhanced surveillance there were 24 non-notified cases of IPD in 2003, largely from hospitals (M. Counahan, personal communication). This failure to notify public health authorities has been observed in the past in Australia [13] and recently in the United Kingdom.[14] Under reporting of cases may also result from changes in surveillance practice and should be taken into account when interpreting the data presented in this report.

Declines in the number of pneumococcal isolates with reduced susceptibility to penicillin and ceftriaxone between 2001 and 2003 in the Northern Territory and among Indigenous cases is important in view of the antibiotic resistance developing during the past 20 years. Reduction of transmission of resistant strains through immunisation of children and lower levels of disease and therefore lower antibiotic use should reduce antibiotic resistance in pneumococcal disease. Continuing high levels of resistance among non-invasive isolates, the lower vaccine efficacy against otitis media and the potential for increased non-vaccine serotype disease make the impact of vaccination on antibiotic resistance uncertain. However it is useful to note the reduction over the past three years in those five years and older with reduced susceptibility for both penicillin and ceftriaxone and the overall proportion of non-Indigenous cases with the reduced susceptibility to ceftriaxone where the majority of these groups are not receiving the 7-valent vaccine. Some of this decrease may relate to other strategies to reduce drug resistance.

As vaccination becomes more widely implemented, concern has been expressed about the incidence of non-vaccine serotype disease increasing. While a trend in this direction may be suggested by 2002–2003 data of the under two year old vaccine eligible Indigenous children it is important that the serotypes continue to be reported in order to ascertain whether this trend will continue reflects the natural fluctuations over time. Therefore, while the overall rates of IPD continue to fall this 'replacement phenomenon' may not pose any threat to disease control, but on-going surveillance and serotyping of all invasive isolates along with anti-microbial resistance patterns is essential.

* The Northern Territory recommend 23vPPV for all Indigenous people 15 and over.

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# Laboratory Surveillance of Invasive Pneumococcal Disease in Australia, 2003 — predicting the future impact of the universal childhood conjugate vaccine program

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## Abstract

**A comprehensive invasive pneumococcal disease (IPD) laboratory surveillance program was carried out in Australia in 2003. This program provided data on the prevalence of pneumococcal serotypes and antimicrobial resistance. There were 1,995 isolates tested with 34 per cent (683) from children aged less than five years and 27 per cent (535) from the elderly aged more than 65 years. One thousand eight hundred and sixty were isolates from blood, 79 from CSF and 56 from other sterile sites. In young children, 84 per cent of isolates were a serotype and 92 per cent a serogroup in the 7-valent pneumococcal conjugate vaccine (7vPCV). Of penicillin resistant isolates in children less than five years of age 85 per cent and 98 per cent were a serotype and serogroup in the 7vPCV respectively. When the universal 7vPCV vaccine program in young children is introduced in 2005, a proportion of cases of IPD should also be prevented in young adults (estimated reduction of 54 cases annually) and elderly Australians (an estimated reduction of 110 cases annually) as a result of improved herd immunity. Pneumococcal serotypes with higher rates of penicillin resistance (19F, 14 and 6B) were more prevalent in the elderly than in young children. In contrast, erythromycin resistance was more common in children less than five years of age (24%) compared to the elderly (15%). The predominant serotype with erythromycin resistance in Australia was serotype 14 and thus there is likely to be a major reduction in erythromycin resistance as a result of 7vPCV vaccination. Continued surveillance of pneumococcal serotype distribution and antibiotic susceptibility will be essential in order to identify serotype replacement by non-vaccine serotypes and to monitor the overall impact of current and future vaccine programs on invasive pneumococcal disease in Australia, not only in young children but also in other age groups.** ***Commun Dis Intell*** **2004;28:455–464.**

*Keywords: invasive pneumococcal disease, vaccination, surveillance*

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## Introduction

*Streptococcus pneumoniae* is a major cause of morbidity and mortality worldwide.[1–4] It is a common cause of life threatening invasive disease (e.g. bactaeremia and meningitis) as well as non invasive disease (e.g. otitis media). It is important that comprehensive laboratory surveillance of invasive pneumococcal disease (IPD) is undertaken to assess the success of universal childhood 7-valent conjugate pneumococcal (7vPCV) vaccination which will be implemented in Australia in 2005. Laboratory surveillance of IPD has been conducted in various Australian states and territories prior to 2002.[5–9] This report summarises the results of laboratory surveillance for all Australian jurisdictions in 2003 and includes comprehensive pneumococcal serotyping of these isolates and antimicrobial resistance data.

Antimicrobial resistance in invasive pneumococci is an emerging problem in Australia.[10] Laboratory data on resistance to penicillin and erythromycin and the key serotypes responsible for antimicrobial resistance in each state and territory are reported. The potential benefits of the universal childhood immunisation program for adults are discussed.

## Methods and Materials

### Case definition

For the purposes of laboratory surveillance, a case of IPD was included when *Streptococcus pneumoniae* was isolated by culture from a normally sterile body site (blood, cerebrospinal fluid (CSF), joint fluid etc). Only one isolate was tested from each patient episode. A new episode was deemed to occur if an isolate was cultured more than 14 days after a previous positive culture.

### Data sources and collection

A network of laboratories in Australia (see list of participating laboratories) obtained pneumococcal isolates referred from all major private and public microbiology laboratories in Australia. Isolates were stored for later serotyping at one of the three designated pneumococcal typing laboratories. Indigenous status data was linked to laboratory data only in the Northern Territory in 2003 and detailed analysis by Indigenous status was not performed in this year's report in contrast to the 2002 report.[11] Enhanced data on IPD including information on pneumococcal serotypes in Indigenous people are collected as an extension of the National Notifiable Diseases Surveillance System (NNDSS) and the 2003 data are provided in the accompanying surveillance report.[12]

### Serotyping

Pneumococcal serotyping was performed at the Pneumococcal Reference Laboratory of Queensland Health Scientific Services (for Western Australia, Northern Territory and Queensland), the Children's Hospital at Westmead's NSW Pneumococcal Reference Laboratory (for New South Wales and the Australian Capital Territory) and the Microbiological Diagnostic Unit (for Victoria, Tasmania and South Australia). Serotyping was performed by the Quellung reaction using antisera from the Statens Seruminstitut, Copenhagen, Denmark.[13]

Analysis of serotypes included the prevalence of vaccine serotypes and vaccine serogroups (that is, pneumococci with serotypes within the same serogroups as vaccine types).[14] The pneumococcal serotypes in the three vaccines referred to in this paper are shown in Table 1.

### Susceptibility testing

Susceptibility testing was performed by a range of different methods. In New South Wales, Victoria, Tasmania, Australian Capital Territory and South Australia the available results were from routine diagnostic laboratories. These laboratories used National Committee for Clinical Laboratory Standards (NCCLS) disc diffusion,[15] Calibrated Dichotomous Susceptibility (CDS) disc diffusion[16] or agar dilution susceptibility testing methods. Most laboratories also confirmed penicillin resistance using the E test method.[17] Results from Queensland, Northern Territory and Western Australia were performed using NCCLS disc diffusion and E test methods in a reference laboratory.

Isolates were categorized as fully sensitive to penicillin or resistant (includes intermediate and high level resistance using NCCLS breakpoints (Minimum Inhibitory Concentration (MIC) ≥0.125mg/L). Erythromycin was categorized as either sensitive or resistant (MIC ≥1mg/L).

**Table 1. Pneumococcal vaccines and constituent serotypes referred to in this report**

| Vaccine | 7-valent conjugate vaccine (7vPCV) | 11-valent conjugate vaccine (11vPCV) | 23-valent polysaccharide vaccine (23vPPV) |
|---|---|---|---|
| Pneumococcal Serotypes | 4, 6B, 9V, 14, 18C, 19F, 23F | 1, 3, 4, 5, 6B, 7F, 9V, 14, 18C, 19F, 23F | 1, 2, 3, 4, 5, 6B, 7F, 8, 9N, 9V, 10A, 11A, 12F, 14, 15B, 17F, 18C, 19A, 19F, 20, 22F, 23F, 33F |

### Statistical analysis

Yates corrected Chi square test was used for univariate analysis using Epi info statistical software Version 6.02 (CDC, USA).

## *Results*

### Cases under laboratory surveillance

There were 1,998 pneumococcal isolates forwarded to the three pneumococcal reference laboratories for serotyping and 1,995 were successfully serotyped. This represents 92 per cent of the 2,174 notified cases of invasive pneumococcal disease in Australia in 2003.[12] The number of isolates by state and territory and specimen type is shown in Table 2.

**Figure 1. Pneumococcal isolates, Australia, 2003 by age and sex**

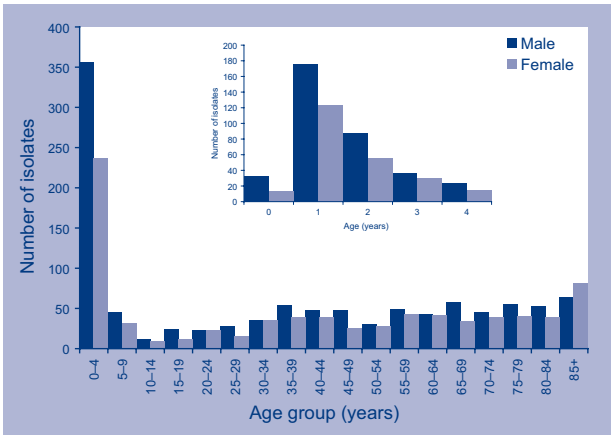



**Table 2. Pneumococcal isolates analysed in this report, by reporting jurisdiction and specimen type**

| | Blood | Cerebrospinal fluid | Other sites* | Total |
|---|---|---|---|---|
| ACT | 45 | 1 | 6 | 52 |
| NSW | 609 | 23 | 22 | 654 |
| NT | 66 | 2 | 1 | 69 |
| Qld | 412 | 24 | 5 | 441 |
| SA | 160 | 4 | 2 | 166 |
| Tas | 33 | 2 | 0 | 35 |
| Vic | 400 | 21 | 15 | 437 |
| WA | 135 | 2 | 5 | 142 |
| Australia | 1,860 | 79 | 56 | 1,995 |

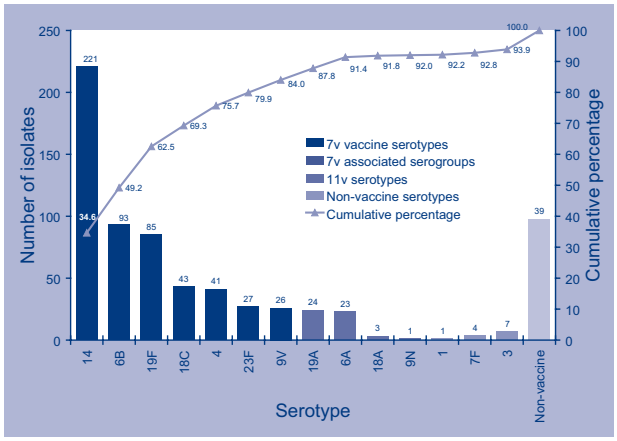
* Other sites includes joint, pleural, peritoneal and pericardial fluid.

The number of isolates by the age and sex of the patient is shown in Figure 1. There were more isolates from males than females (male to female ratio 1.3:1), which was the same sex ratio as seen in the notification data. The largest number of isolates were from children aged 1 year (Figure 1).

**Serotypes responsible for invasive pneumococcal disease in Australian children less than five years of age and proportion represented in conjugate vaccines.**

Six hundred and thirty-eight pneumococcal isolates from children less than five years of age were serotyped. The serotype distribution proportion of isolates from this age group represented in the 7vPCV and prototype 11vPCV conjugate pneumococcal vaccines are illustrated in Figure 2. Eighty-four percent of isolates were a serotype match for the 7vPCV vaccine and 92 per cent of isolates were a serogroup match. The future 11vPCV vaccine (addition of serotypes 1, 7F, 5 and 3) would add another 1.9 per cent of isolates belonging to vaccine serotypes.

**Figure 2. Serotypes responsible for invasive pneumococcal disease in children less than five years, Australia, 2003**



**Pneumococcal serotypes with reduced susceptibility to penicillin and erythromycin in Australian children less than five years of age**

Of the 638 isolates from children less than five years of age that were serotyped, 622 also had penicillin susceptibility results recorded. Overall, 71/622 (11%) had reduced susceptibility to penicillin. Sixty of these (85%) were serotypes and 70 (99%) were serogroups in the 7vPCV (Table 3).

Of the 638 isolates from children less than five years of age that were serotyped, 567 also had susceptibility results for erythromycin recorded. Overall, 138/567 (24%) were resistant to erythromycin. One hundred and thirty-four of the 138 erythromycin resistant isolates were a serotype match for the 7vPCV and the remaining four isolates were a serogroup match (Table 4). The majority (70%) of erythromycin resistant isolates in children less than five years of age in Australia were serotype 14.

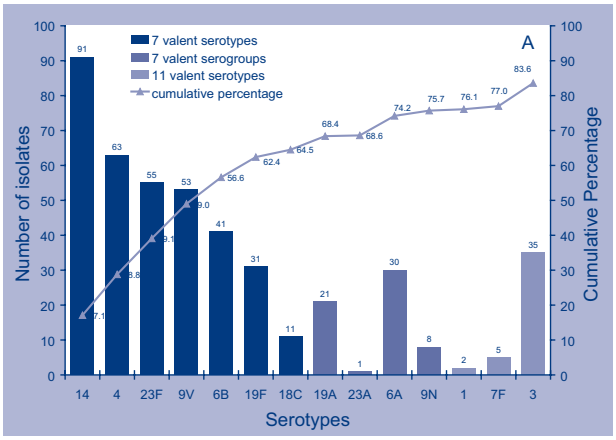
The rates of penicillin and erythromycin resistant pneumococci in children aged less than five years of age by state and territory are shown in Table 5. The overall rate of penicillin resistance and erythromycin resistance varied widely between states. There were no penicillin resistant isolates identified in the Northern Territory or Tasmania and rates ranged from five per cent in South Australia to 14 per cent in New South Wales. Erythromycin resistance was found in all states ranging from six per cent in the Northern Territory to 75 per cent in Tasmania, although the latter rate was based on only four isolates.

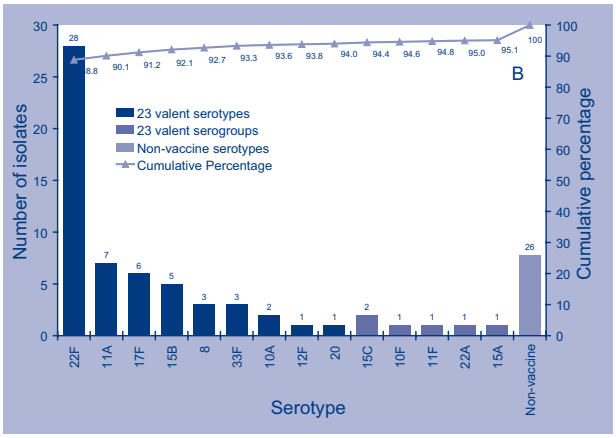
Differences in the prevalence of serotype 14—which accounted for 70 per cent of erythromycin resistance—largely reflected differences in erythromycin resistance rates between jurisdictions.

**Serotypes responsible for IPD in Australian adults over 65 years of age.**

Five hundred and thirty-five isolates from adults aged more than 65 years were analysed. Sixty-five percent of isolates were 7vPCV serotypes and 76 per cent 7vPCV serogroups (Figure 3, Panel A). 84 per cent of serotypes were in the 11vPCV conjugate vaccine and 94 per cent were serotypes in the 23vPPV polysaccharide vaccine (Panel B).

**Figure 3. Serotypes responsible for invasive pneumococcal disease in adults aged more than 65 years, Australia, 2003. Panel A serotypes in 7- and 11vPCV vaccines; Panel B Serotypes in the 23vPPV vaccine**




The likely impact (via herd immunity) on incidence of IPD in the adult population over 65 years of age following the introduction of the universal childhood immunisation program with the 7vPCV vaccine in Australia was assessed based on the reductions seen in the USA.[14] Based on the serotype distribution in the elderly in 2003 it was estimated that 110 cases of IPD would be prevented in adults over 65 years of age as a result of herd immunity associated with universal 7vPCV vaccination in Australian children (Table 6).

Further, based on the serotype prevalence in the 20 to 39 year age group (n=263), approximately 54 cases in this age group should be prevented by the 7vPCV vaccination of Australian children (data not shown).

**Table 3. Serotypes of isolates with reduced susceptibility to penicillin in children aged less than five years, Australia, 2003 (N = 71)**

| Serotype | 19F* | 9V* | 14* | 6B* | 23F* | 19A† | 6A† | 33F‡ | Total |
|---|---|---|---|---|---|---|---|---|---|
| Number of isolates | 19 | 16 | 16 | 8 | 1 | 6 | 4 | 1 | 71/622 |
| Cumulative % | 27% | 49% | 72% | 83% | 85% | 93% | 99% | 100% | 11% |

* 7vPCV conjugate vaccine serotype.

† 7vPCV conjugate vaccine serogroup.

‡ 23vPPV polysaccharide vaccine serogroup.

**Table 4. Serotypes with reduced susceptibility to erythromycin in children aged less than five years, Australia, 2003 (n=138)**

| Serotype | 14* | 19F* | 6B* | 18C* | 23F* | 4* | 19A† | 6A† | Total |
|---|---|---|---|---|---|---|---|---|---|
| Number of isolates | 97 | 17 | 16 | 1 | 2 | 1 | 1 | 3 | 138/567 |
| Cumulative % | 70% | 83% | 94% | 95% | 96% | 97% | 98% | 100% | 24% |

* 7vPCV conjugate vaccine serotype.

† 7vPCV conjugate vaccine serogroup.

‡ 23vPPV polysaccharide vaccine serogroup.

**Table 5. Penicillin and erythromycin resistance in children aged less than five years, Australia, 2003, by state and territory**

| State | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Australia |
|---|---|---|---|---|---|---|---|---|---|
| **Penicillin** | | | | | | | | | |
| Proportion of isolates tested | 11/11 | 211/212 | 18/19 | 159/160 | 65/66 | 5/5 | 116/128 | 37/37 | 622/638 |
| Number (%) Penicillin reduced susceptibility | 1 (9%) | 30 (14%) | 0 (0%) | 22 (14%) | 3 (5%) | 0 (0%) | 12 (10%) | 3 (8%) | 71 (11%) |
| **Erythromycin** | | | | | | | | | |
| Proportion of isolates tested | 11/11 | 201/212 | 18/19 | 158/160 | 63/66 | 4/5 | 75/128 | 37/37 | 567/638 |
| Number (%) Erythromycin resistant | 3 (27%) | 57 (28%) | 1 (6%) | 49 (31%) | 15 (24%) | 3 (75%) | 5 (7%) | 5 (14%) | 138 (24%) |

**Table 6. Predicted number of cases of invasive pneumococcal disease in adults aged more than 65 years that could be prevented as a result of introduction of 7vPCV vaccine in children in Australia**

| Serotype | Number of isolates | Per cent change post vaccine* | Number of cases prevented |
|---|---|---|---|
| 14 | 91 | -36% | 33 |
| 4 | 63 | -26% | 16 |
| 23F | 55 | -31% | 17 |
| 9V | 53 | -36% | 19 |
| 6B | 41 | -16% | 7 |
| 19F | 31 | -4% | 1 |
| 18C | 11 | -31% | 3 |
| 19A | 21 | -22% | 5 |
| 23A | 1 | -22% | <1 |
| 6A | 30 | -22% | 7 |
| 9N | 8 | -22% | 2 |
| Overall | 535 | -20% | 110 |

* Based on data from Reference 14.

**Differences in penicillin and erythromycin resistance rates by age and serotype**

The proportion of penicillin resistant pneumococci in Australians over 65 years of age was significantly higher (17%) than the proportion in children less than five years of age (11% Table 7).

The converse was true for erythromycin resistance with a significantly higher proportion of resistant isolates (24%) in children less than five years of age compared to adults over 65 years of age (15%, Table 8). This difference was contributed to by the larger proportion of erythromycin-resistant serotype 14 isolates in children.

Differences in serotype distribution and penicillin resistance of CSF isolates in patients less than five years of age compared those over five years of age

Pneumococcal isolates from the CSF of patients over five years of age were more likely to be resistant to penicillin than those from patients less than five years of age, but this difference did not reach statistical significance. Serotypes 6B and 14 accounted for a significantly higher proportion of pneumococcal isolates from the CSF of patients under five years of age than those over five years of age. Serotype 19F was more common in patient aged five years or more but the difference in proportion was not significant (Table 9).

## *Discussion*

The impact of a universal 7vPCV program for young children on invasive pneumococcal disease has now been clearly demonstrated in the United States of America (USA).[14] The vaccine program has benefited not only young children but it has also benefited their parent's age group (20 to 39 years) and the elderly in whom the rates of IPD have also decreased. Recently the National Health and Medical Research Council has recommended 7vPCV for all children in Australia as part of their primary immunisation series[18] and the Australian government has undertaken to fund this initiative from January 2005. Reliable baseline data on serotype prevalence in Australian children is essential to measure the impact of this new vaccine program. This study has examined serotype distribution and antimicrobial resistance in more than 90 per cent of the notified cases of IPD in Australia in 2003. These data allow us to predict the likely benefits which will be seen as a result of this new vaccine initiative.

A large proportion of young Australian children with IPD in 2003 were infected with pneumococcal serotypes (84%) or serogroups (92%) in the 7vPCV vaccine. While still not conclusive, some cross-protection for serogroups contained in the vaccine have been reported.[14] It is therefore reasonable to predict that Australia will see a significant decline in IPD in young children in the coming years when the new 7vPCV vaccination program is fully implemented. This could be of the order seen in the USA, where IPD declined by 69 per cent in the under 2-year olds in the first two years of a universal childhood vaccination program in this age group.[14]

The serotype distribution of penicillin resistant pneumococcal strains in young Australian children showed that 85 per cent of penicillin resistant isolates were a serotype and 99 per cent were a serogroup in 7vPCV. There is evidence from the USA that the rate of IPD due to penicillin resistant strains can be expected to fall by as much as 35 per cent with introduction of the 7vPCV.[14]

There are significant regional differences in penicillin and erythromycin resistance in Australia. Victoria in particular appears to have relatively low rates of erythromycin resistance and a low prevalence of serotype 14 which is frequently erythromycin resistant. A recent study by the NSW Pneumococcal Reference Laboratory has identified the predominant penicillin susceptible, erythromycin resistant clone of serotype 14 in NSW to be multi-locus sequence type (MLST) 9 (M. Watson, unpublished observations). The molecular mechanism of resistance to erythromycin in these MLST9 strains in New South Wales appears to be due to the macrolide efflux gene (*mef*) which does not confer cross resistance to the lincosamides such as clindamycin (M. Watson, unpublished observations). Serotype 14 erythromycin resistant isolates accounts for over 70 per cent of macrolide resistant isolates in children in Australia. By contrast the Northern Territory has a very low prevalence of macrolide resistance in children probably due to the virtual absence of serotype 14 following the introduction of the 7vPCV vaccination program in 2001 for all indigenous children in the NT and non-Indigenous children in Central Australia. Variations in rates of antibiotic prescribing and consumption would also explain variation in the prevalence of antimicrobial resistance across Australia and between age groups.

In the first two years of the childhood 7vPCV vaccine program in the United States there were declines in IPD disease rates in adults (32% in the 20 to 39 year age group and 18% in the aged 65 years or older).[14] Extrapolating the reductions in the prevalence of 7vPCV vaccine serotypes seen in the elderly population in the USA,[14] we predict that 110 (20%) episodes of IPD in adults over 65 years of age and 54 (20%) cases in the 20–39 year age group in Australia could be prevented as a result of the paediatric immunisation program. These calculations, based as they are on incomplete data, may under-estimate the eventual impact of universal 7vPCV vaccination

**Table 7. Proportions of penicillin resistant pneumococcal isolates by age group and serotype, Australia, 2003**

| Serotype | Children aged less than five years | | Adults aged over 65 years | | Significance of difference |
|---|---|---|---|---|---|
| | Proportion | Percentage | Proportion | Percentage | |
| Total isolates tested | 622/638 | 97 | 514/535 | 96 | NS |
| Resistant serotypes | 71/622 | 11 | 85/514 | 17 | p<0.05 |
| 7v vaccine serotypes* | 60/71 | 84 | 76/85 | 89 | NS |
| Serotype 19F | 19/85 | 22 | 15/31 | 48 | p<0.05 |
| Serotype 14 | 16/221 | 7 | 18/91 | 19 | p<0.005 |
| Serotype 6B | 8/93 | 9 | 8/41 | 19 | NS |
| Serotype 9V | 16/26 | 61 | 31/53 | 58 | NS |
| Serotype 23F | 1/27 | 4 | 3/55 | 5 | NS |

* includes serotypes in the 7vPCV conjugate vaccine (14, 19F, 14, 6B, 23F, 18C, 4).

NS = not significant.

**Table 8. Proportion of erythromycin resistant pneumococcal isolates by age group and serotype, Australia, 2003**

| Serotype | Children aged less than five years | | Adults aged over 65 years | | Significance of difference |
|---|---|---|---|---|---|
| | Proportion | Percentage | Proportion | Percentage | |
| Total isolates tested | 567/638 | 89 | 474/535 | 89 | NS |
| Resistant serotypes | 138/567 | 24 | 69/474 | 15 | p<0.001 |
| 7v vaccine serotypes* | 135/138 | 98 | 58/69 | 84 | p<0.001 |
| Serotype 19F | 17/85 | 20 | 11/31 | 35 | NS |
| Serotype 14 | 97/221 | 44 | 28/91 | 31 | p<0.05 |
| Serotype 6B | 16/93 | 17 | 6/41 | 15 | NS |
| Serotype 9V | 0/26 | 0 | 1/53 | 2 | NS |
| Serotype 23F | 2/27 | 7 | 7/55 | 13 | NS |

* includes serotypes in the 7vPCV conjugate vaccine (14, 19F, 14, 6B, 23F, 18C, 4).

NS = not significant.

**Table 9. Proportions of penicillin resistant isolates and common serotypes isolated from cerebrospinal fluid, by age group, Australia, 2003**

| Serotype in CSF | Cases aged less than 5 years (n=34) | | Cases aged 5 years and above (n=45) | | Significance of difference |
|---|---|---|---|---|---|
| | Proportion | Percentage | Proportion | Percentage | |
| Penicillin resistant isolates | 3/32 | 9 | 11/41 | 27 | NS |
| Serotype 19F | 1/34 | 3 | 9/45 | 20 | NS |
| Serotype 6B | 9/34 | 26 | 2/45 | 4 | p<0.05 |
| Serotype 14 | 11/34 | 32 | 3/45 | 6 | p<0.01 |
| Serotype 9V | 2/34 | 6 | 5/45 | 11 | NS |

There may also be additional benefits of 7vPCV vaccination by reductions in the prevalence of antibiotic resistant isolates in the elderly through improved herd immunity. However the prevalence of resistance varies significantly between children and adults. This appears to be associated with variations in the prevalence of penicillin and erythromycin resistant clones rather than existence of genetically distinct molecular clones in the two populations (M. Watson unpublished observations). The relatively higher prevalence of penicillin resistant serotypes in the elderly suggests that a reservoir of penicillin resistance exists in the elderly population in Australia with significant selective pressure towards acquisition of penicillin resistant strains of serotype 19F, 14 and 6B occurring in this age group. It is likely

that immunising children with the 7vPCV will reduce the incidence of penicillin resistant serotypes in the elderly since children would be less likely to pass on this serotype to their 'grandparents'. Reductions in IPD caused by serotype 19F and 6B have not been clearly demonstrated in the USA at this time, however a reduction of serotype 14 IPD in the elderly has been observed.[14] In 2003, serotype 19F in the elderly was a cause of meningitis in the older age group and three of the four penicillin resistant serotypes isolated in CSF from people over 65 years of age in Australia. The 23vPPV polysaccharide pneumococcal vaccine continues to provide good serotype coverage for adults, which supports the recent government decision to fully fund this vaccine for those at risk in Australia.

The continued laboratory surveillance of IPD is a vital component of the pneumococcal vaccine strategy for Australia. The funding of this surveillance has assisted a national approach to surveillance and reporting of this important reference laboratory work. Committed funding for serotype and uniform antibiotic susceptibility testing would assure appropriate monitoring of the impact of the new universal childhood 7vPCV program. Our surveillance to date suggests that introduction of the 7vPCV for all children and the 23vPPV vaccine for the 65 years and over in Australia in 2005 is likely to lead to major benefits for both children and the elderly.

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# Tuberculosis notifications in Australia, 2003

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*(Ral Antic – Chair, Ivan Bastian, Amanda Christensen, Mark Hurwitz, Anastasios Konstantinos, Vicki Krause, Moira McKinnon, Avner Misrachi, Graham Tallis, Justin Waring)*
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## Abstract

**The National Notifiable Disease Surveillance System (NNDSS) received 982 tuberculosis (TB) notifications in 2003, of which 947 were new cases, 33 were relapses and two were cases with unknown history. The incidence of TB in Australia has remained at a stable rate since 1985 and was 4.9 cases per 100,000 population in 2003. The high-incidence groups remain people born overseas and Indigenous Australians at 19.9 and 8.7 cases per 100,000 population, respectively. By contrast the incidence in non-Indigenous Australians was 0.9 per 100,000. Comparison of the 2003 TB notification data against the performance indicators set by National Tuberculosis Advisory Committee highlights that enhanced TB control measures should be considered among these high-risk groups. *Commun Dis Intell* 2004;28:464–473.**

*Keywords: tuberculosis, surveillance*

## *Introduction*

Tuberculosis (TB) control in Australia confronts a paradox. Australia has one of the lowest incidence rates of TB in the world and these rates have remained stable at 5–6 cases per 100,000 population since the mid-1980s.[1] Tuberculosis programs in low-incidence countries face problems in maintaining treatment services (including specially-trained staff, drug supplies and funding) for patients with active TB disease, in providing screening and preventative treatment programs for latent tuberculosis infection (LTBI) among high-risk groups, and in realigning

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policies and procedures towards TB elimination.[2] On the other hand, approximately 60% of the 8.8 million TB cases occurring globally in 2002 live in Australia's neighbouring countries in South-East Asia and the Western Pacific.[3] Those born overseas have accounted for an increasing proportion of Australia's burden over the last decade.[1] Australia's migrant intake includes people from countries with high prevalence of TB.

One crucial step in maintaining TB control in a low-incidence country is the collection of accurate, comprehensive and timely statistics. This data must be compared against performance indicators to ensure that strategic directions are identified, that outcomes are achieved, and that Australia's enviable record of TB control is maintained. This paper presents the TB notification data from the National Notifiable Diseases Surveillance System (NNDSS) in 2003. The data is also compared against the National Tuberculosis Performance Indicators (NTPI) set by the National TB Advisory Committee (NTAC) in the *National Strategic Plan for TB Control in Australia Beyond 2000*.[4] Information about drug susceptibility is published by the Australian Mycobacterium Laboratory Reference Network in an accompanying report.

## *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 authority. Information on notified cases for 2003 was collated by jurisdictions and sent electronically to 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 subcommittee of Communicable Diseases Australia Network (CDNA), was responsible for determining the data set collected in 2003 and for its transmission to NNDSS. Data fields in the enhanced TB data set that were analysed in this report were listed in Table 1 with a brief description of each variable.

### Data processing and quality control

Data on all TB notifications reported in 2003 were received by September 2004. Data received from the jurisdictions was examined for completeness and accuracy. Any invalid or missing entries were returned to the jurisdictions for review and correction.

Most cases of TB in Australia are reported to the surveillance system[5]. 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.

### 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 testing indicating *M. tuberculosis* complex except where it is likely to be due to previously treated or inactive disease. The inclusion of NAAT in this definition is to ensure full case ascertainment and does not endorse NAAT for TB diagnosis. Microscopy and culture remain the mainstays of TB laboratory diagnosis and provide the capacity for assessing 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 and some of these may be reinfections rather than a true relapse of prior disease.

### Population estimates for 2003

The rates presented in this report were calculated using population data produced by the Australian Bureau of Statistics (ABS). The estimated resident population (ABS, 2003)[6] as at 30 June 2003, 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[7] estimate of the Indigenous population in Australia (ABS, 2001). The ABS calculated the projections based on assumptions about future births, deaths and migrations in the Indigenous population and a 'low' and 'high' estimate were provided. For the

**Table 1. Description of some of the data fields in the enhanced tuberculosis data set of the National Notifiable Disease Surveillance System***

| Data field | Description |
|---|---|
| Country of birth | Country in which the notified case was born |
| Extrapulmonary site | Details of any extrapulmonary site involved |
| New or relapse case | Options include:<br>New case (without known previous treatment),<br>Relapse of disease following full treatment in Australia,<br>Relapse of disease following partial treatment in Australia,<br>Relapse of disease following full treatment overseas<br>Relapse of disease following partial treatment overseas |
| TB Outcomes | Options include:<br>Cured (bacteriologically confirmed),<br>Completed treatment,<br>Interrupted treatment for less than 2 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 |
| Age | Age of notified case at diagnosis |
| Indigenous status | Whether notified case is self-identified Indigenous (Aboriginal and/or Torres Strait Islander) Australian or not |
| Selected risk factors | Options include:<br>Close contact with a TB patient,<br>Currently/recently residing in a correctional facility,<br>Currently/recently residing in an aged care facility,<br>Currently/previously employed in an institution,<br>Currently/previously employed in the health industry,<br>HIV status (positive or negative)<br>Past residence (3 months or more) in a high risk country |

* Other data collected on each case included diagnosis details, therapy & susceptibility. These were analysed in the accompanying TB lab report.

purpose of this report, the 'low' estimate has been used, which is consistent with previous annual reports for TB notifications in Australia.

The 2001 census data were used to calculate incidence rates of TB in people born overseas. The estimated resident population of overseas-born people (total and by country of birth) in 2001 was used as the denominator in calculating rates.

To estimate the non-Indigenous Australian-born population, the Indigenous population estimate and the overseas-born population estimate were subtracted from the total Australian population. Since some of the TB notifications in the report may include non-permanent residents of Australia in 2003, the rates may be overestimated.

## *Results*

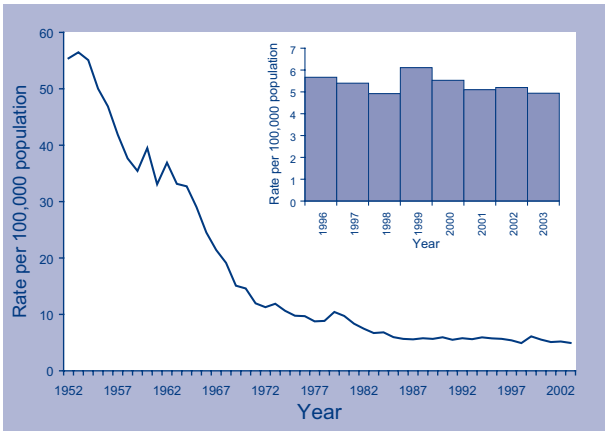
### Data quality

The majority of data fields were well reported. Information on age and sex for all notifications were complete. Country of birth was recorded for 980 (99.8%) of the total TB notifications. Indigenous status was reported for 159 (94.6%) of the 168 people born in Australia. The site(s) of TB disease were reported for 980 cases and whether the case being new or relapse was also reported for 980 cases. Therefore, the total for analysis was 980. Overall reporting of risk factors for TB improved for this period with 82 per cent complete compared with 48.7 per cent complete in 2002. The outcome from treatment was reported for 756 (77%) of cases. HIV status was not well reported (32.2%).

### TB notification rates

The total number of cases reported across Australia in 2003 was 982 (4.9 cases per 100,000 population) compared with 1,028 cases (5.2 cases per 100,000 population) in 2002. The national rate has remained relatively stable since 1985 except for an increase in 1999 due to the large number of TB cases identified in the East Timorese population evacuated to Darwin (Figure 1).

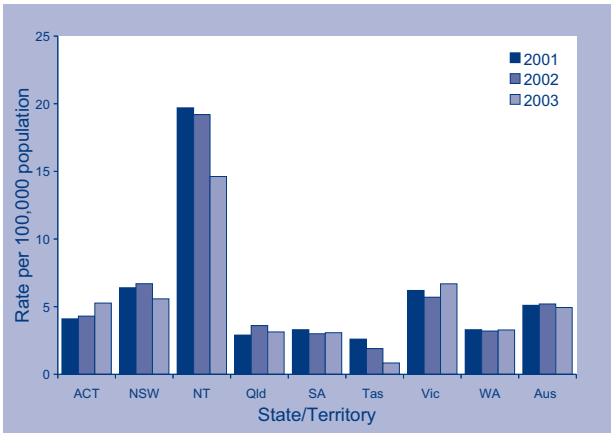
**Figure 1. Incidence rates for TB notifications, Australia, 1952 to 2003**




### TB notifications by jurisdiction

New South Wales reported the largest number of TB cases however the highest rate was recorded in the Northern Territory (Table 2). Figure 2 presents the notification rates by jurisdiction for 2001–2003. The small increases and decreases over time are often difficult to interpret due to the small number of cases within jurisdictions.

**Figure 2. TB notification rates by jurisdiction, Australia, 2001 to 2003**




Of the 33 relapsed cases, 14 were identified following full treatment in Australia, one following partial treatment in Australia, 12 following full treatment overseas and six following partial treatment overseas.

### TB notifications in the Australian-born population

In 2003, the Indigenous status of nine cases was unknown and these cases were added to the non-Indigenous Australian-born category for the calculations of rates (Table 3). One hundred sixty-eight (17%) cases of TB occurred in the Australian-born population, of whom 130 (77%) were non-Indigenous and 38 (23%) were Indigenous Australian.

The TB incidence rate in the non-Indigenous Australian-born population (0.9 cases per 100,000 population) has remained stable over the past 12 years. The incidence of TB in Indigenous Australians for 2003 was 8.7 cases per 100,000 population, the

**Table 2. New and relapsed cases and rates per 100,000 population by jurisdiction, Australia, 2003***

| State/territory | New cases | New cases rate | Relapsed cases | Relapsed cases rate | Total | Total rate |
|---|---|---|---|---|---|---|
| Australian Capital Territory | 17 | 5.3 | 0 | 0.0 | 17 | 5.3 |
| New South Wales | 363 | 5.4 | 10 | 0.1 | 373 | 5.6 |
| Northern Territory | 26 | 13.1 | 3 | 1.5 | 29 | 14.6 |
| Queensland | 114 | 3.0 | 5 | 0.1 | 119 | 3.1 |
| South Australia | 46 | 3.0 | 1 | 0.1 | 47 | 3.1 |
| Tasmania | 3 | 0.6 | 1 | 0.2 | 4 | 0.8 |
| Victoria | 321 | 6.5 | 6 | 0.1 | 327 | 6.6 |
| Western Australia | 57 | 2.9 | 7 | 0.4 | 64 | 3.3 |
| Australia | 947 | 4.8 | 33 | 0.1 | 980 | 4.9 |

* There were two cases where relapse status was unknown.

**Table 3. TB notifications and incidence rates in all Australian-born by jurisdiction, Australia 2003**

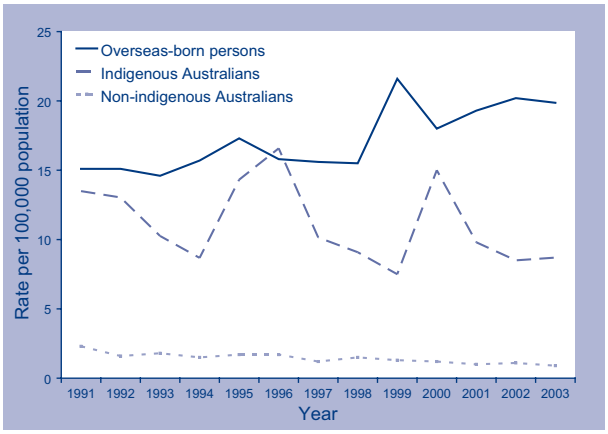
| State/territory | Indigenous Australian-born | Rate | Non-Indigenous Australian-born | Rate | Total Australian-born | Rate |
|---|---|---|---|---|---|---|
| Australian Capital Territory | 0 | 0.0 | 5 | 2.0 | 5 | 1.9 |
| New South Wales | 5 | 4.1 | 43 | 0.8 | 48 | 0.9 |
| Northern Territory | 20 | 34.9 | 2 | 1.8 | 22 | 12.9 |
| Queensland | 6 | 4.9 | 25 | 0.8 | 31 | 1.0 |
| South Australia | 2 | 8.1 | 10 | 0.8 | 12 | 1.0 |
| Tasmania | 0 | 0.0 | 2 | 0.5 | 2 | 0.5 |
| Victoria | 0 | 0.0 | 39 | 1.0 | 39 | 1.0 |
| Western Australia | 5 | 8.0 | 4 | 0.3 | 9 | 0.6 |
| Australia | 38 | 8.7 | 130 | 0.9 | 168 | 1.1 |

second lowest rate reported for this population since 1991. However, the TB incidence among Indigenous Australians remains almost ten times higher than among non-Indigenous Australian-born people. Twenty of 38 cases in Indigenous Australians were also reported from the Northern Territory, a jurisdiction where 28 per cent of the population are Indigenous Australians as compared to two per cent nation wide.

### TB notifications in the overseas-born population

The rate of notification in the overseas-born was 19.9 cases per 100,000 population in 2003, which is similar to the previous two years (20.2 and 19.3 cases per 100,000 population in 2002 and 2001, respectively) (Figure 3). Overseas-born population have represented an increasing proportion of new TB cases over the last decade; 637 (66.4%) of 960 incident cases in 1994 compared with 812 (82.7%) of 982 TB notifications in 2003.

**Figure 3. TB incidence rates by Indigenous status and country of birth, Australia 1991 to 2003**




### TB notifications by age and sex

One of the most important measures of TB control is the incidence in children less than 15 years of age because these cases are markers of recent TB transmission. TB was notified in 43 children under 15 years of age and the overall notification rate for this age group was 1.1 case per 100,000 population (target less than 0.1 per 100,000 population for all groups). The rate was highest in overseas-born children, and high in Indigenous Australian-born children (Table 5). The rate of 0.4 per 100,000 population in non-Indigenous Australian-born children remains low, close to the target of the National Performance Indicators of TB (<0.1 per 100,000 population).

The age and sex-stratified incidence rates for TB in overseas-born, Indigenous Australian-born and non-Indigenous Australian-born populations are shown in Figure 4. The TB distribution pattern in the overseas-born population was different to that of the Australian-born population. In the non-Indigenous Australian-born there was approximately one case per 100,000 population for people up to the 45–54 year age range for both males and females, after which the incidence rate increased gradually for both sexes. The highest rates for the non-Indigenous Australian-born population was in the over 65 year age group, where the rate for males was 4.9 cases per 100,000 population and 2.4 cases per 100,000 population for females. The overall male:female ratio in non-Indigenous Australian-born TB cases was 1.4:1.

Age-specific peaks in TB incidence are evident among overseas-born population (i.e. among infants 0–4 years, among young adults in the 15–34 year age groups, and in those aged over 65 years) (Figure 4). Similar but smaller peaks are discernible in the age-specific incidence rates for the Indigenous Australian-born population. The overall male: female ratio of TB cases in the overseas-born population was 1:1. The overall male: female ratio of TB in the Indigenous Australian-born population was 0.7:1.

## Table 4. Notification of tuberculosis and estimated rate per 100,000 population for selected countries of birth, Australia, 2003

| Country of birth | New cases | Relapsed cases | Total cases | Estimated Australian resident population by country of birth, 2001 | Rate per 100,000 population in Australia by country of birth, 2002* | WHO incidence rate per 100,000 population for country, 2001† |
|---|---|---|---|---|---|---|
| Viet Nam | 106 | 5 | 111 | 154,833 | 71.7 | 192 |
| India | 58 | 1 | 59 | 95,455 | 61.8 | 168 |
| China‡ | 50 | 5 | 55 | 142,778 | 38.5 | 113 |
| Morocco‖ | 46 | 0 | 46 | 1,169 | § | 114 |
| Mongolia | 41 | 1 | 42 | 126 | § | 209 |
| Philippines | 32 | 0 | 32 | 103,942 | 30.8 | 320 |
| Sudan | 24 | 0 | 24 | 4,900 | 489.8 | 217 |
| Somalia | 22 | 0 | 22 | 3,713 | 592.5 | 405 |
| Cambodia | 20 | 2 | 22 | 22,979 | 95.7 | 549 |
| Libya | 22 | 0 | 22 | 1,442 | 1,525.7 | 21 |
| Hong Kong (SAR) | 18 | 0 | 18 | 67,121 | 26.8 | 93 |
| Indonesia | 17 | 0 | 17 | 47,156 | 36.1 | 256 |
| Papua New Guinea | 15 | 2 | 17 | 23,618 | 72.0 | 254 |
| Italy | 16 | 0 | 16 | 218,718 | 7.3 | 8 |
| Others | 301 | 9 | 310 | 3,201,141 | | |
| Overseas | 786 | 26 | 812 | 4,087,928 | 19.9 | |
| Australia | 160 | 8 | 168 | 15,619,272 | 1.1 | |
| Not stated | | | 2 | | | |
| Total | 946 | 34 | 982 | 19,707,200 | 5.0 | |

* Country of birth for denominator is from the 2001 census.

† Rates from the World Health Organization 2004 Global tuberculosis report.

‡ China excludes Hong Kong SAR and Taiwan.

§ Crude rate for people born in Morocco and Mongolia were not estimated as their population estimates in 2001 was small. A small increase in the number of cases could sharply increase the crude rate that does not necessarily reflect the magnitude of the true increase.

‖ Morocco includes people who were born in Western Sahara.

Note: There were two cases where relapse status was unknown.

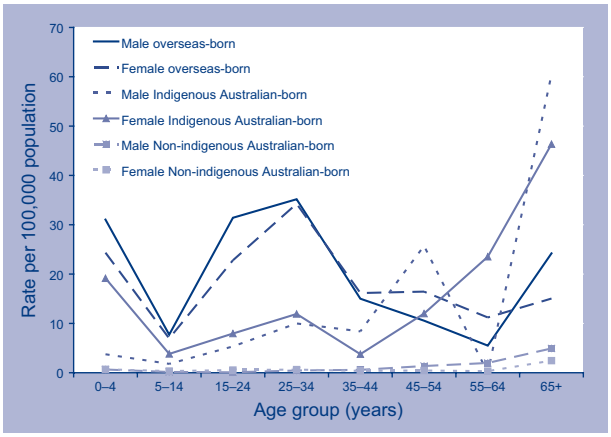
## Table 5. TB notifications and estimated incidence rate by age group, Indigenous status and country of birth, Australia, 2003

| Age group | Indigenous Australian-born | | Non-Indigenous Australian-born | | Overseas-born | |
|---|---|---|---|---|---|---|
| | n | Rate | n | Rate | n | Rate |
| 0–4 | 6 | 11.4 | 8 | 0.7 | 7 | 27.8 |
| 5–14 | 3 | 2.8 | 6 | 0.2 | 13 | 7.3 |
| **Sub total for <15 years** | 9 | 5.6 | 14 | 0.4 | 20 | 9.9 |
| 15–24 | 5 | 6.6 | 7 | 0.3 | 107 | 27.2 |
| 25–34 | 7 | 11.0 | 12 | 0.5 | 210 | 34.7 |
| 35–44 | 3 | 5.9 | 10 | 0.5 | 135 | 15.6 |
| 45–54 | 6 | 18.6 | 16 | 0.9 | 118 | 13.5 |
| 55–64 | 2 | 12.4 | 14 | 1.1 | 57 | 8.2 |
| 65+ | 6 | 52.5 | 57 | 3.5 | 165 | 19.5 |

Note: The denominator used for total non-Indigenous Australian-born population is from the 2001 census, whilst age group breakdowns use denominators from estimated resident population in 2000 based on the 1996 census results.

There were two cases where country of birth was unknown and nine cases where indigenous status was unknown.

**Figure 4. TB incidence in Australian-born and overseas-born by age and sex, 2003**




Note: There were two cases where country of birth was unknown.

### TB and selected risk factors

Information on risk factors for TB disease excluding HIV were reported for 492 (50%) of the 982 cases. Caution must be taken in interpreting these results as it is unclear whether there were no risk factors identified in the other TB notifications or if the information was not recorded. Where risk factors were reported, the majority (433 cases) identified as having previously resided for three or more months in high risk countries as defined by the Department of Immigration, Multicultural and Indigenous Affairs (DIMIA). Among these 433 cases, seven were Australian-born and 426 were overseas-born. An additional 174 cases were household members or close contacts of TB cases, seven cases either resided or had recently resided in a correctional service and nine cases either resided or recently resided in an aged care facility. For individuals working in high risk settings, four cases were employed or recently employed in institutions such as correctional facilities or aged care facilities and 30 cases were employed or recently employed in the health industries. Among these 30 cases, three were Australian-born and 27 were overseas-born.

### TB and HIV status

Information on HIV status was reported in only one-third of cases. Twelve people were identified with HIV infection at the time of diagnosis with TB; five Australian-born and seven overseas-born. The National Strategic Plan recommends that HIV status of all TB cases be reported. The reporting of HIV status has not improved appreciably since 2002 when only 27 per cent cases had HIV status reported.

### Anatomical site of disease

Five hundred sixty-three (57%) of notified cases had pulmonary disease either alone or accompanying disease at an extrapulmonary site; 417 cases (43%) had TB limited to an extrapulmonary site only. The sites of disease in new and relapse cases are shown in Table 6. Pulmonary TB was most commonly reported in the Australian-born populations (73.8%) and less commonly in the overseas-born (54.1%). More cases in 2003 reported lymph nodes as the site of infection (16% in 2002; 24% in 2003).

### Treatment outcomes

Treatment outcomes were reported for 756 (77%) of the cases from 2003 by September 2004. The remaining individuals were either still undergoing treatment or their treatment status was unknown. Satisfactory outcomes were reported for 87.3%, including those with bacteriologically confirmed cure and those who completed treatment without bacteriological evidence of cure (Table 7). There were no treatment failures recorded. Eleven cases (1.5%) were reported as defaulting treatment. The proportion of cases cured or who completed treatment were 96.6% among Indigenous Australians, 90.4% among non-Indigneous Australian born and 86.4% among overseas born. Death from TB is rare in Australia. While there were 53 reported deaths in the notified cases from 2003, only 11 were reported to be due to TB with a case fatality rate of 1.1 per cent. A number of these cases were identified at post-mortem.

The following treatment outcomes were excluded from the analysis: deaths (53), cases transferred out of Australia (63), cases with unknown outcome (19), and cases still undergoing treatment at the time report (130).

### National Performance Indicators

The National Tuberculosis Performance Indicators (NTPI) were set by NTAC in 2002 and reviewed in 2003 (Table 8). As in last year's TB annual report, the performance criteria for people born overseas applies to people who have been living in Australia for more than five years. Of the 812 cases born overseas, 416 (51.2%) had been living in Australia for more than five years. The TB incidence rate for people born overseas who have been living in Australia for more than five years was 10.2 cases per 100,000 population.

The incidence of TB in children less than 15 years of age in the Indigenous population increased from the previous year (5.6 cases per 100,000 in 2003 and

**Table 6. New and relapsed TB cases by site of disease, Australia, 2003**

| Site | New cases | Relapse cases | Total cases | Percent of cases |
|---|---|---|---|---|
| Pulmonary only | 461 | 22 | 483 | 49.2 |
| Pulmonary and other sites | 77 | 3 | 80 | 8.1 |
| Extrapulmonary | 409 | 8 | 417 | 42.5 |
| Lymph Nodes | 235 | 1 | 236 | 24.0 |
| Other | 87 | 5 | 92 | 9.4 |
| Pleural | 63 | 3 | 66 | 6.7 |
| Bone/Joint | 36 | 1 | 37 | 3.8 |
| Genito/Urinary | 30 | 0 | 30 | 3.1 |
| Milliary | 16 | 0 | 16 | 1.6 |
| Meningeal | 14 | 1 | 15 | 1.5 |
| Peritoneal | 14 | 0 | 14 | 1.4 |

Note: Only the first three categories of site that add up to 980 (99.8% of cases).

For the subsequent categories, they were included in either pulmonary & other sites or extrapulmonary site.

**Table 7. Outcomes of TB treatment by population group, Australia, 2003**

| Treatment outcomes | Indigenous Australian-born | Non-Indigenous Australian-born | Overseas-born | Unknown | Total | Percent of cases |
|---|---|---|---|---|---|---|
| Cured (bacteriologically confirmed) | 18 | 12 | 39 | 0 | 69 | 9.6% |
| Completed treatment | 10 | 73 | 474 | 0 | 557 | 77.7% |
| Interrupted treatment* | 0 | 0 | 3 | 0 | 3 | 0.4% |
| Defaulted§ | 1 | 1 | 9 | 0 | 11 | 1.5% |
| Failed‖ | 0 | 0 | 0 | 0 | 0 | 0.0% |
| Missing | 0 | 8 | 69 | 0 | 77 | 10.7% |
| Total | 29 | 94 | 594 | 0 | 717 | 100.0% |

* 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.

Note: The following treatment outcomes were excluded from the analysis: deaths (53), cases transferred out of Australia (63), outcome unknown (19) and cases still undergoing treatment at the time of report (130).

4.3 cases per 100,000 population in 2002), but this represented only two additional cases in this age group in 2003.

## *Discussion*

The incidence of TB in Australia has remained between five and six cases per 100,000 population since the mid-1980s, and represents one of the lowest incidence rates in the world.[3] Other developed countries that have reported rates of less than six per 100,000 in 2002 include Iceland, Sweden, and United States of America. Tuberculosis control in low-incidence countries faces specific problems and challenges,[2] such as: the reduced awareness of TB among healthcare professionals, the increasing importance of imported TB among migrants, the recognition of sub-groups at high risk of TB (e.g. Indigenous Australians).

Doctors and other healthcare professionals in Australia must maintain an index of suspicion for TB. The demographic data presented in this paper highlights that doctors and other healthcare workers (HCWs) must "Think TB" particularly when caring for migrants, Indigenous Australians, and elderly non-Indigenous Australian-born patients (Figure 4 and Table 5). This awareness of TB among healthcare professionals depends on adequate undergraduate and postgraduate training in TB epidemiology, diagnosis, management and control measures for doctors, nurses, laboratory staff and migrant health workers.

**Table 8. National tuberculosis performance indicators, performance criteria and the current status of tuberculosis in Australia, 2003**

| National TB Performance Indicator | Performance criteria | 2002 | 2003 |
|---|---|---|---|
| **Annual Incidence of TB (per 100,000 population)** | | | |
| Crude incidence | | | |
| Indigenous Australians | <1 | 8.5 | 8.7 |
| Non-indigenous Australian-born | <1 | 1.1 | 0.9 |
| Overseas-born persons* | † | 11.5 | 10.2 |
| Relapse cases initially treated in Australia | <2% of total treated cases | 2.3 | 1.1 |
| Incidence in children <15 years, by risk group | | | |
| Indigenous Australian children | <0.1 | 4.3 | 5.6 |
| Non-indigenous Australian-born children | <0.1 | 0.5 | 0.4 |
| Overseas-born children* | † | 7.6 | 9.9 |
| Collection of HIV status in TB cases (% of cases with data collected) | 100% over next 3 years | 27.3 | 32.2 |
| **Treatment outcome measures (%)** | | **(%)** | **(%)** |
| Cases evaluated for outcomes‡ | 100 | 78 | 89.3 |
| Cases that have treatment completed and are cured | >90 | 80 | 87.3 |
| Cases recorded as treatment failures‡ | <2 | 0.1 | 0 |

* The performance criteria for overseas born are applied to people who have been living in Australia for more than 5 years. The denominator for this rate is the total overseas born population living in Australia in 2002.

† Performance criteria currently under review.

‡ The denominator used for both 2001 and 2002 was the number of cases evaluated for treatment outcome.

The overseas-born population represented an increasing proportion of new TB cases. This group are at high risk of TB for numerous reasons. Overseas-born people may come from countries with a high incidence of TB and are likely to have acquired latent infection prior to migration. Many are refugees who have been living in camps where overcrowding, poor sanitation and malnutrition increase their risk of progressing to active disease. Finally, resettlement conditions may be socio-economically stressful to migrants, which may contribute to the progression of latent TB to active TB. Social contact with other migrants from high incidence countries may also increase the risk of exposure to TB.

Australian TB services continue to support pre-migration screening for active TB and to participate in post-migration follow-up programs in cooperation with DIMIA and other organisations. Migrants must have ready access to cost-free, non-threatening and culturally-appropriate TB assessment and treatment. People from Morocco, Mongolia, Sudan and Libya were reported as high-incidence sub-populations in Australia for the first time in 2003, reflecting another change in the composition of Australia's migrant intake. Tuberculosis clinics are producing educational materials in additional languages and are adapting to the specific cultural and social needs of these new patient populations. Community leaders in the new migrant populations must also be identified and encouraged to assist with TB control efforts. These TB control measures have proved successful in other migrant populations and are likely to succeed again. However, as Australia and other low-incidence countries move towards TB elimination, overseas-born population will continue to account for an increasing proportion of incident cases. Additional measures, such as active case finding and increased detection and treatment of LTBI, should be considered in migrant populations with a high incidence of TB.

Similarly, Indigenous Australians are at increased risk of TB with incidence rates nearly ten times higher than among non-Indigenous Australian-born people. This disparity has remained evident for the last decade despite the efforts of TB control programs (Figure 3). Some of the known risk factors that explain the high incidence of TB in the Indigenous Australians are socio-economic disadvantage (reflected in overcrowding), co-morbidities (such as diabetes and renal diseases), smoking, alcohol abuse and poor nutrition.[8] A nihilist would argue that TB cannot be controlled in Aboriginal communities until these causative factors are addressed. However, additional TB control interventions must be attempted in the meantime in collaboration with

Aboriginal health services. Tuberculosis cases tend to be restricted to a small number of Aboriginal communities.[8]

Comparison of the 2003 TB notification data against the NTPI provides some gratifying results, such as the TB incidence in the non-Indigenous Australian-born population (0.9 case per 100,000 population), the incidence among non-Indigenous Australian-born children (0.4 per 100,000 population), the proportion of relapsed cases initially treated in Australia (1.1%), and the proportion of cases recorded as treatment failures (0%)(Table 8). Other performance indicators suggest that further action is required. The NTPI aim for Indigenous communities to have the same low TB incidence as the non-Indigenous Australian-born population. The above paragraph suggested interventions to achieve this goal. The reporting of HIV status for TB cases remains at an unacceptable low level (i.e. 27% in 2002 and 32% in 2003). Studies in the United States show that the rate of TB disease among HIV-infected, tuberculin skin test (TST)-positive persons is approximately 200–800 times higher than the rate of TB for the general population.[9] Despite incomplete reporting, twelve cases of HIV/TB were recognised in Australia in 2003. Australian migrant intake includes people who come from countries where HIV and TB are prevalent. Privacy laws in some states confound efforts to collect information on the HIV status of TB patients. Alternative acceptable strategies must be found to obtain this essential public health information.

One final observation from the 2003 TB notification data deserves comment. Thirty TB cases occurred among HCWs, of whom 27 were overseas-born. Health services in Australia are increasingly reliant upon attracting medical and nursing staff from overseas, including from countries where TB is prevalent. State TB services and staff induction programs should be aware of this trend and ensure that new employees are screened and followed-up appropriately for TB.

In conclusion, easy access to effective TB treatment programs, contact tracing, and provision of health education in appropriate languages remain the essential elements for TB control. Australia also needs to remain alert to the growing global threat of TB and to contribute to TB control efforts in Southeast Asia and the Pacific region.

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# Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2003

## A report of the Australian Mycobacterium Reference Laboratory Network

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## Abstract

**The Australian Mycobacterium Reference Laboratory Network collected and analysed laboratory data on new cases of disease caused by *Mycobacterium tuberculosis* complex in the year 2003. A total of 784 cases were identified by bacteriology, representing an annual reporting rate of 3.9 cases of laboratory confirmed tuberculosis per 100,000 population. The most commonly encountered culture-positive specimens were sputum (n=351), lymph node (n=176) and from bronchoscopy (n=97). Smears containing acid fast bacilli were present in sputum (53.0%), bronchoscopy (32.0%) and lymph node (23.3%). Five children (female n=3, male n=2) under 10 years of age had bacteriologically confirmed tuberculosis. Eighty isolates of *M. tuberculosis* and one of *Mycobacterium africanum* (10.3%) were resistant to at least one of the standard anti-tuberculosis agents. Mono-resistance to isoniazid, ethambutol, rifampicin, and pyrazinamide was detected in 45, three, two, and one isolates respectively. Multidrug-resistance (MDRTB) defined as resistance to both isoniazid and rifampcin was observed in seven (0.9%) isolates. Of the seven MDRTB isolates, six were from the respiratory tract and four were from smear positive specimens. Of the 81 patients with drug resistant isolates, 78 (96.3%) were classified as having initial resistance; two had acquired resistance and no information was available for one isolate; five were Australian-born; and 76 (93.8%) had migrated from a total of 30 countries. *Commun Dis Intell* 2004;28:474–480.**

*Keywords:* Mycobacterium tuberculosis, Mycobacterium bovis, *laboratory diagnosis, tuberculosis, drug resistance, nucleic acid amplification test*

## *Introduction*

The annual incidence of tuberculosis (TB) diagnosed clinically in Australia has fallen from 55 cases per 100,000 population in the mid 1950s to a current level around 5 to 6 cases per 100,000 population. As part of the Western Pacific region of the World Health Organization, Australia enjoys one of the lowest rates of disease compared with the rest of the region which reported an overall notification rate of 47 per 100,000 population in year 2002. This rate has shown no significant variation since 1993.[3] The Western Pacific region contains several countries (China, Philippines, Viet Nam, Cambodia and Papua New Guinea) with a high burden of TB. Another regional neighbour, the Republic of Indonesia, has the third highest burden of TB in the world.[2]

There are two sources of TB-related data for Australia. Since 1991, the National Notifiable Diseases Surveillance System (NNDSS) has provided statistics on cases of tuberculosis reported to public health authorities in Australia's states and territories. The second source, 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 whereas NNDSS data will have a proportion of cases that are identified on the basis of clinical and epidemiological information, or on non-bacteriological laboratory investigations. This report describes the bacteriologically confirmed TB diagnoses for the year 2003.

## *Methods*

The data are based on clinical specimens that were culture-positive for *Mycobacterium tuberculosis* complex (MTBC). Although the BCG strain of *Mycobacterium bovis* is a member of the MTBC, no information on this organism is included in the

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2. Australian Mycobacterium Reference Laboratory Network

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present report. Almost all isolates of MTBC were referred to one of the five laboratories comprising the AMRLN for specific identification and drug susceptibility testing. Comparable methodologies are used in the reference laboratories. Relapse cases, as defined by the *National Strategic Plan for TB Control in Australia beyond 2000* prepared by the National TB Advisory Committee, were included in the laboratory data as laboratories are generally unable to differentiate relapse cases from new cases.[3] Temporary visitors to Australia were included as were illegal aliens within correctional services facilities and asylum seekers located in detention centres or on temporary visas within Australia.

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

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

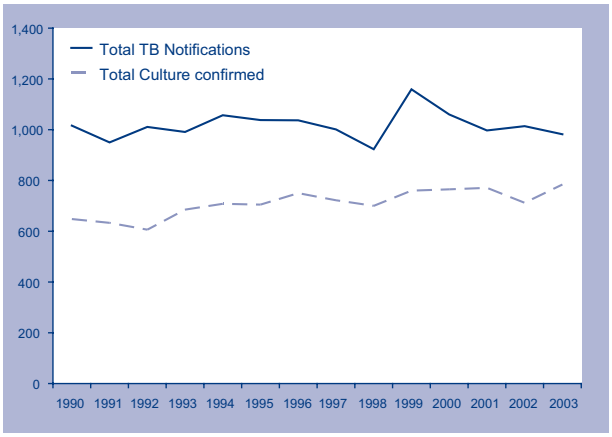
Data from contributing laboratories were submitted in standard format to the scheme coordinator for collation and analysis. Duplicate entries (indicated by identical patient identifier and date of birth) were deleted prior to analysis. Rates were calculated using mid-year estimates of the population for the year 2003 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 considered to indicate pulmonary disease. Cases with multi-site isolations, provided a sputum or bronchoscopy specimen was culture-positive, were listed as having pulmonary disease, the most important category for public health purposes. Cases for which there were multiple-site isolations were not categorised as having miliary or disseminated disease as differentiation is based on clinical findings that are generally not available to the reporting laboratories. Initial drug resistance was defined as the presence of drug resistant strains of *M. tuberculosis* and *M. africanum* in cases of tuberculosis in which there was no known history of anti-tuberculosis treatment. Patients who had begun anti-TB treatment and had developed resistance to one or more of the drugs used during treatment were recorded as having acquired drug resistance.[5]

## *Results*

There were 784 bacteriologically confirmed cases of tuberculosis in 2003 (Figure 1), representing an annual rate of 3.9 per 100,000 population. State-specific reporting rates varied from 0.8 cases (Tasmania) to 10.1 cases per 100,000 population (Northern Territory) (Table 1).

**Figure 1. Comparison between tuberculosis notifications and laboratory data, Australia; 1990 to 2003**




### Causative organism

Almost all isolates were identified as *M. tuberculosis* (n=782), the remaining two isolates being a single *M. africanum* and a *M. bovis*.

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

Complete information for gender and age were submitted for all patients, due to additional information provided by state and territory Tuberculosis Centres. Five children (female n=3 male n=2) under 10 years of age had bacteriologically confirmed tuberculosis (lymph node n=2, tracheal aspirate n=1, gastric aspirate n=1, biopsy n=1).

The relationship of tuberculosis to age and gender are shown in Figure 2. For males, there were two distinct age groups; a rise to 6.9 cases of tuberculosis per 100,000 population at 20–24 and 25–29 years, and in the elderly male where the rate rose from 5.6 at age grouping 65–69 to a peak of 17.1 per 100,000

**Table 1. Bacteriologically confirmed cases of tuberculosis in Australia, 1994 and 2000 to 2003, cases and rate per 100,000 population by state or territory***

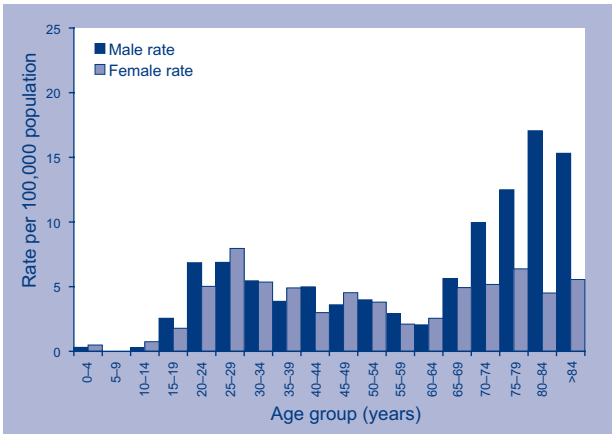
| State or territory | 2003 | | 2002[16] | | 2001[15] | | 2000[14] | | 1994[10] | |
|---|---|---|---|---|---|---|---|---|---|---|
| | n | Rate | n | Rate | n | Rate | n | Rate | n | Rate |
| New South Wales† | 325 | 4.6 | 301 | 4.3 | 327 | 4.8 | 307 | 4.5 | 278 | 4.4 |
| Victoria | 254 | 5.2 | 208 | 4.3 | 222 | 4.6 | 231 | 4.8 | 217 | 4.8 |
| Queensland | 91 | 2.4 | 97 | 2.6 | 81 | 2.2 | 76 | 2.1 | 88 | 2.8 |
| Western Australia | 54 | 2.8 | 46 | 2.4 | 68 | 3.6 | 63 | 3.3 | 53 | 3.1 |
| South Australia | 36 | 2.4 | 26 | 1.7 | 38 | 2.5 | 41 | 2.7 | 41 | 2.8 |
| Tasmania | 4 | 0.8 | 8 | 1.7 | 12 | 2.8 | 2 | 0.4 | 10 | 2.1 |
| Northern Territory | 20 | 10.1 | 26 | 13.0 | 23 | 11.6 | 45 | 23.0 | 21 | 12.3 |
| **Total** | 784 | 3.9 | 712 | 3.6 | 771 | 4.0 | 765 | 4.0 | 708 | 4.0 |

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

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

population for the 80–84 age group. Females in the 25–29 year age group had a peak rate of 8.0 per 100,000 population but in contrast to males, the rate for tuberculosis in the elderly female was more modest rising only to 6.4 cases per 100,000 population. In part, these differences are due to the site of infection. Overall, the male:female ratio was 1.16:1, for sputum isolates, but the ratio was reversed for lymph node isolates (1:1.4). The median age group for patients with respiratory disease was 35–39 for females and 45–49 for males, and for lymph node cases, the median age group for both genders was 35–39 years.

**Figure 2. Laboratory confirmation of *Mycobacterium* tuberculosis complex disease, Australia 2003, by age and sex**




The predominant specimen type was sputum, including three gastric aspirates (n=351, 44.7%); bronchoscopy (n=97, 12.4%), lymph node (n=176, 22.4%) and pleural (n=35, 4.5%) (Table 2).

**Table 2. Site of specimens smear- and culture-positive for *Mycobacterium tuberculosis* complex, in the year 2003**

| | n* | Smear positive (%)† |
|---|---|---|
| Sputum | 351 | 186 (53.0) |
| Bronchoscopy | 97 | 31 (32.0) |
| Lymph node | 176 | 41 (23.3) |
| Pleural | 35 | 2 (5.7) |
| Genito-urinary | 18 | 9 (50.0) |
| Bone/Joint | 25 | 9 (36.0) |
| Peritoneal | 24 | 2 (8.3) |
| Skin | 11 | ND† |
| CSF | 6 | ND† |

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

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

### Association with HIV

The AMRLN database recorded the HIV status for only 55 (7.0%) patients. Only two patients were identified as HIV seropositive; one had smear-positive respiratory disease and the other patient had genitourinary TB.

### Microscopy

Results of microscopy were available for 751 of 784 (95.8%) of specimens; microscopy was not performed on seven specimens and no results were provided for the remaining 26 specimens. Smears were positive for 186 of 351 (53.0%) sputum and 31 of 97 (32.0%) bronchoscopy specimens respectively (Table 2). A total of 35 pleural specimens (8 biopsy and 27 fluids) were culture positive for *M. tuberculosis*, but only one of each specimen type was smear positive. Lymph node specimens were smear positive for only 41 of 176 (23.3%) cases.

### Drug susceptibility testing

Results of *in vitro* drug susceptibility testing were available for all 784 isolates for isoniazid (H), rifampicin (R) and ethambutol (E) and for 783 isolates for pyrazinamide (Z). A total of 81 isolates (10.3%) of *M. tuberculosis* (n=80) and *M. africanum* (n=1) were resistant to at least one of the above anti-tuberculosis agents. Results of testing for streptomycin (S) were available for 222 of 784 (28.3%) of isolates with nine demonstrating S mono-resistance and another eight were resistant to S + H. Resistance to at least both H and R (defined as multidrug resistance) was detected in seven (0.9%). All of the MDR isolates were *M. tuberculosis* (Table 3). Of the 7 MDRTB isolates, six were from the respiratory tract (sputum n=4, bronchoscopy n=2); the remaining isolate was from a lymph node. Three of the MDRTB-positive sputum specimens were smear positive as was one of the bronchoscopy specimens and the single isolate from lymph node tissue. A single isolate of *M. bovis* from a smear-positive sputum was not included in the above results.

Mono-resistance to isoniazid, ethambutol, rifampicin, and pyrazinamide was detected in 45, three, two, and one isolates respectively. There were 75 isolates that demonstrated resistance to H at a concentration of 0.1 mg/L. Of these, 41 (54.7%) demonstrated resistance to H at the higher level of 0.4 mg/L. Thirty-seven of 81 (45.7%) specimens culture-positive for drug resistant *M. tuberculosis*, including 26 of 55 (47.3%) sputum or bronchoscopy specimens, were smear-positive for AFB. Six of the seven MDRTB isolates had high level isoniazid resistance.

### Initial or acquired resistance, and country of origin

There were 80 *M. tuberculosis* and one *M. africanum* resistant to at least one of the standard drugs (H, R, E, Z). Of these, 78 of 81 (96.3%) were classified as having initial resistance, two had acquired resistance, and no data was available for one isolate on the presence or absence of previous treatment. The country of birth was known for all patients with drug resistant strains; five were Australian born, and 76 (93.8%) had migrated from a total of 30 countries.

Of the 76 migrants with drug-resistant disease, 49 (64.5%) had migrated from one of six countries; Viet Nam (n=18), India (n=8), Philippines (n=7), Indonesia (n=5), Sudan (n=5), and China (n=4).

### Use of nucleic acid amplification tests

Nucleic acid amplification testing (NAAT) was performed on 201 of 784 (25.6%) specimens, all of which subsequently grew *M. tuberculosis* on culture. Of these, 123 specimens were of respiratory origin (sputum, n=90, bronchoscopy, n=26, tissue, n=4, aspirate, n=3), and 112 (91.1%) were NAAT positive. For smear positive respiratory specimens, 80 of 83 (96.4%) were NAAT positive whilst 26 of 32 (81.3%) of smear negative respiratory specimens were NAAT positive (Table 4A). Seven specimens did not record a smear result and one smear negative tissue specimen recorded an equivocal result.

There were 78 specimens of non-respiratory origin (tissue, n=50, aspirate, n=14, fluid, n=13, swab, n=1) and only 47.4 per cent were NAAT positive. For smear positive non-respiratory specimens, 19 of 22

**Table 3. Drug resistance patterns in MDR strains, Australia, 1993 to 2003**

| Resistance pattern (standard drugs)* | 2003 | 2002[16] | 2001[15] | 2000[14] | 1999[13] | 1998[13] | 1997[12] | 1996[11] | 1995[10] | 1994[10] | 1993[9] |
|---|---|---|---|---|---|---|---|---|---|---|---|
| H+R‡ only | 4 | 8 | 8 | 3 | 2 | 2 | 6 | 10 | 3 | 2 | 7 |
| H+R+E‡ | 2 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 0 | |
| H+R+Z‡ | 1 | 1 | 3 | 3 | 1 | 2 | 5 | 4 | 1 | 0 | |
| H+R+E+Z‡ | 0 | 2 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| **Total (%)** | 7 (0.9) | 12 (1.7) | 12 (1.6) | 8 (1.0) | 4 (0.5) | 6 (0.9) | 14 (1.9) | 15 (2.0) | 5 (0.7) | 2 (0.3) | 10† (1.5) |

* The streptomycin result was not considered for this table.

† The multi-drug profiles for all 10 strains were not identified.

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

**Table 4A. Results for nucleic acid amplification tests performed on respiratory specimens, Australia, 2003**

| NAAT result | Culture positive respiratory specimens | |
|---|---|---|
| | Smear positive | Smear negative |
| Positive | 80 | 26 |
| Negative | 3 | 6 |
| **Total* (115)** | 83 | 32 |

* Seven specimens did not record a smear result and one smear negative tissue specimen recorded an equivocal result.

**Table 4B. Results for nucleic acid amplification tests performed on non-respiratory specimens, Australia, 2003**

| NAAT result | Culture positive NON-respiratory specimens | |
|---|---|---|
| | Smear positive | Smear negative |
| Positive | 19 | 18 |
| Negative | 3 | 33 |
| **Total* (73)** | 22 | 51 |

* Four specimens did not record a smear result and one smear-positive spinal tissue specimen recorded the presence of NAAT inhibitors.

(86.4%) were NAAT positive and 18 of 51 (35.3%) of smear negative non-respiratory specimens were NAAT positive (Table 4B). Four specimens did not record a smear result and one smear-positive spinal tissue specimen recorded the presence of NAAT inhibitors.

## *Discussion*

The finding of 784 cases of bacteriologically confirmed tuberculosis representing 3.9 cases per 100,000 population in 2003 is consistent with the results of previous AMRLN reports. Since the network began collecting data in 1986, the range for bacteriologically confirmed cases has remained between 3.5–4.1 per 100,000 population.[6–16]

For 2003, the NNDSS reported 982 notified cases of TB, a difference between the two datasets of 198 (25.3%).[17] The NNDSS has consistently recorded a higher number of notifications than the AMRLN data (range 22.7–44%). Possible reasons for the gap between the two data sources have been discussed previously.[14] Furthermore, the handling of multiple sites of disease differs also. The NNDSS database documents all sites of disease, whereas the AMRLN database lists only one site, and when multi-site disease is present, prioritises respiratory disease over non-respiratory sites. Although comparison of the unlinked databases is problematic, there were 483 and 236 notifications of respiratory and lymph node disease respectively in 2003.[17] The AMRLN dataset recorded 351 respiratory and 176 lymph node cases. If the two datasets are compared, then 74.7 per cent and 74.6 per cent of respiratory and lymph node notifications respectively were bacteriologically confirmed. Over the period, 2000–2003, the range of bacteriologically confirmed respiratory or lymph node disease was 70.5–88.5 per cent or 63.5–86 per cent respectively.[14–16, 18–20]

In 2003, almost all isolates were identified as *M. tuberculosis* (n=782), the remaining two isolates being a single *M. africanum* and an *M. bovis*. In the past decade, the absolute number of cases caused by *M. bovis* has fallen from a high of 10 and nine cases in 1996 and 1997 respectively down to four, two, one, zero, and one cases in the years 1999–2003. The number of cases caused by *M. africanum* has remained at a steady, low level between zero and seven cases per year over the past decade. Hence, a positive result by a rapid method that detects the presence of MTBC in a clinical specimen most likely indicates *M. tuberculosis* rather than any other member of the MTBC.[8–16]

A total of 81 isolates (10.3%) of *M. tuberculosis* (n=80) and *M. africanum* (n=1) were resistant to one at least one of H, R, E, or Z. This finding is consistent with previous reports provided by the AMRLN where drug resistance has remained between a high of 17.7 per cent in 1989 and a low of 7 per cent in 1994.[6–16] For 2003, mono-resistance to isoniazid, ethambutol, rifampicin, and pyrazinamide was detected in 45, three, two, and one isolates respectively. Again, this finding is consistent with previous data.

The level of acquired resistance in Australia remains low with only 2/81 (2.5%) cases with a drug resistant strain being described as such. Interestingly, both cases were MDRTB, one from Papua New Guinea and the other from India. Most cases with drug resistant strains (93.8%) occurred in the overseas born and reflects previous data.[14–16] These findings reflect more upon the performance of the TB program from their country of origin rather than the clinical management of these patients in Australia. Therefore, as a measure of performance of Australia's TB control program, the national drug resistance data has limited usefulness.

Results of NAAT were evaluated with smear result and whether the sample was from respiratory or non-respiratory sites. Consistent with previous reports, 96.4 per cent of smear- and culture- positive respiratory specimens were NAAT-positive.[21–23] Importantly, 3/83 (3.6%) of smear positive respira-

tory specimens that subsequently grew MTBC were NAAT negative and only 35.3 per cent of smear-negative culture positive non-respiratory specimens were NAAT-positive. Inhibitors of amplification enzymes may be present in any specimen, especially those of non-respiratory origin. Clinicians must recognise the limited sensitivity of NAAT particularly on non-respiratory samples and laboratorians must remember that NAAT should have an internal amplification inhibitor control to validate a negative result.[23,24] NAAT should be considered a supplemental test that does not replace microscopy or culture. Culture also remains the priority because an MTBC isolate is required for specific identification to species level, drug susceptibility testing and genotyping.

The decision to perform NAAT on a specimen needs to consider several factors, including whether a sufficient amount of specimen has been set aside for microscopy and culture, the degree of clinical suspicion for TB, and the specimen type.[21,24] Public health considerations can also influence the decision to perform NAAT. For respiratory smear-positive with no risk factors for TB, the differential diagnosis also includes disease caused by environmental mycobacteria. A negative NAAT result in this setting supports the diagnosis of NTM disease for which the drug treatment is different, and the public health actions of isolation and contact tracing may be unnecessary. Smear-negative patients may also be suitable candidates for NAAT when the clinical suspicion of TB is moderate to high and multiple sputum specimens are smear negative NAAT may clarify the diagnosis without resorting to further, more-invasive investigations such as bronchoscopy. In contrast, smear negative respiratory specimens from patients with a low probability of TB are not suitable candidates for NAAT due to the test's low sensitivity for the diagnosis of smear negative pulmonary TB.[21,22,23]

For the first time, sufficient data was available to evaluate results of NAAT on non-respiratory specimens. As expected, the correlation for smear positive, non-respiratory specimens that were MTBC culture positive and NAAT positive was lower at 86.4 per cent, most likely due to the presence of inhibitors. For smear negative, non-respiratory specimens that were MTBC culture positive, only 18/51 (35.3%) were NAAT positive. The level of sensitivity for NAAT lies somewhere between that of culture (~10-100 colony forming units per mL) and microscopy (~10,000 acid fast bacilli per mL) and the majority of false-negative results are due to low concentrations of MTBC.[25] Non-respiratory specimens generally have a far lower smear-positivity rate than respiratory specimens (e.g. Table 2). Specimens from non-respiratory sites such as tissue samples or fluids from usually sterile sites (e.g. cerebrospinal, meningeal, pleural, ascitic, pericardial) tend to be paucibacilliary and also have a higher proportion of specimens containing amplification inhibitors. There are circumstances, most notably when meningeal TB is suspected, that requests for NAAT are received. Only when sufficient specimen has been processed for microscopy and culture should NAAT be considered.[25,26]

There is no place for using NAAT for checking the response to treatment. NAAT does not differentiate nucleic acid from viable and non-viable MTBC and furthermore, MTBC nucleic acid may remain *in situ* for an extended period of time. The Centers for Disease Control and Prevention also recommended that NAAT should not be used on specimens from patients who have received greater than seven days of specific anti–TB treatment or have been on treatment within the previous two months.[24]

In summary, the 2003 AMRLN database on positive TB cultures shows a steady rate of laboratory-proven TB disease in Australia. The prevalence of drug-resistant disease also remains unchanged. Most patients with drug-resistant TB were migrants hence the rate of drug-resistant disease in Australia is an unreliable performance indicator for our national TB control program. Finally, the AMRLN database has provided further evidence on the performance characteristics of NAAT. These findings confirm that NAAT should not be performed automatically on every TB specimen or TB suspect. Furthermore, as with all mycobacterial investigations, the decision to perform NAAT and the result interpretation requires close liaison between the clinician and laboratory staff.

## *Acknowledgements*

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

- Institute of Medical and Veterinary Science, Adelaide, South Australia.
- Queensland Health Pathology Services, The Prince Charles Hospital, Chermside, Queensland.
- Victorian Infectious Diseases Reference Laboratory, North Melbourne, Victoria.
- Western Australian Centre for Pathology and Medical Research, The Queen Elizabeth II Medical Centre, Nedlands, Western Australia.
- Institute of Clinical Pathology and Medical Research, Westmead Hospital, Westmead, New South Wales.

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# Report of the Australian Rotavirus Surveillance Program 2003–2004

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## Abstract

**The National Rotavirus Reference Centre together with collaborating laboratories Australia-wide has conducted rotavirus surveillance since June 1999. This report describes the serotypes of rotavirus strains responsible for the hospitalisation of children with acute gastroenteritis during the period 1 July 2003 to 30 June 2004. We examined 688 faecal samples using monoclonal antibody immunoassays, reverse transcription-polymerase chain reaction and polyacrylamide gel analysis. This revealed that serotype G1 has re-emerged as the major serotype nationally, representing 40 per cent of all strains, followed by serotype G3 (25.7%) serotype G2 (17.1%) and serotype G9 (11.7%). However, there is substantial geographic variation in the prevalence of rotavirus serotypes. These findings have implications for vaccine development strategies which have targeted prevention of disease due to serotypes G1-G4. *Commun Dis Intell* 2004;28:481–485.**

*Keywords: rotavirus, surveillance*

## *Introduction*

Group A rotaviruses are the single most important cause of severe gastroenteritis in young children worldwide. An estimated 400,000–500,000 children die annually of severe diarrhoea, however few deaths occur in developed countries.[1] Rotavirus induced disease accounts for between 25–50 per cent of all hospitalisations for diarrhoea, with 10,000 Australian children hospitalised each year.[2] There is wide acceptance of the need for a vaccine to prevent rotavirus disease in children under five years of age throughout the world, with several vaccines under development. National rotavirus surveillance provides an understanding of the epidemiology of rotavirus in Australia, an important component for success in vaccine development and implementation.

The previous rotavirus surveillance report from the National Rotavirus Surveillance Program, covering the period July 2002–June 2003, highlighted the potential importance of uncommon serotypes such as serotype G9. Serotype G9 was first described in Australia in 1997[3] and since then has steadily increased in prevalence to become the dominant serotype nationally during the 2002/2003 period, representing 74.7 per cent of samples.[4] This was the first time since surveillance began in 1993, that serotype G1 was not the dominant type in Australia.

The surveillance and characterisation of rotavirus strains causing annual epidemics of severe diarrhoea in young children in Australia continues to be undertaken by the National Rotavirus Reference Centre in Melbourne, together with seven collaborating centres. In this report we describe the results of the Australian Rotavirus Surveillance program for the period 1 July 2003 to 30 June 2004, and identify the geographic distribution of the predominant rotavirus serotypes.

## *Methods*

Rotavirus detection was undertaken by enzyme immunoassay (EIA) or latex agglutination in collaborating laboratories. Rotavirus positive specimens were collected, stored frozen and forwarded to Melbourne, together with relevant age and sex details. Specimens were then tested using an in-house monoclonal antibody (MAb) based serotyping EIA. The EIA employed a panel of MAbs specific for the major glycoprotein VP7 of the outer capsid of the five major group A human rotavirus serotypes (G1, G2, G3, G4 and G9).[5] Strains which could not be assigned a serotype were genotyped by reverse transcription/polymerase chain reaction (RT/PCR) using serotype specific oligonucleotide primers.[6] Polyacrylamide gel electrophoresis (PAGE) was used to classify rotavirus strains genetically into electropherotypes and to confirm the sharing of the same electropherotype between collaborating centres.

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## *Results*

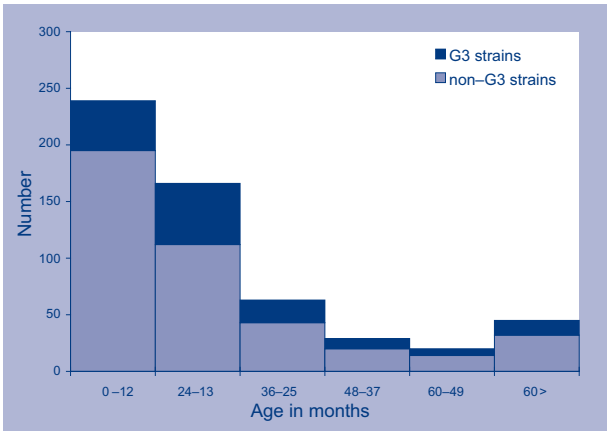
### Number of isolates

Six hundred and eighty-eight specimens were received for analysis from Melbourne and the collaborating centres in Western Australia, Northern Territory and New South Wales. During the sampling period, no samples were collected from South Australia, Queensland or Tasmania. Collections in those states resumed in the financial year beginning July 2004. A total of 608 specimens were confirmed as rotavirus positive using our in-house EIA assay. Specimens containing insufficient specimen for testing (n=29) or specimens that were not confirmed to be positive for rotavirus (n=51) were not analysed further.

### Age distribution

The overall age distribution of the children with acute gastroenteritis was typical of rotavirus infection (see Figure 1). In the reporting period, 42.5 per cent of cases were from infants 12 months of age or less, 29.5 per cent were from patients 13–24 months of age, and 11.2 per cent were from patients 25–36 months of age. Overall, 83.3 per cent of samples were from children three years or less, and 92 per cent were from children five years or less. When the age distribution was broken down according to serotypes, patients aged more than 12 months were significantly more likely to have a serotype G3 infection (69.9%) than infants aged less than 12 months of age (30.1% Chi-square = 11.87, P<0.001, Figure 1).

**Figure 1. Age distribution versus infecting serotype**




Slightly more male children than female were admitted to hospital during the year, (male to female ratio 1.3:1).

### Serotype distribution

The rotavirus serotypes identified in Australia from July 1, 2003 to June 30, 2004 are shown in the Table. Serotype G1 was the most common type identified, representing 40 per cent of all specimens. It was the dominant strain in only two centres (Melbourne and Sydney), and was identified in six of the seven centres. Serotype G3 was the second most common serotype nationally and represented 25.7 per cent of specimens over all. It was identified in four centres and was the dominant type in Perth and Western Australian Path Centre The two Western Australian centres represent different geographic locations, one urban (Perth) and one remote, north western Western Australia (WA Path Centre). Serotype G2 was identified in three centres and represented 17.1 per cent of all specimens. It was the dominant type in Alice Springs. Serotype G9 was the third most common serotype and represented 11.7 per cent of all specimens. It was identified in all seven centres, but, was the dominant type in only two centres (Darwin and Gove). A single serotype G4 isolate was identified in Melbourne.

During the reporting period, 1.3 per cent of the rotavirus samples analysed contained multiple serotypes, and in 4.1 per cent of the samples a serotype was not identified. The latter could be samples with virus numbers below the detection limits of our assays. Alternatively, these could represent unusual serotypes not identified using standard methods. For example, we identified five specimens from Alice Springs which exhibited a super short RNA electropherotype but were not typeable. Future studies will include further characterisation of the genes encoding the outer capsid proteins of these strains.

## *Discussion*

National rotavirus surveillance from 1 July 2003 to 30 June 2004 was highlighted by the finding that serotypes G1, G2, G3 and G9 were each the dominant type in at least one of the collaborating centres.

Serotype G1 was the dominant serotype nationally comprising 40 per cent of all strains. This replaced serotype G9, which was the dominant strain nationally for the previous two years.[4] Serotype G9 persisted as the dominant strain in Darwin, but not elsewhere in Australia. Serotype G1, G2 and G3 were the common types in other centres. Serotype G1 was identified in six centres and was the dominant type in Melbourne and Sydney and the second most common serotype in two other centres Perth and WA Path centre. The re-emergence of serotype G1 as the dominant strain reinforces the importance of this serotype. G1 was the dominant serotype in

**Table. Rotavirus G serotypes in Australia, 1 July 2003 to 30 June 2004**

| Centre | Total number | Serotype percentage (number) | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | G1 | G2 | G3 | G4 | G9 | Mixed serotypes | No result‡ |
| Melbourne | 131 | 86.2 (113) | 0 | 2.3 (3) | 0.8 (1) | 3.8 (5) | 0.8 (1) | 6.1 (8) |
| Sydney | 40 | 75 (30) | 0 | 0 | 0 | 20 (8) | 2.5 (1) | 2.5 (1) |
| Perth* | 148 | 31.1 (46) | 4 (6) | 56.1 (83) | 0 | 5.4 (8) | 2 (3) | 1.4 (2) |
| WA PathCentre* | 137 | 32.1 (44) | 6.6 (9) | 49.7 (68) | 0 | 5.8 (8) | 0.7 (1) | 5.1 (7) |
| Alice Springs | 115 | 4.4 (5) | 77.4 (89) | 0 | 0 | 10.4 (12) | 1.7 (2) | 6.1 (7) |
| Darwin | 33 | 15.1 (5) | 0 | 6.1 (2) | 0 | 78.8 (26) | 0 | 0 |
| Gove | 4 | 0 | 0 | 0 | 0 | 100 (4) | 0 | 0 |
| | 608† | 40 (243) | 17.1 (104) | 25.7 (156) | 0.2 (1) | 11.7 (71) | 1.3 (8) | 4.1 (25) |

* The two Western Australian centres represent different geographic area, one urban (Perth) and one remote Western Australia (WA Path Centre).

† An additional 80 specimens were omitted from analysis due to insufficient sample or specimen was not confirmed to be rotavirus positive.

‡ No result - unable to be sertoyped with monoclonal antibodies or genotyped by RT/PCR.

surveys conducted in Australia from 1993 to 1996[7], and during the 1999/2000 and 2000/2001 surveys.[8, 9] These findings are supported by epidemiological studies conducted throughout the world which have continued to identify serotype G1 as the dominant serotype.[10–14]

The decline in prevalence of serotype G9 has been as dramatic as its emergence. Serotype G9 was first identified during Australia-wide surveillance in 1997,[3] and became the second most prevalent serotype nationally during the 1999/2000 and 2000/2001 surveys, representing 10 per cent and 18.1 per cent respectively of specimens collected in those years.[8, 9] G9 became the dominant strain nationally in 2001/2002, comprising 40 per cent of the strains[15] and 2002/2003 comprising 74.7 per cent.[4] However, during the current survey, G9 while present in each centre, represented only 11.7 per cent of all strains. The decline in the prevalence of G9 strains around Australia in 2003/2004 was associated with an increase in the prevalence of G1 and G3 strains.

The increase in prevalence of serotype G3 in Australia has been dramatic. During the four previous surveys conducted Australia-wide, (1999/2000, 2000/2001, 2001/2002 and 2002/2003) serotype G3 represented less that two per cent of all strains. However during this survey, the prevalence of serotype G3 has increased to 25.7 per cent and was the dominant strain in West Australia. The high prevalence of G3 in Australia is remarkable when compared with the low prevalence rates of this serotype reported in other countries.[10–14] Interestingly, the emergence of serotype G3 has also been recently identified in two regions (Qinhuangdao and Zheng zhou) in a recent study from China,[16] and represented 45 and 80 per cent of isolates from each region. Whether these G3 strains move eastward from Western Australia to Sydney and Melbourne, and have an Australia-wide impact similar to serotype G9, will be followed with interest during the next rotavirus season. The increase in prevalence of serotype G3 appears to have been associated with changes in the age distribution of children infected with rotavirus, when compared to non-G3 strains. While the majority of children (87%) infected with rotavirus were under three years of age, the G3 strains infected children aged 13– 24 months more frequently than children aged 12 months or less ($p<0.001$). In contrast, over 50 per cent of the children infected with the other rotavirus serotypes were under 12 months of age. This data suggests that pre-existing antibodies may not protect against subsequent severe re-infection with the serotype G3 strain.

An outbreak of severe gastroenteritis again swept through Alice Springs in Central Australia causing a major impact on health care facilities in January 2004. Serotype G2 was responsible for this year's outbreak. The previous G2 outbreak in Alice Springs in 1993, was shown to be due to an unusual G2 strain derived by reassortment between subgroup I and subgroup II human strains.[17] The 2004 strain possessed the standard short pattern electropherotype and subgroup I antigenicity. Serotype G2 strains have previously been responsible for intermittent epidemics in several of the other centers during the past 12 years, including in Perth in 1993, Melbourne in 1994, and Sydney in 2001.[7,9]

These results together with those of previous years highlight the continuing change in the prevalence and emergence of new rotavirus serotypes. Multi-centre surveillance of rotavirus is important to continue to monitor strains in Australia. These results contribute to worldwide knowledge of rotavirus epidemiology and essential to inform the development of new rotavirus vaccines.

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**Northern Territory**

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B Truscott and members of the Pathology Department, Western Diagnostic Pathology, Tiwi, NT, 0810.

F Morey and members of the Microbiology Department, Alice Springs Hospital, Alice Springs, NT, 0971.

K Carter and members of Pathology Department, Gove District Hopsital, Nhulunbuy, NT 0880.

**New South Wales**

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**Victoria**

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# OzFoodNet: enhancing foodborne disease surveillance across Australia: quarterly report, July to September 2004

*The OzfoodNet Working Group*

## *Introduction*

The Australian Government Department of Health and Ageing established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease. This quarterly report documents investigations of outbreaks of gastrointestinal illness and clusters of disease potentially related to food occurring around Australia. For information on sporadic cases of foodborne illness, see Communicable Disease Surveillance, Highlights for 3rd quarter 2004 in this issue of *Communicable Diseases Intelligence*.

This report summarises the occurrence of foodborne disease outbreaks and cluster investigations between July and September 2004. Data were received from OzFoodNet representatives in all Australian states and territories and a sentinel site in the Hunter region of New South Wales. The data in this report are provisional and subject to change, as results of outbreak investigations can take months to finalise. We would like to thank the investigators in the public health units and state and territory departments of

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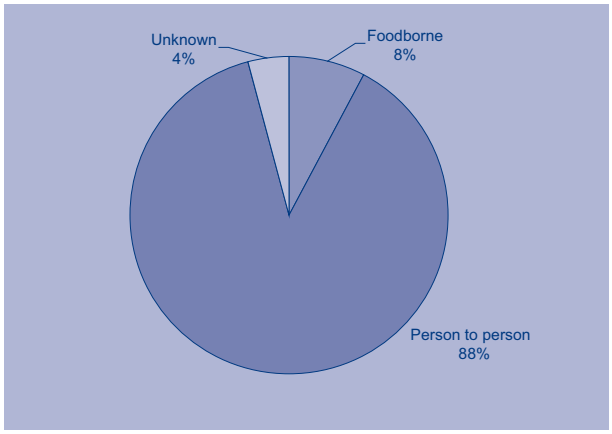
All data are reported using the date the report was received by the health agency.

health as well as public health laboratories and local government environmental health officers who collected data used in this report.

## *Foodborne disease outbreaks*

During the third quarter of 2004, OzFoodNet sites reported 313 outbreaks of foodborne or enteric illness. As usual the vast majority of these (87%, n=274) resulted from person-to-person spread of infection. The figure shows the proportion of the different modes of transmission. In total, 6,994 people were affected with 113 people hospitalised. Twelve deaths were reported. Ten of the deaths occurred in aged care facilities during outbreaks of norovirus infection while the remaining two deaths were associated with cases of *Listeria* infections in severely ill hospitalised patients.

**Figure. Mode of transmission for outbreaks of gastrointestinal illness reported by OzFoodNet sites, July to September 2004**




There were 25 outbreaks of illness where food was suspected or proven to be the primary mode of transmission. This compares with 24 and 37 outbreaks in the first and second quarters of 2004, respectively. *Salmonella* Typhimurium was the causative agent for six outbreaks, while *Campylobacter* and norovirus were each responsible for three outbreaks. Of the remaining outbreaks, one each was caused by *Clostridium perfringens*, Ciguatera toxin, *Salmonella* Enteritidis, *Salmonella* Virchow, *Salmonella* Stanley and *Listeria monocytogenes*. An aetiological agent was not identified for seven of the outbreaks.

Seven of the outbreaks were associated with meals served in restaurants and another seven with food served in private residences. Two were associated with commercial caterers or take away food outlets. Nine of the outbreaks occurred in July, six in August and 10 in September.

To investigate these outbreaks, sites conducted ten cohort studies and two case control studies. For 11 outbreaks, only descriptive data were collected and in two outbreaks no individual case data was collected. In three outbreaks, investigators obtained microbiological evidence linking a food vehicle to illness, and analytical epidemiological evidence in a single outbreak. For the remaining outbreaks, investigators obtained descriptive epidemiological evidence implicating the food vehicle or suggesting foodborne transmission.

In New South Wales there were six outbreaks of foodborne illness, three of which were associated with different phage types of *Salmonella* Typhimurium. One of these was caused by *S.* Typhimurium U290 linked to homemade Chinese style minced fish balls, which affected 11 people. *Salmonella* Typhimurium 126 was associated with homemade tiramisu in an outbreak affecting 11 people. A sample of a wash from a raw egg used to make the dessert tested positive for *S.* Typhimurium 126. The eggs were traced back to an 'organic' egg farm. There were two outbreaks associated with restaurants and one with a school where no agent or food vehicle were identified.

Victoria reported four outbreaks of foodborne disease. One outbreak of *Salmonella* Stanley in a boarding school affected 33 people, with four admitted to hospital. Food served at the school was considered the most likely source of the infection but there was possible person-to-person spread later in the outbreak. *Salmonella* Typhimurium 126 was associated with an outbreak at a conference centre. There were a total of 24 cases from three groups who attended the conference centre. No food vehicle was identified, although tiramisu made with raw eggs was suspected as the source of illness amongst guests at a wedding reception at the centre. Twenty-four cases were associated with an outbreak of campylobacteriosis in an aged care facility. Most of the cases appeared to contract their illness at a barbecue, although no specific food was identified and there may have been some secondary spread. There was an outbreak of illness associated with a restaurant that affected 45 people. The time of onset of illness and the pattern of illness suggested *Clostridium perfringens* infection and this organism was isolated in high numbers from one faecal sample from a restaurant patron but toxin testing was not carried out. Curries at the restaurant were served banquet style and may not have been kept hot enough to prevent bacterial proliferation.

In Queensland, there were nine outbreaks of foodborne illness investigated. Six people were ill from *Clostridium perfringens* after a meal of take away pizza. *C. perfringens* was isolated from various meats used as toppings and from the stool of

**Table. Outbreaks of foodborne disease reported by OzFoodNet sites,* July to September 2004**

| State | Month | Setting | Agent responsible | Number exposed | Number affected | Evidence† | Responsible vehicles |
|---|---|---|---|---|---|---|---|
| NSW | July/August | Home | *S.* Typhimurium U290 | 11 | 11 | M | Chinese style minced fish balls |
| | August | School | Unknown | 300 | 108 | D | Unknown |
| | September | Restaurant | Unknown | Unknown | 11 | D | Unknown |
| | September | Restaurant | Unknown | Unknown | 13 | D | Unknown |
| | September | Home | *S.* Typhimurium 126 | 14 | 11 | M | Tiramisu dessert |
| | September | Institution | *S.* Typhimurium 135a | 50 | 5 | D | Unknown |
| Qld | July | Home | Ciguatoxin | 4 | 4 | D | Grey Mackerel |
| | July | Caterer | Norovirus | Unknown | 26 | D | Unknown |
| | July | Bakery | *S.* Typhimurium 135a | Unknown | 5 | D | Custard Fruit Tarts |
| | August | Home | *C. perfringens* | 11 | 6 | M | Meat lovers pizza |
| | August | Camp | *S.* Virchow 8 | Unknown | 5 | D | Unknown |
| | August | Home | Norovirus | 9 | 7 | D | Pizza |
| | September | Home | *S.* Enteritidis 26 | 60 | 17 | D | Unknown |
| | September | Caterer | Norovirus | 96 | 16 | D | Unknown |
| | September | Take-away | *Campylobacter* | Unknown | 2 | D | Chicken kebab |
| SA | July | Restaurant | Unknown | Unknown | 4 | D | Unknown |
| | July | Restaurant | *Campylobacter* | Unknown | 4 | D | Unknown |
| | July | Home | *S.* Typhimurium 9 | 5 | 4 | D | Home made icecream |
| | August | Restaurant | Unknown | 47 | 8 | D | Unknown |
| | September | Home | Unknown | Unknown | 15 | D | Pizza |
| | September | Hospital | *Listeria monocytogenes* | Unknown | 2 | D | Unknown |
| Vic | July | Caterer | *S.* Typhimurium 126 | 139 | 21 | D | Unknown |
| | July | Institution | *S.* Stanley | unknown | 33 | D | Unknown |
| | August | Restaurant | Unknown | unknown | 45 | D | Unknown |
| | September | Aged Care | *Campylobacter* | 79 | 24 | D | Barbecue |

* No foodborne outbreaks reported from the Australian Capital Territory, the Northern Territory, Tasmania or Western Australia.

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

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

one case. Inadequate refrigeration of the meats may have contributed to the proliferation of the organism in the food. *Salmonella* Virchow 8 was isolated from the faeces of five people attending a school camp, although no food vehicle was identified. An environmental health inspection of the camp kitchen found several food hygiene issues including time-temperature abuse during food preparation and storage. After sharing a takeaway pizza meal seven of ten people became ill. While pizza was the common food, no particular variety was consumed by cases and person-food-person transmission of norovirus appeared to be the mode of transmission. There was one outbreak of ciguatera poisoning affecting four people after a meal of grey mackerel. There was one outbreak of norovirus associated with a catered wedding. Food handlers working for the catering company reported family members with similar illness, indicating a mixture of person-food-person spread. An ill food handler was suspected as the source of illness in another outbreak of norovirus affecting 16 people.

Queensland also reported five cases of *Salmonella* Typhimurium 135a following consumption of apple tarts and custard fruit tarts from a single bakery. An almond sauce containing raw eggs on the tarts was suspected as the source of the outbreak. An investigation of the farm supplying the eggs found no *Salmonella*, but the farm had no quality assurance program and eggs were inadequately cleaned. Seventeen of 60 people became ill with *Salmonella* Enteritidis 26 infection following a wedding reception held at a private home. A wide variety of foods were served, but no particular vehicle was identified. A kebab shop was associated with two cases of campylobacteriosis. Cases purchased the food during busy periods, possibly indicating inadequate cooking and cross contamination.

A total of six outbreaks were investigated in South Australia during the quarter. Two cases of listeriosis were associated with the same hospital. Pulsed field gel electrophoresis profiles of isolates from patients suggested the strains were related but a review of food histories did not identify a common food exposure. Four out of five people became ill with *Salmonella* Typhimurium 9 infection after eating homemade ice cream which contained raw eggs. Tracing back the eggs to an individual farm was not possible due to many farms supplying a single facility. There were four cases of campylobacteriosis associated with a restaurant. No definitive food vehicle was established but an inspection of the restaurant revealed a number of food safety breaches including inadequate temperature control. There were three other outbreaks where foodborne transmission was suspected but no pathogen was identified. One of these was an outbreak associated with a national franchised pizza chain, where illness was investigated in three cohorts of people. The other two involved outbreaks at restaurants.

## *Comments*

During the quarter there were two outbreaks of *S.* Typhimurium 126 in two different states with a tiramisu dessert being a possible vehicle of the pathogen for both outbreaks. The raw eggs used to make the dessert for both outbreaks were sourced from different organic egg farms located in each state. A South Australian outbreak of *S.* Typhimurium 9 associated with homemade ice cream also implicated raw eggs. These and other egg-related outbreaks highlight the need for health departments to thoroughly document the sources of contamination. The fact that the South Australian investigation was unable to trace back products to their source highlights a common problem where food ingredients are suspected as the cause of gastroenteritis outbreaks.

The outbreak of *Salmonella* Enteritidis 26 in Queensland was unusual in that outbreaks of this phage type are rare. The vehicle of pathogen in this outbreak was not identified, despite intensive investigation. *Salmonella* Enteritidis is a serious concern for primary industry due to the ability of some Enteritidis phage types to cause intra-ovarian infection in egg-laying poultry and egg infection has major cost implications.[1] In Australia, OzFoodNet investigates all cases of *Salmonella* Enteritidis to monitor for the emergence of invasive phage types, such as phage type 4.

There was an outbreak of *Salmonella* Stanley during the quarter in a Victorian boarding school where the source was not identified. *Salmonella* Stanley infections are commonly acquired in Asia, although a small number of infections are acquired in Australia each year. In 2001, this serotype was the cause of an international outbreak associated with Chinese peanuts.[2]

The outbreak of *C. perfringens* associated with a meal of take away pizza highlights that the length of cooking time for pizza may not kill this anaerobic spore forming organism. It also demonstrates the need to keep ingredients chilled to prevent growth of bacteria. *C. perfringens* is a hardy organism and vegetative organisms can grow at temperatures between 15–50ºC.[3] There were three outbreaks of illness due to pizza during the quarter. OzFoodNet sites carefully reviewed the results of these investigations to determine if there were any links between the outbreaks, as some franchised chains centralise food preparation and distribution.

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# A report from the Communicable Diseases Network Australia, July to September 2004

*The Communicable Diseases Network Australia (CDNA) consists of communicable disease authorities from various Australian Government agencies and state and territory health authorities, in addition to expert bodies and individuals in the specific areas of communicable disease epidemiology, clinical management, disease control and laboratory diagnosis. The CDNA provides national public health leadership and co-ordination on communicable disease surveillance, prevention and control, and offers strategic advice to governments and other key bodies on public health actions to minimise the impact of communicable diseases in Australia and the region.*

**Summary of major CDNA activities for the period July to September 2004**

During this quarter, CDNA identified several projects/areas to be addressed including:

- proposed changes to the *Australian Immunisation Handbook* 8th Edition 2003 relating to the use of Japanese encephalitis (JE) vaccine for people travelling to Papua New Guinea (PNG);
- acute rheumatic fever and rheumatic heart disease (ARF-HD) as a notifiable disease;
- development of norovirus and meningococcal guidelines;
- policies for the management of health care workers and blood-borne viruses;
- sexually transmitted infection (STI) surveillance in states and territories;
- the management of avian influenza; and
- a recently reported case of Creutzfeld-Jakob disease (CJD) in Victoria.

**Changes to *Australian Immunisation Handbook* on Japanese encephalitis and travel to Papua New Guinea**

CDNA reconsidered available evidence in relation to JE vaccination travellers to PNG, i.e. a report provided by the Australian Technical Advisory Group on Immunisation (ATAGI) titled '*Japanese Encephalitis Virus: Its potential risk to human and animal health in Australia*', and agreed to accept the ATARGI recommendation to amend the *Australian Immunisation Handbook* to recommend JE vaccination for '*travellers intending to spend a month or more in PNG, particularly if the travel is during the wet season'.*

**Acute rheumatic fever and rheumatic heart disease as a notifiable disease**

The report *Should Acute Rheumatic Fever and Rheumatic Heart Disease be Notifiable*?, prepared by Northern Territory MAE student Philippa Burns, was submitted to CDNA in July 2004, for consideration to make ARF-HD a nationally notifiable disease (ARF-RHD is already a notifiable disease in Northern Territory and Queensland). The North Queensland Tropical Health Unit is preparing a report on the last five years of ARF-RHD data, which may have some bearing on the argument of national notification. This issue will be further discussed at the upcoming CDNA face-to-face meeting on 28–29 November 2004.

**Development of norovirus guidelines**

In response to continuing reporting of norovirus outbreaks in most states and territories, CDNA agreed to undertake the development of national norovirus guidelines to reduce the incidence of norovirus outbreaks, particularly in settings such as nursing homes and child care facilities. It is anticipated that the guidelines will be available in the second half of 2005.

**Development of meningococcal guidelines**

A CNDA sub-committee, the Meningococcal Disease Committee (MDC), has recently been reformed. A primary task for the MDC is to review the *Guidelines for the Early Clinical and Public Health Management of Meningococcal Disease in Australia*. Consideration of the meningococcal data fields in relation to enhanced surveillance national datasets is another MDC priority.

### Review of sexually transmitted infection surveillance at the state and territory level

CDNA recently endorsed the report, provided by the National Centre on HIV Epidemiology and Clinical Research on behalf of the STI Surveillance Committee (a sub-committee of the Inter-governmental Committee on AIDS/HIV, hepatitis C and Related Diseases. The report was commissioned to document and compare the current state and territory health authority surveillance activities for the four notifiable bacterial STIs, chlamydia, donovanosis, gonorrhoea and syphilis, and to provide procedural recommendations for future national STI surveillance within the framework of the National Notifiable Diseases Surveillance System. The findings of the report indicate the need for a nationally coordinated STI surveillance program. The report will shortly be made available on the CDNA website.

### Management of avian influenza

In September 2004, the National Influenza Pandemic Action Committee (NIPAC), advised CDNA that they were developing operational guidelines for implementation of the *National Pandemic Influenza Action Plan*. The initial strategy is containment via use of anti-virals, quarantine isolation and contact tracing. If and when an epidemic is declared in Australia the second part of the strategy, maintaining essential services, will come into effect. NIPAC will provide a draft of the operational response plan to CDNA in October 2004 for comment.

### Classical Creutzfeld-Jakob disease case in Victoria

In September 2004, the Jurisdictional Executive Group of CDNA convened to inform members of a classical CJD case in a cranial surgery patient (now deceased) at Royal Melbourne Hospital. Issues covered included notification of the 1,056 surgical patients who were potentially exposed, instrument sterilisation procedures, the impact on future surgery and blood and organ donation. Investigations on the possible cause of transmission continue.

### How to contact CDNA

Key activities of CDNA will be reported quarterly in *Communicable Diseases Intelligence*. For further information, please contact the CDNA Secretariat at: CDNA@health.gov.au, or telephone +61 2 6289 7983 or refer to the CDNA webpages at http://www.health.gov.au/internet/wcms/Publishing.nsf/Content/cda-cdna-index.htm and http://www.nphp.gov.au/workprog/cdna/index.htm

# Surveillance of adverse events following immunisation for children aged less than 7 years, 1 January to 30 June 2004

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Surveillance of adverse events following immunisation (AEFI) is an integral component of the management of immunisation programs. In Australia, national AEFI surveillance data have been collated in the Adverse Drug Reactions Advisory Committee (ADRAC) database since 2000. AEFIs are notified to ADRAC by state and territory health departments, health care professionals, vaccine companies and the public. Two reports summarising national AEFI data have been published in *Commun Dis Intell* for vaccines received between January 2000 and September 2002,[1] and between October 2002 and December 2003.[2]

This report summarises national AEFI surveillance data for children aged less than seven years who received vaccines between 1 January and 30 June 2004 and were reported to ADRAC by 30 September 2004. The average annual population-based AEFI reporting rates were calculated using mid-2003 population estimates. Reporting rates per 100,000

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doses of vaccine were calculated for seven vaccines that are funded by the National Immunisation Program using denominator data from the Australian Childhood Immunisation Register (ACIR) for the period 1 January to 30 June 2004. Reporting rates could not be estimated for some vaccines due to inadequate denominator data. These include the pneumococcal, varicella, influenza and monovalent hepatitis B vaccines.

The data reported here are provisional only. It is important to note that AEFIs are defined as medically important events that are temporally associated with immunisation but are not necessarily causally associated with immunisation. Readers are referred to previous reports for a description of the national AEFI surveillance system,[1] methods used to analyse the data[1,2] and information regarding limitations and interpretation of the data.[2] Often, more than one vaccine is listed as suspected of involvement in the reported adverse event, so the number of vaccines will be greater than the number of AEFI records analysed.

There were a total of 219 records of adverse events following immunisation (AEFI) (24.5 per 100,000 population) for children aged less than seven years where suspected vaccines were administered in the first six months of 2004 to children aged less than seven years. This was a 54 per cent reduction on the 474 records (53.0 per 100,000 population) for the corresponding six month period in 2003. Sixty-six percent (n=144) of records were for children aged 2 to <7, with 11 per cent (n=23) for children aged 1 to <2 years and 24 per cent (n=52) for children aged <1 year. The relative contribution of children aged 1 to <2 years is lower (down from 33%) compared with the first six months of 2003, while that of children aged 2 to <7 years is higher (up from 50%). The male to female ratio was 1.3:1.0, and similar to that seen previously.

Of the 219 records analysed, 19 (8.7%) were defined as having 'serious' outcomes (recovery with sequelae, hospitalisation or death), and was similar to previously reported (9%).[2] Two deaths were reported: neither was thought to be causally related to vaccination. Other serious or potentially life-threatening AEFIs reported were hypotonic-hyporesponsive episode (n=4) and seizure (n=1). The most commonly reported reaction categories were injection site reaction (n=117; 53%) and fever (n=46; 21%).

One or more of the seven vaccines shown in the Table were recorded as being suspected of involvement in the reported adverse event for 205 of the 219 records analysed. The 14 records that listed other

**Table. Reporting rates of adverse events following immunisation (AEFI) per 100,000 vaccine doses,* children aged less than seven years, ADRAC database, January to June 2004**

| Suspected vaccine or AEFI category† | AEFI records‡ (n) | Vaccine doses* (n) | Reporting rate per 100,000 doses§ | Difference‖ |
|---|---|---|---|---|
| Diphtheria-tetanus-pertussis | 122 | 255,758 | 47.7 | -16.6 |
| Diphtheria-tetanus-pertussis-hepatitis B | 32 | 226,240 | 14.1 | -4.8 |
| *Haemophilus influenzae* type b | 44 | 227,364 | 19.4 | -2.4 |
| *Haemophilus influenzae* type b-hepatitis B | 8 | 127,501 | 6.3 | -5.0 |
| Poliovirus (oral or inactivated) | 50 | 478,488 | 10.4 | -4.4 |
| Measles-mumps-rubella | 78 | 239,256 | 32.6 | +1.4 |
| Meningococcal C conjugate | 51 | 179,379 | 28.4 | -10.3 |
| Total† | 205 | 1,749,075 | 11.7 | -8.1 |
| 'Certain' or 'probable' causality rating† | 73 | 1,749,075 | 4.2 | -5.8 |
| 'Serious' outcome† | 17 | 1,749,075 | 1.0 | -0.2 |

* Number of vaccine doses recorded on the Australian Childhood Immunisation Register (ACIR) and administered between 1 January and 30 June 2004.

† Records where at least one of the 7 vaccines shown in the table was suspected of involvement in the reported adverse event. AEFI category includes all records (i.e. total), those assigned 'certain' or 'probable' causality ratings, and those with outcomes defined as 'serious'. Causality ratings were assigned using the criteria described previously.[1,2] A 'serious' outcome is defined as recovery with sequelae, hospitalisation or death.[1,2]

‡ Number of AEFI records in which the vaccine was coded as 'suspected' of involvement in the reported adverse event and the vaccination was administered between 1 January and 30 June 2004. More than one vaccine may be coded as 'suspected' if several were administered at the same time.

§ The estimated adverse events reporting rate per 100,000 vaccine doses recorded on the ACIR.

‖ Difference in reporting rate per 100,000 doses for vaccines administered during January–June 2004 compared with October 2002–December 2003.[2]

suspected vaccines, for which adequate denominator data are not available, included pneumococcal (n=8), monovalent hepatitis B (n=8), varicella (n=7) and influenza (n=1) vaccines.

The AEFI reporting rates per 100,000 vaccine doses, both overall and for specific vaccines, were generally similar to those for January 2000–September 2002[1] and significantly lower than observed for the October 2002–December 2003 period[2] (Table). The largest reductions in reporting rates were for diphtheria-tetanus-pertussis (acellular) (DTPa) vaccine and meningococcal C conjugate vaccine (MenCCV) (Table, Figures 1 and 2). The reporting rate for AEFIs with outcomes defined as 'serious' for the seven vaccines declined slightly from 1.2 to 1.0 per 100,000 doses (Table).

The observed reduction in the number of AEFI reports received for children aged less than seven years during the first half of 2004, and the change in the age distribution, corresponds in time with removal of the fourth dose of DTPa vaccine (previously due at 18 months of age) from the Australian Standard Vaccination Schedule in September 2003 (Figure 1, Table), and with the completion of the MenCCV catch-up campaign for children born before 2002 (Figure 2). The lower AEFI reporting rates per 100,000 doses of MenCCV may also be due to more accurate denominator data recorded on the ACIR and/or to increased familiarity among providers about the more common, less serious side-effects of the vaccine resulting in reduced reporting to ADRAC.

## *Conclusion*

There was a marked decline in the overall AEFI reporting rates for children aged less than seven years during January–June 2004 compared with the previous year. Reporting rates of AEFIs with outcomes defined as 'serious' were stable. Annual reports summarising all AEFI data collated in the ADRAC database are planned for the future with supplementary reports summarising AEFI data for children aged less than seven years for vaccines received in first six months of each year.

**Figure 1. Reports of injection site reaction following diphtheria-tetanus-pertussis (acellular) vaccine, ADRAC database, January 2000 to June 2004, by age group and quarter of vaccination**

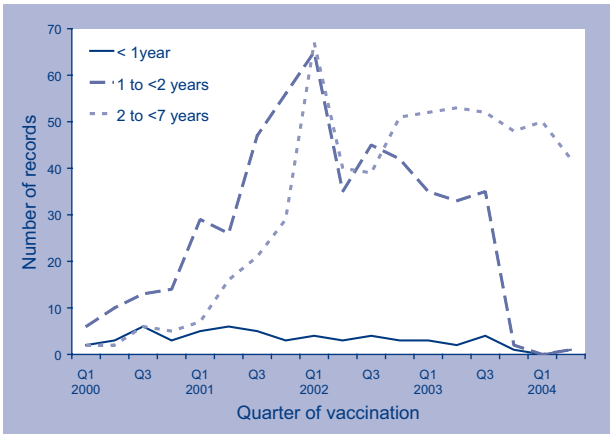
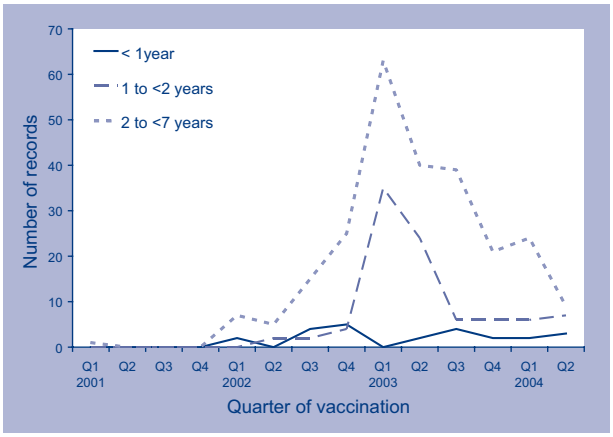



**Figure 2. Reports of adverse events reports following meningococcal C conjugate vaccination, ADRAC database, January 2001 to June 2004, by age group and quarter of vaccination**

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# The acceptance of three simultaneous vaccine injections recommended at 12 months of age

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## Abstract

**Since January 2003, vaccination with Meningococcal C conjugate vaccine (MenCCV) is recommended at 12 months of age, at the same time as the measles-mumps-rubella (MMR) and *Haemophilus influenzae* type b (Hib) vaccines. Most (83%) of a cohort of 751 children in north Queensland born in January 2003 received the three injectable vaccines simultaneously. Of the 122 children who had not received MenCCV with the other two vaccines, 88 (72%) had received it by 18 months of age. The median age of receipt of MenCCV in the children who had received the three vaccines simultaneously was 12.3 months, whereas the median age in the children who had not received it at the same time as the other two vaccines was 14.0 months. This study suggests that non-simultaneous vaccination puts children at-risk of receiving MenCCV late, or not at all, and has implications for the introduction of universal infant pneumococcal vaccination program, starting in January 2005. *Commun Dis Intell* 2004;28:493–496.**

*Keywords: multiple vaccine injections, simultaneous vaccinations, meningococcal C conjugate vaccine*

## *Introduction*

The meningococcal C conjugate vaccine (MenCCV) was introduced into the Australian Standard Vaccination Schedule (ASVS) in January 2003.[1] The vaccine is recommended to be administered at 12 months of age, at the same time as the administration of the first dose of measles-mumps-rubella (MMR) and the third dose of *Haemophilus influenzae* type b (Hib) vaccine.

Since mid-2001, with the introduction of the seven-valent pneumococcal conjugate vaccine (7vPCV) it has been recommended that Indigenous children receive three vaccine injections at two and four months of age.[2] However, the introduction of MenCCV is the first time that three simultaneous vaccine injections are recommended for all Australian children.

Little is known about the acceptability of three simultaneous injectable vaccines to Australian vaccine providers and parents of young children.[3] If three simultaneous injections are considered 'unacceptable' they might be 'split', with two vaccines given at a first visit and the third at a second visit some time later. If this occurs, it is likely that the most recently introduced vaccine (in this case MenCCV) would be the vaccine given later and there is the possibility that the return visit might either occur late or not occur at all.

The aims of this study were to determine the percentage of a cohort of children in north Queensland that had received the three vaccines simultaneously, and to describe some of the characteristics of the children that did not receive the three vaccines simultaneously.

## *Methods*

The vaccination records of all children born in January 2003, who were resident in north Queensland when they were eligible for the three vaccines (i.e. from 12 months of age onwards), were extracted from the state-wide immunisation register (VIVAS). For each child the age when the three vaccines were administered, and whether they were given simultaneously, was recorded, as was the child's Indigenous status.

Six vaccines—including three doses of Japanese encephalitis (JE) vaccine—are recommended on three visits in the 12th month of life for children on the outer islands of the Torres Strait. MenCCV is recommended for these children with the second dose of JE vaccine (one week after MMR and the

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first dose of JE vaccine). Therefore any outer island child who either had MenCCV simultaneously with the MMR and the first dose of JE vaccine, or (as recommended) simultaneously with the second dose of JE vaccine, was defined for the purpose of this study as having had the three vaccines simultaneously.

In cases where the Indigenous status of a child was not recorded, it was assumed that the child was Indigenous if he/she had received both BCG at birth and 7vPCV. The last known vaccine provider was contacted for any further clarification of a child's Indigenous status.

If one or more of the three vaccines was not recorded as having been given to any child, the relevant vaccine provider was contacted and asked for further details. If the vaccine(s) had not been given, the provider was requested to recall the child for vaccination, and if that occurred to ensure that the relevant details were forwarded for entry onto VIVAS. If none of the three vaccines had been given, the relevant Public Health Nursing Officer was informed and requested to attempt to locate the child and, if successful, to request a vaccine provider to vaccinate the child.

The vaccination record extraction for the study commenced in March 2004, and was concluded at the end of July. By this time all the study children would have reached 18 months of age.

## *Results*

The study cohort consisted of 751 children, 165 (22%) of whom were Indigenous children. Seventeen of the Indigenous children were from the outer Torres Strait islands, only eight of whom had received the three injectable vaccines simultaneously (as defined in Methods above).

Four children, all of whom were Indigenous, had not received any of the three injectable vaccines by 18 months of age. Two children (both Indigenous) who were very overdue for various infant vaccines had had four simultaneous injectable vaccines including MMR and Hib but not MenCCV, as part of catch-up schedules, and the parent of another child had refused MMR (but not MenCCV or Hib) for her child. These latter three children therefore could not have had the three recommended vaccines—MMR, Hib and MenCCV—administered simultaneously.

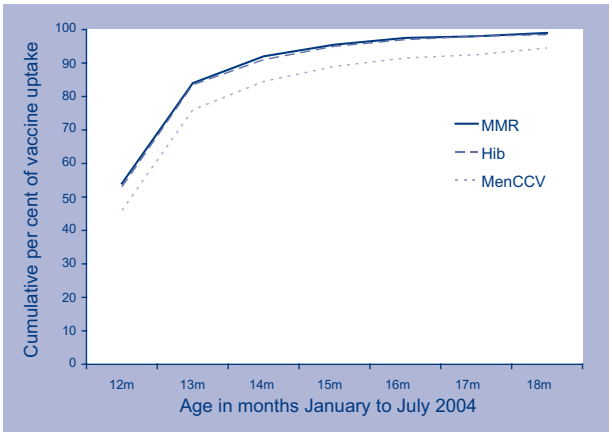
Five children had received privately-purchased MenCCV in age-appropriate schedules in the first year of life. It was assumed that these children would otherwise have had MenCCV simultaneously with the MMR and Hib vaccines, which had indeed been given at the same time. Another seven children had been given a third dose of Hib vaccine at about six months of age, but all had received a fourth dose simultaneously with MMR and MenCCV.

Altogether, 99 per cent of the 751 children had received MMR, 98.5 per cent Hib, and 94.5 per cent MenCCV by 18 months of age. Very similar percentages of Indigenous (93%) and non-Indigenous children (95%) had received MenCCV by 18 months of age.

Altogether, 622 (83%) of the children had received the three injectable vaccines simultaneously. Virtually the same percentage of Indigenous (82%) and non-Indigenous (83%) children received the vaccines simultaneously.

Of the 122 children who had not received MenCCV simultaneously with the other two vaccines, 88 (72%) had received it by 18 months of age. The median age of receipt of MenCCV in the children who had received the three vaccines simultaneously was 12.3 months, whereas the median age of receipt of MenCCV in the children who had not received it at the same time as the other two vaccines was 14.0 months. The cumulative uptake of the three vaccines by age is shown in the Figure.

**Figure. The cumulative percentage uptake of measles-mumps-rubella, *Haemophilus influenzae* type b and Meningococcal C conjugate vaccines in the study cohort, North Queensland, by month January to July 2004**




The vaccine providers who gave MMR to the 122 children who did not receive the three vaccines simultaneously were general practitioners (90; 74%), Queensland Health community health staff (29; 24%) and other providers (3; 2%).

## Discussion

A survey of parental and general practitioner attitudes to multiple vaccine injections in New South Wales in 1997 found that 'only 54 per cent of parents and 28 per cent of GPs said they would allow three injections to be given at one visit'.[3] However, this current study has demonstrated not only that most (83%) children received the three vaccines simultaneously, but also that the multiple vaccine recommendation (introduced in January 2003) had been rapidly accepted and implemented. This difference between the hypothetical attitudes and the practical realities is very similar to that seen in the United States of America prior to, and after, the introduction of inactivated polio vaccine (IPV) not combined with any other antigens.[4–6]

There are two possible reasons that might explain this difference between attitude and practice. Firstly, two (MMR and Hib) of the three vaccines were already relatively well known to parents and vaccine providers, and the other (MenCCV) was for the prevention of a disease that has attracted considerable adverse publicity in Australia in recent years. Secondly, attitudinal surveys indicated that many parents were likely to accept a recommendation of multiple simultaneous injections for their children from a vaccine provider,[3,4] and clearly most providers are comfortable not only with recommending multiple vaccine injections but also administering them.

However, it is of considerable concern that only 72 per cent of the 122 children who did not receive the three vaccines simultaneously had received the MenCCV by 18 months of age. Indeed, this figure is likely to be higher than expected for two reasons: a letter from the Australian Government's Chief Health Officer was sent in June 2004 to all parents of young children who had (apparently) not received MenCCV informing them of the availability of this vaccine free for their children, and the study process itself involved directly contacting vaccine providers and informing them to recall children who had not yet received the vaccine. Without these two reminders, it is likely that even fewer children would have received MenCCV by 18 months of age.

This study has demonstrated that non-simultaneous vaccination puts children at-risk of receiving MenCCV late, or not at all. It also confirms that it is the most recently recommended vaccine, in this case MenCCV, is the most likely to be administered separately from the other vaccines, so as to avoid more than two injections at the same time.

The findings of this study have implications for the introduction of universal infant 7vPCV, to commence in January 2005. This change to the ASVS will mean that three vaccine injections (in Queensland: DTPa-hepB, Hib, 7vPCV) are recommended for all children at two and four months of age. The exception would be if an infant's parents were prepared to purchase the hexavalent DTPa-hepB-IPV-Hib vaccine, to be administered simultaneously with 7vPCV at two, four and six months of age. This option is not considered suitable for Indigenous children.

Severe invasive pneumococcal disease can occur early in life, even in apparently low-risk infants.[7] For example, in 21 cases of pneumococcal meningitis in low-risk non-Indigenous infants in north Queensland, the median age of these children was 11 (range 4–24) months of age. Two children developed the meningitis before six months of age, and 10 (48%) had onsets before eight months of age. Therefore, it is imperative that there not be any unnecessary delays in administering 7vPCV if the vaccine is to have an optimal impact in preventing invasive pneumococcal disease.

The recommendations to administer MenCCV simultaneously at 12 months of age, and 7vPCV simultaneously at two and four months of age were made after consideration of evidence concerning adverse events and immune responses following the simultaneous administration of the relevant vaccines.[8] Vaccine providers, in particular general practitioners, need to be reassured that the available evidence indicates that these vaccines, when given simultaneously as recommended, are safe and effective. Quite simply, the most important factor influencing parents to agree to multiple injectable vaccines for their children is a confident and positive recommendation from the vaccine provider.

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# A family cluster of serogroup C meningococcal disease

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The Brisbane Southside Public Health Unit, received notification of a case of probable meningococcal septicaemia in a 22-year-old female on the 25th May 2004. Onset of illness was the 23rd May. Symptoms included lethargy, malaise and headache. On presentation, the patient was febrile and hypotensive, with an extensive purpuric rash. The patient responded to appropriate treatment and made a full recovery complicated by post infectious polyarthritis. The diagnosis was confirmed with positive blood cultures for *Neisseria meningitidis.* Five household contacts received prophylactic antibiotics on the 25th May, including the case's 2-year-old child. Other (non-household) contacts were provided with information after confirmation of the diagnosis on the 26th May.

On the 27th May 2004, the public health unit received notification that *Neisseria meningitidis* had been isolated from an eye swab. The swab was taken on the 22nd May from the right eye of the case's 2-year-old child. Investigation revealed that this child had developed purulent conjunctivitis on the 21st May after an upper respiratory illness of approximately one week's duration.

The parent case had been interstate for the duration of the child's conjunctival symptoms. The child had been taken by carers to the GP on the 22nd May in response to increasing respiratory symptoms. The GP prescribed Cefaclor (Ceclor®) for the child's respiratory infection and chloramphenicol eye drops for the conjunctivitis. At the time of notification, the child was well and had completed a two day course of rifampicin in addition to the chloramphenicol eye drops. The course of Cefaclor (Ceclor®) had yet to be completed. The child was up to date with vaccinations including the conjugate meningococcal C vaccination that had been administered six months earlier.

Two additional close contacts were identified in relation to the child case. Both had received prophylaxis at the time of the parent case's diagnosis, although this had not been initially recommended by public health. Two other social contacts were given information.

Isolates from the parent and child were confirmed as *Neisseria meningitidis* serogroup C 2a, p 1.5. The blood isolate from the parent case and the con-

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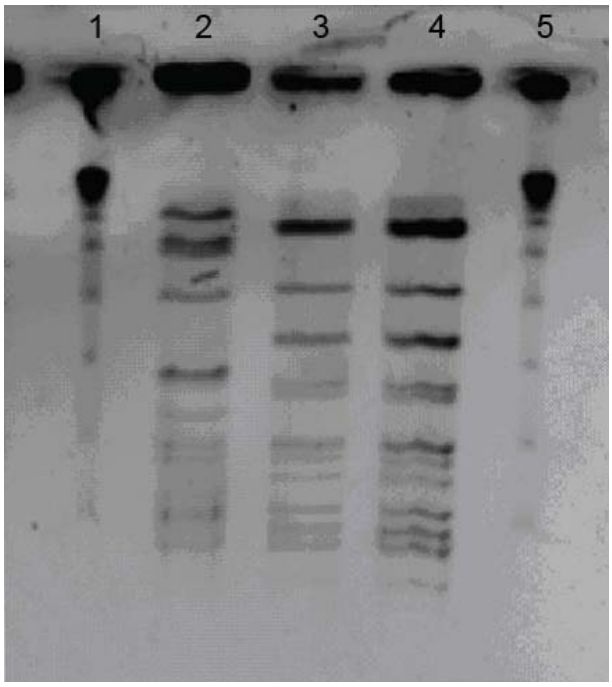
junctival isolate from the child were compared using pulsed field gel electrophoresis. They were found to be identical (Figure).

**Figure. Pulsed field gel electrophoresis patterns of *Neisseria meningitidis* isolates from child and parent cases**




Legend: Lane 1 Marker 48.5kb; Lane 2 N. meningitidis control; Lane 3 isolate from child; Lane 4 isolate from parent; Lane 5 Marker 48.5kb.

This family cluster highlights a number of points of public health importance. The first concerns the protection afforded by the conjugate meningococcal C vaccine. The efficacy of this vaccine has been estimated as 90 per cent and 96 per cent by two different groups in relation to invasive disease in the United Kingdom.[1,2] Immunological memory after vaccination has been well documented using serum antibody responses.[3,4] The efficacy of the vaccine with respect to generating mucosal antibody response is unknown. Zhang *et al* found significant increases in salivary IgA and IgG antibody titres in adolescents at one month after vaccination with the conjugate vaccine.[5] However, these titres decreased considerably towards baseline within six to 12 months. No studies on antibody titres to *Neisseria meningitidis* serogroup C in tears after immunisation, or vaccine efficacy in relation to conjunctivitis could be found in the literature.

Vaccination is known to reduce nasopharyngeal carriage of *Neisseria meningitidis* serogroup C in the community, with protection against carriage of up to 63 per cent after vaccination programs.[6] However, carriage is not eliminated in all vaccinated individuals. Extrapolating what is known about the efficacy of the vaccine against nasopharyngeal carriage to the conjunctiva, as another vascular mucosal surface, it is likely that vaccine efficacy against primary meningococcal conjunctivitis is considerably less than that for invasive disease. The point should be made however, that in the absence of invasive disease in the child case, this was not a case of 'vaccine failure'.

The second issue related to this cluster is the sequence of transmission. The parent case was notified first, but the disease onset occurred two days after the onset of the child's conjunctivitis. Primary meningococcal conjunctivitis may result from inoculation of the conjunctival sac with meningococci that are either airborne or mechanically transmitted.[7] If we assume that the child had pharyngeal colonisation prior to the onset of purulent conjunctivitis, the parent may have acquired meningococci from the child, particularly in light of the child's ongoing respiratory symptoms at the time. The parent had no contact with the child for the duration of purulent eye discharge, and did not self-report conjunctival symptoms, so mechanical transmission appears less likely.

As the incubation period for invasive meningococcal disease varies from two to seven days, it is possible, although not likely, that meningococci from the parent's nasopharynx were transmitted to the child whilst she was asymptomatic. The third and most likely possibility, in relation to the sequence of transmission, is that both parent and child acquired meningococci from a common close contact.

The third issue signified in this cluster involves the identification of related cases of disease. While this question is routinely asked during the investigation of meningococcal cases in Queensland, clinicians, patients, their families and public health officers alike assume the question is about other cases of meningitis or septicaemia; invasive disease that causes the symptoms public health messages warn the community about. This cluster highlights a much less distinctive presentation that may be relevant to some cases.

Finally, the issue of contact definitions in relation to meningococcal conjunctivitis is raised by this cluster. Close contacts for prophylaxis have been defined by the Communicable Diseases Network Australia working party on meningococcal disease[8] and include contacts of meningococcal conjunctivitis. These guidelines for the control of meningococcal disease do not discuss the possibility of transmission of meningococci from conjunctival exudate.[8] Intra and extracellular meningococci have been consistently found in conjunctival exudate.[9]

Meningococci have also been isolated from the nasopharynx of cases of primary meningococcal conjunctivitis, and have proved to be the same serogroup, serotype and subtype as those isolated from the conjunctiva.[10] There are published reports of possible transmission of meningococci to close contacts from cases with primary meningococcal conjunctivitis.[9,11] In both cases, the contact was a member of the case's household. It seems likely, in view of the possibility of organisms colonising the case's nasopharynx, and the absence of conjunctival symptoms in the contacts, that they acquired meningococci from the cases via droplet spread. However, the possibility exists that meningococci present in conjunctival exudate could also be mechanically transferred to the conjunctiva of a contact, perhaps causing a secondary case of meningococcal conjunctivitis. The incidence of any such phenomenon is not currently known however, and further delineation of the role of conjunctivitis in transmission of *Neisseria meningitidis* should be sought. Because any secondary case of meningococcal conjunctivitis would have the potential to go on to develop invasive disease[7], perhaps future consideration should be given to the provision of specific information about the possibility of mechanical transmission of meningococci to the contacts of a case of primary meningococcal conjunctivitis. If deemed appropriate, this information should recommend contacts seek medical advice if they develop symptoms of conjunctivitis, in addition to being alert for, and seeking medical attention if they develop symptoms of invasive disease.

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# Top End rural and remote Indigenous women: an Australian population group vulnerable to rubella

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## Abstract

**Australian efforts to prevent cases of congenital rubella syndrome have been largely successful, although concerns that eradication has not yet been achieved are ongoing. This paper describes an Australian population group with a vulnerability to rubella which has not previously been reported. Fewer than 75 per cent of Indigenous women from rural and remote communities who gave birth at Royal Darwin Hospital in 1999 and were tested for rubella antenatally had adequate levels of immunity. By comparison Indigenous and non-Indigenous women living in the Darwin urban area had a prevalence of adequate immunity to rubella on antenatal testing of >90 per cent, similar to estimates for other Australian-born population groups. Action is required to reduce the risk of cases of congenital rubella syndrome occurring in rural and remote Indigenous communities in the Top End, and may be needed in rural and remote settings elsewhere in Australia. Ensuring each child, adolescent and young adult has received two doses of Measles Mumps Rubella vaccine as part of their primary immunization course will provide increased protection. In addition, more women lacking adequate rubella immunity need to be vaccinated postnatally than was found in this study. Providers of women's care would be assisted in this task if laboratories adopted a standardized approach to reporting the results of antenatal rubella serology tests. *Commun Dis Intell* 2004;28:499–503.**

*Keywords: rubella, Indigenous, vaccination, congenital rubella syndrome*

## *Introduction*

The primary objective of rubella immunization programs is to prevent cases of congenital rubella syndrome (CRS).[1] In Australia, an organized approach to CRS prevention began in 1971 with the introduction of the schoolgirl rubella vaccination program, for girls aged 10–16 years.[2] In 1989, Measles Mumps Rubella (MMR) vaccine was recommended for all 12-month old children.[3] In 1993, a second dose of MMR was added to the standard vaccination schedule for 10–16 year olds, and its timing was shifted to 4–5 years in 1998, and to 4 years in 2000.[4] These Australian rubella immunization programs have had a major impact on reducing the incidence of CRS. Before they began, about 120 cases were diagnosed annually, whereas between 1993 and 1997 a total of only 19 children were diagnosed with CRS.[2] There were no locally acquired cases of CRS reported between 1997 and 2002, and no cases at all were detected between 1998 and 2000.[5]

Despite the apparent success of these efforts to prevent CRS, there remain several areas of concern. The first is illustrated by a recent report of two cases of CRS in 2003 for babies of Australian-born women living in south-eastern Queensland.[5] Low levels of rubella immunity among young men and lack of universal rubella immunity among childbearing women have been identified as likely contributory factors.[6] These cases of CRS prompted calls for a greater effort to ensure that all children are immunized with two doses of MMR and that all women are screened antenatally and vaccinated postnatally if not immune to rubella, as well as the suggestion that an adult male rubella vaccination campaign may be necessary to interrupt rubella virus transmission and prevent further cases of CRS.[6,7]

Another ongoing concern is the occurrence of CRS cases among the babies of women who have migrated to Australia from the many countries where there are no universal rubella immunization programs.[5,8,9] Seroprevalence studies have demonstrated high levels of vulnerability to rubella infection

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for some groups of overseas-born women, prompting recommendations for both targeted vaccination programs and ongoing surveillance.[1,8,10]

This paper reports for the first time on a high level of vulnerability to rubella infection for another Australian population sub-group—Indigenous women living in rural and remote communities in the Top End of the Northern Territory. Antenatal screening for rubella immunity for Indigenous and non-Indigenous women living in the Top End is reviewed here, together with documentation about postnatal vaccination for women with inadequate immunity.

## *Methods*

The results presented are from a project evaluating a broad range of aspects of pregnancy care for Indigenous and non-Indigenous women giving birth at Royal Darwin Hospital, which is located in the Top End of the Northern Territory.[11] More than 90 per cent of Indigenous women and 60–65 per cent of non-Indigenous women who usually reside in the Darwin urban area or the rural and remote communities around Darwin give birth at Royal Darwin Hospital.[12]

All women giving birth at Royal Darwin Hospital in 1999 and usually resident in the Northern Territory were identified from the Northern Territory Midwives Data Collection data set. Four Indigenous women whose names could not be matched with existing Royal Darwin Hospital files were excluded; the remaining 516 Indigenous women giving birth were included. Available resources limited the number of files of non-Indigenous women able to be reviewed. A random sample of 150 of the 1,035 births occurring in 1999 for non-Indigenous women was selected by matching a computer-generated list of random numbers against the list of births, sorting the random numbers and selecting the first 150 births. Four women could not be matched with existing hospital files, two were 'duplicates' relating to twin births and one woman was excluded because she had delivered at home. This left a final sample of 143 non-Indigenous women.

One of the authors (JH) reviewed hospital files for all included women, and extracted a large number and range of data items about pregnancy care. Royal Darwin Hospital files usually include records of antenatal care provided in other settings as well as at the hospital, and this is particularly the case for the results of antenatal screening tests.[11] No additional information was sought from community clinics or other health providers as part of this project.

The analyses presented here report on data collected about rubella serology tests and results, and evidence of rubella vaccination being given following delivery for women who lacked immunity. All data were entered directly into an Microsoft Access[13] database which was used for subsequent analyses. Analysis involved calculating simple proportions, and the proportions of women in each group with documented immunity to rubella were compared statistically using Epi Info.[14]

Place of usual residence was used to allocate women to one of three groups used for analyses, based on the regions defined and commonly used in administrative health data collections in the Northern Territory:[12,18]

1. The 'Darwin urban' area (159 Indigenous women, 127 non-Indigenous women);

2. The rural and remote area surrounding Darwin ('Darwin Rural' region). Women from this region usually came to Darwin to give birth (220 Indigenous women, 10 non-Indigenous women);

3. Outside the Darwin region. Women in this group were most often referred from other parts of the Top End to Darwin to give birth because of pregnancy problems (137 Indigenous women, 6 non-Indigenous women).

Results are presented here for Indigenous women from each of these groups, and for non-Indigenous women from the Darwin urban area. Results for the 16 women classified as both non-Indigenous and living outside the Darwin urban area are not reported. This is because of their small numbers, and also because hospital records for six of these women contained information suggesting they were Indigenous. Evidence available in hospital records suggested Indigenous status misclassification was not a significant issue for the other groups studied.[11]

The project evaluating pregnancy care for women giving birth at Royal Darwin Hospital was developed in consultation with local practitioners, policy makers, Indigenous community members and the local Aboriginal community controlled health service, and was approved by the Top End Joint Institutional and La Trobe University Human Ethics Committees.

## *Results*

Records and results of antenatal testing for rubella are shown in Table 1. More than 90 per cent of Indigenous and non-Indigenous women had records in their hospital files of antenatal rubella serology tests having been performed, and more than 97 per cent of these had results recorded. Laboratories reported the results of rubella serological tests in

a variety of formats. These included a quantified rubella antibody titre; a categorically reported rubella antibody titre, for example <16 IU; and/or a descriptive statement about rubella immunity such as 'low level', 'doubtful' or 'adequate'. The level of quantitative antibody titre used by different laboratories to report whether a woman's rubella immunity was adequate was not consistent, varying from 16 to 25 IU/ml. Because of this variability, results are presented in three categories, 'low/no immunity', 'immune', and a 'borderline' category which includes reports where this term was used or where quantitative titres in the 16 to 25 IU range were reported.

Indigenous women from the Darwin urban area had a similar frequency of documented immunity to rubella (90.3% immune) to non-Indigenous women from the Darwin urban area (94.2% immune, Chi-square=1.45, p=0.23). By contrast, Indigenous women from rural and remote communities were significantly less likely to have documented immunity to rubella. Compared to urban Indigenous women, lower levels of rubella immunity were recorded for both Darwin rural region Indigenous women (72.9% immune, Chi-square=16.4, p<0.001) and for Indigenous women from outside the Darwin region (65.6% immune, Chi-square=24.6, p<0.001).

Vaccination following delivery was recorded inconsistently in hospital notes for Indigenous and non-Indigenous women regardless of their place of usual residence. As shown in Table 2, only 62–75 per cent of women with low/no immunity had records of vaccination in hospital, and vaccination was even less likely to be documented for women with 'borderline' rubella immunity. All postnatal vaccinations were with MMR rather than a monovalent rubella vaccine, and there were no documented cases of women refusing vaccination. Hospital discharge summaries rarely recorded the need for community follow up and vaccination when women with low or no immunity to rubella had not been vaccinated postnatally in hospital.

## *Discussion*

Women giving birth at Royal Darwin Hospital almost universally had rubella tests and results recorded antenatally, consistent with rubella immunity testing being a common and longstanding recommendation in Australian protocols about routine antenatal care.

Because 90 per cent of Indigenous women living in Darwin urban and Darwin rural regions give birth at Royal Darwin Hospital, estimates of the prevalence of rubella immunity for these groups made in this study are likely to approximate population-based measures. More than 90 per cent of urban Indigenous and non-Indigenous women having tests indicates immunity to rubella similar to reported estimates for other Australian population groups. More than 90 per cent of women giving birth at Victorian hospitals between 1976 and 1990 were immune to rubella,[10] and a 1998 national seroprevalence survey reported 97 per cent of women aged 16 to 39 years as immune.[9] Indigenous women living in rural and remote Top End communities were much

**Table 1. Antenatal rubella serological tests and results**

| | Number in group | Per centage with antenatal rubella tests | Results (% women with tests)* | | | |
|---|---|---|---|---|---|---|
| | | | Results not recorded | Low/no immunity | Borderline immunity | Immune |
| Urban Indigenous | 159 | 91.2 | 0.7 | 5.5 | 3.4 | 90.3 |
| Darwin rural Indigenous | 220 | 95.5 | 1.9 | 17.6 | 7.6 | 72.9 |
| Indigenous out of Darwin | 137 | 93.4 | 2.4 | 25.6 | 6.4 | 65.6 |
| Urban non-Indigenous | 127 | 96.8 | 0.0 | 3.3 | 2.5 | 94.2 |

* Women without tests recorded have been excluded from results columns.

**Table 2. Postnatal vaccination for women with low or borderline rubella immunity**

| | Low/no immunity | | Borderline immunity | |
|---|---|---|---|---|
| | Number | Percentage vaccinated | Number | Percentage vaccinated |
| Urban Indigenous | 8 | 62.5 | 5 | 0.0 |
| Darwin rural Indigenous | 37 | 64.9 | 16 | 37.5 |
| Indigenous out of Darwin | 32 | 71.9 | 8 | 12.5 |
| Urban non-Indigenous | 4 | 75.0 | 3 | 33.3 |

less likely than this to be immune to rubella. Women from outside the Darwin region had a similarly low prevalence of immunity to rubella to that of women from the Darwin rural region, despite being a highly selected high risk group. This suggests that vulnerability to rubella may be widespread among Indigenous women usually living in Top End rural and remote communities. Whether or not this is true in other similar regions of Australia could be investigated relatively easily through hospital or community based audits of rubella immunity for women tested antenatally.

There are several possible explanations for the high levels of non-immunity to rubella reported here for Top End rural and remote Indigenous women. Residents in a rural and remote community may have limited past exposure to the rubella virus, resulting in an immune response to a previously acquired infection being less likely. All but a few of the rural and remote Indigenous women giving birth at Royal Darwin Hospital in 1999 would have been of an age eligible to receive rubella vaccination through school-based immunization programs. However, it appears that a significant proportion may have missed out, perhaps because they were not at school. Another possibility is that women were vaccinated but did not achieve an adequate or persistent immune response to rubella. Cold-chain breaches have previously been identified as a problem with vaccine transport to rural and remote communities in the tropical climate of the Top End.[15] As rubella vaccines are sensitive to both heat and light,[16] cold-chain and other transport problems in past years may have impacted on its immunogenicity and the consequent strength or duration of women's immune responses. The contributions of these and other possible explanations could be further explored by determining the vaccination histories of non-immune women.

Although a rural and remote setting may confer some protection against exposure to rubella virus to its residents, high and increasing levels of population mobility mean it is probably only a matter of time before exposure occurs. Only three cases of rubella have been notified from the Northern Territory in the years since the 1999 data reported here.[17] However, only limited reassurance can be gained from this report. Notification rates for rubella are acknowledged as unreliable because the notification is primarily based on the results of serological testing for what is often a mild disease not investigated with tests.[2] In addition, there are other factors in Northern Territory settings that may act to reduce the likelihood of cases of rubella being notified, particularly in rural and remote Indigenous communities. These include rubella's relatively recent inclusion as a notifiable disease in the Northern Territory (in 1994),[2] barriers to accessing health services particularly for Indigenous people,[18] the high prevalence of other more serious health problems experienced by Indigenous people taking priority, and the high workloads and turnover of clinic staff. It is fortunate there have been no cases of CRS reported from the Northern Territory in recent years.[2,5] However, the level of vulnerability to rubella demonstrated here for women from rural and remote Indigenous communities suggests a future outbreak of rubella in the Northern Territory could have tragic consequences in terms of the potential for rubella infection to result in cases of CRS.

It is disappointing that many Top End women considered to lack adequate immunity to rubella on antenatal testing in this study were not vaccinated in hospital following delivery, or did not have clear documentation of the need for vaccination recorded on their hospital discharge summary. Working with hospital and community providers to promote the importance of offering non-immune women vaccination, and improving communication between hospital and community based services are measures that may help improve postnatal vaccination rates. In addition the presentation by laboratories of the results of antenatal rubella serology tests could be improved. Laboratories using different methods to test for rubella antibodies may account for some of the variability in test reporting formats noted in this study, and the most recent *Australian Immunization Handbook* notes the lack of an Australian standard for levels of rubella antibodies required to confer adequate levels of immunity.[16] However, the lack of consistency of laboratory reporting practices for rubella has been noted elsewhere.[19] Australian laboratories agreeing on a standard approach for presenting the results of antenatal rubella serology tests may result in less confusion for antenatal care providers, and more women lacking adequate rubella immunity being vaccinated postnatally.

MMR vaccination has recently been promoted in the Northern Territory, and elsewhere in Australia, for young adults who have not received two previous doses as part of efforts to improve measles control.[16,20] This measure may also have the effect of improving levels of immunity to rubella, and ongoing efforts to encourage young adults to be vaccinated with MMR are justified for both reasons. In addition, increased promotion of the importance of postnatal vaccination of non-immune women, including system changes to ensure opportunities for postnatal vaccination are not missed, and ongoing monitoring of levels of rubella non-immunity among pregnant women are recommended.

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# Influenza A associated morbidity and mortality in a Paediatric Intensive Care Unit

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## Abstract

**This paper reports the clinical features and outcome of all children with a laboratory proven diagnosis of influenza A virus infection admitted to a major Paediatric Intensive Care Unit (PICU) in 2003. Eight of the 22 patients with influenza A virus infection (A/Fujian/411/2002-like type) presented with encephalopathy and three of the 22 patients died. This can be compared with 44 admissions and seven (16%) deaths of patients with influenza virus admitted in the same PICU in the preceding 15 years. In the present cohort, four (18%) of the 22 patients, including one child who died, should have received influenza vaccine according to the current Australian immunisation recommendations. We have no documented evidence that any of the 22 children received influenza vaccination. During the 2003 influenza season there was an increased number of children admitted to our PICU with influenza A infection and an increased number of deaths compared with previous years. Influenza infection causes significant morbidity and mortality in young children, most of whom are not currently recommended for annual influenza vaccination. *Commun Dis Intell* 2004;28:504–509.**

*Keywords: Influenza A, influenza immunisation, Paediatric Intensive Care Unit, Influenza A (H3N2) Fujian/411/2002 type*

## *Introduction*

Influenza is a common disease of childhood with the highest morbidity and mortality occurring in pre-school aged children.[1,2,3] In Australia between 1 July 1998 and June 2000 the rate of hospitalisations of children aged 0–4 years with influenza infection was 70.6/100,000 population, far exceeding rates in all other age groups. The number of deaths attributed to influenza in children are 12/100,000 and 2/100,000 for 0–4 year olds and 5–12 year olds respectively.[3] Annual influenza immunisation is currently recommended in the *Australian Immunisation Handbook* for children at risk of severe influenza, but not routinely for healthy children.[4] However, recent reports of increased morbidity and mortality in children with influenza associated illnesses in North America has led to updating the United States of America (USA) recommendations for influenza vaccination to include all healthy children six–23 months during the influenza season to the extent that is logistically and economically feasible.[5, 6]

In this study we describe the experience of a tertiary children's hospital Intensive Care Unit in Sydney, during the 2003 influenza season. The Children's Hospital at Westmead is one of two Sydney paediatric teaching hospitals which serve New South Wales, with a current population of 6.7 million people. The PICU is a 23 bed intensive care unit which has over 1,000 admissions each year. Of this number, 25–30 per cent are admitted with general medical problems. Almost 70 per cent of the PICU admissions are from the Sydney metropolitan area, the remainder from rural areas.

## *Methods*

We reviewed the medical records of all patients identified with laboratory-proven influenza A virus infection admitted to the PICU during 2003. We compared the 2003 outbreak with the number of admissions and deaths of patients in PICU with laboratory proven influenza virus infection over the last 15 years. Ethical approval for this study was obtained from the hospital ethics committee. Patients were identified by reviewing the Intensive Care Unit database and virology records from 1988–2002. Data collected from the 2003 cohort of patients included date of birth, age, sex, underlying medical condition, clinical presentation, diagnosis, source of isolation of influenza, other positive cultures, vaccination status

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**Table. Clinical features of patients with influenza associated illness admitted to Paediatric Intensive Care Unit in 2003**

| n | Sex | Age years | Pre-existing morbidity | Diagnosis | LOS.p | LOS.h | Outcome |
|---|---|---|---|---|---|---|---|
| 1 | F | 3 | DD/ Seizures | Pneumonia/seizures | 4.6 | 6 | d/c |
| 2 | F | 8 | Seizures/GOR | Septic shock | 2.3 | 7.5 | Died |
| 3 | M | 1.4 | CHD | Pneumonia | 9.1 | 10.8 | d/c |
| 4 | M | 2.3 | Nil | Laryngotracheobronchitis | 2.1 | 4.1 | d/c |
| 5 | M | 2 | Nil | Laryngotracheobronchitis | 1.5 | 2.3 | d/c |
| 6 | F | 3 | Liver disease | Bleeding varices | 0.8 | 10.5 | d/c |
| 7 | M | 1.9 | Nil | Meningococcal septicaemia | 6.5 | 9.9 | d/c |
| 8 | M | 0.75 | CLD | Pneumonitis | 0.7 | 4.8 | d/c |
| 9 | F | 4 | Nil | Cardiac arrest | 4.8 | 4.8 | Died |
| 10 | M | 0.8 | Nil | Bronchiolitis/pneumonia | 1 | 5.5 | d/c |
| 11 | M | 7 | CP hemiparesis | Encephalopathy/pneumonitis | 1.3 | 1.3 | Died |
| 12 | F | 8 | Nil | Pneumonitis/pneumothorax | 2.8 | 4.1 | d/c |
| 13 | M | 13 | Mild DD | Pneumonia/pancreatitis/encephalopathy | 11.4 | 27.3 | r/a |
| 14 | M | 8 | Nil | Meningoencephalitis | 1.1 | 2.5 | d/c |
| 15 | F | 0.4 | DD/CAH | Status epilepticus | 1.7 | 11 | d/c |
| 16 | F | 1.1 | Nil | Pneumonitis/asthma | 0.7 | 8.6 | d/c |
| 17 | M | 2.8 | Nil | Pneumonia/empyema | 0.7 | 15.5 | d/c |
| 18 | F | 3 | Asthma | Asthma | 0.8 | 3.8 | d/c |
| 19 | F | 2.6 | Nil | Encephalopathy/ICH | 7.1 | 25 | d/c |
| 20 | M | 5 | Asthma | Encephalitis/seizures | 0.7 | 4.5 | d/c |
| 21 | M | 1.8 | Nil | Pneumonia/hepatoblastoma | 8.9 | 16.5 | d/c |
| 22 | F | 2.6 | Asthma | Gastroenteritis/encephalopathy | 0.6 | 4 | d/c |

CAH - Congenital adrenal hypoplasia; CHD - Congenital heart disease; CLD - Chronic Lung disease; CP - Cerebral palsy; DD - Developmental delay; GOR - Gastroesophageal reflux; ICH - Intracranial heamorrhage; LOS.p - Length of stay in PICU in days; LOS.h - Length of stay in hospital in days; Outcome - d/c - discharged from PICU, - r/a - readmitted and subsequently d/c

if documented, length of stay in PICU and the outcome. These patients were compared with groups targeted for influenza immunisation according to the current *Australian Immunisation Handbook*.[4]

A laboratory proven case was defined as a child with influenza virus identified either by direct immunoflourescence (DFA) or from viral culture from a nasopharyngeal aspirate (NPA) or by a 4-fold rise in influenza antibody titre in paired sera. Routine practice in the PICU is that all children admitted with respiratory symptoms or possible viral infections are tested for viral antigens on a NPA and viral culture. In our hospital NPAs are tested for respiratory syncytial virus (RSV), parainfluenza viruses 1, 2 and 3, influenza viruses A and B and adenoviruses using DFA (Imagen kit by Dako). Isolates with positive DFAs for an influenza virus are then cultured. Nasopharyngeal aspirates with negative DFAs were also routinely set up for viral culture for three weeks. Cultured isolates were sent to the World Health Organization Collaborating Centre for Reference and Research on Influenza, in Melbourne, Australia for typing by haemagglutination inhibition assay.

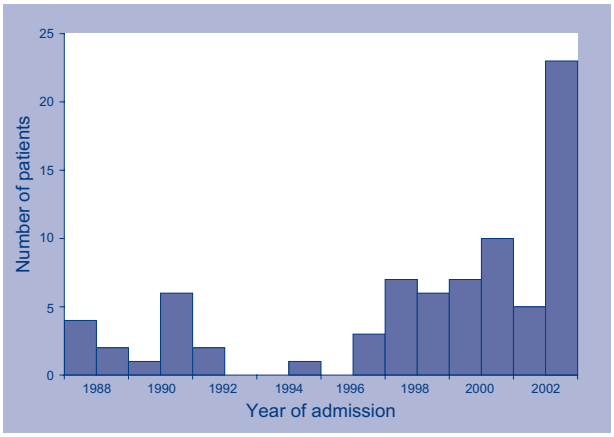
## *Results*

Twenty-two patients (23 admissions) with laboratory-proven influenza A virus were admitted to the Intensive Care Unit in 2003. This was five per cent of the total medical admissions to PICU in 2003. The virus strain was isolated as Influenza A (H3N2) of the Fujian/411/2002-like type. Three (14%) patients died. All admissions occurred during a 3 week period in August, except for one patient who was admitted in late September. In comparison, over the 15 years from 1988 to 2002, there were 44 PICU admissions and seven (16%) deaths of children with laboratory proven influenza infection (Figure).

Clinical features of the 22 patients admitted to PICU with influenza A infection are set out in the table. Patients are listed in order of admission. The mean age in this cohort was 4.1 years (range 4 months–13 years) of whom 16 (73%) patients were under four years of age, six (27%) between six and 23 months of age and one under six months of age. Of the 22 patients there were 12 (55%) males and

10 (45%) females. Eleven (50%) of the patients had a pre-existing condition, including two of the three patients who died.

**Figure. Number of patients admitted to Paediatric Intensive Care Unit with influenza associated illness between 1988 and 2003**




All patients presented with fever and 15 (68%) with clinical evidence of either an upper respiratory tract infection or lower respiratory tract infection defined as tachypnoea, cough, wheeze or respiratory distress. Eight (36%) patients presented with encephalopathy, defined as altered mental status of any duration, including seizures but not including simple febrile convulsions.

During admission, pneumonia or pneumonitis was reported in 10 (45%) patients. One patient had Gram negative diplococci on skin scraping and clinical features of meningococcal septicaemia concurrent with influenza infection. There were no patients with positive blood cultures. The mean length of stay in PICU was 3.2 days (range 0.6 to 11.4 days) compared with the mean length of stay for all patients in this unit of 3.1 days.

Influenza A was detected by DFA from NPAs in all patients. There were no isolates of influenza B virus. Influenza A virus was recovered from 14 samples, all which were positive for influenza A (H3N2) of the Fujian/411/2002-like type.

According to the 7th edition of the *Australian Immunisation Handbook*,[4] routine annual influenza immunisation was recommended for four (18%) of the 22 patients including one patient who died. There was no documented evidence that any of the patients were vaccinated against influenza.

## *Case reports*

Clinical details of the three patients who died are presented as brief case reports below.

### Patient 1

A four-year-old girl, who was previously well, developed coryza, lethargy and rash 48 hours prior to presentation. She was seen by the local doctor who prescribed oral penicillin. She became increasingly lethargic and collapsed at home. Cardiopulmonary resuscitation was commenced by her father and continued until the ambulance arrived. At this time she had no respirations and was pulseless. She was intubated, ventilated, given endotracheal and intravenous adrenaline and atropine after which her circulation returned. On arrival at the emergency department she had evidence of haemodynamic shock, a Glasgow Coma Score of 4 and temperature of 36.2°C. The chest x-ray (CXR) showed bilateral infiltrates. She was transferred to PICU and subsequently developed evidence of acute lung injury. Influenza A was isolated from NPA collected on day 1 of admission. Her respiratory state improved over the initial 24 hours but she developed signs of cerebral oedema and at 72 hours of the admission, brain stem testing revealed brain death. No other pathogens were isolated. Post mortem was not performed.

### Patient 2

An eight-year-old girl with a background of severe developmental delay, seizures, gastroesophageal reflux and gastrostomy feeds presented to hospital with a 4-day history of fever, cough and respiratory distress. She was admitted to intensive care after worsening in her respiratory status requiring intubation and ventilation. Her CXR showed prominent interstitial markings with right lower lobe consolidation. Influenza A was detected from an NPA collected on day 1 of admission. She required artificial ventilation, went on to develop haemodynamic shock, intractable multi-organ failure and died. Post mortem was not performed.

### Patient 3

A seven-year-old boy with a background of right sided hemiparesis from congenital cerebral atrophy presented with a 48 hour history of cough, coryza and vomiting. He was only mildly affected by his hemiparesis, being able to ride a bike and attend a normal school. After feeling unwell at midday, he went to rest and was found obtunded seven hours later. On arrival at the emergency department his Glasgow Coma Score was 3 and temperature was 35°C. He was intubated, ventilated, commenced on intravenous cefotaxime, acyclovir and transferred to

PICU. CXR showed opacification of the right lung field and patchy consolidation of the left. Influenza A was detected from NPA collected on day 1 of admission. Electroencephalogram showed encephalopathic changes. A magnetic resonance imaging scan of his brain showed widespread cerebral oedema, ischaemia and bilateral uncal and transtentorial herniation. Brainstem testing confirmed brain death. Post mortem was not performed.

## *Discussion*

Influenza infections cause substantial morbidity and mortality in children every year.[3, 7] Our data have shown an increase in admissions to PICU and an increased number of deaths of children with influenza associated illnesses in 2003 compared to the past 15 years and with a similar study of admissions from 1974–1994.[8] We believe that influenza A infection was the principal cause of mortality and acute morbidity in our 22 patients. One patient admitted with laboratory-proven influenza infection also had meningococcal septicaemia. Previous reports have shown an association between outbreaks of influenza and meningococcal disease.[9] Whilst we do not have post mortem data in those patients who died, we have no other identified cause of their admitting illness despite intensive anti-mortem investigations. *The New South Wales Influenza Surveillance Annual Report* states that the 2003 influenza season was moderate,[10] suggesting that the increase in paediatric disease severity was out of keeping with the overall experience. The National Notifiable Diseases Surveillance System reported in comparison to 2002, notification rates of influenza declined in the over 65 age group but increased among the 0–4 year age group and remained unchanged in the rest of the age groups.[11] The majority of influenza A isolates identified during the peak period was A (H3N2) viruses of the A/Fujian/411/2002 type.[10] This same strain was isolated in the cohort of patients described in this paper.

Thirty-six per cent of our patients presented with encephalopathy associated with influenza illness. This finding is similar to reports of the 2003 Michigan influenza season, when surveillance identified four deaths and 10 severe illnesses among children and adults less than 21 years with influenza associated illness. Eight (57%) of these 14 cases, had evidence of encephalopathy and one had evidence of myocarditis; two of the children with encephalopathy died.[5] Similarly, in Virginia, five unexplained deaths associated with influenza infection in children aged two to seven years were reported in the 2003 influenza season.[12] An increased number of cases of influenza-associated encephalopathy in children less than five years of age was also been reported from Japan.[13,14,15] The Centres for Disease Control in the USA recently published a report of 142 influenza associated deaths among children less than 18 years in their current winter season as of 27th March 2004.[6] Over half of these deaths were in children under five years of age, and only 21 had high-risk medical conditions that put them at risk for complications of influenza.[16]

In Australia, although influenza hospitalisation rates in children aged 0–4 years far exceed the rates in all other age groups,[3] the only children targeted for annual influenza immunisation include those with chronic cardiac or pulmonary disorders, children residing in chronic care facilities, and children with chronic conditions such as diabetes mellitus and other metabolic diseases, cancer, immunodeficiency, immunosuppression, renal disease, anaemia and haemoglobinopathy.[4] A recent study in Melbourne reported the impact of influenza A (H3N2) during the 2003 outbreak on children with disabilities with an increased hospital admission rate, PICU admission and length of stay.[17] However over 80 per cent of patients in our cohort and two of the three patients who died, did not belong to the specific high-risk groups currently recommended for annual influenza vaccination. Similarly, a study by Quach *et al* in Montreal Children's Hospital found that the majority of children hospitalised for influenza did not belong to specific risk groups targeted by current recommendations and one third were less than six months of age, hence too young to be vaccinated.[1]

In 2003 antigenic drift was detected in the H3N2 virus strains circulating in Australia and New Zealand.[10] The A/Fujian-like virus is related to the A/Moscow-like strain included in the 2003 vaccine. Perhaps this A/Fujian virus caused an increase in the influenza associated childhood morbidity and mortality throughout the country which will be apparent when the national data are available for 2003. If so, a similar pattern of illness could be predicted in 2004 and beyond.

There are relatively few data on the efficacy of inactivated influenza vaccine in the paediatric population.[18] Even though two doses at least one month apart are recommended for children aged less than nine years who are receiving influenza vaccine for the first time,[4] it is possible that the cost effectiveness of this vaccine would compare favourably with the vaccination program in this age group including conjugated pneumococcal and meningococcal vaccines.

In response to the increased morbidity and mortality of influenza associated illnesses in the USA, the Advisory Committee on Immunization Practices and the American Academy of Pediatrics now recommend influenza immunisation of all healthy children who will be six to 23 months of age during the influenza season as well as encourage vaccination of

household contacts and out-of-home caregivers of children younger than 24 months of age.[19,20,21] This decision is based on the higher hospitalisation rates within this age group. These new recommendations however may not have changed the outcome of most of the deaths previously described in the USA nor indeed the four, seven and eight year old that died in our cohort. Delivery of inactivated influenza vaccine on an annual basis to Australian children under the age of 23 months would be difficult and could potentially compromise the existing recommended schedule. Despite these difficulties, we believe that in view of our experience in 2003, Australia should now seriously reconsider recommendations for influenza vaccination to include not only children at risk of severe complications but also healthy children. Should we follow the American guidelines and recommend vaccination of all healthy six to 23 month olds, or should we extend this to 48 months in order to cover the age most affected by influenza associated illnesses? In the mean time, we must improve coverage by actively encouraging paediatricians and local doctors to vaccinate children who belong to risk groups according to the current Australian recommendations.

Undoubtedly, heightened awareness of the severe complications and deaths associated with influenza among children is necessary. Mandatory notification of influenza associated deaths among children, as in the USA, needs to be considered to provide more immediate surveillance of the severity of influenza outbreaks.[20] In reporting this cohort of children, we aim not only to emphasis the need to provide annual vaccination to children in high-risk groups, but also to stimulate discussion regarding change in Australian vaccination recommendations to include all healthy children aged six–23 months.

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# The cost of seasonal respiratory illnesses in Australian children: the dominance of patient and family costs and implications for vaccine use

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## Abstract

**Respiratory viral infections are one of the next group of diseases likely to be targeted for prevention in childhood by the use of vaccines. To begin collecting necessary epidemiology and cost information about the illnesses caused by these viruses, we conducted a prospective cohort study in 118 Melbourne children between 12 and 71 months of age during winter and spring 2001. We were interested in calculating an average cost per episode of community-managed acute respiratory disease, in identifying the key cost drivers of such illness, and to identify the proportion of costs borne by the patient and family. There were 202 community-managed influenza-like illnesses identified between July and December 2001, generating 89 general practitioner visits, and 42 antibiotic prescriptions. The average cost of community-managed episodes (without hospitalisation) was $241 (95% CI $191 to $291), with the key cost drivers being carer time away from usual activities caring for the ill child (70% of costs), use of non-prescription medications (5.4%), and general practice visits (5.0%). The patient and family met 87 per cent of total costs. The lowest average cost occurred in households from the highest income bracket. Acute respiratory illness managed in the community is common, with the responsibility for meeting the cost of episodes predominantly borne by the patient and family in the form of lost productivity. These findings have implications for preventive strategies in children, such as the individual use of, or implementation of public programs using, currently available vaccines against influenza and vaccines under development against other viral respiratory pathogens. *Commun Dis Intell* 2004;28:509–516.**

*Keywords: vaccine use, respiratory illnesses*

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## *Introduction*

For some time now there has been a divergence between what vaccines the National Health and Medical Research Council (NHMRC), more recently on the advice of the Australian Technical Advisory Group on Immunisation (ATAGI), recommends Australians should receive, and what is paid for by the National Immunisation Program (NIP). The NIP is a Commonwealth, State and Territory Governments' initiative that provides certain vaccines free of charge to Australians.

This divergence previously only applied to recommendations for older Australians, in particular, influenza and 23-valent polysaccharide pneumococcal vaccines. Influenza vaccine was first recommended for older Australians in the third edition of, what is now called, the *Australian Immunisation Handbook* in 1986[1] but was only funded nationally in 1999.[2] A general recommendation for use of polysaccharide pneumococcal vaccine in older Australians was first made in the fifth edition of the immunisation handbook (1994),[3] and the Commonwealth Government has announced funding for a national program to commence in 2005.[4]

But as Burgess and McIntyre reported recently,[5] the release of the eighth edition of the handbook[6] has seen this divergence between recommended and funded vaccines extend to children. Varicella vaccine is on the Australian Standard Vaccination Schedule at 18 months of age; there is a universal recommendation for a primary course of the relatively expensive seven-valent conjugate pneumococcal vaccine; and inactivated poliomyelitis vaccine is recommended when appropriate combination vaccines become available. An infant program and a catch-up program for children under the age of two years for pneumococcal vaccine commenced at the beginning of 2005,[4] but there is currently no provision to fund either universal childhood varicella vaccination or a transition to inactivated poliomyelitis vaccine.

This emerging discrepancy between recommended and funded vaccines is only likely to widen. One of the next major groups of diseases preventable by use of vaccines is likely to be the respiratory viral infections of childhood. Injectable influenza vaccines are currently licensed for Australian children down to the age of six months, but are currently recommended only for children in high-risk groups.[6] A trivalent, cold-adapted, influenza vaccine (CAIV-T), containing live-attenuated virus and delivered intranasally, was licensed in the United States of America (USA) in 2003 for healthy five to 49-year-olds,[7] with the likelihood of younger and older age indications in the future. Other vaccines against respiratory viral infections, including respiratory syncytial virus and parainfluenza viruses, are currently under development. Information about the epidemiology and costs of acute community-based respiratory illness in children, particularly those costs borne by the patient and family, are required to guide future vaccine use and other control measures. Given the circulation patterns of these viruses, particularly respiratory syncytial virus and influenza virus, we collected information about respiratory illness in winter and spring.

We report here burden information for community-based respiratory illnesses in urban Australian children, and use these to calculate an average cost for these episodes.

## *Method*

We conducted a prospective cohort study of healthy children in metropolitan Melbourne, Victoria, between 1 July and 1 December 2001. The Royal Children's Hospital Ethics in Human Research Committee approved the study and written informed consent was obtained from parents/guardians. Methods for this study have been described elsewhere.[8] Eligible children were between 12 and 71 months of age at enrolment without pre-existing chronic respiratory or other medical problems. Children aged between 12 and 23 months were recruited largely via maternal and child health nurses (MCHNs) and immunisation providers in 23 local council areas across greater Melbourne. We invited participation from families with older children who had previously participated in a (non-respiratory pathogen) vaccine study conducted by our group, and also distributed flyers and posters through childcare centres. More than one child per family could be enrolled. We collected household demographic features at enrolment. Gross household income was collected in four brackets: bracket 1, ≤ $21,000; bracket 2, $21,101 to $33,000; bracket 3, $33,001 to $56,000; and bracket 4, > $56,000.

Parents/guardians completed a symptom diary card for each day the child was on the study. We designated an important respiratory illness to be an influenza-like illness (ILI) using the criteria described by Belshe *et al* in the CAIV-T efficacy study conducted in the USA during 1996 and 1997.[9] An ILI was defined as having occurred if a child had at least one category A symptom or at least two category B symptoms (Table 1). All information about symptoms was from parental report only. Individual episodes began on the first day on which there were sufficient symptoms to meet the definition of an ILI, and finished on the final day there were any documented symptoms associated with the ILI. A new episode was deemed to have commenced if there were three or more symptom-free days since the last day with any symptoms of the previous episode. Number and duration of ILIs was ascertained;

**Table 1. Defining symptoms of an influenza-like illness**

| Category A symptoms | Category B symptoms |
|---|---|
| • fever (either identified without measurement or a measured temperature of 37.6°C or higher by axillary thermometer)<br>• wheezing<br>• shortness of breath<br>• pulmonary congestion (moist cough)<br>• pneumonia (diagnosed by a healthcare provider)<br>• ear infection (suspected by parent/guardian or diagnosed by healthcare provider) | • runny nose/nasal congestion<br>• sore throat<br>• cough<br>• muscle aches<br>• chills<br>• headache<br>• irritability<br>• decreased activity (lethargy/weakness)<br>• vomiting |

incidence rates were calculated using child-months (person-time) as the denominator, and 95 per cent confidence intervals (CI) were produced using the standard method for incidence rate data.[10]

We used incident-based costing to derive an average cost of community-managed episodes (not including illnesses in which there was a hospitalisation). Once a child developed an ILI we asked parents to complete a burden diary on healthcare use, travel costs seeking healthcare (including car used and kilometres travelled), medication usage, investigations performed, time spent seeking healthcare during the episode, and excess time spent caring for a sick child—that is, time over and above that normally required for the care of the child when well.

Cost data were calculated from a societal perspective using 2001–2002 financial year Australian dollar values (Table 2). Discounting is not relevant as costs were collected in a single year. Direct and indirect costs were included, and we allocated costs as being borne either by the patient and family, the healthcare sector, or by another sector.[11] Details of sources for all costs are provided (Tables 2 and 3). An average cost per episode was calculated using the total number of illnesses, not just those where burden information was available, as the denominator.

Carer time spent seeking healthcare and excess time spent caring for an ill child were collected in three categories: time away from work with pay lost; time away from work with no pay lost; and time away from usual activities. We applied a sex-weighted hourly rate derived from the Australian Bureau of Statistics average weekly earnings (females: $19.69 per hour; males: $22.44 per hour) for reported times.[12] For time away from work with pay lost and time away from usual activities we allocated the cost to the patient and family sector; and for time away from work with no pay lost, we allocated the cost to the employer (other sector), who was paying for working hours not performed.

We identified the key cost drivers for illness, and calculated an average resource unit used per episode and 95 per cent confidence intervals (95% CI) using standard methods.[13] Where information was not available for an illness, we applied a zero value for missing data when calculating means and CIs. One-way sensitivity analyses were undertaken by using the 95 per cent confidence limits for these key cost drivers, and we calculated an average cost for all episodes, by including those illnesses where there was a hospitalisation. We also used the confidence limits to perform multi-way sensitivity analyses, with a least expensive and most expensive scenario for community-managed episodes.

Calculations were performed using Microsoft Excel.

## *Results*

One hundred and twenty-one children from 80 households were enrolled; complete individual and household demographic data about 118 children (98%)—52 females and 66 males—from 78 households (97.5%) were available and these are included in this analysis. These 118 children provided 14,430 child-days (477.3 child-months) of follow-up between 1 July and 1 December 2001. Most study households came from the highest annual income bracket: 73 per cent from bracket 4 (income > $56,000); 15 per cent from bracket 3; 6 per cent from bracket 2; and 5 per cent from bracket 1. There were 15 households with a couple and one child, 41 with a couple and two children, 17 with a couple and three children, three with a couple and four children, one household with a single parent and two children, and one household with a single parent and three children. Eight children (7%) were one year of age at enrolment, 62 (53%) were two years of age, 19 (16%) were three years of age, 18 (15%) were four years of age, and 11 (9%) were five years of age.

**Table 2. Summary of resources consumed during 202 influenza-like illnesses in 118 Melbourne children during winter and spring 2001**

| Resource | Units consumed | Patient and family sector | Healthcare sector | Other sectors | % ILI cost |
|---|---|---|---|---|---|
| General practice visits* | 89 | $255.43 | $2,174.94 | – | 5.0% |
| Other healthcare provider visits† | 10 | $156.42 | $115.00 | – | 0.2% |
| Hospital emergency department visit (no admission)‡ | 4 | – | $160.00 | – | 0.3% |
| Diagnostic tests§ | 1 | $5.00 | $28.31 | – | 0.1% |
| Antibiotics‖ | 42 | $579.47 | – | – | 1.2% |
| Other prescription medication‖ | 24 | $336.92 | – | – | 0.7% |
| Over-the-counter and other medication¶ | 244 | $2,617.40 | – | – | 5.4% |
| Paid childcare for other children** | 11 episodes | $133.00 | – | – | 0.3% |
| Travel costs seeking healthcare†† | | | | | |
| *Car* | 460.6 kms | $209.96 | – | – | 0.4% |
| *Parking* | 3 episodes | $13.00 | – | – | 0.0% |
| Time seeking healthcare‡‡ | | | | | |
| *Time away from work, pay lost* | 34.5 hours | $679.31 | – | – | 1.4% |
| *Time away from work, no pay lost* | 22.25 hours | – | – | $438.10 | 0.9% |
| *Time away from usual activities* | 67.04 hours | $1,329.18 | – | – | 2.7% |
| Excess time caring for ill child‡‡ | | | | | |
| *Time away from work, pay lost* | 81.50 hours | $1,604.74 | – | – | 3.3% |
| *Time away from work, no pay lost* | 178.60 hours | – | – | $3,609.03 | 7.4% |
| *Time away from usual activities* | 1682.54 hours | $34,212.06 | – | – | 70.3% |
| **Sector total§§** | | $42,131.87 | $2,478.24 | $4,047.14 | 100% |
| *Sector cost per ILI* | | $208.57 | $12.27 | $20.04 | |
| *Sector per cent* | | 86.6% | 5.1% | 8.3% | |
| **Total** | | $48,657.25 | **Total cost per ILI** | $240.88 | |

* Based on 2001 Medicare Benefits Schedule rates for healthcare sector costs[28] (85% of code 23—$28.75) and mean patient cost per GP/vocationally registered GP visit for 2001 ($2.87) for patient and family cost.[2]

† Based on parent-reported costs for visits to naturopaths (2 visits) and chiropractors (6 visits), and 2001 Medicare Benefits Schedule[28] fee rates for 2 specialist visits (85% of MBS code 104—$57.50) and mean patient cost per specialist visit for 2001 ($15.71) for patient and family cost.[29]

‡ Hospital emergency department (ED) visits based on the cost for emergency department presentation for the Australian Ambulatory Classes group 23: Other respiratory diseases without procedure ($40).[14]

§ Actual cost paid by parent charged not uniformly available. Government cost based on 85% of 2001 Medicare Benefits Schedule[28] fees for one chest x-ray (MBS code 58500—$33.30). The cost allocated to patient and family was the difference between the Medicare rebate and the schedule fee (15% of the Medicare Benefits Schedule Fee).[28]

‖ Given the high proportion of households in the study from the highest income bracket, we have costed all prescription medication for general PBS beneficiaries (no concessional beneficiaries); we have also assumed they were purchased without a Safety Net Entitlement Card. Prescription medication costs were the maximum recordable value for the Safety Net from the Schedule of Pharmaceutical Benefits for Approved Pharmacists and Medical Practitioners.[30] None of these individual costs exceed the maximum cost for a pharmaceutical benefit item ($21.90), so all costs were allocated to the patient and family sector.

¶ Over-the-counter medication from pharmacies is the MIMS Australia cited cost,[31] and other medication (for example, natural therapies) based on parent-report.

** Parent-reported childcare costs for other children whilst seeking care for ill child.

†† Car running costs per kilometre (business cost) from the Royal Automobile Club of Victoria (RACV) based on type, age, and engine size of car used.[32] Parking costs as reported by parents in seeking healthcare.

‡‡ All time based on parent-reported hours. Cost applied from sex-weighted Australian Bureau of Statistics (ABS) average weekly earnings, November 2001:[12] male ($852.70 per 38 hour week) and female ($748.20 per 38 hour week). Cost allocated to the employer (other sector) for time away from work, no pay lost, and to patient and family sector for time away from work, pay lost and time away from usual activities.

§§ Columns do not add exactly to total due to rounding.

There were 202 ILI community-managed episodes identified, giving an incidence rate of 0.42 ILIs per child-month (95% CI 0.36 to 0.48). There were three episodes that resulted in hospitalisation,[8] and these were not included in general calculations. During the period, 21 children had no episodes of ILI, 35 children had one episode, 30 children had two episodes, 24 children had three episodes, five children had four episodes, and three children had five episodes. We received costing information for 180 (89%) of these illnesses (Table 2). The illnesses where we did not receive burden data were shorter (median duration: 2.5 days versus 5 days) and less likely to have parent-reported fever or ear infection[8] (proportion with uncomplicated illness: 77% versus 48%), compared to those illnesses where burden data were available. Parents may have been less likely to report burden information for illnesses they felt were trivial, or resulted in no excess resource consumption.

Using the costs from these 180 for all 202 illnesses gave an average cost per ILI episode of $241 (95% CI $191 to $291). The average cost using only those illnesses we had information on was $270. The key cost driver for ILI in children was carer time spent caring for the ill child away from usual activities, making up 70 per cent of total costs. Females spent an average of 6.38 hours per episode (95% CI 4.61 to 8.15) caring for the ill child away from their usual activities, and males an average of 1.95 hours per episode (95% CI 1.05 to 2.84). The next most important non-carer time related drivers were use of non-prescription medication (5.4% of total costs, 244 episodes of use, 95% CI 215 to 273), and general practitioner visits (5.0% of total costs, 89 visits, 95% CI 68 to 110 visits).

The average cost per episode was lowest for those illnesses occurring in households from the highest income bracket: bracket 4, $208; bracket 2, $290; bracket 1, $377; and bracket 3, $449. These rankings remained the same when illnesses where there was no information available were removed from average calculations (bracket 4, $235; bracket 2, $327; bracket 1, $431; and bracket 3, $474).

Funding the resource use during illness was predominantly the responsibility of the patient and family, with this sector being responsible for meeting 87 per cent of total costs. The healthcare sector met five per cent of costs, and other sectors met eight per cent of costs.

As key costs drivers, carer time away from usual activities, non-prescription medication, and general practice visits were individually varied in one-way sensitivity analyses, according to the upper and lower 95 per cent confidence limits. The average cost per episode varied little for the sensitivity analyses involving non-prescription medication and general practice visits (Table 3), but ranged from $186 to $296 when carer time away from usual activities was varied. The one-way sensitivity analysis which included the three illnesses with hospitalisations increased average cost per episode to $287 (Table 3). Two scenarios were tested producing a least expensive average cost per episode of $177, and a most expensive average cost per episode of $304 (Table 3). Unsurprisingly, these values varied little from those generated in the one-way analyses of carer time away from usual activities.

## *Discussion*

As demonstrated by our findings, acute respiratory illness in healthy, urban children during winter and spring is common, with the costs borne largely by the patient and family. These findings have implications for preventive strategies in Australian children, particularly vaccine use. The impact carer time away from usual activities has on the average cost per episode can be seen in a number of ways: the proportion of total costs made up by this single variable (70%); and in the multi-way sensitivity analyses producing least and most expensive cost per episode scenarios varying little from the one-way sensitivity analysis of this variable alone. Not including carer time away from usual activities, as recommended for submissions to have drugs listed on the Pharmaceutical Benefits Scheme,[14] would substantially under-estimate the true impact of community-managed disease of this nature. In this regard, these illnesses may be similar to chickenpox, being common and usually community-managed, with the direct costs of a proposed infant vaccination program in Australia outweighing the direct costs associated with not implementing such a program.[15]

A significant proportion of the illnesses identified in this study are likely to have been caused by respiratory viral infections, including respiratory syncytial virus, influenza virus, parainfluenza viruses, human metapneumovirus, coronaviruses, adenoviruses, and rhinoviruses. The Victorian Infectious Diseases Reference Laboratory identifies 2001 as a year of normal seasonal activity for influenza from the collaborative sentinel influenza surveillance scheme.[16] Injectable influenza vaccine is licensed in Australia for children down to six months of age. The recent licensing in the USA of the intranasal CAIV-T vaccine provides the possibility of better access, acceptability, and delivery of public influenza vaccination programs, especially if the license for use extends to a lower age-group. The current price of the vaccine, though set to fall to USA$23.50 for the 2004/2005 influenza season in the United States of America,[17] will remain an impediment to its wider use. Vaccines against other respiratory viruses are

**Table 3. One-way and multi-way sensitivity analyses for average cost of episodes**

| Sensitivity analyses | Modification | Values used | Average cost per episode |
|---|---|---|---|
| ***One-way analyses*** | | | |
| General practice visits | Number of general practice visits and dependent variables* | Lower value: 68 visits<br>Upper value: 110 visits | $233.55<br>$247.94 |
| Over-the-counter and other medication | Number of episodes of over-the-counter and other medication use | Lower value: 215 episodes<br>Upper value: 273 episodes | $239.34<br>$242.42 |
| Carer time away from usual activities | Time spent caring from ill child away from usual activities | Lower value: 5.67 hours (4.61 female, 1.05 male)<br>Upper value: 10.99 hours (8.15 female, 2.84 male) | $186.03<br>$295.73 |
| Hospitalisation | Addition of three ILIs with a hospitalisation | All costs for these ILIs added to total costs† | $287.03 |
| ***Multi-way analyses*** | | | |
| Least expensive scenario | General practice visits*<br>Over-the-counter and other medication<br>Carer time from usual activities | 68 visits<br>215 episodes of use<br>Female carers: 4.61 hours per episode<br>Male carers: 1.05 hours per episode | $177.17 |
| Most expensive scenario | General practice visits*<br>Over-the-counter and other medication<br>Carer time from usual activities | 110 visits<br>273 episodes of use<br>Female carers: 8.15 hours per episode<br>Male carers: 2.84 hours per episode | $304.33 |

* Changes in the number of general practice visits included proportionate changes in the cost of other variables that rely on a general practice visit: diagnostic tests, antibiotics and other prescription medication, travel seeking healthcare, parking, and time seeking healthcare.

† Costing for non-hospital related costs in three additional ILIs as per Table 1. Extra diagnostic tests performed outside of hospital: urine microscopy, culture, and sensitivity (MBS code 69312—$33.00—with 2 performed), full blood evaluation (MBS code 65070—$16.70—with 2 performed), and serum biochemistry (MBS code 66515—$19.20—with 2 performed). Healthcare sector cost based on 85% of 2001 Medicare Benefits Schedule;[28] the cost allocated to patient and family for diagnostic tests was the difference between the Medicare rebate and the schedule fee (15% of the Medicare Benefits Schedule Fee).[28] Ambulance cost from Victorian Ambulance Service for emergency transport to hospital less than 10kms away (one transfer). Public hospital admission National Hospital Cost Data Collection code E62C ($2,395).[33] Private hospital admission costs as reported by the two private hospitals: overnight admission for respiratory infection $228 paid by patient and family to private hospital; overnight admission for febrile convulsion $400 paid by health insurance company (other sector cost), and $222 paid by patient and family to private hospital. Private health insurance fees not included.

under development, but still likely to be some way off; the possibility for preventing such illnesses at present is limited to influenza. Beginning in 2004 the Advisory Committee on Immunisation Practices (ACIP) in the USA have made injectable influenza vaccine part of the routine childhood immunisation schedule—for children from six months up to two years of age. This recommendation extends to household contacts (including older children) and out-of-home caregivers of all children less than two years of age.[7] Interest in this recommendation was driven by the USA Centers for Disease Control and Prevention initiating national surveillance for paediatric influenza-associated deaths.[18] It is also possible that use of vaccine in this age group may lead to reduced incidence of disease in other age-groups due to herd protection, similar to effects seen from vaccinating school-aged children against influenza,[19,20] and more recently, seen in vaccinating USA infants with conjugate pneumococcal vaccine.[21]

Vaccination programs against illnesses that are largely managed in the community may not appear cost-effective if the impact of lost productivity is ignored. Vaccines against influenza and other viral respiratory pathogens may be recommended for young children in the near future, but may not pass the cost-effectiveness hurdle for public funding. There are few published studies looking at the cost-effectiveness of influenza vaccine specifically in children. Given the findings of our study, it will not be surprising that a childhood influenza vaccine program could be potentially cost-saving if indirect costs are included.[22] The reduction in indirect costs is central to the economic benefits of vaccina-

tion,[23–25] with previous studies showing that these benefits are greatest when parents are prevented from missing work to care for an ill child.[24]

The majority of households (73%) in our study came from the highest income bracket. This compares with approximately 40 per cent of Victorian family households being in this income range, according to 2001 Census data.[26] This may have had a number of impacts: members of lower income households have been shown to have a higher incidence of respiratory viral infections, thought to be due to the impact of crowding;[27] but in this study we did not find a lower rate of illness in children from high income families.[8] It could be argued that parents from relatively higher income households might be more likely to expend more resources in caring for a sick child, as compared with those from a lower income household; but we found that the average cost per episode was lowest in households from the highest income bracket. In our study, high-income households were more likely to have both parents spending some time working outside the home. Parents in these households might have different thresholds for seeking medical attention or using medication for illnesses that are perceived to be mild or of minor significance. If anything, due to the lower average cost per episode in higher income households, the over-representation of such households in our study may have made our cost estimate conservative.

Costing studies such as this, together with studies that measure the relative role of specific pathogens, will not only inform local cost-effectiveness studies, but failing public funding of programs, will provide important information for vaccine providers and parents about the likely benefits of paying for available vaccines themselves.

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# Hepatitis C prevalence — a nationwide serosurvey

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## Abstract

**Hepatitis C is the most commonly notified disease in Australia. In 1998 the Hepatitis C Virus Projections Working Group (HCPWG) estimated that there were approximately 210,000 people who had been infected by hepatitis C virus (HCV) in Australia by 2001. Population-based serosurveys are required to validate this estimate. Here we estimate HCV prevalence on the basis of HCV antibody seroprevalence in the Australian national serosurvey. Between 1996 and 1998, 2,800 sera opportunistically collected from pathology laboratories throughout Australia were tested for HCV antibody. National HCV notifications reported from 1991 through 1998 were also assessed. Eighty-one sera were HCV antibody positive, giving an age standardised prevalence of 2.3 per cent (95% CI 1.8%–2.9%). The 20–24 year age group had the highest HCV prevalence, 5.3 per cent (95%CI 3.3%–8.1%) and the male to female ratio was 1.8:1.0. Approximately 111,000 HCV notifications were received from 1991 through 1998. HCV prevalence estimated by the serosurvey is approximately three times higher than cumulative HCV notifications. Age and sex distributions of seroprevalence are broadly consistent with cumulative notification data. These distributions are consistent with the majority of HCV infections in Australia being transmitted by injecting drug use. Very low age specific seroprevalence estimates in the over 50 years age group indicate that there is not a large pool of undiagnosed infection in this age group. The serosurvey provides an estimate of Australian HCV prevalence and baseline data to determine incidence trends, both of which are required for health-care planning. *Commun Dis Intell* 2004;28:517–521.**

*Keywords: hepatitis C, prevalence, serosurvey*

## *Introduction*

Over the last decade hepatitis C (HCV) has been the most commonly notified infectious disease in Australia.[1] The large population of people with HCV, together with estimates of continued high HCV incidence and the often long latency of HCV related disease, will produce an escalating health burden for at least the next two decades.[2] The already increasing incidence of hepatocellular carcinoma is thought to be related to the expanding HCV epidemic and cirrhosis due to chronic HCV infection is already the most common underlying reason for liver transplantaion.[3,4] The true extent of this epidemic is however not known.

To date estimates of HCV prevalence and incidence have primarily relied on data from specific populations such as prison entrants, injecting drug users, blood product recipients, antenatal populations and blood donors.[5–13] In 1998 the Hepatitis C Virus Projections Working Group (HCPWG) was formed with a brief to determine estimates of HCV incidence and prevalence in Australia. The HCPWG estimated that there were approximately 210,000 people who had been infected by HCV in Australian by 2001.[2] This estimate was primarily derived from estimates of the prevalence of injecting drug use (IDU) and the incidence of HCV infection among IDUs. Population-based serosurveys are required to validate this estimate.

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The first Australian national serosurvey of selected infectious diseases was conducted using sera collected in 1996–1998. Here we report the results for HCV antibody testing of specimens from the national serosurvey. We also compare the HCV seroprevalence estimates with the cumulative number of HCV notifications, to assess the potential extent of undiagnosed and unreported HCV infection in Australia.

## *Methods*

### Serosurvey

Details of the serosurvey are presented elsewhere.[14] The samples were collected between July 1996 and December 1998. All major public and private diagnostic laboratories, including reference laboratories, throughout Australia were invited to contribute sera that had been submitted for diagnostic testing and would otherwise have been discarded. Forty-five of the 52 invited laboratories agreed to participate. Sera submitted to laboratories from sexual health clinics were excluded from this study.

All sera were tested by an indirect enzyme immunoassay (EIA), MONOLISA anti-HCV PLUS Version 2 (BIO-RAD, Marnes la Coquette, France) according to the manufacturer's directions. Samples with an optical density (OD) below that of the cut-off less 10 per cent were deemed negative. All other samples were retested. On retesting, samples giving an OD less than the cut-off were reported as negative. Samples with an OD between that of the cut-off and the cut-off plus 10 per cent recorded as weak positive and all others as positive. The reactivity of samples in the latter two categories was confirmed by a strip immunoblot (using two encoded antigens and three encoded synthetic peptides), CHIRON* RIBA* HCV 3.0 SIA (Chiron Corporation, Emeryville, California, United States of America. Distributed by Ortho-Clinical Diagnostics, Incorporated, Raritan, New Jersey, United States of America) according to the manufacturer's guidelines. The band patterns were interpreted as negative, indeterminate or positive for HCV infection. Indeterminate sera were subsequently assigned as negative for HCV infection.

Sample sizes were calculated for the following age groups: 1–4, 5–9, 10–14, 15–19, 20–24, 25–29, 30–39, 40–49, 50–59, 60–69 and 70+ years, based on the expected prevalence of hepatitis C antibodies. Within each age group, states and territories were sampled proportionally to their population size. Sample sizes were calculated to achieve confidence intervals of approximately +/-5 per cent for each age group. Approximately equal numbers of sera from males and females were tested. Australian Bureau of Statistics (ABS) end of 1997 population estimates were used to age standardise seroprevalence and to estimate the number of people exposed to HCV.[15]

### Notifications

Hepatitis C notification data (acute and unspecified) was extracted from the National Notifiable Diseases Surveillance System in August 2003 for cases with report date between 1 January 1991 and 31 December 1998 (Personal communication - Communicable Diseases Network Australia - National Notifiable Diseases Surveillance System, 2003). For comparison with the results of the serosurvey, the age distribution of cumulative notifications up to 1998 was determined by calculating age on the 1st of July 1998 based on date of birth or, if not available, the age at notification for each notification. Cumulative notification rates were calculated using the ABS end of 1997 population estimates.[15]

Confidence intervals for HCV seroprevalence and cumulative notification rates by age group were calculated using exact binomial methods.

### Ethics approval

The study was approved by appropriate institutional ethics committees and the State-wide Health Confidentiality and Ethics Committee of the New South Wales Health Department.

## *Results*

Of the 2,800 sera tested, 81 were HCV antibody positive, giving an age standardised seroprevalence of 2.3 per cent (95% CI 1.8%–2.9%). This corresponds to approximately 433,000 people (95% CI 336,000 to 530,000) with HCV antibodies, in Australia, in 1998. Approximately 111,000 HCV notifications were reported from 1991 through 1998.

The age distribution for HCV seroprevalence and cumulative notification rate were similar, with the highest rates in the 20 to 49 year age groups and lowest rates in the age groups under 15 years and 50 years and older (Figure 1). However, the highest seroprevalence was in the 20 to 24 year age group, 5.3 per cent (95% CI 3.3%–8.1%) while the highest cumulative notification rate was in the 30 to 39 years age group, 1.1 per cent. There were more seropositive males than females overall (male: female ratio 1.8:1; 95% CI 1.3–3.0). This sex difference was greatest in the 25 to 29 years age group (Figure 2). The cumulative notification rate was also higher in males than in females (male:female ratio 1.7:1).

**Figure 1. Age distribution of hepatitis C virus prevalence (percentage of population HCV positive) as determined by serosurvey and cumulative (1991 to 1998) notification rate**

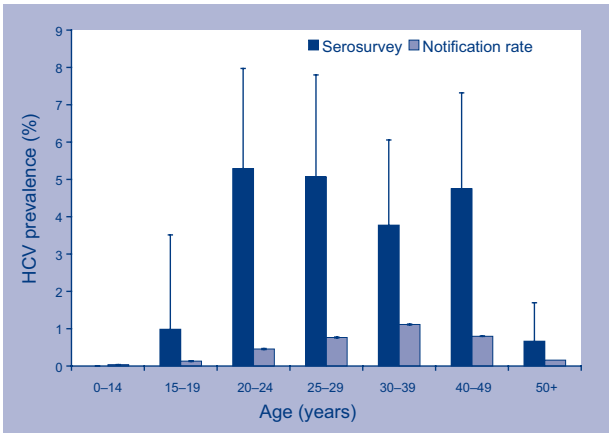
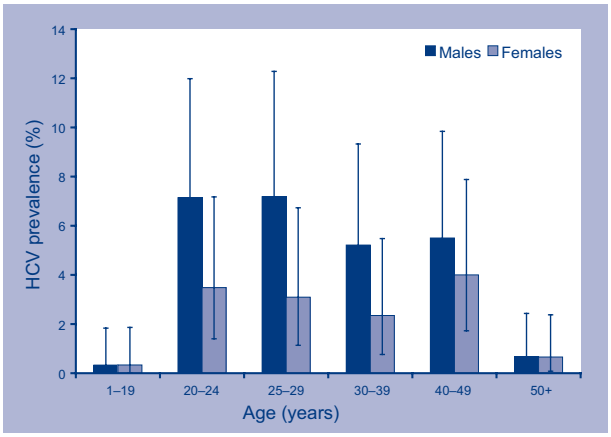



**Figure 2. Age distribution of hepatitis C virus prevalence by sex in the HCV serosurvey**




## *Discussion*

The prevalence estimate from the serosurvey of approximately 433,000 persons with HCV antibodies in Australia in 1998 is almost four fold greater than the 110,000 cumulative notifications reported from 1991 to the end of 1998 and more than double the 196,000 of people estimated to be seropositive in 1997 by mathematical modelling.[16]

The cumulative HCV notification rate, and to a lesser extent the modelled estimates, are likely to underestimate HCV prevalence. Diagnostic testing for HCV only became available in late 1989 and while notification of HCV commenced in 1991, all states and territories did not report until 1995.[1,17] Also, cumulative notifications from 1991 through 1998, underestimate HCV prevalence because of under reporting in the early years of HCV surveillance, low levels of testing in some at risk populations, the generally asymptomatic nature of acute HCV infection, and the long latency of HCV related disease. The modelled estimates of HCV incidence were primarily based on the prevalence of IDU and HCV incidence amongst IDUs.[16] Underestimates of either measure would result in an under estimate of population HCV prevalence. An under estimate of the proportion of HCV acquired through non-IDU modes of transmission (around 20% in the HCPWG estimates) two would also produce an underestimate of population HCV prevalence. It is also possible that the serosurvey overestimates HCV prevalence.

Anonymous opportunistic testing of remanent sera has been shown to provide estimates of immunity comparable with a random population based survey of some vaccine preventable diseases.[18,19] However, the lack of detailed information about participants in opportunistic serosurveys means that it is not possible to identify or control for various potential biases. In this serosurvey, the potential for selection bias was reduced by enrolling 87 per cent of major laboratories and including primarily ambulatory rather than hospitalised patients.[14] The majority of Australian laboratories are located in major centres which may have resulted in selection bias. However, a comparison of Accessibility and Remoteness Index Australia scores for the population tested in the serosurvey and the Australian population showed that remote areas were not under-represented (data not shown). It is possible that people with chronic HCV were over sampled due to their potentially higher utilisation of health care services. The effect of this bias would be partially ameliorated by exclusion of sera from sexual health clinics but ideally sera from liver clinics should also have been excluded. Further, while this bias would result in an over estimation of overall prevalence, it should not distort the pattern of HCV seroprevalence by age or sex for HCV.

The low seroprevalence in persons over the age of 50 years in this study is consistent with findings from an opportunistic serosurvey in England and Wales and a population based random survey in the United States of America.[20,21] In both of these surveys, seroprevalence was found to be associated with drug use. In contrast, a study from Italy in which seroprevalence was highest in older age groups, showed an association with medical interventions.[22] The low levels of notification and seroprevalence in older age groups in Australia indicate that these age groups are not particularly subject to under notification and there is unlikely to be a hidden epidemic related to medical interventions in older Australians. A more robust estimate of HCV seroprevalence and conclusive evidence that IDU is the main source of infection in Australia, could be ascertained by undertaking a population based random sampled serosurvey in which risk factor information is col-

lected. However, such surveys are costly and can also be biased by non-participation especially by high-risk groups such as IDUs.

The impact on the health system of illness associated with chronic HCV infection over the next decade is likely to increase as those aged 20 to 50 in this study approach the duration of infection, 15–25 years, at which severe complications of hepatitis C arise.[2,23] The projected high prevalence of such complications, makes the planning of health services to provide appropriate and accessible services and treatment, including antiviral therapy, imperative.

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# A multi-jurisdictional outbreak of hepatitis A related to a youth camp — implications for catering operations and mass gatherings.

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## Abstract

**In June 2003, Australian state and territory health departments were notified of an outbreak of Hepatitis A in people who had attended a five-day youth camp. Approximately 350 people attended the event in Central Australia between 24 and 28 April 2003. The public health investigation comprised of case identification, food handler interviews, an environmental health investigation of the campground and associated food premises, laboratory analysis of blood specimens and food/water samples, and an epidemiological study. Twenty-one cases fitted the case definition for the outbreak. A retrospective cohort study involving four states was conducted, with 213 people interviewed. Coleslaw and cordial were significantly associated with illness, however when the two exposures were adjusted for each other to account for confounding, only coleslaw remained significantly associated with illness (adjusted RR 2.5, 95% CI 1.09 – 5.77). The investigation highlighted a number of food hygiene and safety issues relating to the catering of mass gatherings. Implementation of food safety programs in these settings are likely to reduce the occurrence of such outbreaks. The recent proposal by Food Standards Australia New Zealand to mandate food safety programs for catering operations is supported. *Commun Dis Intell* 2004;28:521–527.**

*Keywords: Hepatitis A, camp, multi-jurisdictional, catering operations, mass gatherings*

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## *Introduction*

Hepatitis A is a notifiable disease in every state and territory of Australia. Although the disease can vary in severity, symptoms and duration, the illness is generally mild and characterised by fever, malaise, anorexia, nausea, dark coloured urine, abdominal pain and jaundice.[1] Asymptomatic infection may occur, however this is more predominant in children under five years of age.[1] Infection can occur through person-to-person transmission, or by ingestion of contaminated food or water. In Australia, the illness is usually associated with household or sexual contact with a case, illicit drug use, childcare facilities, institutions or overseas travel, with foodborne disease outbreaks attributed to contaminated shellfish or foods contaminated by infectious food handlers.[2–7]

## *Background*

Between the 29 May and 4 June 2003, the Public and Environmental Health Service of the Department of Health and Human Services in Tasmania, received four separate notifications of laboratory-confirmed Hepatitis A. Initial interviews indicated that the cases had travelled by bus (3) or aeroplane (1) to the Northern Territory in late April to attend a five-day youth camp. Discussions with the camp organisers indicated that approximately 350 people had travelled to the camp by bus from Queensland, Victoria, New South Wales, Western Australia, and Tasmania, which prompted an investigation to identify other possible cases and the source of the infection.

The youth camp was held at a large campground in Central Australia from 24–28 April 2003. People attending the camp were accommodated in three separate areas of the campground. Each state bus group brought their own portable kitchen, which was used to provide all but one meal for their state attendees. The camp program included visits to local tourist attractions, group discussions, and social activities. There were two occasions where all state groups were present at the same time: a social function on the evening of Friday 25 April, and a Saturday (26 April) afternoon festival, which included social activities, a barbeque, concert and water ceremony relating to Indigenous reconciliation.

## *Methods*

### Case identification

Jurisdictional health departments were alerted to the possibility of a nationwide outbreak through Communicable Disease Network Australia and OzFoodNet. Travel histories of hepatitis A notifications received since the start of the year were reviewed to ascertain whether the case had travelled to Central Australia, whilst newly notified cases were asked if they had attended the camp.

A letter advising of the outbreak and symptoms of hepatitis A was forwarded to all people who had attended the camp. As a control measure, household and close contacts of cases were contacted and advised to visit their general practitioner for immunisation with normal human immunoglobulin.

### Environmental health investigation

A Regional Environmental Health Officer from the Northern Territory Department of Health and Community Services conducted an environmental investigation on 11 and 12 June 2003. The investigation focussed on the temporary kitchen used for the preparation of group meals, local food premises used to supply ingredients for the camp, water treatment and supply, wastewater disposal, the constructional and hygiene standards of camp facilities, and campground layout. Samples of water and ice obtained during the inspection were submitted to the Water Microbiological Laboratory (Northern Territory Department of Business, Industry and Resource Development) for microbiological analysis.

### Food handler interview

State and territory health departments interviewed food handlers on their food handling activities at the camp and their knowledge of food hygiene principles.

The interview included questions on preparation methods of high-risk foods and history of illness before, during and after the camp.

### Laboratory analysis

All known food handlers involved in preparing group meals were asked to submit a blood specimen for hepatitis A viral (HAV) serology to determine whether they could have been infectious around the time of the camp. States reporting cases associated with the outbreak were asked to send blood specimens from cases to the Institute of Medical and Veterinary Science, Adelaide—for genotyping using reverse transcriptase polymerase chain reaction methodology.[8]

### Epidemiological investigation

Following a review of notifications and initial case interview, a retrospective cohort study was conducted, involving the four states (Queensland, Tasmania, Victoria and New South Wales) that

reported cases associated with the camp. The cohort was defined as persons who attended the camp between the 24 and 28 April 2003. This epidemiological approach was chosen as the population who attended the camp was well defined and the names and contact details of attendees were obtainable.

The case definition for the outbreak was a person with serologically confirmed hepatitis A, who had attended the camp, with a definitive onset date of hepatitis A-like illness within fifty days of the camp.

From discussions with the organisers of the camp and hypothesis generating interviews with cases, a questionnaire was developed for interviewing the cohort. Questions were designed to obtain information about contact with known or suspected cases of hepatitis A, HAV immunisation status, illness history, food items consumed, drinking water sources, tourist attractions visited, contact with local indigenous communities, camp activities attended, frequency of hand washing and hand washing technique. Potential risk factors associated with hepatitis A infection including sexual contact, sharing of cigarettes and drug paraphernalia, and sharing of eating and drinking utensils were also addressed. State health departments conducted telephone or face-to-face interviews. Risk ratios (RR) were calculated for common food items, tourist attractions visited, drinking water sources, amenities used (including male/female facilities), attendance of events, and other risk factors associated with hepatitis A infection, using Stata Statistical and Data Analysis package, Version 8.0.[9]

## *Results*

### Case identification

Through laboratory notifications, 21 cases of hepatitis A were identified among those people attending the youth camp in the Northern Territory. An additional case was identified as a result of the cohort study. When reviewing the case histories, one case was identified as not fitting the case definition and was excluded from the cohort study. There were no other cases of hepatitis A infection reported in people who visited the locality before, during or after the camp.

### Environmental health investigation

The environmental health investigation was conducted whilst equipment used at the organisation's campsite was being dismantled. It was noted that the temporary kitchen used for food preparation for the camp did not have a designated hand washing facility. Food premises that had supplied ingredients for food and drinks served during the camp were inspected and found to comply with the legislation. Samples from an ice machine that was used to supply the camp with ice complied with microbiological criteria.

The campground and nearby town were supplied with treated bore water. Camp organisers used water from this source to fill bulk containers with water and cordial, which were supplied for group activities. There were no problems identified with the water treatment or water supply system around the time period of the camp, and samples of water taken during the environmental health investigation complied with microbiological requirements for drinking water. Amenity blocks at the time of the inspection were found to be clean and supplied with soap and disposable towels. Constructional standards of other camp facilities were satisfactory.

### Laboratory results

Blood specimens from eight of the nine food handlers were submitted for serology. All specimens were IgM anti-HAV negative. One food handler declined to be tested. Thirteen blood specimens from cases associated with the outbreak were sent to the Institute of Medical and Veterinary Science for genotyping. HAV ribonucleic acid sequence data indicated that at least ten and most likely twelve of the cases were the result of an infection with a common HAV isolate. One sample could not be genotyped. The HAV isolate was closely related to genotype 3A reference sequence.[8]

### Epidemiological investigation

Twenty-one people fitted the case definition for the outbreak, however one case was lost to follow up between the initial case interview and the cohort study, and therefore not included in the epidemiological investigation. In total 213 camp participants were interviewed. Demographics and attack rates for states and the cohort overall were calculated (Table 1). Nausea was reported as the predominant symptom (100%), followed by jaundice (95%), anorexia (75%), body aches, headaches and dark urine (70% each). Onset dates for the cases ranged from the 19 May to the 5 June 2003. (Figure)

Attack rates and risk ratios for food items are outlined in Table 2. None of the non-food variables were found to be significantly associated with illness and have not been shown in the table.

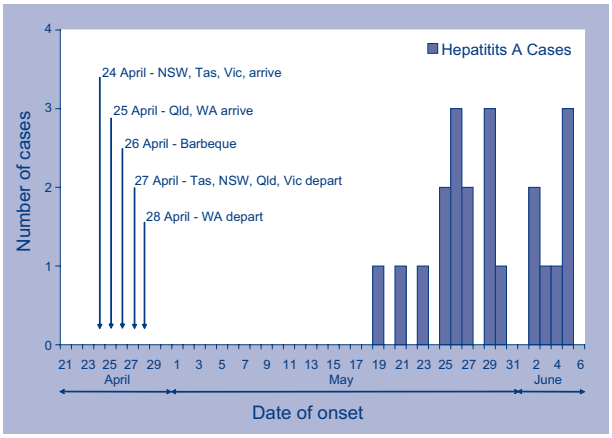
Reported consumption of both coleslaw and cordial served at the barbeque was significantly associated with hepatitis A infection, with crude RRs of 3.42 and 2.46 respectively. When coleslaw and cordial were adjusted for each other to control for confounding, only the association between coleslaw and illness remained statistically significant (Mantel Haenzel adjusted RR 2.5, 95% CI 1.09–5.77).

**Table 1. Attack rates and demographics of states attending a Youth Camp in Central Australia during April 2003 (n=213)**

| State | Number attending | Number interviewed | Number of cases | | Attack rate (%) | Median age (range) - interviewed |
|---|---|---|---|---|---|---|
| | | | Notified * | Interviewed | | |
| State A | 45 | 23 | 1 | 1 | 4.3 | 17 (10–49) |
| State B | 84 | 50 | 8 | 8 | 16.0 | 15 (11–52) |
| State C | 111 | 95 | 8 | 7 | 7.4 | 18 (11–61) |
| State D | 49 | 45 | 4 | 4 | 8.9 | 16 (12–57) |
| Overall | 289 | 213 | 21 | 20 | 9.4 | 16 (10–61) |

* includes only those people who fitted the case definition

**Figure. Epidemic Curve of cases of hepatitis A among people attending a Youth Camp in Central Australia during April 2003, by Onset Date (n=21)**




The Queensland bus group consumed leftover sausages and coleslaw from the barbeque the following day. Variables for the two products served at the barbeque and as leftovers were combined as 'coleslaw (BBQ and/or leftovers) and sausage (BBQ and/or leftovers). Coleslaw (BBQ and/or leftovers) was found to have a significant association with illness (RR 4.92, 95% CI 1.8–13.2). Attributable risk percentages were calculated for coleslaw (BBQ and/or leftovers) (79.7%) and coleslaw served at the barbeque (70.7%).

## *Discussion*

We have found strong evidence that this outbreak of hepatitis A was associated with a five-day youth camp in the Northern Territory. We found a temporal relationship between attendance at the camp and development of symptoms within the incubation period. Genotyping of available sera suggested that the cases were related. Elevated risk ratios were identified for exposure to coleslaw and coleslaw (BBQ and/or leftovers) with disease. Attributable risk percentages for coleslaw and coleslaw (BBQ and/or leftovers) were high (70.7% and 79.7% respectively). This evidence suggests that coleslaw was the likely vehicle for the outbreak.

Two batches of coleslaw were made for the barbeque on the Saturday night. A large batch was prepared in a portable kitchen by two food handlers associated with one of the state groups. A second, smaller batch was prepared by a local resident in their own home. Both batches were made from ingredients (cabbage, carrot, capsicum, apple and commercial mayonnaise) purchased from a local food premises. The leftover coleslaw was given to the Queensland bus group for consumption the following day.

Three cases from Queensland did not attend any of the activities on the Saturday, including the barbeque where the coleslaw was served. However, one case could recall eating leftover coleslaw the following day, whilst the other two cases were unsure whether they consumed the leftover product. The RR for eating coleslaw (BBQ and/or leftover) was 4.92 (95% CI 1.8–13.2), supporting the proposition that coleslaw was the likely vehicle for this outbreak. However five cases did not report eating coleslaw at either the barbeque or as leftovers. This suggests that there may have been other exposures, or that cases who did not report eating coleslaw could not recall, or possibly did not know what the coleslaw was. Some errors in recall are likely given the inevitable time lapse between exposure and the commencement of the investigation, and possibly the age of the cohort.

It is possible that the raw ingredients used to make the coleslaw were the source of the contamination. Overseas, hepatitis A outbreaks associated with fruit and vegetable consumption have been linked to the use of contaminated fertilisers or irrigation supplies, or by people handling the product during harvesting or packing process.[10–16] However, hepatitis outbreaks implicating these products have not been detected in Australia. The ingredients used for the coleslaw could have been contaminated at a farm level, how-

**Table 2. Attack rates and risk ratios for exposure to specific foods in people attending a Youth Camp in Central Australia during April 2003 (n=213)**

| Exposure | Exposed | | | Not exposed | | | | |
|---|---|---|---|---|---|---|---|---|
| | Number ill | Total | Attack rate (%) | Number ill | Total | Attack rate (%) | Risk ratios | 95% CI |
| **Social Activity: Friday 25 April** | | | | | | | | |
| Hot Chocolate | 10 | 125 | 8.0 | 10 | 88 | 11.4 | 0.70 | 0.31–1.62 |
| Marshmallows | 10 | 119 | 8.4 | 10 | 94 | 10.6 | 0.79 | 0.34–1.82 |
| **Barbeque: Saturday 26 April** | | | | | | | | |
| Sausage | 14 | 148 | 9.5 | 6 | 65 | 9.2 | 1.02 | 0.41–2.55 |
| Sausage (BBQ &/or leftovers) | 16 | 153 | 10.4 | 4 | 60 | 6.7 | 1.57 | 0.55–4.50 |
| Vegetable Patty | 3 | 20 | 15.0 | 17 | 193 | 8.8 | 1.70 | 0.55–5.31 |
| Bread | 16 | 160 | 10.0 | 4 | 53 | 7.5 | 1.33 | 0.46–3.79 |
| Sauce | 11 | 135 | 8.1 | 9 | 78 | 11.5 | 0.71 | 0.31–1.63 |
| Coleslaw | 12 | 65 | 18.5 | 8 | 140 | 5.4 | **3.42** | **1.47–7.96** |
| Coleslaw (BBQ &/or leftovers) | 13 | 73 | 17.8 | 5 | 138 | 3.6 | **4.92** | **1.8–13.2** |
| Onion | 9 | 79 | 11.4 | 11 | 134 | 8.2 | 1.39 | 0.60–3.2 |
| Choc. Cake | 9 | 100 | 9.0 | 11 | 113 | 9.7 | 0.92 | 0.40–2.14 |
| Butter Cake | 3 | 31 | 9.7 | 17 | 182 | 9.3 | 1.04 | 0.32–3.33 |
| Cordial at BBQ | 10 | 64 | 15.6 | 9 | 142 | 6.3 | **2.46** | **1.05–5.77** |
| Water at BBQ | 6 | 47 | 12.7 | 13 | 159 | 8.2 | 1.56 | 0.63–3.88 |
| **Water Ceremony: Saturday 26 April** | | | | | | | | |
| Water | 16 | 178 | 9.0 | 4 | 35 | 11.4 | 0.79 | 0.28–2.21 |
| **Afternoon Programme: Saturday 26 April** | | | | | | | | |
| All water | 10 | 102 | 9.8 | 10 | 111 | 9.0 | 1.09 | 0.47–2.51 |
| All cordial | 15 | 141 | 10.6 | 5 | 72 | 6.9 | 1.53 | 0.58–4.05 |

ever if this were the case, an increase in hepatitis A notifications in a region or across Australia would be expected and was not found.

It is most likely that an infectious food handler contaminated the coleslaw. It is possible that there was an infectious food handler not known to the investigating team and therefore missed in the interview and testing process. There were reports of camp participants assisting in serving food at the barbeque, however the designated food handlers did not substantiate this report. Another possibility is that the food handler who refused to submit a blood specimen for serology was infectious at the time of the camp. When interviewed, the food handler denied having hepatitis A symptoms before, during or after the camp. It should be noted that this person had prepared meals for one of the state groups on the bus trip to and from Central Australia and if infectious with hepatitis A, a higher attack rate for this state would've been expected. This was not evident. Another explanation is that the second batch of coleslaw was contaminated. Although the person preparing the product had provided a blood specimen for serology, household contacts were not approached for testing. Whilst these three scenarios may possibly explain the contamination of the coleslaw by an infectious food handler, they are difficult to prove.

Contaminated water was initially a hypothesis of the investigating team, and was a major focus of the environmental health investigation. There were no problems identified with the water treatment facility, and microbiological samples obtained during the inspection complied with drinking water guidelines. Although it is possible that the water supply was the source of the infection, water consumption at any of the activities did not have an association with illness, and there were no cases of hepatitis A reported in people who did not attend the camp.

This outbreak highlights a number of important issues concerning catering operations associated with mass gatherings. Interviews with camp organisers and anecdotal reports together with the environ-

mental health investigation suggested there were a number of concerns associated with construction of the temporary food premises, food handler training, preparation of food in designated food premises and the hygienic preparation of food. It is felt that a food safety program and audit process would have addressed the problems found in relation to food hygiene and food safety, and could have ultimately reduced the likelihood of this outbreak occurring.

Food Standards Australia New Zealand recently released an initial assessment report (Proposal 290) recommending that food safety programs are a mandatory requirement for catering operations.[17] This proposal is based on the report released by the National Risk Validation Project, which identified high-risk food industries, and the costs/benefits associated with implementation of food safety programs in these sectors. Catering operations that served potentially hazardous foods to large numbers of people were classified as high risk, with 30 per cent of food-borne illness outbreaks being attributable to this sector.[18] In consideration of this outbreak, the recommendation by Food Standards Australia New Zealand to mandate food safety programs to this sector should be supported.

## *Acknowledgements*

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# Outbreak of *Salmonella* Singapore associated with eating sushi

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An outbreak investigation of *Salmonella* Singapore associated with sushi consumption in Queensland was conducted in March–April 2004. There appear to be no previously published outbreaks of salmonellosis associated with sushi consumption in Australia.

On 30 March 2004, Queensland Health Scientific Services notified the Queensland OzFoodNet site (Queensland Health Communicable Diseases Unit) of a cluster of six cases of *S*. Singapore infection, with specimen collection dates over a ten day period. All cases were 20–39 years of age and lived in the Brisbane and Gold Coast regions.

Telephone interviews were initially conducted for five cases using a hypothesis-generating questionnaire. These interviews revealed that all five cases worked in the Brisbane Central Business District (CBD) and had eaten takeaway sushi as a lunch meal in the five days prior to their illness. Four cases reported eating takeaway sushi rolls from the same sushi takeaway outlet (outlet A) and one case from a different sushi outlet in the Brisbane CBD (outlet B).

A case control study was commenced to attempt to identify the likely food vehicle or source of infection for this outbreak. A case was defined as either a laboratory confirmed case of *S*. Singapore infection notified from 28 March or an epidemiologically linked case who reported a gastrointestinal illness including diarrhoea within three days of eating sushi at outlet A or after eating sushi for a lunch meal with a laboratory confirmed case from 15 March. Population-based controls were sourced from Queensland Health employees working in corporate office in the Brisbane CBD and were matched to cases by gender and broad age groups. Case-nominated controls who attended the sushi outlet at the same time as the case were also sought, but insufficient numbers excluded their use in the analysis.

Thirteen cases of *S*. Singapore infection were notified to Queensland Health between 29 March and 7 April 2004. Eleven laboratory confirmed and one epidemiologically-linked case were interviewed. Sixteen population-based controls were eligible to participate and were included in the study. All but one case worked in the Brisbane CBD and all cases had eaten a lunch meal in the Brisbane CBD. Cases had a median age of 26 years (age range 20–34 years), five (42%) were male and seven (58%) were female. Controls had a median age of 34 years (age range 25–56 years), four (25%) were male and twelve (75%) were female.

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All 12 cases reported stomach cramps and diarrhoea while only one case reported vomiting. The median duration of illness was 5.5 days (range 3 to 9 days) for the eight cases who were no longer symptomatic when interviewed. No cases were hospitalised.

All 12 cases ate a lunch meal from a sushi outlet in the Brisbane CBD in the seven days prior to their illness. Eleven of these 12 cases consumed a sushi roll for lunch from the same sushi outlet (outlet A) over an 18-day period between the 16 March and 2 April 2004. Seven of the 16 controls ate a lunch meal from a sushi outlet in the Brisbane CBD during the same exposure period but none from outlet A. *S*. Singapore infection was associated with eating a sushi roll from outlet A (odds ratio undefined; p<0.001). An association between specific food items from a sushi outlet and illness could not be established from this study as cases consumed a wide variety of sushi rolls and frequently consumed more than one type. A variety of other takeaway food venues were included in the case control study but none were significantly associated with infection.

An environmental investigation of outlet A was conducted on 6 April. Food items (raw and cooked) were sampled for analysis and swabs were obtained from food preparation equipment and areas. All samples were culture negative for *Salmonella* species. The inspection identified several food hygiene issues including inadequate hand washing facilities, bare hand contact with ready-to-eat food, and mayonnaise prepared on-site with raw egg yolk as an ingredient but separating the yolk from the white using the egg shells. The mayonnaise was prepared weekly and was left at room temperature during opening times. Eggs were traced to a large producer which had quality systems in place including egg washing. The internal temperature of displayed sushi products when measured with a temperature probe varied between 17°C and 24°C. No illness was reported among staff members.

The probable vehicle of *S*. Singapore infection in this outbreak was sushi rolls purchased from outlet A in the Brisbane CBD, however, the environmental investigation could not confirm the source of the *Salmonella*.

Outbreaks of foodborne disease associated with sushi have rarely been described in the literature. Sushi rice is generally handled (moulded or pressed) at temperatures between 21°C and 25°C and usually involves considerable bare hand contact. Traditionally, sushi is not refrigerated and is often displayed and eaten at room temperature. Pathogen growth may be inhibited through acidification of the rice. However sushi may contain potentially hazardous foods used as fillings. Acidification of the rice will not necessarily inhibit pathogen growth in the fillings.

A recent microbiological survey of sushi conducted in the Australian Capital Territory[1] showed that 17/55 (31%) sushi samples were contaminated with potential foodborne pathogens at levels outside of acceptable microbiological limits for ready-to-eat foods[2]. This survey found only 63% of rice samples had a pH below the recommended level[3] of ≤ 4.8 with a median of 4.7 (range 4.3–5.9). The median temperature of 45 sushi samples tested was 15ºC (range <5ºC–25ºC).

The Australia New Zealand Food Standards Code requires that potentially hazardous food (such as sushi) be displayed under temperature control (5ºC or less). However, potentially hazardous food may be displayed at another temperature if the food business demonstrates that maintenance of the food at this temperature will not adversely affect the microbiological quality of the food.

The Victorian Department of Human Services has recently developed a sushi supplement for businesses with a food safety program that prepare, receive and/or display sushi[3]. Food businesses that strictly adhere to the recommended procedures can display sushi (nori rolls) for up to 12 hours out of temperature range if the display temperature is maintained at ≤15ºC and the pH of the rice is ≤ 4.8. For businesses that do not wish to adhere to these guidelines, sushi displayed at >5ºC should be eaten after 2–4 hours or discarded after more than four hours.

As this outbreak involved food prepared and consumed over an 18-day period, it is likely that *Salmonella* was introduced from a contaminated raw product and used directly as an ingredient or was a constituent of one of the sushi ingredients. It is doubtful whether adherence to the above guidelines would have prevented the occurrence of this outbreak.

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# Reporting of communicable disease conditions under surveillance by the APSU, 1 January to 30 June 2004

*Compiled by Elizabeth Elliott, Donna Rose*
*Australian Paediatric Surveillance Unit*

## *Background*

The Australian Paediatric Surveillance Unit (APSU) was established in 1993 and is a unit of the Division of Paediatrics and Child Health, Royal Australasian College of Physicians. The activities of the APSU are funded in part by the Federal Department of Health and Ageing through the communicable diseases program. The APSU is a founding member of the International Network of Paediatric Surveillance Units (INoPSU). INoPSU now has 15 member units who employ a similar methodology.

The APSU conducts national active surveillance of rare diseases of childhood, including infectious and vaccine preventable diseases, genetic disorders, childhood injuries and mental health conditions. Surveillance through the APSU provides the only available method of national data collection for most of the childhood conditions studied.

The primary aim of the APSU is to document the epidemiology of the conditions under surveillance, their clinical features, current management and short-term outcome. The APSU's secondary aims are to provide a mechanism for national collaborative research and to disseminate data acquired by the Unit to inform best practice, appropriate prevention strategies and optimal health resource allocation.

Contributors to the APSU are clinicians known to be working in paediatrics and child health in Australia. In 2003, and average of 1,050 clinicians participated in the monthly surveillance of 14 conditions, with an overall response rate of 96 per cent.

As one-hundred per cent case ascertainment is unlikely to be achieved by any one surveillance scheme, additional data sources are used to supplement or verify case finding through the APSU. For further information please contact the APSU on telephone: +61 2 9845 2200 or email: apsu@chw.edu.au

## *About the APSU communicable diseases studies:*

### Acute flaccid paralysis

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Acute flaccid paralysis (AFP) surveillance in children under 15 years of age was initiated in 1995 to help meet the World Health Organisation (WHO) certification standards for poliomyelitis eradication. To the end of 2003 there were 289 confirmed cases of non-polio AFP. The reported rate of non-polio AFP (1995–2003) is therefore 0.83 (95% CI 0.74–0.94) per 100,000 children under 15 years (the World Health Organization (WHO) AFP case notification target for developed countries (1/100.000 children < 15 years) was reached in 2000 and 2001 but not 2003. In 2003 Guillain-Barre syndrome was again the most common cause of AFP (35% of confirmed cases), followed by transverse myelitis (18%). Adequate faecal specimens were received from 26 per cent of all eligible cases in 2002 and 24 per cent in 2003, well below the WHO target of 80 per cent. However, relevant diagnostic tests and/or 60 day follow up data available for these cases allowed them to be classified as AFP–non-polio.

### Congenital cytomegalovirus infection

*William Rawlinson, Daniel Trincado, Gillian Scott, Sian Munro, Pamela Palasanthiran, Mark Ferson, David Smith, Geoff Higgins, Michael Catton, Alistair McGregor, Dominic Dwyer, Alisson Kesson*

Congenital Cytomegalovirus infection (CMV) surveillance in children up to 12 months of age commenced through the APSU in 1999. Between January 1999 and December 2003 there were 31 confirmed cases of CMV (that is infants with CMV being isolated in blood, urine, saliva or tissue in the first three weeks of life). However, follow-up information available on children reported with CMV was of poor quality in 2003 prohibiting classification of all cases notified to APSU. Thus, the reported rate of confirmed cases

**Table. Confirmed cases of communicable diseases reported to the Australian Paediatric Surveillance Unit between 1 January and 30 June 2004***

| Condition | Previous reporting period Jan–Dec 2003 | Current reporting period Jan–June 2004* |
|---|---|---|
| Acute Flaccid Paralysis | 30 | 12 |
| Congenital cytomegalovirus | 10 | 8 |
| Congenital rubella | 3 | 1 |
| Perinatal exposure to HIV | 14 | 9 |
| HIV infection | 1† | |
| Neonatal herpes simplex virus infection | 6 | 2 |
| Hepatitis C Virus infection | 12 | 5 |

* Surveillance data are provisional and subject to revision.

† HIV virus infection through heterosexual contact.t

in 2003—1.78 (95% CI 1.21–2.52) per 100,000 live births—is likely to be an underestimate of the true rate.

### Congenital rubella

*Margaret Burgess, Jill Forrest, Cheryl Anne Jones, Peter McIntyre*

Surveillance of newly diagnosed congenital rubella in children and adolescents under 16 commenced in 1993. A total of 49 children with congenital rubella were identified through the APSU between May 1993 and December 2003. The national Measles Control Campaign in 1998 aimed to give measles-mumps-rubella (MMR) vaccine to all unvaccinated preschoolers and a second dose to primary schoolchildren. Following the Campaign no children with congenital rubella defects were born to Australian residents during the five years 1998 to 2002. However, during this period five imported cases were reported. Two cases of congenital rubella were reported from Queensland in late 2003. These children were born to young mothers who missed vaccination with rubella in the school programme and highlight the need for continuing education regarding the risks of rubella infection in pregnancy.

### HIV infection, AIDS and perinatal exposure to HIV

*Ann McDonald, John Kaldor, Michelle Good, John Ziegler*

This study monitors new cases of HIV/AIDS infection in children under 16 years and perinatal exposure to HIV. Perinatal exposure to HIV is now the most frequently reported source of HIV infection in Australian children. Between January 1997 and December 2003, 136 children with perinatal exposure to HIV were reported through the APSU and/or the National HIV/AIDS surveillance program. Additionally, in 2003 there was one reported case of HIV infection in a young person, which was attributed to heterosexual contact. The reported rate of perinatal HIV exposure is 7.80 (95% CI 6.54–9.22) per 100,000 live births. Of 39 cases of perinatal exposure to HIV reported through the APSU in 2002–2003, 38 were born to women whose HIV infection was diagnosed antenatally. Only one of 38 (2.6%) children born to these women acquired HIV infection. This reflects the use of interventions (antiretroviral treatment in pregnancy, elective caesarian delivery, and avoidance of breastfeeding) in women whose HIV infection is diagnosed antenatally, which substantially reduce the risk of mother to child HIV transmission.

### Neonatal herpes simplex virus infection

*Cheryl Anne Jones, David Isaacs, Peter McIntyre, Tony Cunningham, Suzanne Garland*

Surveillance of HSV infection in children aged up to 28 days commenced in 1997. There were 59 confirmed cases of neonatal HSV infection in infants up to 28 days of age reported between January 1997 and December 2003. The reported rate is 3.38 (95% CI 2.57–4.36) per 100,000 live births (similar to the United Kingdom but considerably lower than the rate in the United States of America). In contrast to the United States of America, Herpes simplex type 1 is the predominant isolate causing neonatal HSV infection in Australia.

### Hepatitis C virus infection

*John Kaldor, Cheryl Anne Jones, Elizabeth Elliott, Winita Hardikar, Alison Keeson, Susan Polis, Catherine Mews*

Surveillance of Hepatitis C infection in children commenced in January 2003. APSU contributors are asked to report any child less than 15 years of age

newly diagnosed with Hepatitis C infection. Twelve confirmed cases of Hepatitis C virus infection were reported to the APSU in 2003. The reported rate of HCV virus infection in Australian children less than 15 years of age, based on these preliminary data, is very low at 0.30 (95% CI 0.16–0.53) per 100,000 children under 15 years of age. In these children HCV infection was due to vertical transmission from an infected mother or childhood intravenous drug use. Most children were asymptomatic at diagnosis. Although the study findings are consistent with previous global studies, the small number of HCV cases identified nationally to date raises the possibility of under reporting. It is likely that some infected women remain undiagnosed during pregnancy and that some children born to infected mothers are not referred for investigation and paediatric follow-up.

# Communicable Diseases Surveillance

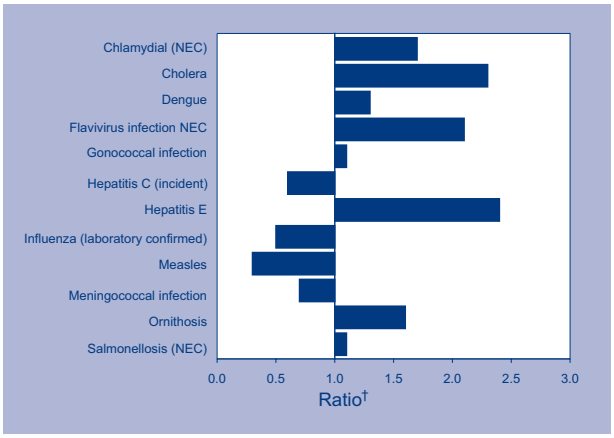
## Highlights for 3rd quarter, 2004

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

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

Figure 1 shows the changes in disease notifications with an onset in third quarter 2004 compared with a 5-year mean for the same period. The number of notifications received in the quarter was above the five year mean for chlamydial infections, cholera, dengue, flavivirus (NEC), gonococcal infections, hepatitis E and ornithosis. The number of notifications received was below the five year mean for hepatitis C (incident), influenza, measles, and meningococcal disease.

**Figure 1. Selected* diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 July to 30 September 2004 with historical data***




* Selected diseases are chosen each quarter according to current activity.

† Ratio of current quarter total to mean of corresponding quarter for the previous five years.

## *Gastrointestinal diseases*

### Hepatitis A

There was a small cluster of five cases of hepatitis A infection in children at a New South Wales primary school in July. One of the cases was a sibling of one of the students. Immunoglobulin prophylaxis was given to students and no further cases were reported. The source of the infection appears to be a confectionary jar.

Later in July, patrons of a city café in Sydney were contacted to receive hepatitis A immunoglobulin therapy after a café employee was diagnosed with hepatitis A. More than 100 people received the prophylaxis and there were no cases reported.

Despite these cases, the number of notifications in the third quarter (n=73) was well below the five year mean for the quarter (172).

### Listeriosis

There were 16 cases of listeriosis reported in the third quarter. These occurred in all states except the Northern Territory and Tasmania (Table 2). Most cases occurred in the elderly (median age 68.5 years).

Two maternal foetal cases of listeriosis were reported at a maternity unit in Victoria. The first baby was very ill and died shortly after birth. *Listeria monocytogenes* was cultured from the placenta and blood culture. A second baby born 27 hours later was well at birth and *Listeria* was cultured only from placenta swabs, while blood cultures were negative. The mother of the second baby had negative cultures of urine and high vaginal swabs. Examination of the *Listeria* isolates indicated that they were the same strain. It was concluded that the placenta of the second baby was contaminated during examination in the delivery suite or in the laboratory and that the second case was not a maternal foetal pair.

## *Quarantinable diseases*

### Cholera

Three cases of cholera were reported in the third quarter, one from New South Wales and the other from Victoria. One was a *Vibrio cholerae* 01 El Tor infection and the other two were both *Vibrio cholerae* 01 Ogawa. All three infections were acquired overseas.

## *Vaccine preventable disease*

### Measles

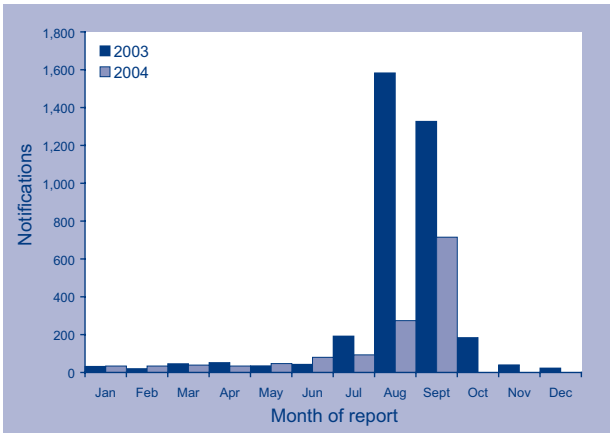
There were 12 cases of measles reported in the third quarter. Notifications were received from New South Wales (2), South Australia (3), Victoria (5) and Western Australia (2). This total is well below the 26 cases reported in the same period last year and the average of 24 cases reported in the period over the past five years.

The reported measles infections were acquired in seven overseas travellers and their contacts and of the remaining five cases, two were an unvaccinated child and its mother; one was a sporadic case in an unvaccinated 21 year-old and the other two cases were partially vaccinated children under two years of age (NSW).

### Influenza

There was a total of 1,082 notifications of laboratory confirmed influenza in the third quarter, which was well below the three year mean for this period (2,233 notifications). In contrast to 2003, the peak in influenza activity appeared later in the year and at a lower level (Figure 2).

**Figure 2. Notifications of laboratory confirmed influenza, Australia 2003 and 2004 (to end September)**




Two outbreaks of influenza-like illness were reported in army barracks, during the quarter. The first in Victoria occurred in new recruits, with a total of 94 cases, eight of which were typed as A/Fujian (H3N2)-like. Although vaccination is not compulsory, influenza vaccine is offered to all new recruits with an uptake rate of 73%. The second outbreak of influenza-like illness occurred during a military exercise in Queensland involving military personnel from around the country.

In September, 13 outbreaks of influenza-like illness were reported from residential institutions in NSW, including 12 aged care facilities (ACFs) and one correctional centre. The outbreaks had high attack rates (up to 76% of residents and 42% of staff) and death rates of up to 20% in residents.

In response to these outbreaks, public health unit staff provided advice to facility managers on control measures. NSW Health developed guidelines to assist ACF managers to minimise the spread of influenza within their institutions. The guidelines document *Controlling influenza outbreaks in aged care facilities* at http://www.health.nsw.gov.au/living/flucontrol_cdfs.html was distributed to ACFs throughout New South Wales.

In previous years, NSW Health has not actively solicited reports of influenza outbreaks from institutions, or systematically collated information on reported outbreaks. The reasons for the apparent large number (13) reported in NSW in September 2004, and the large proportion of these reported from the Hunter Area, are unclear. One explanation could be improved reporting in 2004 following the release of the *Controlling Influenza Outbreaks in Aged Care Facilities* guidelines. The first outbreak was associated with substantial media interest that may in turn have led to improved reporting by other ACFs.

NSW Health provides Australian Government-funded influenza vaccine annually to residents of ACFs. However, in the investigation of these outbreaks, public health units found that residents' immunisation records did not provide clear evidence of vaccination, perhaps because the turnover of residents in ACFs was sometimes high and the immunisation status of new residents was not always assessed on admission.

Annual immunisation of both residents and staff before winter (when the influenza activity usually begins) is essential to limit the extent of such outbreaks. ACF managers should ensure that record systems are in place to document the vaccination status of residents and staff, and flag the records of new residents and staff to ensure that they are offered immunisation. With growing evidence that anti-influenza medicines are effective in slowing outbreaks, ACF managers and clinicians should strongly consider their use to limit the spread of the infection in residential facilities

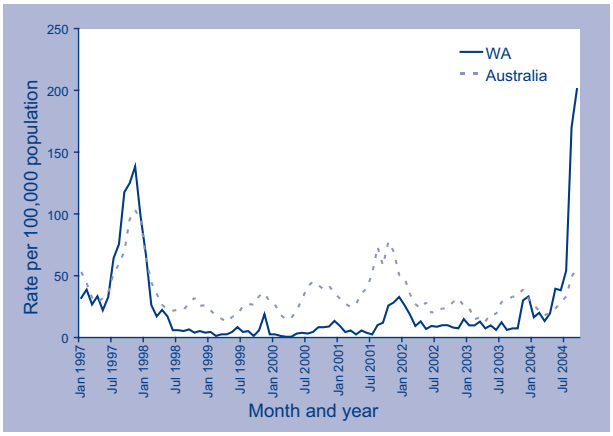
(A full report is available at: http://www.health.nsw.gov.au/living/infectreport.html )

**Pertussis**

There were 2,588 notifications of pertussis in the third quarter. One thousand and ninety-seven were from New South Wales and 779 were from Western Australia. Western Australia had the highest rate (159.6 cases per 100,000 population, Table 3).

An increase in notifications of pertussis in Western Australia has been noted since May. Increased notification rates were first observed in the Great Southern region around Albany and Denmark in May, and subsequently in the Goldfields, South-West, Wheatbelt and metropolitan Perth regions. Notifications are at the highest level recorded in the State since 1997(Figure 3). A relatively high proportion of notifications have been in secondary school aged children (25%) and adults (38% in those aged 25 years and above), compared to primary school aged students (18%) and younger children (12%). Given evidence that secondary school aged students have been pivotal in propagating transmission, a mass vaccination campaign in WA secondary schools using dTpa (Boostrix TM) vaccine was implemented in the 4th school term, in an attempt to limit the extent of the epidemic.

**Figure 3. Notification rates of pertussis, Western Australia and Australia 1997 to September 2004 (per 100,000 population)**




## *Other bacterial infections*

**Meningococcal infections**

There were 125 notifications of meningococcal infection in Australia in the third quarter. This number is well below the average number of notifications for this period received over the past five years (229 notifications).

There was serogroup data available on 89 of the notified cases in the quarter. Fifty seven (64%) were serogroup B, 26 were serogroups C (29%), five were serogroups W135 and there was a single case of serogroup Y.

A community based outbreak of meningococcal group C involving a rural High School in the North East of Tasmania occurred between 26 July, 2004 and 9 August, 2004. The total number of cases involved in this outbreak was four, all of which were later diagnosed as group C meningococcal infection. Of the four cases, three were fourteen year old students at the High School. The strain that caused the outbreak was later identified as C;P1.7-2.4. During the course of the outbreak, staff from the PEHS Hobart office provided antibiotic clearance to 87% of the students at the High School, and in association with the local Council, assisted in the delivery of meningococcal C conjugate vaccine to 84% of the students at the school. This increased coverage for meningococcal C vaccine in the High School to 95%.

A national immunisation program began in New Zealand during the quarter against the meningococcal serogroup B subtype B4P1.4/1.4(7), which has been responsible for a 14-year epidemic in that country. A review of meningococcal serogroup data in Australia (1995–2003) collected through the National Neisseria Network was conducted by the National Centre for Immunisation Research and Surveillance (NCIRS) to determine trends in the prevalence of the 'New Zealand' strain in Australia. The review found that despite over all increases in the incidence of meningococcal disease in Australia, the overall incidence of the New Zealand strain remained low (0.17 per 100,000 population). There was an overall increase in the number of isolates of the New Zealand stain detected in the period 1999–2003 (n=165) compared with the period 1995 to 1998 (n=56) with most this increase seen in New South Wales and Victoria (Puech and McIntyre, 2004 unpublished).

## *Other diseases*

### *M. ulcerans*

There was an outbreak of *Mycobacterium ulcerans* infections at Point Lonsdale in Victoria and two cases reported in Darwin in the Northern Territory during the quarter. *M. ulcerans* infections have been recognised in two geographic foci in Australia—one in the Gippsland region of Victoria and the second in the Daintree region of Queensland. The current outbreak which involved 14 people, appears to be a new endemic focus. There is no information as yet as to whether there is a focus in the Northern Territory.

**With thanks to:** Mark Bartlett (NSW Health), Lynne Brown (DHS, Victoria), David Coleman and Avner Misrachi (DHHS, Tasmania).

# Tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 27,389 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 July and 30 September 2004 (Table 2). The notification rate of diseases per 100,000 population for each State or Territory is presented in Table 3.

There were 7,808 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 July to 30 September 2004 (Tables 4 and 5).

## Table 1. Reporting of notifiable diseases by jurisdiction

| Disease | Data received from: |
|---|---|
| **Bloodborne diseases** | |
| Hepatitis B (incident) | All jurisdictions |
| Hepatitis B (unspecified) | All jurisdictions except NT |
| Hepatitis C (incident) | All jurisdictions except Qld |
| Hepatitis C (unspecified) | All jurisdictions |
| Hepatitis D | All jurisdictions |
| **Gastrointestinal diseases** | |
| Botulism | All jurisdictions |
| Campylobacteriosis | All jurisdictions except NSW |
| Cryptosporidiosis | All jurisdictions |
| Haemolytic uraemic syndrome | All jurisdictions |
| Hepatitis A | All jurisdictions |
| Hepatitis E | All jurisdictions |
| Listeriosis | All jurisdictions |
| Salmonellosis | All jurisdictions |
| Shigellosis | All jurisdictions |
| SLTEC, VTEC | All jurisdictions |
| Typhoid | All jurisdictions |
| **Quarantinable diseases** | |
| Cholera | All jurisdictions |
| Plague | All jurisdictions |
| Rabies | All jurisdictions |
| SARS | All jurisdictions |
| Smallpox | All jurisdictions except ACT, Qld |
| Tularemia | All jurisdictions except ACT, NT, Qld |
| Viral haemorrhagic fever | All jurisdictions |
| Yellow fever | All jurisdictions |
| **Sexually transmissible infections** | |
| Chlamydial infection | All jurisdictions |
| Donovanosis | All jurisdictions |
| Gonococcal infection | All jurisdictions |
| Syphilis (unspecified) | All jurisdictions |
| Syphilis < 2 years duration | All jurisdictions |
| Syphilis > 2 years of unknown duration | All jurisdictions |
| Syphilis - congenital | All jurisdictions |

| Disease | Data received from: |
|---|---|
| **Vaccine preventable diseases** | |
| Congenital Rubella | All jurisdictions |
| 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 |
| Tetanus | All jurisdictions |
| **Vectorborne diseases** | |
| Barmah Forest virus infection | All jurisdictions |
| Flavivirus infection (NEC)† | All jurisdictions |
| Dengue | All jurisdictions |
| Japanese encephalitis | All jurisdictions |
| Kunjin | All jurisdictions except ACT‡ |
| Malaria | All jurisdictions |
| Murray Valley encephalitis | All jurisdictions except ACT‡ |
| Ross River virus infection | All jurisdictions |
| **Zoonoses** | |
| Anthrax | All jurisdictions |
| Australian bat lyssavirus | All jurisdictions |
| Brucellosis | All jurisdictions |
| Leptospirosis | All jurisdictions |
| Lyssavirus unspecified | All jurisdictions |
| Ornithosis | All jurisdictions |
| Q fever | All jurisdictions |
| **Other bacterial infections** | |
| Legionellosis | All jurisdictions |
| Leprosy | All jurisdictions |
| Meningococcal infection | All jurisdictions |
| Tuberculosis | All jurisdictions |

* Laboratory confirmed influenza is not notifiable in the Australian Capital Territory but reports are forwarded to NNDSS.

† Flavivirus (NEC) replaces Arbovirus (NEC) from 1 January 2004.

‡ In the Australian Capital Territory, Murray Valley encephalitis and Kunjin are combined under Murray Valley encephalitis.

## Table 2. Notifications of diseases received by State and Territory health authorities for the period 1 July to 30 September 2004, by date of onset*

| Disease | State or territory | | | | | | | | Total 3rd quarter 2004[1] | Total 2nd quarter 2004 | Total 3rd quarter 2003 | Last 5 years mean 3rd quarter | Year to date 2004 | Last 5 years YTD mean | Ratio† |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | | | | | | | |
| **Bloodborne diseases** | | | | | | | | | | | | | | | |
| Hepatitis B (incident) | 1 | 3 | 2 | 11 | 3 | 6 | 35 | 14 | 75 | 71 | 86 | 97 | 216 | 288 | 0.8 |
| Hepatitis B (unspecified) | 15 | 998 | NN | 204 | 74 | 9 | 370 | 92 | 1,762 | 2,114 | 1,440 | 1,820 | 5,331 | 5,278 | 1.0 |
| Hepatitis C (incident) | 1 | 0 | NN | NN | 13 | 1 | 20 | 38 | 73 | 53 | 125 | 115 | 206 | 362 | 0.6 |
| Hepatitis C (unspecified) | 46 | 1,465 | 54 | 739 | 140 | 91 | 739 | 276 | 3,550 | 3,453 | 3,569 | 4,289 | 10,558 | 12,988 | 0.8 |
| Hepatitis D | 0 | 5 | 0 | 4 | 0 | 0 | 1 | 0 | 10 | 8 | 10 | 8 | 22 | 18 | 1.2 |
| **Gastrointestinal diseases** | | | | | | | | | | | | | | | |
| Botulism | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 |
| Campylobacteriosis[2] | 80 | NN | 56 | 1,079 | 532 | 122 | 1,436 | 441 | 3,746 | 3,010 | 3,333 | 3,493 | 11,022 | 10,364 | 1.1 |
| Cryptosporidiosis‡ | 1 | 21 | 21 | 67 | 23 | 11 | 75 | 21 | 240 | 435 | 190 | 220 | 1,190 | 1,714 | 0.7 |
| Haemolytic uraemic syndrome | 0 | 2 | 0 | 0 | 1 | 0 | 0 | 0 | 3 | 3 | 3 | 2 | 9 | 9 | 1.0 |
| Hepatitis A | 1 | 28 | 0 | 7 | 2 | 0 | 21 | 14 | 73 | 76 | 91 | 172 | 255 | 572 | 0.4 |
| Hepatitis E | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 2 | 8 | 5 | 2 | 21 | 9 | 2.4 |
| Listeriosis | 1 | 5 | 0 | 1 | 2 | 0 | 4 | 3 | 16 | 21 | 12 | 13 | 53 | 49 | 1.1 |
| Salmonellosis (NEC) | 15 | 264 | 69 | 365 | 75 | 15 | 230 | 129 | 1,162 | 1,978 | 1,019 | 1,062 | 5,868 | 5,343 | 1.1 |
| Shigellosis | 0 | 16 | 16 | 11 | 4 | 0 | 22 | 15 | 84 | 147 | 94 | 109 | 384 | 394 | 1.0 |
| SLTEC,VTEC[3] | 0 | 0 | 0 | 2 | 8 | 0 | 0 | 0 | 10 | 8 | 9 | 9 | 30 | 36 | 0.8 |
| Typhoid | 0 | 4 | 0 | 3 | 1 | 0 | 8 | 0 | 16 | 15 | 13 | 15 | 59 | 50 | 1.2 |
| **Quarantinable diseases** | | | | | | | | | | | | | | | |
| Cholera | 0 | 1 | 0 | 0 | 0 | 0 | 2 | 0 | 3 | 2 | 0 | 1 | 6 | 3 | 2.3 |
| Plague | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 |
| Rabies | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 |
| Smallpox | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 |
| Tularemia | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 |
| Viral haemorrhagic fever | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 |
| Yellow fever | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 |

**Table 2. Notifications of diseases received by State and Territory health authorities for the period 1 July to 30 September 2004, by date of onset* *continued***

| Disease | State or territory | | | | | | | | Total 3rd quarter 2004[1] | Total 2nd quarter 2004 | Total 3rd quarter 2003 | Last 5 years mean 3rd quarter | Year to date 2004 | Last 5 years YTD mean | Ratio[†] |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | | | | | | | |
| **Sexually transmissible diseases** | | | | | | | | | | | | | | | |
| Chlamydia | 147 | 2,422 | 356 | 2,250 | 516 | 153 | 1,818 | 1,015 | 8,677 | 8,899 | 7,710 | 5,413 | 26,678 | 15,801 | 1.7 |
| Donovanosis | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 1 | 3 | 1 | 3 | 4 | 6 | 15 | 0.4 |
| Gonococcal infection[4] | 6 | 254 | 337 | 361 | 48 | 7 | 265 | 318 | 1,596 | 1,902 | 1,607 | 1,492 | 5,248 | 4,682 | 1.1 |
| Syphilis (unspecified) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 57 | 118 | 223 | 123 | 646 | 0.2 |
| Syphilis < two years duration | 2 | 60 | 21 | 19 | 4 | 0 | 17 | 8 | 131 | 131 | 129 | 108 | 412 | 311 | 1.3 |
| Syphilis > two years or unknown duration | 0 | 277 | 32 | 47 | 0 | 0 | 91 | 14 | 461 | 397 | 242 | 239 | 1,276 | 753 | 1.7 |
| Syphilis - congenital | 0 | 0 | 1 | 2 | 0 | 0 | 0 | 0 | 3 | 3 | 4 | 3 | 8 | 7 | 1.1 |
| **Vaccine preventable disease** | | | | | | | | | | | | | | | |
| Diphtheria | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 |
| *Haemophilus influenzae* type b | 0 | 1 | 1 | 2 | 1 | 0 | 1 | 0 | 6 | 3 | 6 | 6 | 13 | 22 | 0.6 |
| Influenza (laboratory confirmed)[‡] | 0 | 423 | 19 | 455 | 3 | 2 | 112 | 68 | 1,082 | 161 | 3,102 | 2,233 | 1,350 | 2,629 | 0.5 |
| Measles | 0 | 2 | 0 | 0 | 3 | 0 | 5 | 2 | 12 | 6 | 26 | 24 | 29 | 96 | 0.3 |
| Mumps | 1 | 14 | 0 | 3 | 0 | 0 | 1 | 3 | 22 | 21 | 16 | 35 | 67 | 104 | 0.6 |
| Pertussis | 25 | 1,097 | 0 | 265 | 250 | 10 | 162 | 779 | 2,588 | 1,205 | 1,517 | 1,772 | 4,891 | 4,045 | 1.2 |
| Pneumococcal disease (invasive)[‡] | 17 | 341 | 33 | 244 | 48 | 18 | 135 | 85 | 921 | 612 | 796 | 774 | 1,861 | 1,542 | 1.2 |
| Poliomyelitis | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 |
| Rubella | 0 | 4 | 0 | 3 | 0 | 0 | 0 | 0 | 7 | 11 | 6 | 71 | 24 | 177 | 0.1 |
| Rubella - congenital | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 2.5 |
| Tetanus | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 1 | 0 | 4 | 3 | 1.4 |
| **Vectorborne diseases** | | | | | | | | | | | | | | | |
| Barmah Forest virus infection | 1 | 66 | 5 | 95 | 3 | 0 | 3 | 13 | 186 | 324 | 167 | 124 | 829 | 787 | 1.1 |
| Dengue | 1 | 7 | 3 | 13 | 1 | 0 | 2 | 2 | 29 | 64 | 34 | 29 | 330 | 257 | 1.3 |
| Flavivirus infection NEC | 0 | 4 | 0 | 10 | 0 | 0 | 1 | 0 | 15 | 12 | 20 | 7 | 83 | 39 | 2.1 |
| Japanese encephalitis[‡] | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0.0 |
| Kunjin virus[‡] | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 7 | 7 | 0.0 |
| Malaria | 2 | 31 | 4 | 68 | 3 | 2 | 17 | 11 | 138 | 149 | 132 | 162 | 411 | 542 | 0.8 |
| Murray Valley encephalitis[‡] | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 2 | 0.6 |
| Ross River virus infection | 0 | 39 | 17 | 57 | 3 | 1 | 3 | 19 | 139 | 1,382 | 214 | 189 | 3,958 | 3,001 | 1.3 |

**Table 2. Notifications of diseases received by State and Territory health authorities for the period 1 July to 30 September 2004, by date of onset*** ***continued***

| Disease | State or territory | | | | | | | | Total 3rd quarter 2004[1] | Total 2nd quarter 2004 | Total 3rd quarter 2003 | Last 5 years mean 3rd quarter | Year to date 2004 | Last 5 years YTD mean | Ratio† |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | | | | | | | |
| **Zoonoses** | | | | | | | | | | | | | | | |
| Anthrax‡ | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 |
| Australian bat lyssavirus‡ | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 |
| Brucellosis | 0 | 0 | 0 | 10 | 0 | 0 | 1 | 0 | 11 | 4 | 4 | 10 | 21 | 22 | 1.0 |
| Leptospirosis | 0 | 9 | 0 | 19 | 0 | 0 | 1 | 2 | 31 | 56 | 27 | 32 | 154 | 175 | 0.9 |
| Lyssavirus unspecified‡ | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 |
| Ornithosis | 0 | 25 | 0 | 1 | 0 | 0 | 21 | 0 | 47 | 57 | 67 | 44 | 174 | 106 | 1.6 |
| Q fever | 0 | 55 | 0 | 18 | 6 | 0 | 9 | 1 | 89 | 110 | 88 | 133 | 304 | 453 | 0.7 |
| **Other bacterial infections** | | | | | | | | | | | | | | | |
| Creutzfeldt-Jakob disease | | | | | | | | | | | | | | | |
| Legionellosis | 0 | 12 | 1 | 13 | 9 | 0 | 18 | 11 | 64 | 92 | 60 | 62 | 239 | 254 | 0.9 |
| Leprosy | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 3 | 3 | 0.9 |
| Meningococcal infection | 4 | 42 | 1 | 28 | 4 | 9 | 21 | 16 | 125 | 117 | 208 | 229 | 335 | 471 | 0.7 |
| Tuberculosis | 2 | 70 | 6 | 11 | 0 | 4 | 70 | 16 | 179 | 205 | 246 | 252 | 613 | 726 | 0.8 |
| **Total** | 369 | 8,069 | 1,057 | 6,489 | 1,780 | 461 | 5,737 | 3,427 | 27,389 | 27,386 | 26,549 | 25,099 | 84,684 | 75,153 | 1.1 |

1. Totals comprise data from all states and territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
2. Not reported from New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
3. Infections with shiga-like toxin (verotoxin) producing *Escherichia coli* (SLTEC/VTEC).

* Date of onset = the true date of onset. If this is not available, the 'date of onset' is equivalent to the earliest of two dates: (i) specimen collection date or (ii) the date of notification to a public health unit. Hepatitis B and C unspecified were analysed by date of notification.

† Ratio = ratio of current quarter total to the mean of last 5 years for the same quarter.

‡ Notifiable from January 2001. Ratio and mean calculations are based on the last three years.

NN Not notifiable.

NEC Not elsewhere classified.

## Table 3. Notification rates of diseases by state or territory, 1 July to 30 September 2004 (Rate per 100,000 population)

| Disease[1] | State or territory | | | | | | | | Australia |
|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | |
| **Bloodborne diseases** | | | | | | | | | |
| Hepatitis B (incident) | 1.2 | 0.2 | 4.0 | 1.2 | 0.8 | 5.0 | 2.9 | 2.9 | 1.5 |
| Hepatitis B (unspecified) | 18.6 | 59.7 | NN | 21.5 | 19.4 | 7.5 | 30.2 | 18.8 | 35.8 |
| Hepatitis C (incident) | 1.2 | 0.0 | NN | NN | 3.4 | 0.8 | 1.6 | 7.8 | 1.8 |
| Hepatitis C (unspecified) | 57.0 | 87.6 | 108.9 | 77.9 | 36.7 | 76.3 | 60.3 | 56.5 | 71.5 |
| Hepatitis D | 0.0 | 0.3 | 0.0 | 0.4 | 0.0 | 0.0 | 0.1 | 0.0 | 0.2 |
| **Gastrointestinal diseases** | | | | | | | | | |
| Botulism | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Campylobacteriosis[2] | 99.1 | NN | 112.9 | 113.7 | 139.3 | 102.3 | 117.1 | 90.4 | 113.7 |
| Cryptosporidiosis | 1.2 | 1.3 | 42.3 | 7.1 | 6.0 | 9.2 | 6.1 | 4.3 | 4.8 |
| Haemolytic uraemic syndrome | 0.0 | 0.1 | 0.0 | 0.0 | 0.3 | 0.0 | 0.0 | 0.0 | 0.1 |
| Hepatitis A | 1.2 | 1.7 | 0.0 | 0.7 | 0.5 | 0.0 | 1.7 | 2.9 | 1.5 |
| Hepatitis E | 0.0 | 0.1 | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Listeriosis | 1.2 | 0.3 | 0.0 | 0.1 | 0.5 | 0.0 | 0.3 | 0.6 | 0.3 |
| Salmonellosis (NEC) | 18.6 | 15.8 | 139.1 | 38.5 | 19.6 | 12.6 | 18.8 | 26.4 | 23.4 |
| Shigellosis | 0.0 | 1.0 | 32.3 | 1.2 | 1.0 | 0.0 | 1.8 | 3.1 | 1.7 |
| SLTEC,VTEC[3] | 0.0 | 0.0 | 0.0 | 0.2 | 2.1 | 0.0 | 0.0 | 0.0 | 0.2 |
| Typhoid | 0.0 | 0.2 | 0.0 | 0.3 | 0.3 | 0.0 | 0.7 | 0.0 | 0.3 |
| **Quarantinable diseases** | | | | | | | | | |
| Cholera | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.2 | 0.0 | 0.1 |
| Plague | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Rabies | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Smallpox | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Tularemia | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Viral haemorrhagic fever | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Yellow fever | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| **Sexually transmissible diseases** | | | | | | | | | |
| Chlamydia | 182.1 | 144.9 | 717.9 | 237.0 | 135.1 | 128.3 | 148.2 | 208.0 | 174.7 |
| Donovanosis | 0.0 | 0.0 | 4.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.2 | 0.1 |
| Gonococcal infection | 7.4 | 15.2 | 679.6 | 38.0 | 12.6 | 5.9 | 21.6 | 65.2 | 32.1 |
| Syphilis (unspecified) | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Syphilis < 2 years duration | 2.5 | 3.6 | 42.3 | 2.0 | 1.0 | 0.0 | 1.4 | 1.6 | 2.6 |
| Syphilis > 2 years or unknown duration | 0.0 | 16.6 | 64.5 | 5.0 | 0.0 | 0.0 | 7.4 | 2.9 | 9.3 |
| Syphilis - congenital | 0.0 | 0.0 | 2.0 | 0.2 | 0.0 | 0.0 | 0.0 | 0.0 | 0.1 |

## Table 3. Notification rates of diseases by state or territory, 1 July to 30 September 2004. (Rate per 100,000 population) *continued*

| Disease[1] | State or territory | | | | | | | | Australia |
|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | |
| **Vaccine preventable diseases** | | | | | | | | | |
| Diphtheria | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| *Haemophilus influenzae* type b | 0.0 | 0.1 | 2.0 | 0.0 | 0.3 | 1.7 | 0.0 | 0.0 | 0.1 |
| Influenza (laboratory confirmed) | 0.0 | 2.8 | 2.0 | 2.3 | 2.6 | 0.0 | 1.3 | 2.0 | 2.1 |
| Measles | 0.0 | 0.4 | 0.0 | 0.0 | 0.3 | 0.0 | 0.3 | 0.0 | 0.2 |
| Mumps | 2.5 | 0.9 | 0.0 | 0.1 | 0.0 | 0.0 | 0.2 | 0.8 | 0.5 |
| Pertussis | 53.3 | 28.0 | 2.0 | 16.4 | 11.0 | 10.9 | 17.0 | 14.8 | 20.2 |
| Pneumococcal disease (invassive) | 11.2 | 6.0 | 34.3 | 6.3 | 10.5 | 1.7 | 5.7 | 3.5 | 6.3 |
| Poliomyelitis | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Rubella | 0.0 | 0.3 | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.1 |
| Rubella - congenital | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Tetanus | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| **Vectorborne diseases** | | | | | | | | | |
| Barmah Forest virus infection | 0.0 | 5.7 | 10.1 | 20.2 | 0.0 | 0.0 | 0.2 | 4.5 | 6.4 |
| Dengue | 3.7 | 0.5 | 20.2 | 21.5 | 0.8 | 0.8 | 0.2 | 0.2 | 4.7 |
| Flavivirus infection (NEC) | 0.0 | 0.2 | 2.0 | 5.7 | 0.0 | 0.0 | 0.3 | 0.0 | 1.3 |
| Japanese encephalitis | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Kunjin virus | 0.0 | 0.0 | 0.0 | 0.6 | 0.0 | 0.0 | 0.0 | 0.0 | 0.1 |
| Malaria | 6.2 | 1.2 | 10.1 | 5.9 | 0.5 | 2.5 | 1.1 | 1.0 | 2.2 |
| Murray Valley encephalitis | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Ross River virus infection | 2.5 | 14.1 | 367.0 | 106.1 | 5.0 | 10.9 | 4.1 | 172.9 | 47.4 |
| **Zoonoses** | | | | | | | | | |
| Anthrax | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Australian bat lyssavirus | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Brucellosis | 0.0 | 0.0 | 0.0 | 0.3 | 0.0 | 0.0 | 0.1 | 0.0 | 0.1 |
| Leptospirosis | 0.0 | 0.6 | 0.0 | 5.7 | 0.0 | 0.0 | 0.0 | 0.0 | 1.3 |
| 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 | 0.0 | 0.5 | 0.0 | 4.0 | 0.0 | 1.2 |
| Q fever | 1.2 | 2.8 | 4.0 | 4.3 | 0.8 | 0.0 | 0.2 | 0.2 | 2.0 |
| **Other bacterial infections** | | | | | | | | | |
| Legionellosis | 0.0 | 1.3 | 0.0 | 0.9 | 2.4 | 0.8 | 1.6 | 3.1 | 1.5 |
| Leprosy | 0.0 | 0.1 | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Meningococcal infection | 6.2 | 1.7 | 8.1 | 2.1 | 1.0 | 1.7 | 1.7 | 1.0 | 1.8 |
| Tuberculosis | 3.7 | 3.3 | 12.1 | 2.0 | 4.7 | 0.8 | 5.2 | 2.0 | 3.6 |

1. Rates are subject to retrospective revision.
2. Not reported from New South Wales where it is only notifiable as ‘foodborne disease’ or ‘gastroenteritis in an institution’.
3. Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (SLTEC/VTEC).

NN Not Notifiable.

NEC Not Elsewhere Classified.

**Table 4. Virology and serology laboratory reports by state or territory[1] for the reporting period 1 July to 30 September 2004, and total reports for the year[2]**

| | State or territory | | | | | | | | This period 2004 | This period 2003 | Year to date 2004[3] | Year to date 2003 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | | | | |
| **Measles, mumps, rubella** | | | | | | | | | | | | |
| Measles virus | 0 | 1 | 0 | 1 | 2 | 0 | 3 | 1 | 8 | 10 | 21 | 50 |
| Mumps virus | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 2 | – | 5 | 7 |
| Rubella virus | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 1 | 3 | 7 | 11 | 19 |
| **Hepatitis virus** | | | | | | | | | | | | |
| Hepatitis A virus | 0 | 1 | 0 | 7 | 1 | 0 | 2 | 6 | 17 | 28 | 36 | 64 |
| Hepatitis D virus | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 2 | 3 | 7 | 6 | 15 |
| Hepatitis E virus | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 2 | – | 13 | |
| **Arboviruses** | | | | | | | | | | | | |
| Ross River virus | 0 | 0 | 1 | 9 | 4 | 0 | 0 | 10 | 24 | 71 | 705 | 1,183 |
| Barmah Forest virus | 0 | 2 | 0 | 32 | 5 | 0 | 0 | 3 | 42 | 34 | 168 | 375 |
| Dengue type 2 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 1 | 1 | 2 |
| Dengue not typed | 0 | 0 | 3 | 0 | 0 | 0 | 0 | 3 | 6 | 5 | 7 | 27 |
| Flavivirus (unspecified) | 0 | 0 | 0 | 13 | 0 | 0 | 2 | 0 | 15 | 4 | 93 | 108 |
| **Adenoviruses** | | | | | | | | | | | | |
| Adenovirus typed 40 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 11 | 11 | 8 | 16 | 28 |
| Adenovirus not typed/ pending | 1 | 92 | 2 | 22 | 117 | 0 | 13 | 60 | 307 | 299 | 753 | 754 |
| **Herpesviruses** | | | | | | | | | | | | |
| Herpes virus type 6 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 2 | 2 | 4 | 5 |
| Cytomegalovirus | 3 | 87 | 0 | 29 | 68 | 4 | 13 | 0 | 204 | 194 | 603 | 705 |
| Varicella-zoster virus | 1 | 35 | 6 | 283 | 122 | 3 | 18 | 182 | 650 | 469 | 1,502 | 1,264 |
| Epstein-Barr virus | 0 | 22 | 3 | 177 | 440 | 0 | 11 | 31 | 684 | 469 | 1,852 | 1,308 |
| **Other DNA viruses** | | | | | | | | | | | | |
| Molluscum contagiosum | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 2 | 1 | 3 | 11 |
| Contagious pustular dermatitis (Orf virus) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 2 | 1 | 2 | 3 |
| Parvovirus | 0 | 3 | 0 | 40 | 15 | 0 | 19 | 88 | 165 | 62 | 283 | 168 |
| **Picornavirus family** | | | | | | | | | | | | |
| Echovirus type 9 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 2 | 4 | 11 |
| Echovirus type 11 | 0 | 8 | 0 | 0 | 0 | 0 | 0 | 0 | 8 | 2 | 14 | 4 |
| Echovirus type 30 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | – | 6 | 1 |
| Poliovirus type 1 (uncharacterised) | 0 | 9 | 0 | 0 | 0 | 0 | 0 | 0 | 9 | 6 | 15 | 32 |
| Poliovirus type 2 (uncharacterised) | 0 | 5 | 0 | 0 | 0 | 0 | 0 | 0 | 5 | 5 | 13 | 9 |
| Poliovirus type 3 (uncharacterised) | 0 | 5 | 0 | 0 | 0 | 0 | 0 | 0 | 5 | 3 | 6 | 4 |
| Rhinovirus (all types) | 0 | 79 | 0 | 0 | 22 | 0 | 1 | 106 | 208 | 138 | 395 | 389 |
| Enterovirus type 71 (BCR) | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | – | 3 | – |
| Enterovirus not typed/ pending | 1 | 5 | 2 | 3 | 4 | 0 | 1 | 20 | 36 | 37 | 123 | 126 |

**Table 4. Virology and serology laboratory reports by state or territory[1] for the reporting period 1 July to 30 September 2004, and total reports for the year,[2] *continued***

| | State or territory | | | | | | | | This period 2004 | This period 2003 | Year to date 2004[3] | Year to date 2003 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | | | | |
| **Ortho/paramyxoviruses** | | | | | | | | | | | | |
| Influenza A virus | 0 | 108 | 0 | 33 | 55 | 1 | 7 | 37 | 241 | 1,542 | 312 | 1,746 |
| Influenza B virus | 0 | 7 | 0 | 6 | 33 | 0 | 0 | 36 | 82 | 38 | 119 | 97 |
| Parainfluenza virus type 1 | 0 | 4 | 0 | 0 | 17 | 0 | 0 | 12 | 33 | 9 | 130 | 35 |
| Parainfluenza virus type 2 | 0 | 1 | 0 | 1 | 0 | 0 | 1 | 2 | 5 | 17 | 11 | 66 |
| Parainfluenza virus type 3 | 0 | 13 | 2 | 1 | 85 | 0 | 2 | 109 | 212 | 208 | 409 | 405 |
| Respiratory syncytial virus | 3 | 338 | 2 | 80 | 316 | 46 | 60 | 197 | 1,042 | 900 | 2,435 | 1,579 |
| **Other RNA viruses** | | | | | | | | | | | | |
| HTLV-1 | 0 | 0 | 0 | 0 | 3 | 0 | 0 | 3 | 6 | 2 | 12 | 10 |
| Rotavirus | 0 | 124 | 8 | 1 | 64 | 75 | 36 | 62 | 370 | 433 | 539 | 576 |
| Calcivirus | 0 | 3 | 12 | 0 | 0 | 0 | 0 | 214 | 229 | 23 | 256 | 103 |
| Norwalk agent | 0 | 2 | 0 | 0 | 0 | 4 | 99 | 0 | 105 | 87 | 294 | 130 |
| **Other** | | | | | | | | | | | | |
| *Chlamydia trachomatis* not typed | 9 | 188 | 5 | 539 | 392 | 10 | 7 | 400 | 1,550 | 1,082 | 3,977 | 3,400 |
| *Chlamydia pneumoniae* | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 2 | 1 | 6 | 11 |
| *Chlamydia psittaci* | 0 | 1 | 0 | 0 | 0 | 0 | 26 | 1 | 28 | 45 | 136 | 87 |
| *Chlamydia* species | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 1 | 3 | 1 |
| *Mycoplasma pneumoniae* | 0 | 13 | 3 | 134 | 121 | 9 | 82 | 13 | 375 | 471 | 1,035 | 904 |
| *Mycoplasma hominis* | 0 | 2 | 0 | 0 | 1 | 0 | 0 | 0 | 3 | 4 | 4 | 9 |
| *Coxiella burnetii* (Q fever) | 0 | 0 | 2 | 6 | 25 | 0 | 6 | 1 | 40 | 48 | 120 | 141 |
| *Rickettsia prowazeki* | 0 | 0 | 0 | 0 | 29 | 0 | 0 | 1 | 30 | – | 30 | 2 |
| *Rickettsia tsutsugamushi* | 0 | 0 | 0 | 0 | 18 | 0 | 1 | 1 | 20 | 1 | 21 | 2 |
| *Rickettsia* - spotted fever group | 0 | 0 | 0 | 0 | 43 | 1 | 0 | 0 | 44 | – | 44 | – |
| *Streptococcus* group A | 0 | 2 | 1 | 106 | 0 | 0 | 28 | 0 | 137 | 91 | 360 | 362 |
| *Yersinia enterocolitica* | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 3 | 5 | 5 | 9 |
| *Brucella abortus* | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | – | 5 | 2 |
| *Brucella* species | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 2 | 3 | 5 | 5 |
| *Bordetella pertussis* | 0 | 15 | 0 | 56 | 170 | 2 | 46 | 175 | 464 | 108 | 732 | 364 |
| *Legionella pneumophila* | 0 | 1 | 0 | 0 | 1 | 0 | 9 | 1 | 12 | 60 | 65 | 113 |
| *Legionella longbeachae* | 0 | 0 | 0 | 0 | 4 | 0 | 6 | 9 | 19 | 26 | 57 | 54 |
| *Legionella* species | 0 | 1 | 0 | 0 | 0 | 0 | 3 | 0 | 4 | 6 | 14 | 10 |
| *Cryptococcus* species | 0 | 2 | 0 | 1 | 6 | 0 | 0 | 0 | 9 | 8 | 32 | 20 |
| *Leptospira* species | 0 | 0 | 0 | 3 | 0 | 0 | 0 | 0 | 3 | 10 | 19 | 21 |
| *Borrelia burgdorferi* | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | – | 1 | – |
| *Treponema pallidum* | 0 | 39 | 0 | 154 | 96 | 0 | 0 | 4 | 293 | 284 | 908 | 958 |
| *Entamoeba histolytica* | 0 | 0 | 0 | 2 | 0 | 0 | 2 | 0 | 4 | 4 | 9 | 10 |
| *Toxoplasma gondii* | 0 | 1 | 0 | 0 | 4 | 0 | 3 | 1 | 9 | 11 | 26 | 32 |
| *Echinococcus granulosus* | 0 | 0 | 0 | 0 | 2 | 0 | 1 | 0 | 3 | 3 | 10 | 14 |
| Total | 18 | 1,228 | 52 | 1,742 | 2,287 | 155 | 513 | 1,813 | 7,808 | 7,396 | 18,804 | 17,950 |

1. State or territory of postcode, if reported, otherwise state or territory of reporting laboratory.
2. Data presented are for reports with report dates in the current period.

– No data received this period.

## Table 5. Virology and serology reports by laboratories for the reporting period 1 July to 30 September 2004*

| State or territory | Laboratory | July 2004 | August 2004 | September 2004 | Total this period |
|---|---|---|---|---|---|
| Australian Capital Territory | The Canberra Hospital | – | – | – | – |
| New South Wales | Institute of Clinical Pathology and Medical Research, Westmead | 151 | 125 | 130 | 406 |
| | New Children's Hospital, Westmead | 102 | 116 | 100 | 318 |
| | Repatriation General Hospital, Concord | – | – | – | – |
| | Royal Prince Alfred Hospital, Camperdown | 42 | 33 | 31 | 106 |
| | South West Area Pathology Service, Liverpool | 136 | 129 | 99 | 364 |
| Queensland | Queensland Medical Laboratory, West End | 733 | 582 | 502 | 1,817 |
| | Townsville General Hospital | – | – | – | – |
| South Australia | Institute of Medical and Veterinary Science, Adelaide | 749 | 742 | 785 | 2,276 |
| Tasmania | Northern Tasmanian Pathology Service, Launceston | 45 | 60 | 44 | 149 |
| | Royal Hobart Hospital, Hobart | – | – | – | – |
| Victoria | Monash Medical Centre, Melbourne | 50 | 18 | – | 68 |
| | Royal Children's Hospital, Melbourne | 102 | 35 | 23 | 160 |
| | Victorian Infectious Diseases Reference Laboratory, Fairfield | 96 | 127 | 54 | 277 |
| Western Australia | PathCentre Virology, Perth | 543 | 567 | 720 | 1,830 |
| | Princess Margaret Hospital, Perth | – | – | – | – |
| | Western Diagnostic Pathology | – | – | 37 | 37 |
| Total | | 2,749 | 2,534 | 2,525 | 7,808 |

* The complete list of laboratories reporting for the 12 months, January to December 2004, will appear in every report regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.

– No data received this period.

# Additional reports

## *Australian Sentinel Practice Research Network*

*The Research and Health Promotion Unit of the Royal Australian College of General Practitioners operates the Australian Sentinel Practice Research Network (ASPREN). ASPREN is a 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.*

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

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

*In 2004, nine conditions are being monitored, five of which are related to communicable diseases. These include influenza, gastroenteritis, varicella and shingles. Definitions of these conditions are described in Surveillance systems reported in CDI, published in* Commun Dis Intell *2004;28:99. Note that in 2004, two case definitions for influenza are being recorded in parallel.*

Data from 1 July to 30 September 2004 are shown as the rate per 1,000 consultations in Figures 4, 5, 6 and 7.

**Figure 4. Consultation rates for influenza-like illness, ASPREN, 1 July to 30 September 2004, by week of report**

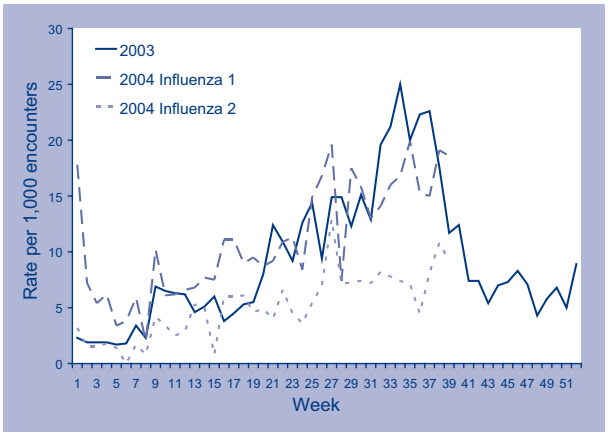



**Figure 5. Consultation rates for gastroenteritis, ASPREN, 1 July to 30 September 2004, by week of report**

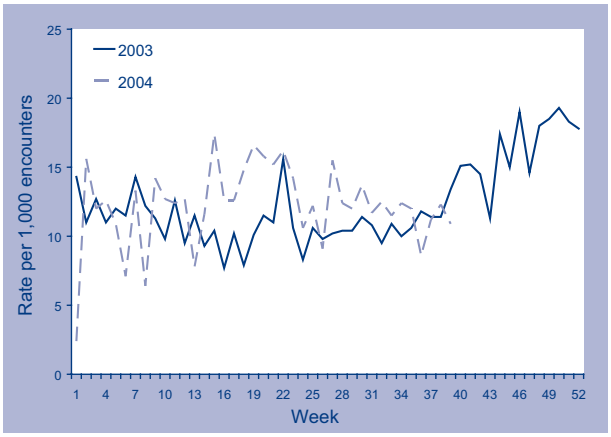



**Figure 6. Consultation rates for chickenpox, ASPREN, 1 July to 30 September 2004, by week of report**

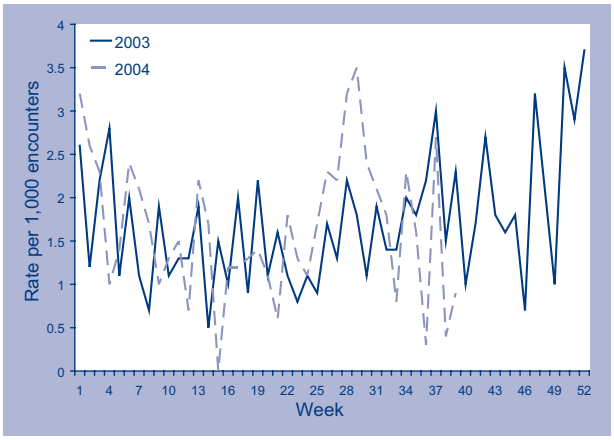
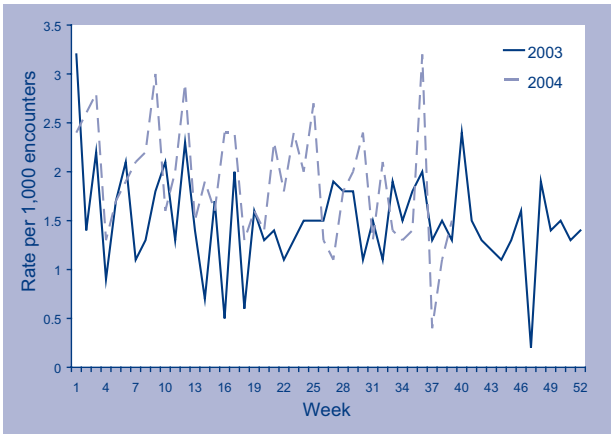



**Figure 7. Consultation rates for shingles, ASPREN, 1 July to 30 September 2004, by week of report**



# *Childhood immunisation coverage*

*Tables 6, 7 and 8 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR).*

*The data show the percentage of children fully immunised at age 12 months for the cohort born between 1 April and 30 June 2003; at 24 months of age for the cohort born between 1 April and 30 June 2002; and at 6 years of age for the cohort born between 1 April and 30 June 1998, according to the Australian Standard Vaccination Schedule.*

*For information about the Australian Childhood Immunisation Register see Surveillance systems reported in CDI, published in* Commun Dis Intell *2004;28:102 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). Telephone: +61 2 9845 1256. Email: brynleyh@chw.edu.au.*

Immunisation coverage for 'fully immunised' at 12 months of age for Australia increased marginally from the last quarter by 0.4 percentage points to 91.3 per cent (Table 6). There was a substantial increase in 'fully immunised' coverage by State and Territory in one jurisdiction, the Northern Territory, with an increase of 5.3 percentage points, whilst all other jurisdictions experienced very little change in coverage. As expected, the Northern Territory also had increases in coverage for individual vaccines. Apparently large changes in coverage in jurisdictions like the Northern Territory and the Australian Capital Territory, who have relatively small populations, can result from small absolute numbers of unimmunised children and should be treated with caution.

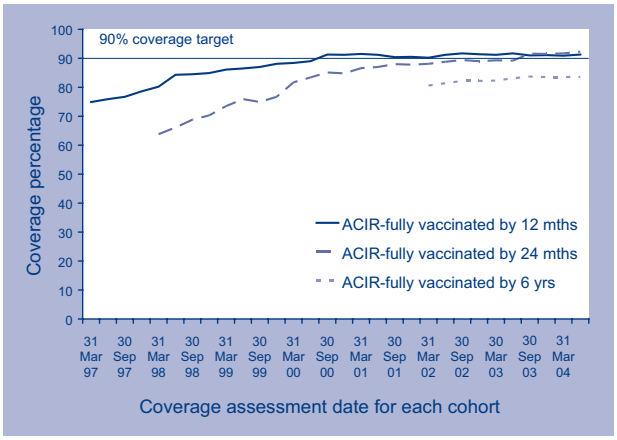
Coverage for 'fully immunised' at 24 months of age for Australia increased marginally from the last quarter by 0.6 percentage points to 92.3 per cent (Table 7). Coverage for individual vaccines increased marginally in most jurisdictions with coverage greater than 95 per cent in almost all jurisdictions for all vaccines except Hib. HepB coverage at 24 months of age is now greater than 98 per cent in the Northern Territory.

Table 8 shows immunisation coverage estimates for 'fully immunised' and for individual vaccines at six years of age for Australia and by state/territory.

'Fully immunised' coverage at six years of age for Australia was unchanged overall, apart from increases in Tasmania (+3.1%) and in the Northern Territory (+4.1%), also reflected in individual vaccines. Coverage for vaccines assessed at six years is at or near 85 per cent in the most jurisdictions, but Western Australia remains well below the average.

Figure 8 shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and six years, although the rate of increase has slowed over the past year for all age groups. The figure shows that there have now been four consecutive quarters where 'fully immunised' coverage at 24 months of age has been greater than 'fully immunised' coverage at 12 months of age, following the removal of the requirement for 18 month DTPa vaccine.

**Figure 8. Trends in vaccination coverage, Australia, 1997 to 2004, by age cohorts**




**Acknowledgement:** These figures were provided by the Health Insurance Commission (HIC), to specifications provided by the Australian Government Department of Health and Ageing. For further information on these figures or data on the Australian Childhood Immunisation Register please contact the Immunisation Section of the HIC: Telephone: +61 2 6124 6607.

**Table 6. Percentage of children immunised at 1 year of age, preliminary results by vaccine and state or territory for the birth cohort 1 April and 30 June 2003; assessment date 30 September 2004**

| Vaccine | State or territory | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Australia |
| Number of children | 959 | 21,653 | 921 | 12,711 | 4,371 | 1,347 | 15,155 | 6,124 | 63,241 |
| Diphtheria, tetanus, pertussis (%) | 94.3 | 92.7 | 91.9 | 92.7 | 92.8 | 93.7 | 93.2 | 90.7 | 92.7 |
| Poliomyelitis (%) | 94.3 | 92.6 | 91.4 | 92.6 | 92.6 | 93.5 | 93.1 | 90.7 | 92.6 |
| *Haemophilus influenzae* type b (%) | 95.6 | 94.6 | 95.7 | 94.8 | 95.7 | 95.7 | 95.1 | 93.8 | 94.8 |
| Hepatitis B (%) | 95.3 | 95.3 | 96.3 | 94.8 | 95.8 | 95.6 | 94.7 | 93.3 | 94.9 |
| Fully immunised (%) | 93.4 | 91.3 | 90.5 | 91.7 | 91.7 | 92.4 | 91.7 | 88.8 | 91.3 |
| Change in fully immunised since last quarter (%) | +2.7 | +0.8 | +5.3 | +0.1 | +0.3 | -1.0 | +0.0 | -0.5 | +0.4 |

**Table 7. Percentage of children immunised at 2 years of age, preliminary results by vaccine and state or territory for the birth cohort 1 April and 30 June 2002, assessment date 30 September 2004***

| Vaccine | State or territory | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas. | Vic. | WA | Australia |
| Number of children | 1,003 | 21,222 | 873 | 12,724 | 4,274 | 1,444 | 15,217 | 6,184 | 62,941 |
| Diphtheria, tetanus, pertussis (%) | 95.7 | 95.4 | 96.9 | 95.0 | 95.4 | 96.3 | 95.7 | 94.4 | 95.3 |
| Poliomyelitis (%) | 95.6 | 95.2 | 97.1 | 94.9 | 95.4 | 96.2 | 95.7 | 94.2 | 95.2 |
| *Haemophilus influenzae* type b (%) | 94.7 | 93.4 | 95.5 | 94.0 | 94.1 | 94.5 | 94.2 | 92.6 | 93.8 |
| Measles, mumps, rubella (%) | 95.0 | 93.5 | 95.5 | 93.9 | 94.5 | 94.7 | 94.6 | 92.8 | 93.9 |
| Hepatitis B(%) | 96.1 | 95.7 | 98.2 | 95.6 | 96.2 | 96.4 | 96.3 | 95.4 | 95.9 |
| Fully immunised (%) | 92.7 | 91.8 | 93.8 | 92.3 | 93.0 | 93.8 | 93.1 | 90.6 | 92.3 |
| Change in fully immunised since last quarter (%) | +2.7 | +0.8 | -0.7 | +0.4 | +0.3 | -1.0 | +0.8 | -0.0 | +0.5 |

**Table 8. Percentage of children immunised at 6 years of age, preliminary results by vaccine and state or territory for the birth cohort 1 April and 30 June 1998; assessment date 30 September 2004**

| | State or territory | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Australia |
| Number of children | 1,049 | 21,810 | 892 | 13,394 | 4,674 | 1,496 | 15,498 | 6,733 | 65,546 |
| Diphtheria, tetanus, pertussis (%) | 85.9 | 85.3 | 83.7 | 85.1 | 85.3 | 85.2 | 87.4 | 81.6 | 85.4 |
| Poliomyelitis (%) | 86.0 | 85.2 | 85.3 | 85.2 | 85.4 | 85.2 | 86.9 | 82.0 | 85.3 |
| Measles, mumps, rubella (%) | 84.9 | 84.3 | 85.2 | 84.9 | 85.0 | 84.3 | 87.1 | 81.4 | 84.8 |
| Fully immunised (%) | 83.8 | 83.1 | 82.7 | 83.7 | 83.9 | 83.4 | 85.7 | 80.1 | 83.6 |
| Change in fully immunised since last quarter (%) | -1.1 | -0.0 | +4.1 | +0.0 | +0.6 | +3.1 | +0.2 | -1.0 | +0.1 |

* The 12 months age data for this cohort was published in *Commun Dis Intell* 2003;27:569.

## *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 which are 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 treatments[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 programme-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. For more information see* Commun Dis Intell *2004;28:100.*

### Reporting period 1 July to 30 September 2004

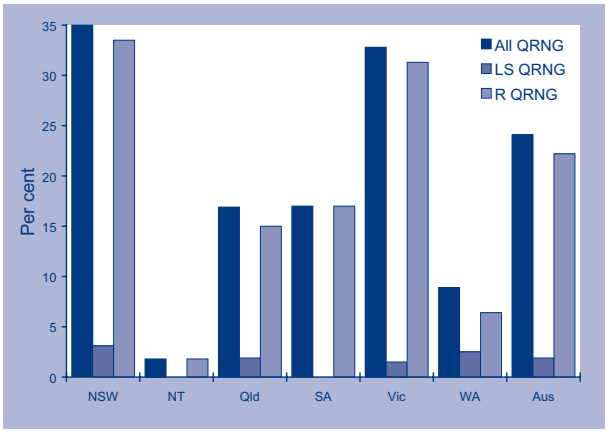
The AGSP laboratories examined a total of 829 isolates in this quarter. The total is slightly less than the 857 isolated or referred in 2003. About 31% of the current total was from New South Wales, 24% from Victoria, 19% from Queensland, 13% from the Northern Territory, 10% from Western Australia and 3% from South Australia. Isolates from Tasmania (6) and the Australian Capital Territory (4) were few.

### Quinolone antibiotics

The total number (200) and proportion (24%) of all quinolone resistant *N. gonorrhoeae* (QRNG) is at an historical high. In the first quarter of 2004 there were 188 QRNG (20.5%), and in the second quarter 172 (20.2%). The numbers here are substantially higher than the corresponding figures in the third quarter of 2003 (136 isolates, 16%). The majority of the QRNG (184 of 200, 92%) exhibited higher-level resistance. 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 again widely distributed. The highest number, 93, was found in New South Wales (36.6% of isolates in that State) while 64 QRNG were 33% of gonococci in Victoria. In Queensland there were 27 QRNG (17%), seven in Western Australia (9%), four in South Australia (17%), two in both the Northern Territory and Tasmania and one in the Australian Capital Territory.

**Figure 9. The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia by jurisdiction, 1 July to 30 September 2004**



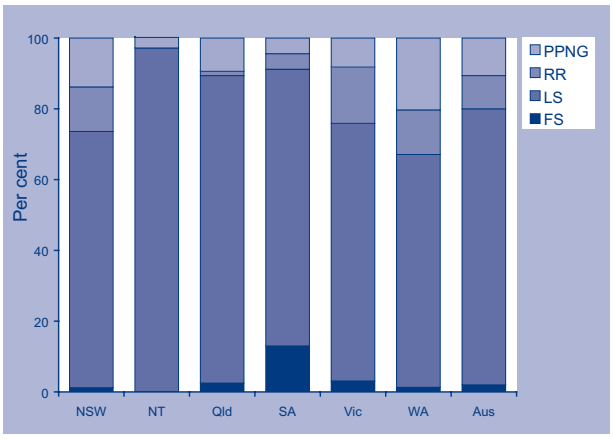

| | |
|---|---|
| LS QRNG | Ciprofloxacin MICs 0.06 – 0.5 mg/L. |
| R QRNG | Ciprofloxacin MICs => 1 mg/L. |

### Penicillins

In this quarter 20% of all isolates examined were penicillin resistant by one or more mechanisms—10.6% penicillinase producing *Neisseria gonorrhoeae* (PPNG) and 9.4% by chromosomal mechanisms (CMRNG). The proportion of all penicillin resistant strains is little changed from the previous quarter and a slight increase from the 18% detected in the third quarter of 2003. The number of PPNG increased to 88 from the 77 seen in the same period in 2003, but the number of CMRNG was essentially unchanged (77 for this period in 2004, 76 in 2003). The proportion of all strains resistant to the penicillins by any mechanism ranged from 3.8% in the Northern Territory to 33% in Western Australia.

Figure 10 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 PPNG, aggregated for Australia and by State and 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.

**Figure 10. Categorisation of gonococci isolated in Australia by penicillin susceptibility and by region, 1 July to 30 September 2004**




| | |
|---|---|
| FS | fully sensitive to penicillin, MIC <,= 0.03 mg/L. |
| LS | less sensitive to penicillin, MIC 0.06 – 0.5 mg/L. |
| RR | relatively resistant to penicillin, MIC >,= 1 mg/L. |
| PPNG | penicillinase producing Neisseria gonorrhoeae. |

The highest proportion of PPNG was found in Western Australia where the 16 PPNG were 20.3% of all isolates. Thirty-five PPNG representing 13.8% of all isolates were found in New South Wales, 15 (9.4%) in Queensland and 16 (8.2%) in Victoria. There was a single PPNG in South Australia, Tasmania and the Australian Capital Territory and three in the Northern Territory. The number of CMRNG was highest in Victoria (31, 16%) and New South Wales (32, 13%) and in Western Australia 10 CMRNG isolates were 12.6% of the total. Elsewhere CMRNG were in low numbers (Queensland, Tasmania, South Australia) or absent (Northern Territory, Australian Capital Territory).

**Ceftriaxone.**

An increased number of isolates (12, 4.7%) with decreased susceptibility to ceftriaxone were detected in New South Wales in this quarter, but none were seen elsewhere. Small numbers of these strains have been present for a number of years, mostly in New South Wales, but only occasionally in other jurisdictions.

**Spectinomycin**

All isolates susceptible to this injectable agent.

**High level tetracycline resistance (TRNG)**

Both the number (121) and proportion (14.6%) of TRNG continued to increase from the 2003 figures (92, 11.5%). TRNG were found in all jurisdictions with 22 (28%) in Western Australia, 56 (22%) in New South Wales, 18 (11%) in Queensland and 19 (10%) in Victoria.

**Reference**

1. Management of sexually transmitted diseases. World Health Organization 1997; Document WHO/GPA/TEM94.1 Rev.1 p 37.

## *Meningococcal surveillance*

*John Tapsall, The Prince of Wales Hospital, Randwick, NSW, 2031 for the Australian Meningococcal Surveillance Programme.*

*The reference laboratories of the Australian Meningococcal Surveillance Programme report data on the number of laboratory confirmed cases confirmed either by culture or by non-culture based techniques. Culture positive cases, where a Neisseria meningitidis is grown from a normally sterile site or skin, and non-culture based diagnoses, derived from results of nucleic acid amplification assays and serological techniques, are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions. Data contained in the quarterly reports are restricted to a description of the number of cases per jurisdiction, and serogroup, where known. A full analysis of laboratory confirmed cases of IMD is contained in the annual reports of the Programme, published in Communicable Diseases Intelligence.*

*Laboratory confirmed cases of invasive meningococcal disease for the period 1 July to 30 September 2004, are included in this issue of* Communicable Diseases Intelligence *(Table 9)*.

**Reporting period 1 July to 30 September 2004**

**Table 9. Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 July to 30 September 2004, by jurisdiction and serogroup**

| Jurisdiction | Year | Serogroup | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | A | | B | | C | | Y | | W135 | | ND | | All | |
| | | Q3 | ytd | Q3 | ytd | Q3 | ytd | Q3 | ytd | Q3 | ytd | Q3 | ytd | Q3 | ytd |
| Australian Capital Territory | 2004 | | | 0 | *3* | 3 | *7* | | | | | | | 3 | 10 |
| | 2003 | | | (2) | (3) | (2) | (2) | | | | | | | (4) | (5) |
| New South Wales | 2004 | | | 22 | *60* | 6 | *15* | 1 | *3* | 2 | *4* | 2 | *12* | 33 | 94 |
| | 2003 | | | (38) | (75) | (19) | (32) | (1) | (4) | (0) | (1) | (3) | (15) | (61) | (127) |
| Northern Territory | 2004 | | | 0 | *5* | 0 | *0* | | | 0 | 1 | | | 0 | 6 |
| | 2003 | | | (3) | (9) | (0) | (0) | | | (1) | (1) | | | (4) | (10) |
| Queensland | 2004 | 0 | *1* | 13 | *36* | 8 | *20* | 0 | *1* | 1 | *2* | 0 | *2* | 22 | 62 |
| | 2003 | (1) | (1) | (17) | (34) | (16) | (31) | (1) | (1) | (0) | (0) | (0) | (8) | (35) | (75) |
| South Australia | 2004 | | | 2 | *11* | 1 | *1* | | | | | | | 3 | 12 |
| | 2003 | | | (7) | (15) | (1) | (2) | (1) | (1) | (1) | (1) | | | (10) | (19) |
| Tasmania | 2004 | | | 3 | *6* | 5 | *5* | | | 0 | *1* | 1 | *3* | 9 | 15 |
| | 2003 | | | (3) | (3) | (4) | (5) | | | | | | | (7) | (8) |
| Victoria | 2004 | | | 17 | *45* | 3 | *12* | 0 | *3* | 2 | *2* | 1 | *3* | 23 | 65 |
| | 2003 | | | (22) | (35) | (17) | (39) | (2) | (2) | (0) | (1) | (1) | (6) | (42) | (83) |
| Western Australia | 2004 | | | 11 | *23* | 2 | *4* | | | 1 | *1* | | | 14 | 28 |
| | 2003 | | | (11) | (22) | (2) | (5) | (0) | (1) | | | | | (13) | (28) |
| Australia | 2004 | 0 | *1* | 68 | *189* | 28 | *64* | 1 | *7* | 6 | *11* | 4 | *20* | 107 | 292 |
| | 2003 | (1) | (1) | (103) | (196) | (61) | (116) | (5) | (9) | (2) | (4) | (4) | (29) | (176) | (355) |

Numbers of laboratory confirmed diagnoses of IMD made in the same periods in 2003 are also shown in parenthesis.

**Q3** = third quarter; *ytd* = year to 30 September 2004; ND = not determined.

## *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 Surveillance systems reported in CDI, published in* Commun Dis Intell *2004;28:99.*

*HIV and AIDS diagnoses and deaths following AIDS reported for 1 April to 30 June 2004, as reported to 30 September 2004, are included in this issue of* Communicable Diseases Intelligence *(Tables 10 and 11).*

**Table 10. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 April to 30 June 2004, by sex and state or territory of diagnosis**

| | Sex | State or territory | | | | | | | | Total for Australia | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | This period 2004 | This period 2003 | Year to date 2004 | Year to date 2003 |
| HIV diagnoses | Female | 0 | 10 | 0 | 7 | 1 | 1 | 10 | 0 | 29 | 23 | 67 | 43 |
| | Male | 0 | 82 | 0 | 28 | 4 | 3 | 41 | 0 | 158 | 208 | 359 | 410 |
| | Not reported | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 2 | 2 |
| | Total[1] | 0 | 93 | 0 | 35 | 5 | 4 | 51 | 0 | 188 | 232 | 429 | 455 |
| AIDS diagnoses | Female | 0 | 2 | 0 | 1 | 0 | 0 | 1 | 0 | 4 | 3 | 7 | 7 |
| | Male | 0 | 11 | 0 | 7 | 2 | 1 | 3 | 0 | 24 | 52 | 62 | 87 |
| | Total[1] | 0 | 13 | 0 | 8 | 2 | 1 | 4 | 0 | 28 | 55 | 70 | 95 |
| AIDS deaths | Female | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 2 | 5 |
| | Male | 0 | 7 | 0 | 3 | 5 | 0 | 0 | 0 | 15 | 14 | 27 | 31 |
| | Total[1] | 0 | 8 | 0 | 3 | 5 | 0 | 0 | 0 | 16 | 15 | 29 | 36 |

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

**Table 11. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 30 June 2004, by sex and state or territory**

| | Sex | State or territory | | | | | | | | Australia |
|---|---|---|---|---|---|---|---|---|---|---|
| | | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | |
| HIV diagnoses | | 30 | 759 | 17 | 222 | 82 | 8 | 303 | 159 | 1,580 |
| | | 247 | 12,507 | 121 | 2,373 | 805 | 88 | 4,664 | 1,049 | 21,854 |
| | | 0 | 238 | 0 | 0 | 0 | 0 | 22 | 0 | 260 |
| | | 277 | 13,532 | 138 | 2,604 | 888 | 96 | 5,008 | 1,215 | 23,758 |
| AIDS diagnoses | Female | 9 | 220 | 1 | 61 | 30 | 4 | 91 | 34 | 450 |
| | Male | 92 | 5,094 | 41 | 964 | 385 | 48 | 1,844 | 407 | 8,875 |
| | Total[1] | 101 | 5,329 | 42 | 1,027 | 416 | 52 | 1,945 | 443 | 9,355 |
| AIDS deaths | Female | 6 | 127 | 0 | 40 | 20 | 2 | 58 | 22 | 275 |
| | Male | 71 | 3,442 | 26 | 625 | 262 | 32 | 1,353 | 277 | 6,088 |
| | Total[1] | 77 | 3,578 | 26 | 667 | 282 | 34 | 1,419 | 300 | 6,383 |

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

## *National Enteric Pathogens Surveillance System*

*The National Enteric Pathogens Surveillance System (NEPSS) collects, analyses and disseminates data on human enteric bacterial infections diagnosed in Australia. These pathogens include* Salmonella, E. coli, Vibrio, Yersinia, Plesiomonas, Aeromonas *and* Campylobacter. Communicable Diseases Intelligence *quarterly reports include only* Salmonella.

*Data are based on reports to NEPSS from Australian laboratories of laboratory-confirmed human infection with* Salmonella. Salmonella *are identified to the level of serovar and, if applicable, phage-type. Infections apparently acquired overseas are included. Multiple isolations of a single* Salmonella *serovar/phage-type from one or more body sites during the same episode of illness are counted once only. The date of the case is the date the primary diagnostic laboratory isolated a* Salmonella *from the clinical sample.*

*Note that the historical quarterly mean counts should be interpreted with caution, and are affected by surveillance artefacts such as newly recognised (such as* S. *Typhimurium 197 and* S. *Typhimurium U290) and incompletely typed* Salmonella.

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

*Reports to the National Enteric Pathogens Surveillance System of* Salmonella *infection for the period 1 July to 30 September 2004 are included in Tables 12 and 13. Data include cases reported and entered by 28 October 2004. Counts are preliminary, and subject to adjustment after completion of typing and reporting of further cases to NEPSS. For more information about NEPSS see Surveillance systems reported in CDI, published in* Commun Dis Intell *2004;28:101.*

**Reporting period 1 July to 30 September 2004**

The total number of reports to NEPSS of human Salmonella infection declined to 1,156 in the third quarter of 2004, 42 per cent fewer than in second quarter of 2004 (Table 12) but 19 per cent more than the final count for the third quarter of 2004. Case counts to 28 October 2004 are expected to comprise more than 95 per cent of the final counts for the quarter.

During the third quarter of 2004, the 25 most common Salmonella types in Australia accounted for 669 cases, 58 per cent of all reported human Salmonella infections (Table 13).

Eighteen of the 25 most common Salmonella infections in the third quarter of 2004 were among the 25 most commonly reported in the previous quarter.

Reports of common salmonellae with counts well above historical averages include S. Typhimurium phage type 197 (in the eastern mainland states), S. Virchow phage type 8 (particularly in Queensland and New South Wales), and S. Stanley (particularly in Victoria). Counts of several typically overseas-acquired phage types of S. Enteritidis were also elevated. While still among the more common salmonellae, reports of S. Typhimurium phage type 170/108 declined in number and relative prominence.

We thank scientists, diagnostic and reference laboratories, State and Territory health departments, and the Australian Government Department of Health and Ageing for their contributions to NEPSS.

**Acknowledgement**: Thanks to contributing laboratories and scientists.

**Table 12. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 July to 30 September 2004, as reported to 28 October 2004**

| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Australia |
|---|---|---|---|---|---|---|---|---|---|
| Total all *Salmonella* for quarter | 17 | 263 | 68 | 355 | 69 | 11 | 238 | 135 | 1,156 |
| Total contributing *Salmonella* types | 12 | 91 | 29 | 102 | 33 | 10 | 88 | 65 | 209 |

## Table 13. Top 25 *Salmonella* types identified in Australia, by state or territories, 1 July to 30 September 2004

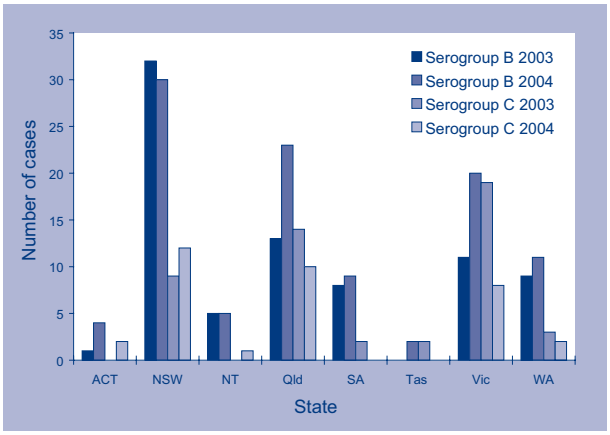
| National rank | *Salmonella* type | State or territory | | | | | | | | Total 3rd quarter 2004 | Last 10 years mean 3rd quarter | Year to date 2004 | Year to date 2003 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | ACT | NSW | NT | QLD | SA | Tas | Vic | WA | | | | |
| 1 | *S* Typhimurium 135 | 0 | 20 | 0 | 31 | 4 | 0 | 20 | 26 | 101 | 77 | 450 | 559 |
| 2 | *S* Typhimurium 197 | 2 | 8 | 0 | 39 | 0 | 0 | 14 | 0 | 63 | 5 | 203 | 134 |
| 3 | *S* Saintpaul | 0 | 8 | 7 | 24 | 1 | 0 | 5 | 9 | 54 | 41 | 290 | 230 |
| 4 | *S* Typhimurium 9 | 1 | 5 | 0 | 4 | 9 | 1 | 18 | 2 | 40 | 74 | 287 | 338 |
| 5 | *S* Virchow 8 | 2 | 12 | 0 | 22 | 1 | 0 | 2 | 0 | 39 | 18 | 269 | 130 |
| 6 | *S* Typhimurium 170 | 4 | 13 | 0 | 7 | 0 | 1 | 10 | 0 | 35 | 19 | 413 | 340 |
| 7 | *S* Birkenhead | 0 | 8 | 1 | 18 | 0 | 0 | 0 | 0 | 27 | 20 | 198 | 140 |
| 8 | *S* Chester | 0 | 7 | 4 | 8 | 5 | 0 | 0 | 3 | 27 | 20 | 156 | 174 |
| 9 | *S* Stanley | 1 | 4 | 1 | 4 | 1 | 0 | 11 | 4 | 26 | 13 | 59 | 35 |
| 10 | *S* Enteritidis 6a | 0 | 3 | 0 | 3 | 1 | 1 | 8 | 10 | 26 | 3.2 | 50 | 15 |
| 11 | *S* Typhimurium RDNC | 0 | 13 | 0 | 3 | 3 | 0 | 5 | 0 | 24 | 15 | 84 | 54 |
| 12 | *S* Infantis | 0 | 5 | 2 | 1 | 1 | 0 | 10 | 3 | 22 | 21 | 113 | 165 |
| 13 | *S* Aberdeen | 0 | 2 | 1 | 13 | 0 | 1 | 2 | 0 | 19 | 11 | 91 | 67 |
| 14 | *S* Weltevreden | 0 | 3 | 2 | 8 | 0 | 0 | 5 | 1 | 19 | 7 | 59 | 34 |
| 15 | *S* Muenchen | 0 | 4 | 2 | 5 | 2 | 0 | 1 | 4 | 18 | 16 | 90 | 109 |
| 16 | *S* Enteritidis 4b | 0 | 1 | 0 | 0 | 0 | 1 | 14 | 2 | 18 | 4.4 | 29 | 11 |
| 17 | *S* Typhimurium 126 | 0 | 5 | 0 | 1 | 1 | 0 | 6 | 1 | 14 | 19 | 58 | 55 |
| 18 | *S* Ball | 0 | 0 | 14 | 0 | 0 | 0 | 0 | 0 | 14 | 6 | 49 | 39 |
| 19 | *S* Typhimurium 12 | 0 | 9 | 0 | 1 | 0 | 0 | 3 | 0 | 13 | 4.3 | 202 | 74 |
| 20 | *S* Litchfield | 0 | 5 | 4 | 4 | 0 | 0 | 0 | 0 | 13 | 3.6 | 34 | 30 |
| 21 | *S* Enteritidis 1 | 0 | 3 | 0 | 2 | 3 | 0 | 3 | 1 | 12 | 6 | 32 | 12 |
| 22 | *S* Enteritidis 1b | 0 | 6 | 0 | 1 | 0 | 0 | 2 | 3 | 12 | 1.2 | 26 | 10 |
| 23 | *S* Agona | 1 | 1 | 1 | 4 | 1 | 0 | 1 | 2 | 11 | 13 | 63 | 53 |
| 24 | *S* Typhimurium untypable | 0 | 4 | 0 | 2 | 0 | 0 | 4 | 1 | 11 | 11 | 22 | 18 |
| 25 | *S* Hvittingfoss | 0 | 2 | 0 | 7 | 1 | 0 | 1 | 0 | 11 | 9 | 117 | 73 |

# Errata

**Figure 3 Highlights for second quarter 2004, CDI 2004;28:3.**

Figure 3 in the Highlights section of *Communicable Diseases Intelligence* 2004;28:3 p 409 'Notifications of serogroup B and C meningococcal infection, January to June 2003 and January to June 2004, by jurisdiction' was published without the data for Western Australia. A corrected Figure 3, including Western Australian data is reproduced below.

**Figure 3. Notifications of Serogroup B and C meningococcal infection, January to June 2003 and January to June 2004, by jurisdiction**




**Communicable Diseases Surveillance data—Table 2 ( p413) and Table 3 (p415), *Communicable Diseases Intelligence* 2004;28:3**

The editors apologise for the errors in the notifications (Table 2) and rates (Table 3) for the period 1 April 2004 to 30 June 2004, presented in the last issue of *CDI* The correct notification rates are shown in the tables on the following pages.

**Table 2. Notifications of diseases received by State and Territory health authorities for the period 1 April to 30 June 2004, by date of onset***

| Disease | State or territory | | | | | | | | Total 2nd quarter 2004[1] | Total 1st quarter 2004 | Total 2nd quarter 2003 | Last 5 years mean 2nd quarter | Year to date 2004 | Last 5 years YTD mean | Ratio† |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | | | | | | | |
| **Bloodborne diseases** | | | | | | | | | | | | | | | |
| Hepatitis B (incident) | 2 | 17 | 2 | 13 | 2 | 10 | 9 | 5 | 60 | 70 | 90 | 99.2 | 130 | 190.6 | 0.6 |
| Hepatitis B (unspecified) | 12 | 1,324 | NN | 216 | 66 | 30 | 386 | 87 | 2,121 | 1,479 | 1,470 | 1,817.2 | 3600 | 3,458.2 | 1.2 |
| Hepatitis C (incident) | 3 | 4 | NN | NN | 8 | 2 | 13 | 18 | 48 | 79 | 110 | 114.0 | 127 | 247.0 | 0.4 |
| Hepatitis C (unspecified) | 58 | 1,218 | 72 | 709 | 182 | 170 | 714 | 275 | 3,398 | 3,572 | 3,292 | 4,237.4 | 6970 | 8,699.0 | 0.8 |
| Hepatitis D | 0 | 2 | 0 | 5 | 0 | 0 | 1 | 0 | 8 | 4 | 5 | 5.4 | 12 | 10.8 | 1.5 |
| **Gastrointestinal diseases** | | | | | | | | | | | | | | | |
| Botulism | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.0 | 0.0 |
| Campylobacteriosis[2] | 77 | NN | 43 | 767 | 401 | 208 | 1,211 | 389 | 3,096 | 4,437 | 3,515 | 3,295.8 | 7533 | 6,870.8 | 0.9 |
| Cryptosporidiosis‡ | 0 | 60 | 27 | 215 | 17 | 6 | 82 | 31 | 438 | 515 | 310 | 452.0 | 953 | 1,493.7 | 1.0 |
| Haemolytic uraemic syndrome | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 1 | 3 | 3 | 2 | 2.0 | 6 | 6.6 | 1.5 |
| Hepatitis A | 0 | 32 | 3 | 7 | 1 | 0 | 15 | 16 | 74 | 106 | 108 | 174.4 | 180 | 399.4 | 0.4 |
| Hepatitis E | 0 | 1 | 0 | 1 | 0 | 2 | 5 | 0 | 9 | 11 | 1 | 4.0 | 20 | 6.2 | 2.3 |
| Listeriosis | 0 | 11 | 0 | 3 | 1 | 0 | 3 | 1 | 19 | 17 | 19 | 15.8 | 36 | 35.2 | 1.2 |
| Salmonellosis (NEC) | 44 | 524 | 102 | 666 | 145 | 48 | 321 | 137 | 1,987 | 2,772 | 1,602 | 1,676.0 | 4759 | 4,281.0 | 1.2 |
| Shigellosis | 1 | 20 | 36 | 23 | 15 | 2 | 15 | 31 | 143 | 154 | 103 | 130.6 | 297 | 285.0 | 1.1 |
| SLTEC,VTEC[3] | 0 | 0 | 0 | 2 | 5 | 0 | 1 | 0 | 8 | 12 | 12 | 9.2 | 20 | 26.6 | 0.9 |
| Typhoid | 0 | 8 | 0 | 1 | 1 | 0 | 2 | 3 | 15 | 27 | 6 | 11.0 | 42 | 35.0 | 1.4 |
| **Quarantinable diseases** | | | | | | | | | | | | | | | |
| Cholera | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 2 | 1 | 1 | 0.6 | 3 | 1.4 | 3.3 |
| Plague | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.0 | 0.0 |
| Rabies | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.0 | 0.0 |
| Smallpox | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.0 | 0.0 |
| Tularemia | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.0 | 0.0 |
| Viral haemorrhagic fever | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.0 | 0.0 |
| Yellow fever | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.0 | 0.0 |

**Table 2. Notifications of diseases received by State and Territory health authorities for the period 1 April to 30 June 2004, by date of onset* *continued***

| Disease | State or territory | | | | | | | | Total 2nd quarter 2004[1] | Total 1st quarter 2004 | Total 2nd quarter 2003 | Last 5 years mean 2nd quarter | Year to date 2004 | Last 5 years YTD mean | Ratio[†] |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | | | | | | | |
| **Sexually transmissible diseases** | | | | | | | | | | | | | | | |
| Chlamydia | 157 | 2,329 | 443 | 2,136 | 666 | 258 | 1,866 | 1,049 | 8,904 | 9,215 | 7,432 | 5,236.8 | 18,119 | 10,387.2 | 1.7 |
| Donovanosis | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 2 | 3 | 4.6 | 3 | 10.4 | 0.2 |
| Gonococcal infection[4] | 6 | 319 | 443 | 318 | 123 | 16 | 257 | 400 | 1,882 | 1,751 | 1,684 | 1,599.0 | 3,633 | 3,190.0 | 1.2 |
| Syphilis (unspecified) | 5 | 0 | 57 | 0 | 0 | 0 | 0 | 0 | 62 | 82 | 134 | 213.8 | 144 | 423.2 | 0.3 |
| Syphilis < two years duration | 0 | 47 | 1 | 24 | 8 | 0 | 23 | 8 | 111 | 142 | 104 | 96.5 | 253 | 202.5 | 1.2 |
| Syphilis > two years or unknown duration | 0 | 220 | 17 | 33 | 1 | 0 | 87 | 27 | 385 | 409 | 249 | 259.5 | 794 | 514.0 | 1.5 |
| Syphilis - congenital | 0 | 0 | 2 | 0 | 0 | 0 | 1 | 0 | 3 | 2 | 3 | 2.8 | 5 | 4.4 | 1.1 |
| **Vaccine preventable disease** | | | | | | | | | | | | | | | |
| Diphtheria | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.2 | 0.0 |
| *Haemophilus influenzae* type b | 0 | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 3 | 7 | 8 | 10.2 | 10 | 15.8 | 0.3 |
| Influenza (laboratory confirmed)[‡] | 1 | 106 | 4 | 26 | 13 | 2 | 8 | 1 | 161 | 107 | 130 | 306.3 | 268 | 395.7 | 0.5 |
| Measles | 0 | 1 | 0 | 0 | 1 | 0 | 4 | 1 | 7 | 11 | 27 | 26.8 | 18 | 71.6 | 0.3 |
| Mumps | 0 | 12 | 0 | 5 | 1 | 0 | 0 | 0 | 18 | 24 | 12 | 39.8 | 42 | 69.0 | 0.5 |
| Pertussis | 13 | 573 | 1 | 140 | 59 | 8 | 112 | 147 | 1,053 | 1,107 | 832 | 1,100.6 | 2,160 | 2,273.0 | 1.0 |
| Pneumococcal disease (invasive)[‡] | 17 | 226 | 18 | 107 | 51 | 23 | 106 | 42 | 590 | 328 | 544 | 510.7 | 918 | 768.3 | 1.2 |
| Poliomyelitis | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.0 | 0.0 |
| Rubella | 0 | 6 | 0 | 4 | 0 | 0 | 0 | 0 | 10 | 6 | 11 | 51.4 | 16 | 105.8 | 0.2 |
| Rubella - congenital | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0.0 | 1 | 0.2 | 0.0 |
| Tetanus | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 2 | 0 | 0.4 | 3 | 2.4 | 2.5 |
| **Vectorborne diseases** | | | | | | | | | | | | | | | |
| Barmah Forest virus infection | 0 | 135 | 8 | 157 | 3 | 0 | 3 | 10 | 316 | 319 | 760 | 397.8 | 635 | 662.6 | 0.8 |
| Dengue | 2 | 6 | 5 | 47 | 0 | 0 | 3 | 1 | 64 | 236 | 264 | 88.6 | 300 | 227.6 | 0.7 |
| Flavivirus infection NEC | 0 | 4 | 0 | 24 | 0 | 0 | 1 | 0 | 29 | 74 | 15 | 11.4 | 103 | 32.0 | 2.5 |
| Japanese encephalitis[‡] | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0.0 | 1 | 0.0 | 0.0 |
| Kunjin virus[‡] | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 6 | 5 | 2.3 | 6 | 7.3 | 0.0 |
| Malaria | 4 | 19 | 8 | 75 | 8 | 8 | 19 | 8 | 149 | 127 | 155 | 169.4 | 276 | 380.8 | 0.9 |
| Murray Valley encephalitis[‡] | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0.0 | 1 | 1.5 | 0.0 |
| Ross River virus infection | 3 | 324 | 17 | 866 | 7 | 10 | 21 | 138 | 1,386 | 2,444 | 2,350 | 1,386.2 | 3,830 | 2,812.2 | 1.0 |

**Table 2. Notifications of diseases received by State and Territory health authorities for the period 1 April to 30 June 2004, by date of onset* *continued***

| Disease | State or territory | | | | | | | | Total 2nd quarter 2004[1] | Total 1st quarter 2004 | Total 2nd quarter 2003 | Last 5 years mean 2nd quarter | Year to date 2004 | Last 5 years YTD mean | Ratio† |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | | | | | | | |
| **Zoonoses** | | | | | | | | | | | | | | | |
| Anthrax‡ | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.0 | 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 | 3 | 0 | 0 | 1 | 0 | 4 | 6 | 7 | 5.0 | 10 | 12.0 | 0.8 |
| Leptospirosis | 0 | 5 | 1 | 46 | 0 | 0 | 0 | 0 | 52 | 65 | 28 | 67.4 | 117 | 143.6 | 0.8 |
| Lyssavirus unspecified‡ | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.0 | 0.0 |
| Ornithosis | 0 | 15 | 0 | 0 | 1 | 0 | 37 | 0 | 53 | 70 | 44 | 37.0 | 123 | 62.8 | 1.4 |
| Q fever | 0 | 52 | 1 | 34 | 6 | 0 | 11 | 2 | 106 | 110 | 121 | 150.2 | 216 | 320.4 | 0.7 |
| **Other bacterial infections** | | | | | | | | | | | | | | | |
| Creutzfeldt-Jakob disease | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.0 | 0 | 0.0 | 0.0 |
| Legionellosis | 1 | 26 | 3 | 3 | 10 | 0 | 34 | 11 | 88 | 86 | 87 | 113.4 | 174 | 191.4 | 0.8 |
| Leprosy | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 2 | 1.2 | 2 | 2.4 | 0.0 |
| Meningococcal infection | 1 | 46 | 4 | 22 | 5 | 6 | 27 | 9 | 120 | 96 | 104 | 136.6 | 216 | 241.8 | 0.9 |
| Tuberculosis | 0 | 52 | 3 | 19 | 18 | 3 | 64 | 16 | 175 | 227 | 192 | 225.8 | 402 | 474.4 | 0.8 |
| **Total** | 407 | 7,747 | 1,323 | 6,721 | 1,825 | 812 | 5,464 | 2,864 | 27,163 | 30,324 | 25,953 | 24,296.5 | 57,487 | 50,053.4 | 1.1 |

1. Totals comprise data from all states and territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
2. Not reported from New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
3. Infections with shiga-like toxin (verotoxin) producing *Escherichia coli* (SLTEC/VTEC).

* Date of onset = the true date of onset. If this is not available, the 'date of onset' is equivalent to the earliest of two dates: (i) specimen collection date or (ii) the date of notification to a public health unit. Hepatitis B and C unspecified were analysed by date of notification.

† Ratio = ratio of current quarter total to the mean of last 5 years for the same quarter.

‡ Notifiable from January 2001. Ratio and mean calculations are based on the last three years.

NN Not notifiable.

NEC Not elsewhere classified.

**Table 3. Notification rates of diseases by state or territory, 1 April to 30 June 2004. (Rate per 100,000 population)**

| Disease[1] | State or territory | | | | | | | | Australia |
|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | |
| **Bloodborne diseases** | | | | | | | | | |
| Hepatitis B (incident) | 2.5 | 1.0 | 4.0 | 1.4 | 0.5 | 8.4 | 0.7 | 1.0 | 1.2 |
| Hepatitis B (unspecified) | 14.9 | 79.2 | NN | 22.8 | 17.3 | 25.2 | 31.5 | 17.8 | 43.1 |
| Hepatitis C (incident) | 3.7 | 0.2 | NN | NN | 2.1 | 1.7 | 1.1 | 3.7 | 1.2 |
| Hepatitis C (unspecified) | 71.9 | 72.9 | 145.2 | 74.7 | 47.7 | 142.5 | 58.2 | 56.3 | 68.4 |
| Hepatitis D | 0.0 | 0.1 | 0.0 | 0.5 | 0.0 | 0.0 | 0.1 | 0.0 | 0.2 |
| **Gastrointestinal diseases** | | | | | | | | | |
| Botulism | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Campylobacteriosis[2] | 95.4 | NN | 86.7 | 80.8 | 105.0 | 174.4 | 98.7 | 79.7 | 94.0 |
| Cryptosporidiosis | 0.0 | 3.6 | 54.4 | 22.7 | 4.5 | 5.0 | 6.7 | 6.4 | 8.8 |
| Haemolytic uraemic syndrome | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.2 | 0.1 |
| Hepatitis A | 0.0 | 1.9 | 6.0 | 0.7 | 0.3 | 0.0 | 1.2 | 3.3 | 1.5 |
| Hepatitis E | 0.0 | 0.1 | 0.0 | 0.1 | 0.0 | 1.7 | 0.4 | 0.0 | 0.2 |
| Listeriosis | 0.0 | 0.7 | 0.0 | 0.3 | 0.3 | 0.0 | 0.2 | 0.2 | 0.4 |
| Salmonellosis (NEC) | 54.5 | 31.3 | 205.7 | 70.2 | 38.0 | 40.2 | 26.2 | 28.1 | 40.0 |
| Shigellosis | 1.2 | 1.2 | 72.6 | 2.4 | 3.9 | 1.7 | 1.2 | 6.4 | 2.9 |
| SLTEC,VTEC[3] | 0.0 | 0.0 | 0.0 | 0.2 | 1.3 | 0.0 | 0.1 | 0.0 | 0.2 |
| Typhoid | 0.0 | 0.5 | 0.0 | 0.1 | 0.3 | 0.0 | 0.2 | 0.6 | 0.3 |
| **Quarantinable diseases** | | | | | | | | | |
| Cholera | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 | 0.0 | 0.1 | 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 |
| Smallpox | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Tularemia | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Viral haemorrhagic fever | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Yellow fever | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| **Sexually transmissible diseases** | | | | | | | | | |
| Chlamydia | 194.5 | 139.3 | 893.4 | 225.0 | 174.4 | 216.3 | 152.2 | 214.9 | 179.3 |
| Donovanosis | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Gonococcal infection | 7.4 | 19.1 | 893.4 | 33.5 | 32.2 | 13.4 | 21.0 | 82.0 | 37.9 |
| Syphilis (unspecified) | 6.2 | 0.0 | 114.9 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 1.2 |
| Syphilis < 2 years duration | 0.0 | 2.8 | 2.0 | 2.5 | 2.1 | 0.0 | 1.9 | 1.6 | 2.2 |
| Syphilis > 2 years or unknown duration | 0.0 | 13.2 | 34.3 | 3.5 | 0.3 | 0.0 | 7.1 | 5.5 | 7.8 |
| Syphilis - congenital | 0.0 | 0.0 | 4.0 | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 | 0.1 |

**Table 3. Notification rates of diseases by state or territory, 1 April to 30 June 2004. (Rate per 100,000 population) , *continued***

| Disease[1] | State or territory | | | | | | | | Australia |
|---|---|---|---|---|---|---|---|---|---|
| | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | |
| **Vaccine preventable diseases** | | | | | | | | | |
| Diphtheria | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| *Haemophilus influenzae* type b | 0.0 | 0.1 | 2.0 | 0.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.1 |
| Influenza (laboratory confirmed) | 1.2 | 6.3 | 8.1 | 2.7 | 3.4 | 1.7 | 0.7 | 0.2 | 3.2 |
| Measles | 0.0 | 0.1 | 0.0 | 0.0 | 0.3 | 0.0 | 0.3 | 0.2 | 0.1 |
| Mumps | 0.0 | 0.7 | 0.0 | 0.5 | 0.3 | 0.0 | 0.0 | 0.0 | 0.4 |
| Pertussis | 16.1 | 34.3 | 2.0 | 14.7 | 15.5 | 6.7 | 9.1 | 30.1 | 21.2 |
| Pneumococcal disease (invasive) | 21.1 | 13.5 | 36.3 | 11.3 | 13.4 | 19.3 | 8.6 | 8.6 | 11.9 |
| Poliomyelitis | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Rubella | 0.0 | 0.4 | 0.0 | 0.4 | 0.0 | 0.0 | 0.0 | 0.0 | 0.2 |
| Rubella - congenital | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Tetanus | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| **Vectorborne diseases** | | | | | | | | | |
| Barmah Forest virus infection | 0.0 | 8.1 | 16.1 | 16.5 | 0.8 | 0.0 | 0.2 | 2.0 | 6.4 |
| Dengue | 2.5 | 0.4 | 10.1 | 5.0 | 0.0 | 0.0 | 0.2 | 0.2 | 1.3 |
| Flavivirus infection (NEC) | 0.0 | 0.2 | 0.0 | 2.5 | 0.0 | 0.0 | 0.1 | 0.0 | 0.6 |
| Japanese encephalitis | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Kunjin virus | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Malaria | 5.0 | 1.1 | 16.1 | 7.9 | 2.1 | 6.7 | 1.5 | 1.6 | 3.0 |
| Murray Valley encephalitis | 0.0 | 0.0 | 2.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Ross River virus infection | 3.7 | 19.4 | 34.3 | 91.2 | 1.8 | 8.4 | 1.7 | 28.3 | 27.9 |
| **Zoonoses** | | | | | | | | | |
| Anthrax | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Australian bat lyssavirus | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Brucellosis | 0.0 | 0.0 | 0.0 | 0.3 | 0.0 | 0.0 | 0.1 | 0.0 | 0.1 |
| Leptospirosis | 0.0 | 0.3 | 2.0 | 4.8 | 0.0 | 0.0 | 0.0 | 0.0 | 1.0 |
| Lyssavirus unspecified | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Ornithosis | 0.0 | 0.9 | 0.0 | 0.0 | 0.3 | 0.0 | 3.0 | 0.0 | 1.1 |
| Q fever | 0.0 | 3.1 | 2.0 | 3.6 | 1.6 | 0.0 | 0.9 | 0.4 | 2.1 |
| **Other bacterial infections** | | | | | | | | | |
| Legionellosis | 1.2 | 1.6 | 6.0 | 0.3 | 2.6 | 0.0 | 2.8 | 2.3 | 1.8 |
| Leprosy | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Meningococcal infection | 1.2 | 2.8 | 8.1 | 2.3 | 1.3 | 5.0 | 2.2 | 1.8 | 2.4 |
| Tuberculosis | 0.0 | 3.1 | 6.0 | 2.0 | 4.7 | 2.5 | 5.2 | 3.3 | 3.5 |

1. Rates are subject to retrospective revision.
2. Not reported from New South Wales where it is only notifiable as ‘foodborne disease’ or ‘gastroenteritis in an institution’.
3. Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (SLTEC/VTEC).

NN Not Notifiable.

NEC Not Elsewhere Classified.

# Overseas Briefs

## *ProMed-mail*

***This material has been summarised from information provided by ProMED-mail (http://www.promedmail.org). A link to this site can be found under 'Other Australian and international communicable diseases sites' on the Communicable Diseases Australia homepage.***

## *Avian influenza — situation in Thailand; status of pandemic vaccine development, 4 October 2004*

The Ministry of Public Health in Thailand has today confirmed a further case of human infection with H5N1 avian influenza. The case, which was fatal, was a 9-year-old girl from the northern province of Phetchabun. She developed symptoms on 23 September, was hospitalized on 27 September, and died of severe respiratory disease on 3 October.

Investigation of the case has identified exposure to diseased chickens as the most likely cause of infection. Following the death of chickens in the child's household, she assisted in preparation of the birds for cooking, including the plucking of feathers.

The World Health Organization (WHO) stresses the importance of educating populations in affected countries, especially those living in remote rural areas, about the danger of contact with diseased birds.

Since the beginning of this year, Thailand has reported 16 laboratory confirmed cases of H5N1 infection, of which 11 have been fatal. Four of these cases have occurred during the past four weeks.

Last week, Thai officials announced a probable case of human-to-human transmission in a family cluster of cases. Analysis of specimens from this cluster is presently under way at a WHO collaborating laboratory to determine whether the virus has changed its genetic make-up. Heightened surveillance for further cases has provided no evidence that efficient and sustained human-to-human transmission is presently occurring in Thailand.

## *Hepatitis E — Sudan (Darfur)*

*Source: World Health Organization, CSR, Disease Outbreak News, 28 September 2004 (edited)*

From 22 May 2004 to 17 September 2004, a total of 6,861 cases and 87 deaths of suspected hepatitis E was reported from health clinics in the Greater Darfur region through the early warning alert and response system. The total number of reported cases per week continues to increase. West Darfur remains the most affected area.

There are ongoing control measures being implemented in three states. Health agencies have been working with WHO to scale up mass hygiene education programs, increase the availability of soap, dig new wells, and ensure effective chlorination of water bladders and wells. South Darfur has the poorest water and sanitation indicators. WHO is working with the Water and Environmental Sanitation Department and the State Ministry of Health to develop an Emergency Environmental Health plan for Internally Displaced Persons camps in South Darfur.

Existing resources remain insufficient to cover the basic water and sanitation needs of the displaced populations in Darfur. Additional efforts are still needed to reduce the number of new hepatitis E infections, and to prevent the spread of other waterborne diseases.

## *Melioidosis — Singapore*

*Source:* Straits Times*, Singapore 17 September 2004 (edited)*

A soil-borne bacterium, causing melioidosis, has killed 24 of the 79 people infected in Singapore so far in 2004, a three-fold rise in the death rate for the disease.

The overall death rate has jumped from 10 per cent of those infected in 2003—four deaths out of the 40—to about 30 per cent this year (2004). The high death rate has led the Ministry of Health to investigate whether the disease was being caused intentionally, since the bacterium (*Burkholderia pseudomallei*) is a known agent for potential biological warfare. Fortunately, it was not caused intentionally: different patients were seen to have different strains of the bacterium, said the director of medical services in the ministry.

The death rate from the disease between January and July 2004 was three times that of SARS. The overall death rate during that period was 40 per cent. In the same time period, SARS, by comparison, had a death rate of 13 per cent. However, 80 per cent of those who died had existing health problems, such as diabetes and hypertension, which are known to reduce immunity. More than 75 per cent of the victims were aged over 45 years.

The biggest outbreak—involving 19 people—occurred during heavy rains in March 2004, which brought the bacterium, usually buried in the soil, to the surface. The rains were the heaviest recorded since 1913 and this may have been a factor.

Doctors and scientists continue to be baffled by the disease, because so little is known about it. The disease has many different strains and often resists common antibiotics. There are no vaccines so far. There is still little evidence to show how the infection could stay dormant for years in some patients and appear quickly in others, and why some people who are exposed to the bacterium get infected, while others do not.

But, some of these questions are likely to be answered soon. Scientists from Britain's Sanger Institute announced that they had, for the first time, identified all the 6,000 genes of the bacterium which causes melioidosis. This 'genetic atlas,' said the institute's Dr Matthew Holden, will help scientists understand how it causes the disease and develop better diagnostic tools, drugs, and vaccines.

Melioidosis is caused through direct contact of bruised skin with the soil, leading to abscesses and conditions such as septicemia, or blood poisoning, in which people with low immunity are felled by bacteria that enter their bloodstream. The melioidosis bacterium lives mostly in clay soils, 25 to 45 cm deep, but monsoon rains can bring it to the surface.

## *Malaria — Papua New Guinea (Southern Highlands)*

*Source:* The National*, Port Moresby 21 August 2004 (edited)*

Health authorities have confirmed that the mysterious illness that killed over 90 people in the Kagua district of the Southern Highlands province early this year was malaria. Four medical teams were sent to Kagua to investigate the cause of the deaths and to administer drugs on a mass basis, after the deaths were reported in *The National* newspaper (in July 2004).

Michael Mombu, acting coordinator for Rural Health Services in Mendi, said yesterday that they discovered from the analysis of blood samples taken that the victims died of malaria. The teams visited affected villages two weeks ago and collected blood samples, while carrying out mass drug administration and spraying DDT, an insecticide that kills mosquitoes, at suspected breeding areas.

Mr Mombu said that because the malaria parasites developed resistance to chloroquine, the unavailability of alternate drugs may have led to the deaths. He said they were currently giving patients strong antimalarial drugs like quinine, artemether, and fansidar. He said the situation appears to have been brought under control, because no more deaths have been reported in the area.

He said that in other parts of the province, like Poroma in the Nipa/Kutubu electorate, where similar outbreaks were reported to have claimed 30 lives, a similar exercise was implemented to bring the situation under control.

ProMED-mail reported an outbreak of a fatal disease in the Southern Highlands of Papua New Guinea around 1 August 2004, and the Papua New Guinea authorities have now resolved the outbreak and determined it was due to malaria. Malaria remains a serious health problem in coastal and inland regions, affecting 15 provinces. It is endemic up to an altitude of 1,200–1,500 metres, where it becomes epidemic. Transmission is persistently high throughout the year, with *Plasmodium falciparum* causing an estimated 75 per cent of infections. Malaria is the third leading cause of hospital admissions and deaths. Among the contributing factors are shortages in health care personnel, breakdowns in drug supplies to rural areas, and lack of vector control. Only the higher mountainous areas and Port Moresby are malaria-free. Chloroquine resistance is regarded as widespread, and chloroquine should not be used for either treatment or prophylaxis. The overall risk of malaria in PNG was 1.7 per 1,000 population in 2000 and 14.3 per 1,000 in 2002 (WHO). The apparent increase may be due to enhanced reporting and not a true rise in incidence.

## *CJD (new variant) — UK: update 2004*

*Source: UK Department of Health, Monthly Creutzfeldt-Jakob Disease 2 August 2004 (edited)*

Deaths from definite vCJD (confirmed): 104

Deaths from probable vCJD (without neuropathological confirmation): 38

Deaths from probable vCJD (neuropathological confirmation pending): 0

Total number of deaths from definite or probable vCJD (as above): 142

Number of probable vCJD cases still alive: 5

Number of definite or probable vCJD (dead and alive): 147

These figures are unchanged from those published on 6 July 2004 for the preceding month, and the total number of vCJD cases (dead and alive) remains at 147. This is an additional indication that the vCJD epidemic in the UK may have passed its peak. Only one new case of vCJD has been confirmed during the past six months. This brings the total number of new cases of vCJD, during 2004, to four, compared to 18 deaths during 2003, and, a peak of 28 deaths in the year 2000. However, the identification of vCJD prions in the spleen of an elderly blood transfusion recipient, who, unlike all previous cases, was heterozygous at codon 129 of the PRNP gene, introduces new uncertainties into estimates of the possible course of the vCJD epidemic.

## *Salmonellosis, Tomatoes, convenience stores — USA (multistate)*

*Source:* The Tribune *Review 7 August 2004 (edited)*

The salmonellosis outbreak that sickened at least 416 people, in five states, might have been caused by four bacterial strains, an unusual occurrence, health officials said

Investigators suspect that all strains were found on contaminated Roma tomatoes served at Sheetz convenience stores, according to the Pennsylvania Department of Health and the CDC. Officials said they believe the tainted tomatoes have been removed from the market and are no longer infecting people.

*Salmonella* Javiana serotype has infected 324 of the 330 people with confirmed salmonellosis in Pennsylvania. Health officials in Ohio, West Virginia, Maryland, and Virginia have confirmed 86 salmonellosis cases linked to the outbreak, and, are investigating at least 51 others.

A rare, 2nd serotype, *S.* Anatum, is the only one found on the more than 260 samples of food that the Pennsylvania Department of Agriculture has tested. Four people, who ate at a Sheetz store, were sickened by that type, which was matched, genetically, to the *Salmonella* in an unopened bag of tomatoes taken from another Sheetz store. A fifth person infected with that strain is still being checked for a potential link.

The health update sent to medical professionals said 'another rare salmonella' type, 'Thompson' might also have been involved in the outbreak. About a dozen people who ate at a Sheetz store have been infected with that strain, but, are not yet counted among Pennsylvania's 330 confirmed cases. A fourth variant, *S.* Muenchen, also infected about a dozen people, potentially from tomatoes in early July 2004.

'Finding multiple strains of salmonella in a single outbreak is unusual, but it has happened several times', said Jennifer Morcone, CDC spokeswoman. Three strains of salmonella were found in beef jerky that sickened 93 people in New Mexico in 1995, and two salmonella strains were associated with orange juice that sickened people in Florida. 'An animal could carry more than a single strain and contaminate food growing in the field', the CDC spokeswoman said.

## *Poliomyelitis — India (Mumbai)*

*Source:* Times of India *4 July 2004 (edited)*

Just when the city thought it had eradicated polio, comes the news that an infant from a slum in Dindoshi, Malad east, has tested positive for the wild polio virus. 'This detection means Mumbai, which was polio-free for three years, will have to face another rigorous anti-polio drive for the next three years,' said an official. To get polio-free status, any area or country should not have a case for three consecutive years.

The detection comes on the eve of the special pulse polio drive on Sunday. About 1.1 million children in Mumbai will be given the oral polio vaccine as part of the fifth pulse polio drive since October. Usually, Mumbai has two or three rounds of polio drives per year.

According to the polio eradication website <http://www.polioeradication.org/casecount.asp>, in 2004, as of the week of 30 Jun, 339 cases of poliomyelitis associated with wild virus infection have been identified globally, 312 of which were reported from six endemic countries: Nigeria (259 cases), Niger (18), Pakistan (17), India (14), Afghanistan (3), and Egypt (1). As the article above mentions, interruption of wild virus transmission is defined as the absence of cases of polio associated with wild poliovirus for three consecutive years. While India as a country has not yet interrupted transmission, the number of annual reported cases has fallen significantly, and there are states within the country that have interrupted transmission with no wild poliovirus identified for three or more years.

Annual reported case counts for India are shown below, with this year to date appearing to have a major reduction in reported cases from prior years. <http://www.who.int/vaccines/casecount/afpextractnew.cfm>.

| Year | Number of confirmed cases of polio |
|---|---|
| 1996 | 1,005 |
| 1997 | 2,275 |
| 1998 | 4,322 |
| 1999 | 2,817 |
| 2000 | 265 |
| 2001 | 268 |
| 2002 | 1,600 |
| 2003 | 225 |
| 2004 | 14 (as of week of 30 Jun 2004) |

Within India, Uttar Pradesh and Bihar are endemic states where poliovirus transmission has not been interrupted. In addition, an outbreak of polio in Karnataka that began in mid-2003 has led to cases in Karnataka in 2004 and additional cases in Andhra Pradesh and Tamil Nadu (National Polio Surveillance Project <http://www.npspindia.org/>).

Mumbai is located in Maharastra state, which borders with Karnataka and Andhra Pradesh. It is a disappointing setback to have re-seeding of geographic areas that had previously interrupted wild poliovirus circulation.

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# CDI author index, 2004

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# CDI reviewers, 2004

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