

### 3. Vaccine preventable diseases

#### Diphtheria

Diphtheria is an acute bacterial toxin-mediated systemic disease caused by *Corynebacterium diphtheriae*. Infection remains localised to the throat or skin but disease is mainly due to local and systemic toxæmia. The major manifestation of pharyngeal diphtheria is a membranous inflammation of the upper respiratory tract, which may be extensive enough to cause laryngeal obstruction. Damage to other organs including the myocardium, nervous system and kidneys, caused by the organism's exotoxin, may complicate pharyngeal or cutaneous diphtheria.<sup>15,16</sup> Non-toxigenic *C. diphtheriae* usually causes mild throat or skin infection, which is occasionally complicated by invasive disease including endocarditis or arthritis.

##### Case definitions

##### Notifications

Isolation of toxigenic *Corynebacterium diphtheriae* and one of the following:

- pharyngitis *and/or* laryngitis (with or without membrane) or
- toxic (cardiac or neurological) symptoms.

##### Hospitalisations

The ICD-10-AM codes used to identify hospitalisations were: A36.0, pharyngeal diphtheria; A36.1, nasopharyngeal diphtheria; A36.2, laryngeal diphtheria; A36.8 + I41.0, diphtheritic myocarditis.

##### Deaths

The ICD-10 code A36 (diphtheria) was used to identify deaths.

#### Notifications, hospitalisations and deaths

There was one notification of, and no deaths due to, diphtheria during the review period. The single notification was of a case of cutaneous diphtheria in a middle-aged man reported from the Northern Territory but acquired in East Timor. For the two year period 2000/2001 and 2001/2002, there was only one hospitalisation meeting the above case definition, coded as pharyngeal diphtheria (A36.0) and primary diagnosis; it occurred in 2000/2001 and the patient was from New South Wales. There were another 53 hospitalisations coded as cutaneous (A36.3; n=35), other (A36.8; n=12) or unspecified (A36.9; n=6) diphtheria. Most were reported from the Northern Territory and South Australia (34/53; 64%).

#### Comment

Diphtheria has become rare in Australia. The cutaneous toxigenic case notified in 2001 is the first case reported since 1993. Cutaneous diphtheria is known to occur in the Northern Territory, where *C. diphtheriae* is endemic and non-toxigenic strains are regularly cultured from wound and nasopharyngeal swabs.<sup>17</sup> The criteria, other than isolation of toxigenic *C. diphtheriae*, that meant that the case met the current definition for notification, are not apparent in the available NNDSS data. It is possible that the only hospitalised pharyngeal case was not due to toxigenic *C. diphtheriae* as it was not notified and the length of stay was only one day.

From 2004, all toxigenic isolates, including those from cutaneous cases, will be notifiable. Future reports will require the inclusion of all ICD codes for diphtheria in hospitalisation data to be consistent with notification data. It is therefore noteworthy that there were 35 hospitalisations in the two year period (2000/2001 and 2001/2002) coded as cutaneous diphtheria and 18 hospitalisations coded as other or unspecified diphtheria.

The epidemiology of diphtheria in Australia is similar to that in other developed countries. Almost all recent cases in the United Kingdom, the United States of America (USA) and countries bordering the Newly Independent States of the Soviet Union, where a prolonged outbreak commenced in the early 1990s, have been associated with imported infections.<sup>18</sup> The United Kingdom has recently reported imported cases of cutaneous toxigenic diphtheria, which are important as they can cause respiratory and cutaneous infections in contacts.<sup>19</sup> Hence, as occurred in the notified case in 2001 who acquired disease in East Timor, there is still the possibility of an imported case occurring in Australia, particularly from developing countries.<sup>20</sup> It is therefore important for Australia to retain high levels of immunity through high vaccination coverage.

Analyses of the recent epidemiology of diphtheria suggest that adults are a susceptible group, with a shift in recent outbreaks from children to the adult age group.<sup>21</sup> International and Australian (NCIRS, unpublished data) serosurveys have shown that many adults in developed countries are now susceptible to diphtheria.<sup>22,23</sup> With 25 countries in Asia, South America, Africa and Europe reporting 10 or more cases of diphtheria to the World Health Organization (WHO) in 2002,<sup>24</sup> it is important that adult travellers to these areas have been immunised against diphtheria. Disruption of vaccination programs and reduction in vaccination coverage following the collapse of the Soviet Union resulted in over 50,000 cases and 4,000 deaths from diphtheria.<sup>25</sup> The experience of the Newly Independent States of the former Soviet Union illustrates the importance of maintaining high levels of vaccination coverage against diphtheria.

## Haemophilus influenzae type b (Hib) disease

*Haemophilus influenzae* is a fastidious Gram-negative bacterium which occurs in both encapsulated and unencapsulated forms. Before Hib vaccines became available one encapsulated serotype, type b (Hib), caused at least 95 per cent of infections due to *H. influenzae* in children.<sup>26,27</sup> Prior to the introduction of Hib vaccination the most common manifestation of invasive Hib disease was meningitis, with children aged less than 18 months most at risk.<sup>27-29</sup> Aboriginal children had a particularly high risk of Hib meningitis with rates among the highest recorded anywhere in the world.<sup>30</sup> Survivors of Hib meningitis commonly had neurological sequelae such as deafness and intellectual impairment. Epiglottitis was the other major category of infection, most often occurring in children over the age of 18 months. Less common manifestations of Hib disease include cellulitis, septic arthritis, pneumonia, pericarditis, osteomyelitis and septicaemia.

### Haemophilus influenzae type b disease

#### Case definitions

#### Notifications

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

- isolation of *Haemophilus influenzae* type b from blood; *or*
- detection of Hib antigen (in a clinically compatible case); *or*
- detection of Gram-negative bacteria where the organism fails to grow in a clinical case.

**or**

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

**Note:** In 2001 Victoria used the above case definition while in 2002 the surveillance case definition was changed to exclude clinical criteria and only include cases where Hib was laboratory confirmed.<sup>31</sup>

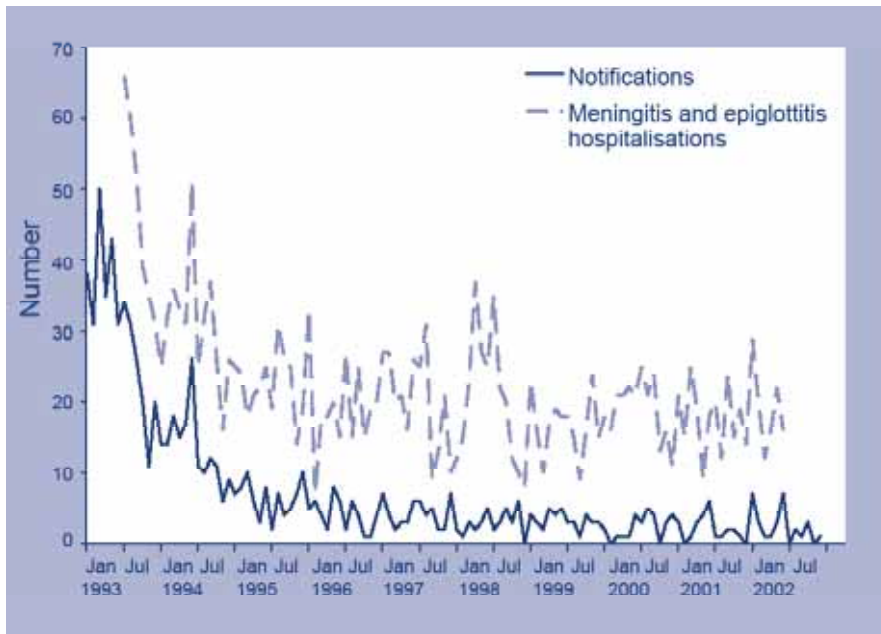
#### Hospitalisations and deaths

There were no ICD-10-AM/ICD-10 codes which specified Hib as a causative organism. Two ICD-10-AM/ICD-10 codes were used to identify presumed Hib cases: G00.0 (*Haemophilus meningitis*), and J05.1 (acute epiglottitis). The ICD-10-AM/ICD-10 codes for *H. influenzae* pneumonia, *H. influenzae* septicaemia and *H. influenzae* infection were not included as these were thought to be less specific for invasive *H. influenzae* type b disease.

## Secular trends

During the two years from 2001 to 2002 there were a total of 53 Hib notifications. The average annual notification rate has halved from the previous review period, 1999 to 2000, to 0.1 per 100,000 population (Table 3). A median of 2 cases (range 0–7) were notified per month (Figure 1). There were 440 hospitalisations (average annual rate 1.1 per 100,000) for presumed Hib disease, with a median of 18 cases (range 4–29) hospitalised per month. Despite a decrease in notification rates, the average annual hospitalisation rate remains fairly constant and the proportion due to acute epiglottitis has slightly increased during this review period. Acute epiglottitis accounted for 386 (88%) of these hospitalisations and meningitis for 54 (12%). Hospitalisations occurred throughout the year but were slightly more frequent during the winter months.

**Figure 1. *H. influenzae* type b (Hib) notifications and presumed Hib hospitalisations for all ages,\* Australia, 1993 to 2002,† by month of onset or admission**



\* Hospitalisations for *H. influenzae* meningitis and acute epiglottitis.

† Notifications where the month of onset was between January 1993 and December 2002; hospitalisations where the month of admission was between 1 July 1993 and 30 June 2002.

**Severe morbidity and mortality**

At all ages, the number and rate of hospitalisations were higher than the number and rate of notifications (Table 3). The principal diagnosis was *H. influenzae* meningitis or acute epiglottitis in 344 (78%) of the hospitalisations. Over the review period a total of 2,683 hospital bed days (average 1,342 days per year) was recorded for patients with presumed Hib. The median length of stay for meningitis hospitalisations was longer than for epiglottitis hospitalisations in all age groups. In the two years 2001 to 2002, *H. influenzae* meningitis was recorded as the underlying cause of death for one child (less than 15 years old) and acute epiglottitis for two patients (Table 3).

**Table 3. *H. influenzae* type b (Hib) notifications, presumed Hib hospitalisations\* and deaths, Australia, 2000 to 2002,† by age group**

Age group (years)	Notifications 2 years (2001–2002)		Hospitalisations 2 years (July 2000–June 2002)				LOS‡ per admission (days)	Deaths 2 years (2001–2002)	
	n	Rate§	n	(**)	Rate§	(**)	Median	n	Rate§
0–4	24	0.9	60	(51)	2.3	(2.0)	5	1	0.0
5–14	9	0.2	31	(27)	0.6	(0.5)	1	1	0.0
15–24	2	0.0	34	(30)	0.6	(0.6)	3	0	–
25–59	10	0.1	215	(164)	1.1	(0.9)	7	1	0.0
60+	8	0.1	100	(72)	1.6	(1.1)	9	0	–
All ages	53	0.1	440	(344)	1.1	(0.9)	5	3	0.0

\* Hospitalisations for *H. influenzae* meningitis and acute epiglottitis.

† Notifications where the month of onset was between January 2001 and December 2002; hospitalisations where the month of separation was between 1 July 2000 and 30 June 2002; deaths where the date of death was recorded between 2001 and 2002.

‡ LOS = length of stay in hospital.

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

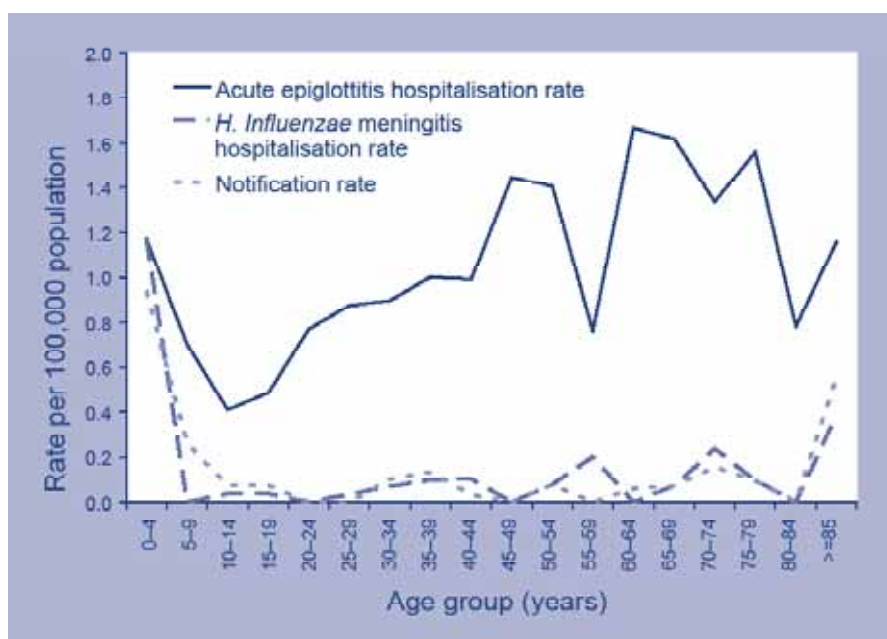
|| Includes cases with unknown ages.

\*\* Principal diagnosis (hospitalisations).

## Age and sex

The most significant reduction from the previous review period in both notification and hospitalisation rates is in the 0–4 year age group, while rates have remained relatively stable in other age groups. Hospitalisations for presumed Hib disease were higher in males than females, with a male:female ratio of 1.7:1, while Hib notifications were more common in females, with a male to female ratio of 0.7:1. *H. influenzae* related deaths occurred in two males and one female. In children aged 0–4 years, *H. influenzae* meningitis and acute epiglottitis hospitalisations were equally common (30 cases of each). Overall, children aged 0–4 years accounted for 45 per cent (24/53) of all notifications, 56 per cent (30/54) of all meningitis hospitalisations and 33 per cent (1/3) of all deaths, but only eight per cent (30/386) of all epiglottitis hospitalisations (Figure 2). The highest epiglottitis hospitalisation rates were in those over 60 years of age. The age-specific notification rate closely matched the age-specific *H. influenzae* meningitis hospitalisation rate.

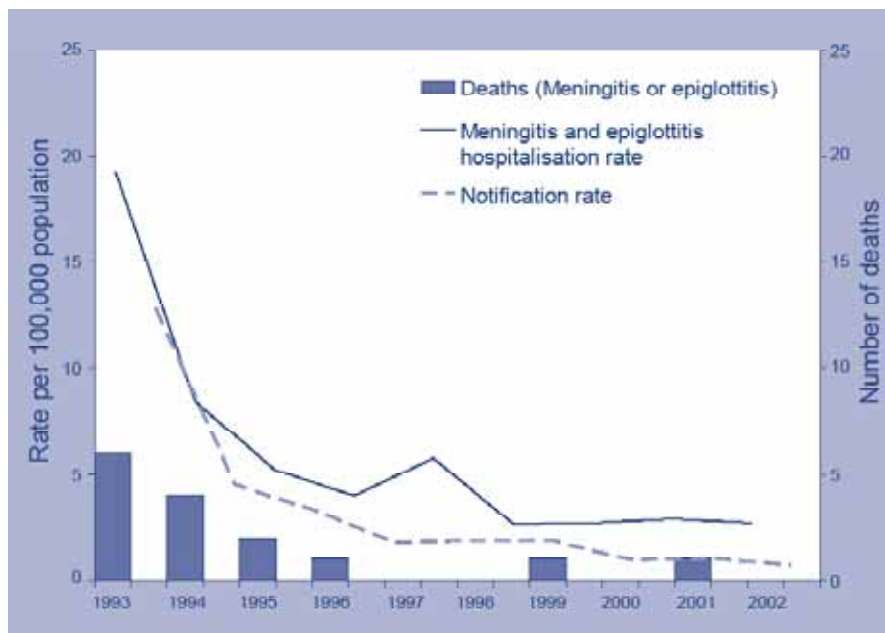
**Figure 2. *H. influenzae* type b (Hib) notification and presumed Hib hospitalisation rates, Australia, 2000 to 2002,\* by age at admission**



\* Notifications where the month of onset was between January 2001 and December 2002; hospitalisations where the month of separation was between 1 July 2000 and 30 June 2002.

Since 1993, all measures of invasive Hib disease in children aged 0–4 years have fallen (Figure 3). Average annual notification rates have decreased 25 per cent from 1.2 per 100,000 population in 1999/2000 to 0.9 per 100,000 population in 2001/2002. Despite a small rise in 1997/1998, meningitis and epiglottitis hospitalisation rates fell from a rate of 20.6 per 100,000 in 1993/1994 to 2.9 per 100,000 in 1999/2000 and have fallen 20 per cent further to 2.3 per 100,000 in 2001/2002. Six deaths were recorded in this age group in 1993 and none in 2002.

**Figure 3.** *H. influenzae* type b (Hib) notification and presumed Hib hospitalisation\* rates and numbers of deaths for children aged 0–4 years, Australia, 1993 to 2002†



\* Hospitalisations for *H. influenzae* meningitis and acute epiglottitis.

† Notifications with onset dates between July 1993 and June 2002; hospitalisations with separations between July 1993 and June 2002; deaths reported between 1993 and 2002.

## Geographical variation

There was little variation in notification and hospitalisation rates between the States and Territories. Tasmania and the Australian Capital Territory had no notifications during the review period. The Northern Territory continued to have higher notification and hospitalisation rates for all ages than other jurisdictions, but the absolute number of cases was small (Appendices 2 and 3).

## Comment

The dramatic reduction in the incidence of invasive Hib disease seen following the introduction of conjugated vaccines in 1993 has been maintained. In 2000 Australia instituted a new Hib immunisation schedule for all children, comprising PRP-OMP vaccine at two and four months of age with a booster at 12 months of age. This meant that the primary immunisation schedule was completed earlier, at four months rather than at six months of age. Following this change there has been a 20–25 per cent reduction in the average annual Hib notification and hospitalisation rates in 0–4 year olds between this review period and the previous period. Hib remains a rare disease in children and deaths are very rare.

Hib notifications are very specific and may underestimate Hib cases. There is evidence from the United Kingdom that enthusiasm for reporting of Hib disease may decline following the successful implementation of an immunisation program.<sup>32</sup> However, it is likely that notifications, because they are usually linked to laboratory identification of Hib, more closely represent the true incidence of Hib disease than hospitalisations. In 2002, Victoria changed its Hib surveillance case definition to include only cases where Hib was isolated from a normally sterile site confirmed at an approved reference laboratory or where Hib was detected in cerebrospinal fluid when other laboratory parameters was consistent with meningitis.<sup>31</sup> The case definition for national notification has recently been modified to include only those with laboratory definitive evidence as outlined above.<sup>33</sup> This new case definition is being implemented nationally during 2004. Enhanced Hib meningitis surveillance in Far North Queensland has detected only one case in a child under five years of age in the 10 years 1994–2003, following the implementation of the National Hib Immunisation Program.<sup>34</sup>

Epiglottitis hospitalisations are an especially important example of these problems. Since the introduction of Hib vaccination, the assumption that almost all hospitalisations for acute epiglottitis and *H. influenzae* meningitis are due to Hib infection is no longer reliable.<sup>35</sup> The highest epiglottitis hospitalisation rate in Australia is in those over 60 years of age, and has shown little reduction following the introduction of childhood Hib immunisation. In addition, epiglottitis hospitalisation rates in adults have been reported to be increasing overseas.<sup>36</sup> However, most cases of epiglottitis in adults have no identifiable cause or may be due to organisms other than *H. influenzae*.<sup>37,38</sup> Epiglottitis hospitalisation data also overestimate incidence if cases are counted twice when a patient is transferred between hospitals. Epiglottitis hospitalisations in Sydney during 1998 to 2000 were reviewed by the National Centre for Immunisation Research and Surveillance. The review found no cases caused by Hib, one case due to *Streptococcus pneumoniae* and 32 per cent incorrectly coded as epiglottitis.<sup>39</sup> Therefore the use of epiglottitis hospitalisations as one of the markers of Hib disease is probably no longer appropriate.

The surveillance data presented in this report suggest that invasive Hib disease remains rare. It is therefore important to have laboratory confirmation of all suspected cases, ideally by polymerase chain reaction (PCR) in a reference laboratory. This is particularly important in an era of widespread Hib vaccination when Hib vaccine failures have been reported internationally. In the United Kingdom there has been a recent increase in the incidence of invasive Hib disease, including Hib epiglottitis, predominantly in appropriately vaccinated children, emphasising the importance of ongoing surveillance even when disease rates have become very low.<sup>40-42</sup> In contrast to the United Kingdom, Australia's Hib vaccine schedule includes a booster in the second year of life and there has been a decrease rather than an increase in Hib disease, as documented here.

## Hepatitis A

Infection with the hepatitis A virus (HAV), a picorna virus, may produce a wide range of symptoms from malaise and diarrhoea to acute hepatitis with jaundice to fulminant liver failure. Onset of clinical symptoms is usually abrupt with fever, anorexia, malaise, nausea and abdominal discomfort followed by jaundice. The single most important factor in determining the clinical presentation and outcome of HAV infection is age. Over 90 per cent of infections acquired before the age of five years are silent, with the proportion of infected individuals showing symptoms increasing to 9 per cent in adults.<sup>15,16</sup>

### *Case definitions*

#### **Notifications**

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

**or**

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

#### **Hospitalisations and deaths**

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

## Secular trends

There were 909 hepatitis A notifications in 2001 to 2002 (average annual notification rate 2.3 per 100,000) (Table 4). A median of 37.5 cases (range 17–59) were notified per month. There were 671 hospitalisations (average annual hospitalisation rate 1.7 per 100,000) with a median of 28 admissions (range 16–41) per month.

Notification and hospitalisation rates declined in 2001 and again in 2002 compared with previous years (Figure 4). There was no apparent seasonality in notifications or hospitalisations.

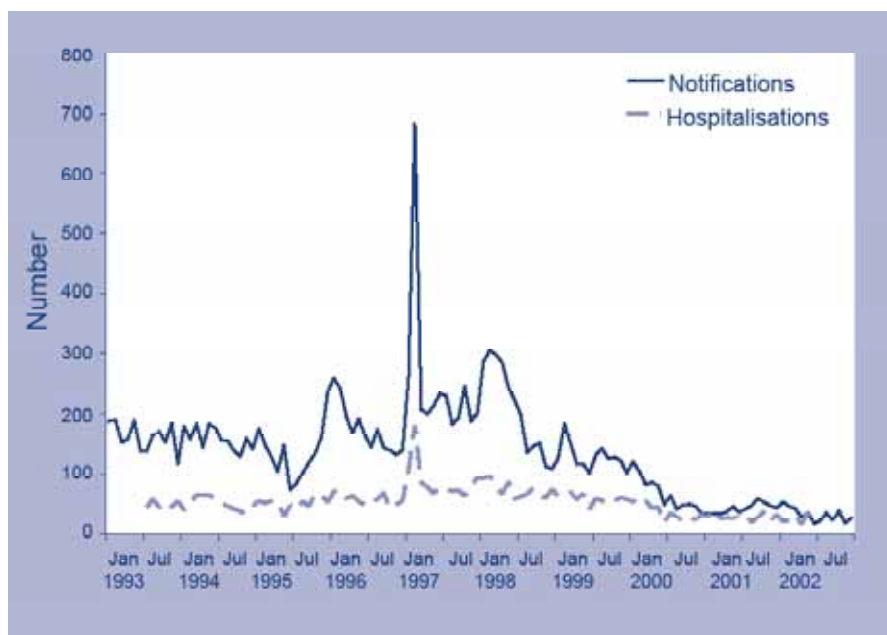
## Severe morbidity and mortality

There were 3,930 hospital bed days (average 1,965 per year) recorded for patients with an ICD-10-AM code for hepatitis A. Hepatitis A was the principal diagnosis in 42 per cent of these hospitalisations (282 cases, average annual rate 0.7 per 100,000). The median length of stay was longer for those aged 60 years or more than for younger age groups (Table 4). In 2001 to 2002, hepatitis A was recorded as the underlying cause of two deaths (0.01 per 100,000). One death occurred in the 60-year and over age group and one in Western Australia in an Indigenous child aged 0–4 years.

Hepatitis A with hepatic coma (ICD-10-AM B15.0) was recorded for five hospital admissions, all aged five years and over, and for the child aged 0–4 years who died.



**Figure 4. Hepatitis A notifications and hospitalisations, Australia, 1993 to 2002,\* by month of onset or admission**



\* Notifications where the month of onset was between January 1993 and December 2002; hospitalisations where the month of admission was between 1 July 1993 and 30 June 2002.

**Table 4. Hepatitis A notifications, hospitalisations and deaths, Australia, 2000 to 2002,\* by age group**

Age group (years)	Notifications 2 years (2001–2002)		Hospitalisations 2 years (July 2000–June 2002)				LOS <sup>†</sup> per admission (days)	Deaths 2 years (2001–2002)	
	n	Rate <sup>‡</sup>	n	(  )	Rate <sup>‡</sup>	(  )	Median	n	Rate <sup>‡</sup>
0–4	56	2.2	18	(11)	0.7	(0.4)	3.5	1	0.0
5–14	107	2.0	27	(23)	0.5	(0.4)	2.0	0	–
15–24	153	2.9	84	(58)	1.6	(1.1)	2.0	0	–
25–59	528	2.8	396	(156)	2.1	(0.8)	3.0	0	–
60+	65	1.0	146	(34)	2.3	(0.5)	6.0	1	0.0
All ages <sup>§</sup>	909	2.3	671	(282)	1.7	(0.7)	3.0	2	0.0

\* Notifications where the month of onset was between January 2001 and December 2002; hospitalisations where the month of separation was between 1 July 2000 and 30 June 2002; deaths where the date of death was recorded between 2001 and 2002.

† LOS = length of stay in hospital.

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

§ Includes cases with unknown ages.

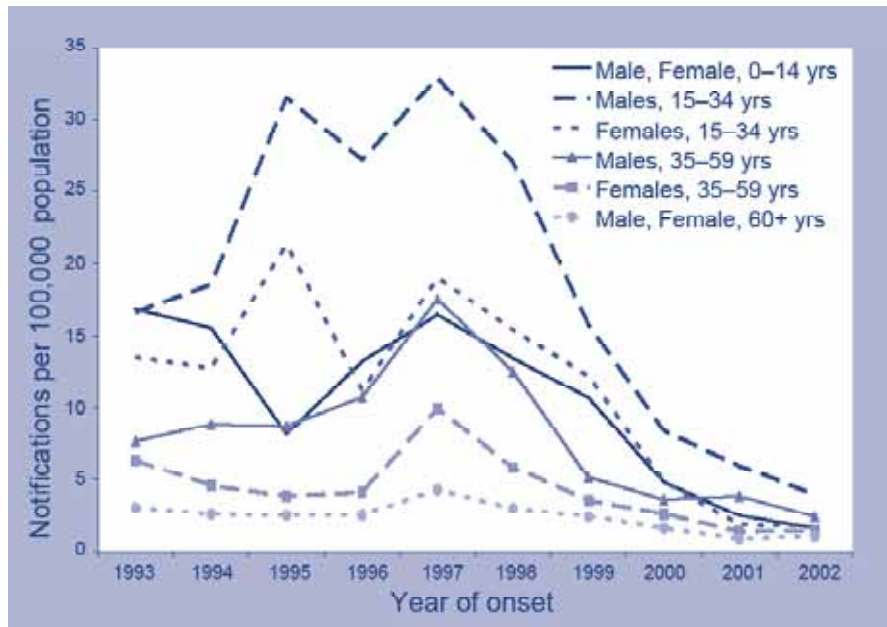
|| Principal diagnosis.

### Age and sex distribution

The overall male to female ratio was 2.1:1 for notifications and 1.1:1 for hospitalisations. Both deaths were in females. The sex ratio differed between age groups for notifications and hospitalisations. It was highest for notifications aged 15–34 (2.7:1) years and for hospitalisations among adults aged 35–59 years and children 0–14 years (1.2:1 for both age groups).

Notification and hospitalisation rates for all age and sex groups declined in the two year review period compared with previous years (Figures 5 and 6). The highest notification rate occurred among males aged 15–34 years (average annual rate, 4.9 per 100,000), while the highest hospitalisation rates occurred among males aged 34–59 years and 60 years and over (average annual rates of 2.3 per 100,000 and 2.2 per 100,000, respectively).

**Figure 5. Hepatitis A notification rates, Australia, 1993 to 2002,\* by age group, sex and year of onset**



\* Notifications where the month of onset was between January 1993 and December 2002.

### Geographical distribution

Notification and hospitalisation rates varied by jurisdiction (Appendices 2 and 3). Overall, the highest rates occurred in the Northern Territory (average annual rates 21.2 per 100,000 for notifications and 6.9 per 100,000 for hospitalisations). Notification rates were lower in all jurisdictions except the Northern Territory and Tasmania in 2002 compared with 2001 (Appendix 2). Hospitalisation rates increased in the Northern Territory and New South Wales in 2002 compared with 2001 (Appendix 3).

### Comment

In Australia, as in other industrialised countries, hepatitis A occurs sporadically with epidemic peaks related to point-source outbreaks and large community-wide outbreaks which occur at greater than five year intervals. The overall patterns are evident in hepatitis A notification and hospitalisation rates over the 10 years 1993–2002. There was a decline in rates in 2001–2002 following peaks in total hepatitis cases during the 1990s due to a large point-source epidemic associated with consumption of contaminated oysters in February 1997<sup>43</sup> and large community-wide epidemics mainly among men who have sex with men and illicit drug users.<sup>44–47</sup> The decline in hepatitis A cases following the large outbreaks during the 1990s is likely to represent an inter-epidemic period, due to a reduction in the number of people in high risk groups who were susceptible to hepatitis A virus infection, rather than to changes in vaccination policy or coverage.

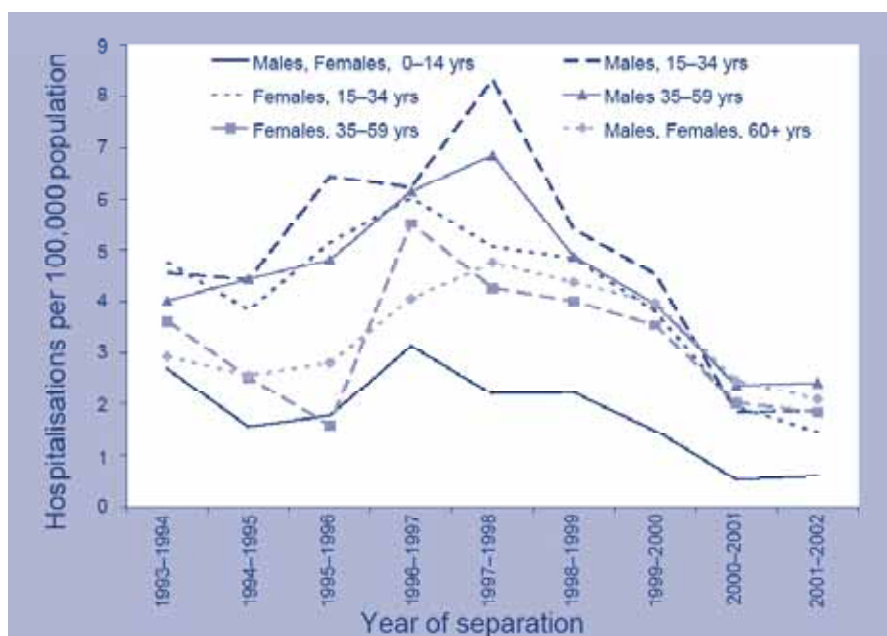
In Australia, the groups most at risk of acquiring and transmitting hepatitis A are travellers to countries where hepatitis A is endemic, children attending child care and preschool, people who use illicit drugs, men who have sex with men, sewage workers, food handlers and Indigenous Australians.<sup>48–50</sup>

The epidemiology of hepatitis A differs significantly for the Indigenous population, where it remains endemic, compared with the non-Indigenous population. Among non-Indigenous Australians, like other developed countries, adolescents and young adults have a lower seroprevalence than older adults.<sup>46</sup> In contrast, hospitalisation and notification rates are higher among Indigenous Australians, with rates in Indigenous children

aged less than five years over 20 times higher than those of non-Indigenous children in the same age group.<sup>50</sup> During 1999–2002 there were three deaths due to hepatitis A among children aged less than five years; all were Indigenous.<sup>45,50</sup>

Hepatitis A vaccines are effective in preventing disease in individuals<sup>49</sup> and in controlling outbreaks in some settings.<sup>49,51</sup> In Australia, vaccination is recommended for selected at-risk groups and occupations. In 1999 an immunisation program commenced for Indigenous children aged 18 months to 6 years living in north Queensland. Data indicate that this program has had a significant impact on reducing hepatitis A across the community.<sup>52</sup> In the United States of America, hepatitis A cases have decreased following the introduction of hepatitis A vaccine into the routine vaccination schedule for States with high hepatitis A notification rates in 1999.<sup>53</sup> The data presented here show that hepatitis A contributes to infectious disease morbidity and mortality in Australia and may warrant further general or targeted public health intervention.<sup>48</sup>

**Figure 6. Hepatitis A hospitalisation rates, Australia, 1993 to 2002,\* by age group, sex and year of separation**



\* Hospitalisations where the month of separation was between 1 July 1993 and 30 June 2002.

## Acute hepatitis B

Acute infection with hepatitis B virus (HBV), a hepadnavirus, may produce a range of conditions from subclinical infection to acute hepatitis with jaundice and, rarely, fulminant hepatitis. Only a small proportion of HBV infections are clinically recognised, with less than 10 per cent of children and 30–50 per cent of adults experiencing jaundice.<sup>16,54</sup> Onset of illness, when it occurs, is usually insidious with anorexia, vague abdominal discomfort, nausea and vomiting, sometimes arthralgia and rash, often progressing to jaundice. The main burden of disease is related to chronic HBV infection. The risk of an acute infection becoming chronic varies inversely with age: chronic HBV infection occurs in about 90 per cent of infants infected at birth, 20–50 per cent of children infected at 1–5 years of age, and about 1–10 per cent of persons infected as older children and adults.<sup>16</sup> Of people chronically infected with HBV, 15–40 per cent develop cirrhosis of the liver and/or hepatocellular carcinoma.<sup>55,56</sup>

HBV transmission occurs by percutaneous or permucosal exposure to infective body fluids such as blood, semen, vaginal secretions and any other body fluid containing blood.<sup>16</sup> Major modes of transmission include sexual or household contact with an infected person, perinatal transmission from mother to infant, injecting drug use and nosocomial exposure of health care workers.<sup>16</sup> In countries with a high burden of hepatitis B, such as Taiwan, universal hepatitis B vaccination programs have had a profound impact on the incidence of chronic infection and hepatocellular carcinoma.<sup>57</sup> From 1988 to 1999, a targeted hepatitis B vaccination program was recommended in Australia. A publicly funded universal infant hepatitis B vaccination program commenced in Australia in 2000, as part of global efforts to eradicate hepatitis B.

The summary below is restricted to acute hepatitis B. Reviews of the burden of disease related to chronic hepatitis B infection in Australia have been published elsewhere.<sup>56,58,59</sup>

### *Case definitions*

#### **Notifications**

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

a) hepatitis B core antibody (Anti-HBc) IgM

**or**

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

#### **Hospitalisations**

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

As in the previous report, hospitalisations were included only where the relevant ICD code was the principal diagnosis. Acute hepatitis B was the principal diagnosis in 34.5% of all hospitalisations with acute hepatitis B. Although this proportion has markedly increased compared to previous analyses of hepatitis B hospitalisations, it remains lower than for the other diseases.<sup>2</sup>

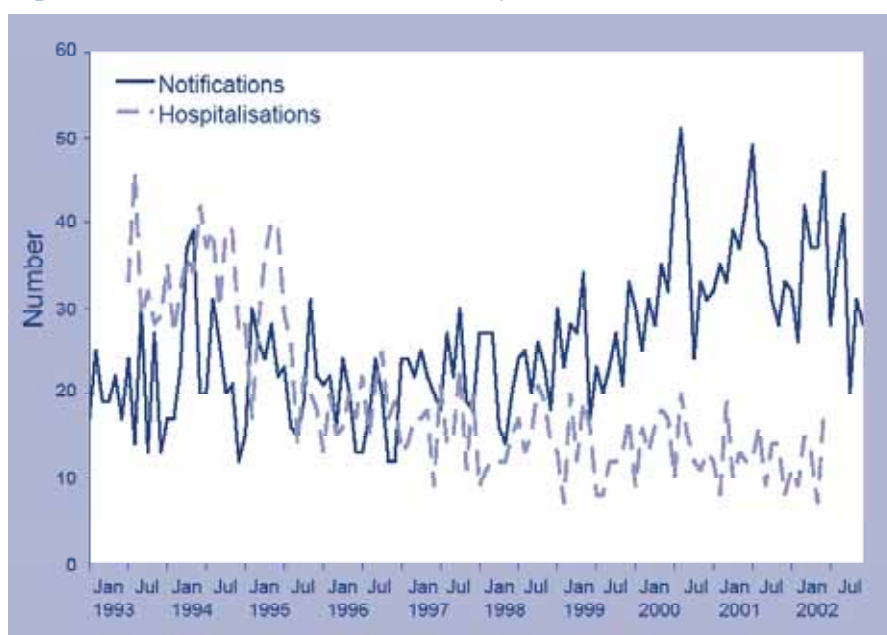
#### **Deaths**

The ICD-10 code B16 (acute hepatitis B) was used to select deaths from acute hepatitis B

## Secular trends

In the two years from January 2001 to December 2002, there were 837 notifications (average annual rate 2.1 per 100,000) with a median of 35 notifications per month (range 20–49) (Figure 7, Table 5). The peak notification rate was in the age group 15–24 years (average annual rate 5.3 per 100,000). Between 2000/2001 and 2001/2002 there were 305 hospitalisations with a principal diagnosis of acute hepatitis B (average annual rate 0.8 per 100,000) with a median of 12 hospitalisations per month (range 7–20). Ninety-eight per cent (300/305) of these hospitalisations were coded as ‘acute hepatitis B without delta-agent and without hepatic coma’ (ICD-10-AM B16.9). While nationally there has been an upward trend for notifications, particularly since 1999, hospitalisations have generally declined every year from 1993/1994 to 1998/1999 with stabilisation of the national hospitalisation rate at about 0.8 per 100,000 since 1999/2000. The national notification rate peaked in 2001 at 2.2 per 100,000, with more notifications of acute hepatitis B recorded (n=434) than for any other year since surveillance began in most States and Territories in 1993 (Appendices 2 and 3).

**Figure 7. Acute hepatitis B notifications, and hospitalisations with a principal diagnosis of acute hepatitis B,\* Australia, 1993 to 2002,† by month of onset or admission**



\* Prior to July 1994, hospitalisations for acute hepatitis B could not be distinguished from hospitalisations for chronic hepatitis B infection.

† Notifications where the month of onset was between January 1993 and December 2002, hospitalisations where the month of admission was between 1 July 1993 and 30 June 2002. Note that the number of jurisdictions notifying acute hepatitis B increased over the review period until 1996 when acute hepatitis B became notifiable in all States and Territories. The Australian Capital Territory did not report in 1994 and Western Australia did not report in 1994 and 1995.

## Severe morbidity and mortality

For patients with a principal diagnosis of acute hepatitis B, 1,516 hospital bed days (866 and 650 bed days in 2000/2001 and 2001/2002, respectively) were recorded. The median length of stay was four days, with longer stays for adults aged 60 years and over (Table 5). There were 20 deaths from acute hepatitis B recorded in the two years 2001 to 2002, 17 in males and three in females. None of the deaths occurred in children aged less than 15 years while 70 per cent (14/20) occurred in individuals aged 15–59 years; eight of these 14 deaths were in people aged 40 years and over, and seven out of eight were males (data not shown). There was only one case of hepatic coma recorded among hospitalisations with a principal diagnosis of acute hepatitis B (Table 6).

**Table 5. Acute hepatitis B notifications, hospitalisations and deaths, Australia, 2000 to 2002,\* by age group**

Age group (years)	Notifications 2 years (2001–2002)		Hospitalisations† 2 years (July 2000–June 2002)		LOS‡ per admission (days)	Deaths 2 years (2001–2002)	
	n	Rate§	n	Rate§	Median	n	Rate§
0–4	2	0.1	0	–	–	0	–
5–14	13	0.2	6	0.1	1	0	–
15–24	283	5.3	63	1.2	3	2	0.0
25–59	512	2.7	211	1.1	4	12	0.1
60+	24	0.4	25	0.4	5	6	0.1
All ages	837	2.1	305	0.8	4	20	0.1

\* Notifications where the month of onset was between January 2001 and December 2002; hospitalisations where the month of separation was between 1 July 2000 and 30 June 2002; deaths where the date of death was recorded between 2001 and 2002.

† Hospitalisations with a principal diagnosis of acute hepatitis B.

‡ LOS = length of stay for hospitalisations with a principal diagnosis of acute hepatitis B.

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

|| Includes cases with unknown ages.

**Table 6. Hepatic coma in hospitalised cases with principal diagnosis of acute hepatitis B, Australia, 2000–2002,\* by age group**

Age group (years)	Hepatic coma†	
	n	% total
0–4	–	–
5–14	0	0.0
15–24	0	0.0
25–59	1	0.5
60+	0	0.0
All ages	1	0.3

\* Measured using National Hospital Morbidity data where the month of hospital separation was between 1 July 2000 and 30 June 2002.

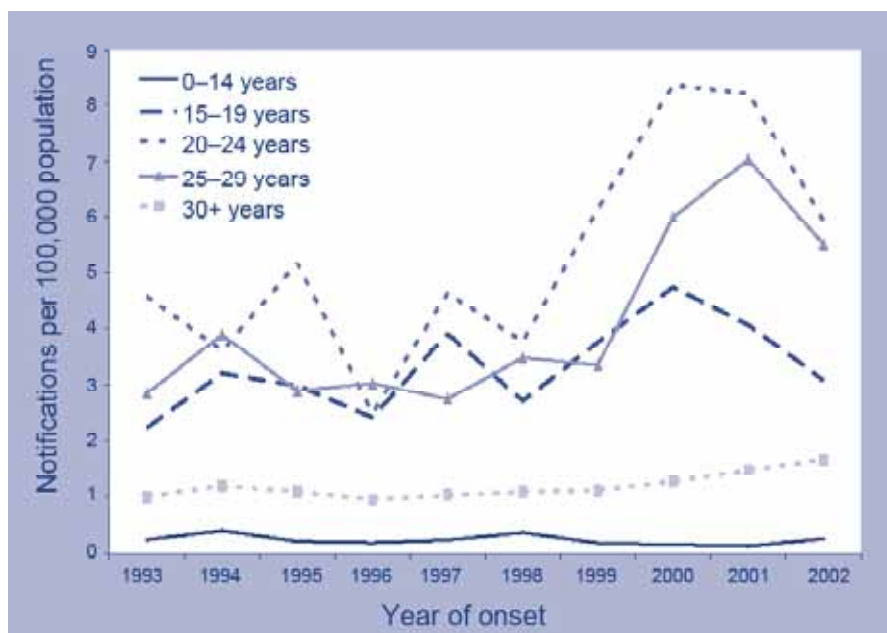
† ICD-10-AM codes B16.0 and B16.2.

### Age and sex distribution

Over the years, notification rates have consistently been highest in young adults aged 15–19 years, 20–24 years and 25–29 years (Figure 8). While there was an upward trend in the 15–29 year old notification rates between 1998 and 2000, these rates seemed to peak around 2000–2001. Notification rates have remained fairly stable in the other age groups from 1993 to 2002. As in previous years, there were more male than female notifications in almost all age groups in 2001 and 2002, with an overall male:female ratio of 1.9:1.

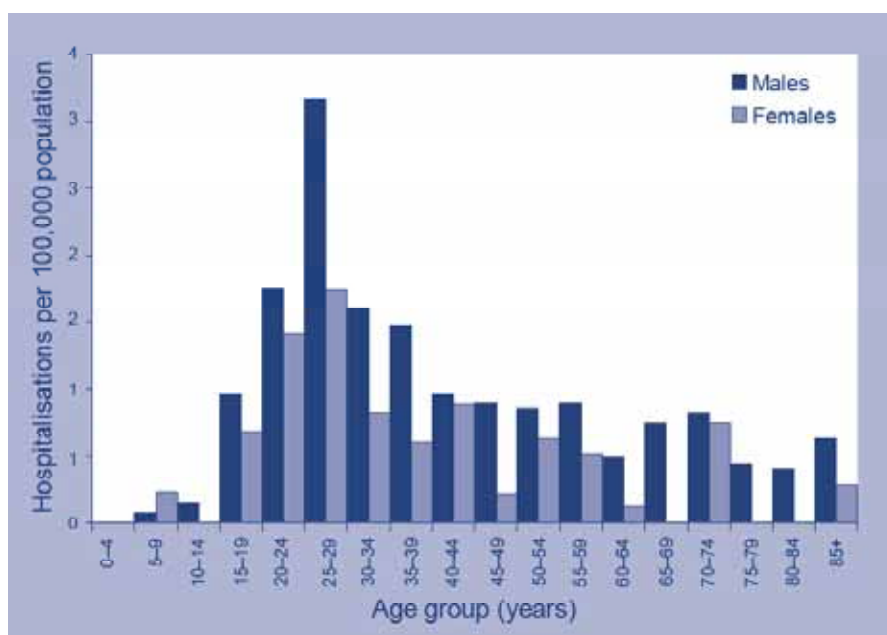
During 2000/2001 and 2001/2002, rates for hospitalisations with a principal diagnosis of acute hepatitis B were the highest in adults aged 25–29 years (2.5 per 100,000) and 20–24 years (1.6 per 100,000) (Figure 9). Like notifications and as in previous years, hospitalisations occurred predominantly in males with an overall male:female ratio of 1.7:1 for the years 2000/2001 and 2001/2002.

Figure 8. Acute hepatitis B notification rates, Australia, 1993 to 2002,\* by age group



\* Notifications where the month of onset was between January 1993 and December 2002.

Figure 9. Acute hepatitis B hospitalisation rates, Australia, 2000/2001 to 2001/2002,\* by age group and sex



\* Hospitalisations where the principal diagnosis was acute hepatitis B and the month of separation was between 1 July 2000 and 30 June 2002.

### Geographical distribution

For 2001 and 2002, Victoria recorded the highest number of notifications (n=383; 46%), followed by New South Wales (n=177; 21%). The Northern Territory had the highest average annual notification rate at 6.3 per 100,000. Tasmania and Victoria were next at 4.2 and 4.0 per 100,000 respectively while rates were 2.0 per 100,000 or less in the other jurisdictions (Appendix 2).

For the same period, Victoria also had the highest number of hospitalisations (n=126; 41%) followed by New South Wales (n=82; 27%). As for notifications, the Northern Territory had the highest average annual hospitalisation rate at 1.8 per 100,000, with Victoria and Tasmania at 1.3 and 1.2 per 100,000 respectively and rates in other jurisdictions were 1.0 per 100,000 or less (Appendix 3).

### Comment

Overall there were more hospitalisations than would be expected given the number of notifications and the epidemiology of the disease. It is likely that this is caused by a combination of (a) misclassification of hospitalisations due to chronic infection as acute infection and (b) under-reporting of notifications.

At both national and jurisdictional levels, notifications have generally increased since 1993 while hospitalisations have decreased. The decline in hospitalisations is likely to be a reflection of changes to coding practices. Up to 1997/1998, the four ICD-9-CM codes used to select hospitalisations included 'acute or unspecified' hepatitis B. In 1998/1999, ICD-10-AM, which can differentiate between acute and unspecified hepatitis B, replaced ICD-9-CM, although some States and Territories continued to use ICD-9-CM in 1998/1999. These coding changes, more specific for acute HBV disease, are therefore likely to have been responsible for the initial reduction in hospitalisation rates from 1998/1999, followed by their stabilisation, observed nationally since 1999/2000, once changes were established. Improved coding practices are also likely to be responsible for the significant decrease in deaths related to acute hepatitis B from an average 50 per year for the period 1993–1997 to about 10 per year in 2001 and 2002. Misclassification is likely to still be a problem, as only one of the 20 deaths recorded for the two years 2001 and 2002 had acute hepatitis B with hepatic coma (B16.0 or B16.2) as the underlying cause of death, when it would be expected to be more frequent for acute hepatitis B deaths.

The increase in acute hepatitis B national notification rate observed between 1998 and 2001 is largely confined to young adults aged 15–29 years. This selective increase could represent a real increase in new infections; it could also be due to increased testing in this age group rather than improved reporting, which should affect all age groups. The national notification rate appears to have peaked in 2001 at 2.2 per 100,000, mirrored by similar profiles for 15–29 year old incidence rates. A consolidation of these downwards trends in the coming years would reflect the impact of the national adolescent immunisation program started in Australia in 1997.<sup>60</sup>

The variation in notification rates between States and Territories may be due to differences in surveillance methods, but could also be a real difference resulting from differences in the proportion of the population at increased risk of hepatitis B infection. The Australian Capital Territory and Victoria instituted enhanced surveillance of acute hepatitis B in January 2000 and July 2001 respectively, and this can be expected to influence notification rates in these jurisdictions.

In the Northern Territory hepatitis B vaccine has been routinely given at birth to Aboriginal infants since 1988, and to all infants since August 1990. In the rest of Australia, at-risk infants have been given hepatitis B vaccine since 1987 (except in South Australia, which began in 1996) while universal infant hepatitis B immunisation was introduced in May 2000. The effect of this policy on the reported incidence of acute hepatitis B would not be expected to become apparent until the first cohort of vaccinated infants reaches adolescence (around 2015).

Acute hepatitis B is only one measure of the burden of disease caused by HBV. The current prevalence of chronic HBV infection reflects historical transmission patterns and in the longer term the impact of immunisation policies will be reflected in trends in chronic infection and its complications, such as liver cirrhosis and hepatocellular carcinoma.<sup>58,61</sup> The data presented here suggest that, in the interim period before the impact of adolescent vaccination is seen, greater attention to prevention of hepatitis B among young adults is warranted.



## Influenza

Influenza A and B viruses can cause major epidemics of respiratory disease. Often indistinguishable on a clinical basis from disease caused by other respiratory viruses, symptoms can include abrupt onset of fever, myalgia, headache, sore throat and acute cough. Influenza epidemics usually occur during the winter months in temperate climates, causing an increase in hospitalisations for pneumonia and exacerbation of chronic diseases and also resulting in increased mortality, particularly among the elderly and those with chronic diseases. In tropical climates influenza infection is often observed to be endemic with two annual peaks, as illustrated in the Northern Territory.<sup>62</sup> Pandemics of influenza are caused by major antigenic shift, but antigenic drift occurs more regularly, causing smaller epidemics.

### *Case definitions*

#### **Notifications**

Laboratory confirmed influenza became a nationally notifiable disease in 2001 with all states implementing notification during 2001 except Tasmania.

Laboratory confirmed infections are those in which influenza virus is isolated by cell culture, detected by nucleic acid testing, by influenza antigen testing or serological methods.

#### **Hospitalisations and deaths**

The ICD-10-AM codes used to identify hospitalisations were: J10 (influenza due to identified influenza virus) and J11 (influenza, virus not identified). In this report, no distinction was made between admissions where a virus was identified and those where it was not.

#### **Deaths**

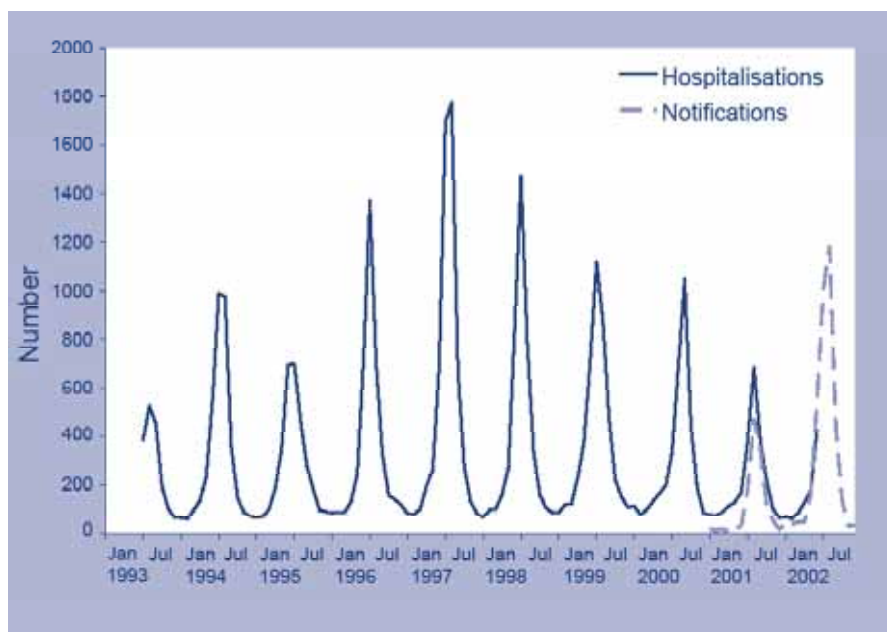
The ICD-10 codes used to identify deaths were: J10 (influenza due to identified influenza virus) and J11 (influenza, virus not identified).

## Secular trends

Although only some States and Territories were notifying influenza for the entire period in 2001, there were 1,283 notifications received. If only States and Territories which notified for the entire calendar year are included (New South Wales, the Australian Capital Territory, South Australia and Western Australia), there were 624 notifications, giving a rate for the populations of these areas of six per 100,000. In 2002, when all jurisdictions were notifying, there were 3,676 notifications (rate of 19 per 100,000). There was a clear seasonal distribution of notifications in both years with most notifications in 2001 received in August and September and most in 2002 received in July and August.

In 2000/2001–2001/2002 there were 6,275 hospitalisations coded as influenza (an average annual rate of 16.3 per 100,000), with most of the hospitalisations recorded in the earlier period (2000/2001; n=3,468.) There was a clear seasonal pattern with dramatic increases over the winter months (Figure 10). The median number of admissions per month was 149 (range 59–1,047) with annual maximums of 1,047 and 686 admissions occurring in September 2000 and August 2001, respectively.

**Figure 10. Influenza hospitalisations and notifications,\* Australia, July 1993 to December 2002, by month**



\* Notifications where the month of onset was between January 2001 and December 2002, hospitalisations where the month of admission was between 1 July 1993 and 30 June 2002. Note that the Northern Territory, Queensland, Tasmania and Victoria did not notify influenza for the complete year in 2001.

### Severe morbidity and mortality

A total of 42,248 hospital bed days were recorded for people with an ICD-10-AM code for influenza. The median length of stay was at least twice as long for older people than it was for any other age group: six days among people aged 60 years or over (Table 7). Influenza was the principal diagnosis for 67.2 per cent of the hospitalisations.

From 1 January 2001 to 31 December 2002, there were 87 deaths for which influenza was recorded on the death certificate as the underlying cause. Of these, 73 (84%) were aged 60 years or more, 11 (13%) were aged 25–59 years and three (3%) were aged 0–4 years.

**Table 7. Influenza hospitalisations and deaths, Australia, 2000 to 2002,\* by age group**

Age group (years)	Hospitalisations 2 years (July 2000–June 2002)				LOS <sup>†</sup> per admission (days)	Deaths 2 years (2001–2002)	
	n	(  )	Rate <sup>‡</sup>	(  )	Median	n	Rate <sup>‡</sup>
0–4	1,269	965	49.5	37.7	3	3	0.1
5–14	477	364	8.8	6.8	2	0	–
15–24	618	410	11.7	7.8	1	0	–
25–59	2,239	1,431	11.8	7.6	2	11	0.1
60+	1,672	1,046	26.0	16.3	6	73	1.1
All ages <sup>§</sup>	6,275	4,216	16.3	10.9	2	87	0.2

\* Hospitalisations where the month of separation was between 1 July 2000 and 30 June 2002; deaths where the date of death was recorded between 2001 and 2002.

† LOS = length of stay in hospital.

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

§ Includes cases with unknown ages.

|| Principal diagnosis (hospitalisations).

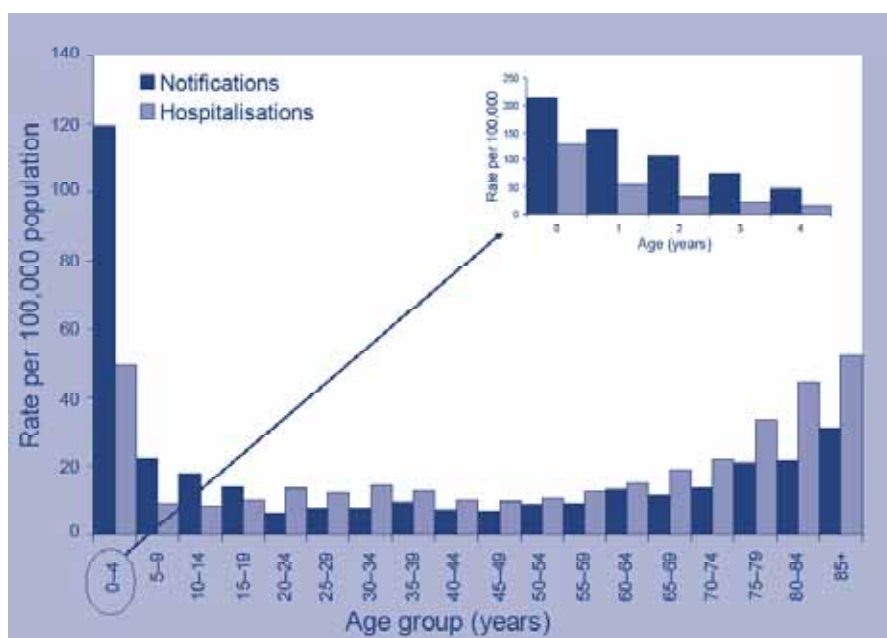
### Age and sex distribution

The pattern of notifications in 2002 has a striking age distribution, with a substantial peak rate in notifications in the under-five year age group (Figure 11). In this age group the highest rates of notifications are in those under one year of age and the rate declines with each year of increasing age. The overall male to female ratio was 1.2:1.

Among the age groups specified in Table 7, hospitalisation rates were highest in children aged under five years (49.5 per 100,000). Although overall hospitalisation rates were lower among people aged 60 years or more, the rates increased with increasing age, ranging from 15 per 100,000 for those aged 60–64 years to 52 per 100,000 for those aged 85 years or more (data not shown). Among children aged less than five years, the hospitalisation rates were highest among infants (138 and 116 per 100,000 population aged less than one year in 2000/2001 and 2001/2002, respectively).

The overall male to female hospitalisation ratio was 0.82:1; however, this was not consistent across all age groups. In children under 10 years of age males predominated.

**Figure 11. Influenza notification rates 2002 and hospitalisation rates 2000 to 2002, Australia,\* by age group**



\* Hospitalisations where the month of separation was between 1 July 2000 and 30 June 2002.

### Geographical distribution

There was a wide variation in the average crude hospitalisation rate recorded for the two year review period, ranging from 4.4 per 100,000 in the Australian Capital Territory (n=28) to 27.5 per 100,000 in Western Australia (n=1,038) (Appendix 3). In 2002, notification rates were similarly varied, with the highest rates reported in Western Australia and the Northern Territory (both reporting 28 per 100,000 population) and the lowest in the Australian Capital Territory and Tasmania (6 per 100,000 and 1 per 100,000, respectively).

In regard to hospitalisation rates the 2000/2001 period saw hospitalisations in States and Territories in the mid-range compared with previous years, and the 2001/2002 rates were below average in all areas. Historically the winter of 1997 remains the period between 1993/1994 and 2001/2002 during which most States and Territories recorded the highest number of hospitalisations.

## Comment

The timing and absolute numbers of laboratory notifications received are consistent with hospitalisation data. Undoubtedly higher rates in children, especially those under one year of age, reflect patterns of health care use and diagnostic testing for respiratory viruses in this age group. Vaccination, which is currently targeted at older Australians, may also play a role in reducing the number of notifications received in older age groups. On the other hand, the role of influenza in exacerbating chronic cardiac and respiratory disease in the elderly may not be reflected adequately in these surveillance data. Notification data for 2001 are likely to be incomplete for some jurisdictions due to the phasing in of mandatory notifications. It should be noted that there is no specialised diagnostic influenza laboratory in Tasmania or the Northern Territory, with specimens positive on direct fluorescent antibody testing referred interstate.

Hospitalisation data referred to in this report are based on discharge coding and it is possible that some of those with less specific influenza codes (e.g. J11) may be due to other respiratory pathogens such as respiratory syncytial virus (RSV)<sup>63</sup> or picornavirus.<sup>64,65</sup> The apparent differences in hospitalisation rates between States and Territories should be treated with caution as they may reflect differences in coding practices or rates of virological testing of inpatients between jurisdictions. Deaths and hospitalisations coded as influenza are widely acknowledged to underestimate deaths and hospitalisations due to influenza.<sup>66-68</sup> Deaths reported here underestimate manyfold the number of deaths due to influenza infection, which may exacerbate underlying cardiorespiratory disease. The proportion of deaths due to influenza in people aged 60 years and over (84%) is lower than in most other published studies.<sup>69,70</sup> This may be due to competing causes of death in this age group, as we only included influenza deaths where influenza was cited as the principal cause of death, or to lower rates of virological testing.

Influenza A and B viruses are known to cause major epidemics of respiratory disease resulting in severe morbidity and increasing numbers of deaths. Annual influenza vaccination is the primary method of prevention and is currently recommended for all people aged 65 years or more, all Aboriginal and Torres Strait Islander people aged 50 years or more, and people aged six months or more who are considered to be at high risk, such as those who have chronic disorders of the pulmonary or circulatory systems or other chronic illnesses requiring regular follow-up or hospitalisation.<sup>49</sup> Vaccination uptake in Australians aged 65 and older was estimated at 76.9 per cent in 2001, 2002<sup>71</sup> and 2003.<sup>72</sup> Health care workers and others caring for or living with high risk people should also be vaccinated, not only to protect themselves, but also because they can act as a vehicle for introduction of the virus.<sup>73</sup> Recently the US Centers for Disease Control and Prevention, on the advice of the Advisory Committee on Immunization Practices, and based on a high burden of illness,<sup>67,74,75</sup> has also recommended the routine vaccination of healthy American children aged six to 23 months with influenza vaccine.<sup>76,77</sup> Whilst available Australian data also suggest that there is a significant burden of illness in this age group, examination of the feasibility of recommending influenza vaccination for all children and cost-effectiveness analysis are required before recommending and implementing such a population level strategy.<sup>78,79</sup>

In 2001 the predominant influenza isolate was influenza A (81%), with a majority of subtype H1N1 (81%) and a minority of H3N2 (19%). All the H1N1 viruses analysed were A/New Caledonia/20/99 strain and the 2001 influenza vaccine was a good match to circulating viruses.<sup>62</sup> In 2002 the predominant influenza isolate was again A (77%), 99 per cent of which was of subtype H3N2. Most of the H3N2 strains were closely related to the A/Moscow/10/99 reference strain and the A/Panama/2007/99 vaccine strain. The 2002 influenza vaccine was a good antigenic match for the circulating influenza A viruses but only for a minority of the influenza B strains.<sup>80</sup> With the ongoing presence of avian influenza in Asia and elsewhere, there is increasing concern regarding the likelihood of an influenza pandemic. In this context, it has been suggested that high vaccination coverage could help prevent the emergence of pandemic influenza, by protecting humans against co-infection and hence re-assortment of animal and human influenza viruses. It could also increase local influenza vaccine production capacity by increasing annual demand for influenza vaccine.<sup>81-84</sup>

## Measles

Measles is an acute and highly communicable disease caused by a morbillivirus. The clinical picture includes a prodromal fever, rash, conjunctivitis, coryza, cough and Koplik spots on the buccal mucosa. Complications include otitis media, pneumonia and encephalitis. Subacute sclerosing panencephalitis (SSPE) occurs very rarely as a late sequel.<sup>16</sup>

### *Case definitions*

#### **Notifications**

a) An illness characterised by all the following features:

- a generalised maculopapular rash lasting three or more days, *and*
- a fever (at least 38°C if measured), *and*
- cough or coryza or conjunctivitis or Koplik spots

**or**

b) Demonstration of measles-specific IgM antibody

**or**

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

**or**

d) Isolation of measles virus from a clinical specimen

**or**

e) A clinically compatible case epidemiologically related to another case.

#### **Hospitalisations and deaths**

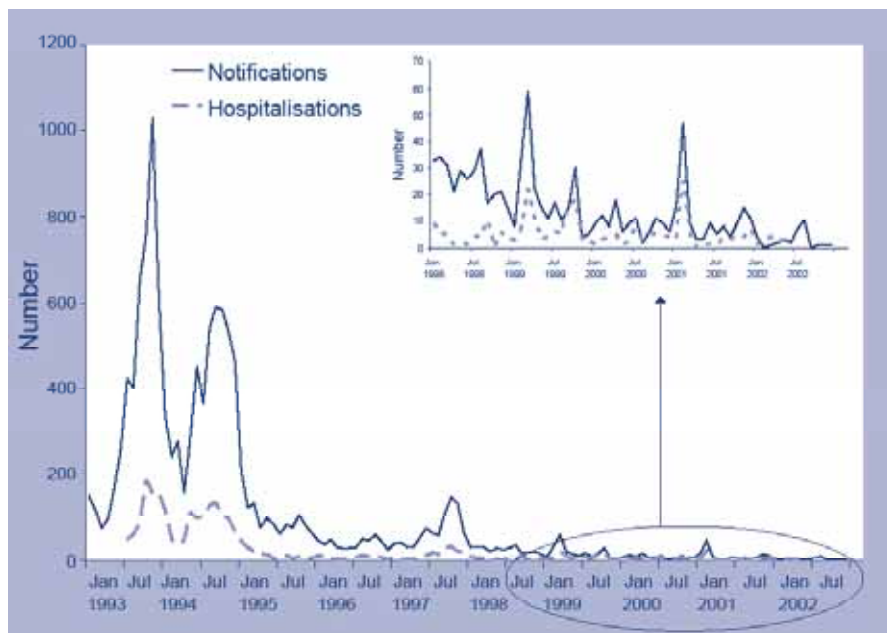
The ICD-10-AM/ICD-10 code B05 (measles) was used to identify hospitalisations and deaths. SSPE was not included in these analyses.

## Secular trends

In the two year review period there were 171 notified cases of measles, an average annual notification rate of 0.4 per 100,000 (Table 8). Although there were slightly more notifications in 2001 (n=139) compared with the previous year (n=107), in 2002 they were the lowest on record (n=32; Figure 12, Appendix 2). In the two year review period the median number of notifications per month was four (range 0–47).

In 2000/2001 and 2001/2002 there were 105 hospitalisations with the ICD-10-AM code B05 (measles). This equates to an average annual rate of 0.3 per 100,000. Annual hospitalisation rates have been declining since 1997/1998 and in 2001/2002 were the lowest on record at 41 separations, rate 0.2 per 100,000 (Appendix 3). The median number of hospitalisations per month was four (range 1–25). As with notifications, hospitalisations peaked in February 2001 and have been considerably lower since then (Figure 12).

Figure 12. Measles notifications and hospitalisations, Australia, 1993 to 2002,\* by month of onset or admission



\* Notifications where the month of onset was between January 1993 and December 2002; hospitalisations where the month of admission was between 1 July 1993 and 30 June 2002.

### Severe morbidity and mortality

In the two year review period, hospital separations for measles accounted for 419 hospital bed days. The median length of stay (LOS) was two days, with little variation across the age groups (Table 8). Of the 105 hospitalisations, 96 (91%) had measles recorded as the principal diagnosis. Complications arising from measles infection were recorded for 12 (11%) separations. There were no hospitalisations coded as having otitis media, or intestinal or neurological (encephalitis or meningitis) complications (Table 9). Six (6%) hospitalisations were coded as having pneumonia, seven (7%) as other complications, and one hospitalisation had both these codes. Adults aged 15 years and over accounted for five of the six (83%) hospitalisations coded with pneumonia.

There were no deaths recorded from measles between 2001 and 2002 (Table 8).

Table 8. Measles notifications, hospitalisations and deaths, Australia, 2000 to 2002,\* by age group

Age group (years)	Notifications 2 years (2001–2002)		Hospitalisations 2 years (July 2000–June 2002)				LOS† per admission (days)	Deaths 2 years (2001–2002)	
	n	Rate‡	n	(  )	Rate‡	(  )	Median	n	Rate‡
0–4	36	1.4	26	(23)	1.0	(0.9)	2	0	–
5–14	14	0.3	8	(6)	0.1	(0.1)	2	0	–
15–24	63	1.2	28	(26)	0.5	(0.5)	2	0	–
25–59	57	0.3	40	(38)	0.2	(0.2)	3	0	–
60+	1	0.0	3	(3)	0.0	(0.0)	2	0	–
All ages§	171	0.4	105	(96)	0.3	(0.2)	2	0	–

\* Notifications where the month of onset was between January 2001 and December 2002; hospitalisations where the month of separation was between 1 July 2000 and 30 June 2002; deaths where the date of death was recorded between 2001 and 2002.

† LOS = length of stay in hospital.

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

§ Includes cases with unknown ages.

|| Principal diagnosis (hospitalisations).

**Table 9. Indicators of severe morbidity for hospitalised cases of measles, Australia, 2000 to 2002,\* by age group**

Age group (years)	Measles encephalitis		Measles pneumonia	
	n	% total	n	% total
0–4	0	0.0	1	3.8
5–14	0	0.0	0	0.0
15–24	0	0.0	2	7.1
25–59	0	0.0	3	7.5
60+	0	0.0	0	0.0
All ages	0	0.0	6	5.7

\* Measured using National Hospital Morbidity data where the month of hospital separation was between 1 July 2000 and 30 June 2002.

### Age and sex distribution

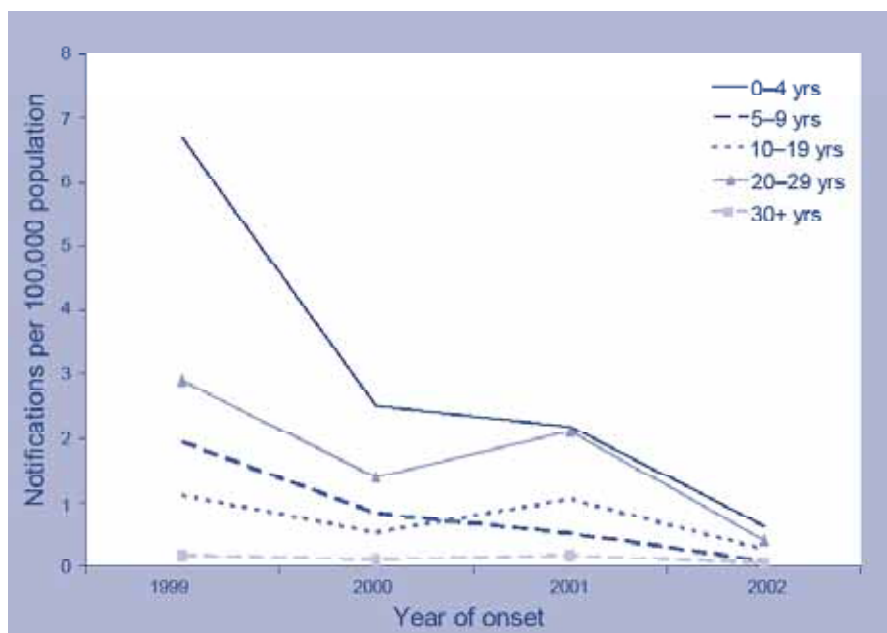
In the two year review period, notification and hospitalisation rates for children under 10 years of age continued a downward trend (Figures 13 and 14). In 2002, the notification rate for the 0–4 year age group (0.6 per 100,000) and 5–9 year age group (0.07 per 100,000) was the lowest on record. Similarly, in 2001/2002 hospitalisation rates were at an all time low for 0–4 year olds (0.8 per 100,000) and there were no hospitalisations in 5–9 year olds. The greatest rate reductions since the Measles Control Campaign (MCC) in 1998 have been in the 0–4 year age group, especially in children aged less than two years.

For ages 10 years and over, notification and hospitalisation rates increased in the first year of the review period, in contrast to the trend for younger children, but were lower in the second year (Figures 13 and 14). In the first year reviewed, all ages of 10 years and over had higher notification rates and all ages except 15–19 year olds had higher rates of hospitalisation. The greatest increase was in 20–34 year olds, and for the first time on record the 20–24 year age group had the highest notification (2.1 per 100,000) and hospitalisation rates (1.4 per 100,000) of any 5-year age group. In the second year of the review period, both notification and hospitalisation rates were lower, with notification rates declining to record low levels in all ages except 30–34 year olds (n=3; rate 0.2 per 100,000). Hospitalisation rates for 10–19 year olds were also the lowest on record, and rates for 20–24 year olds declined (0.5 per 100,000). However, rates for 25–29 year olds (0.8 per 100,000) remained high and were the highest of any 5-year age group except 0–4 year olds. Over the two year period, 4 per cent of the notifications and 52 per cent of the hospitalisations were aged 20–34 years.

Since the MCC, there have been declining notification and hospitalisation rates in children. This has led to an increase in the median age of both notifications and hospitalisations. In the most recent year reviewed, the median age for notified cases was 21 years, 19 years higher than the lowest figure of two years of age in 1998 (data not shown). Similarly, the median age of hospitalised cases in 2001/2002 was 25 years compared with five years in 1997/1998.

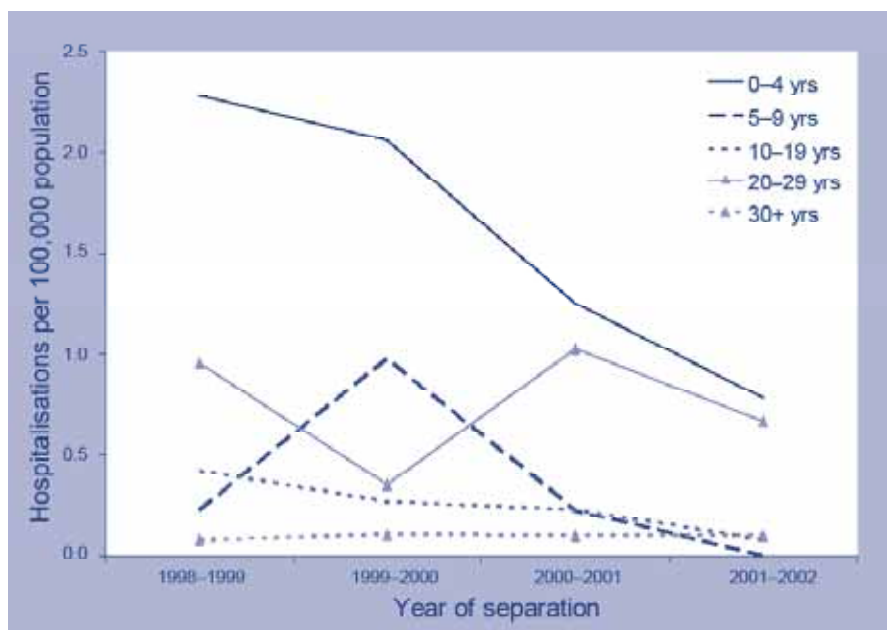
Over the two year review period there were slightly more notifications for females than males (male:female ratio 1:1.1). Conversely, there were more hospitalisations of males than females (male:female ratio 1.4:1).

Figure 13. Measles notification rates, Australia, 1999 to 2002,\* by age group and year of onset



\* Notifications where the month of onset was between January 1999 and December 2002.

Figure 14. Measles hospitalisation rates, Australia, 1998/1999 to 2001/2002\* by age group and year of separation



\* Hospitalisations where the month of separation was between 1 July 1998 and 30 June 2002.

### Geographical distribution

Victoria had the highest notification rate in 2001 (1.7 per 100,000) and hospitalisation rate in 2000/2001 (0.8 per 100,000). The notification rate was four times higher, and the hospitalisation rate twice as high, as the previous year. All other jurisdictions showed similar or lower numbers of notifications and hospitalisations than in the past year and there were no notified cases from the Australian Capital Territory or the Northern Territory (Appendices 2 and 3). The increased rates in 2001 in Victoria were mainly due to two outbreaks. The largest outbreak involved 51 cases between January and March 2001 (90% aged 15-34 years).<sup>8,85,86</sup> A smaller



outbreak of 18 cases occurred later between October and December 2001.<sup>8,86</sup> The only other outbreak to be reported for 2001 was in Sydney and involved seven cases.<sup>8</sup> In all, Victoria contributed 59 per cent of the notifications and 56 per cent of the hospitalisations in the first year of this review period.

In the second year of the review, notification rates declined to record low levels in all jurisdictions, and hospitalisation rates were lower in all except Western Australia and Tasmania. In Victoria, notification rates were 83 per cent lower than in 2001, but were still the highest of any jurisdiction (0.3 per 100,000). Victoria also continued to contribute the highest proportion of both notifications in 2002 and hospitalisations in 2001/2002 (44% for both). In 2002, there were no notifications from the Australian Capital Territory, Tasmania, the Northern Territory or Western Australia. The largest reported outbreak in 2002 involved seven cases. It began in the Whitsunday region of north Queensland, but also spread to New South Wales.<sup>87</sup>

### Comment

In the two year review period, measles notifications and hospitalisations continued to decline to new record lows. Measles accounted for only 32 notifications in 2002 and 41 hospitalisations in 2001/2002 and there have been no reported deaths from measles since 1995. This trend is similar to that seen in other countries with high coverage, such as the Americas and Finland.<sup>88,89</sup>

Now that measles is rare, enhanced surveillance including a high level of confirmation is required and recommended by the WHO.<sup>90</sup> All cases need to be confirmed (either by laboratory tests or by linkage to a chain of transmission that includes a laboratory-confirmed case) because a high proportion of clinically diagnosed cases is now unlikely to be measles.<sup>91</sup> Enhanced surveillance for measles during an inter-epidemic period in Victoria (July 1997–December 1998) found that only seven per cent of the 258 suspected cases tested for measles were laboratory confirmed and the positive predictive value (PPV) of the clinical case definition for notification was only 14 per cent.<sup>92</sup> Since 1999, over 80 per cent of notified cases have been confirmed with most of the improvement in ages less than 15 years.<sup>93</sup> This means we can be more confident that notifications represent true cases, even though the level of confirmation in some States and Territories still requires improvement.

The record low rates of measles could be partly due to better efforts to confirm cases, but are also likely to be due to several vaccination initiatives. Improved coverage with a two-dose schedule has led to increased herd immunity and this probably explains why rates overall, and especially those for less than one year olds (who are not targeted by vaccination), have declined. The mass vaccination of primary school aged children as part of the MCC,<sup>94</sup> together with additional cohorts being eligible for the second dose of measles-mumps-rubella (MMR) vaccine prior to school entry, continues to result in low rates of measles in 5–9 year olds, and now also in 10–14 year olds as two-dose vaccinated cohorts move into this age group. In the second year of the review period there were only two notifications and one hospitalisation from the 5–14 year age group.

High coverage in children has left a residual cohort of susceptible young adults. Since the MCC, most outbreaks have involved a high proportion of young adults, especially those born in the 1970s and early 1980s, when measles vaccine was first introduced but coverage was low. To improve immunity in this age group the young adult MMR vaccination campaign was conducted during 2001.<sup>95</sup> The Campaign may help to explain the lower rates in young adults in 2002. However, there is evidence to suggest that coverage did not improve significantly<sup>96</sup> and further studies are under way to more formally evaluate uptake using serosurveillance data.

In September 2003, the WHO Regional Committee for the Western Pacific confirmed that measles elimination should be a regional goal and that each country should set a target date for elimination.<sup>97</sup> In Australia, local measles transmission may have already been interrupted, as in most outbreaks the index case has been infected overseas. Molecular genotyping of measles isolates from the resulting outbreaks also supports this conclusion.<sup>98</sup>

Despite evidence to suggest that elimination has been achieved, high coverage with a two-dose childhood program needs to be maintained and indeed improved. Even though adults make up a higher proportion of cases than ever before, children aged 0–4 years continue to have the highest notification and the second highest hospitalisation rates of any age group. Therefore, better timeliness and completeness of childhood vaccinations remains an important goal of Australia's measles control strategy.

## Meningococcal disease

Meningococcal disease is defined as isolation of *Neisseria meningitidis* from cerebrospinal fluid (CSF), blood and other normally sterile sites including skin lesions. Clinical manifestations include meningitis, meningococcaemia without meningitis (which varies in presentation from fulminant to chronic) and septic arthritis. In culture-negative cases with a compatible clinical picture, a diagnosis of meningococcal disease can be supported by a range of laboratory evidence. This includes the identification of Gram-negative intracellular diplococci or meningococcal antigen in blood or cerebrospinal fluid (CSF), the identification of nucleic acid from *Neisseria meningitidis* in body fluids or demonstration of a serological response to *Neisseria meningitidis*.

### Case definitions

#### Notifications

In jurisdictions apart from New South Wales and the Northern Territory, a notification of meningococcal disease requires supportive laboratory evidence, although the nature of this varies. In New South Wales, Queensland and the Northern Territory, a clinical diagnosis of meningococcal disease without laboratory evidence is accepted as a presumptive (New South Wales) or probable (Queensland, Northern Territory) case. The serogroup of meningococcal cases is not currently routinely available from notification data but is reported annually by the National Neisseria Network in *Communicable Diseases Intelligence*.

#### Hospitalisations

The ICD-10-AM code used to identify hospitalisations was A39 (meningococcal infection). This includes meningococcal meningitis (A39.0), Waterhouse-Friderichsen syndrome (A39.1), acute meningococcaemia (A39.2), chronic meningococcaemia (A39.3), meningococcaemia unspecified (A39.4), meningococcal heart disease (A39.5), other meningococcal infections (A39.8), and meningococcal infection unspecified (A39.9). As all cases with one of these codes, not just principal diagnoses, were included, cases were identified in a hierarchical fashion to avoid double counting. First, those with code A 39.0 (meningitis), then those without A 39.0 but with A39.1 or A39.2 or A39.3 or A39.4 (septicaemia without meningitis), then those with none of these codes but with codes in any other subsection of A39 were selected. However, as re-admissions and inter-hospital transfers are separate records, duplication may occur for a condition such as meningococcal disease where complications are frequent.

#### Deaths

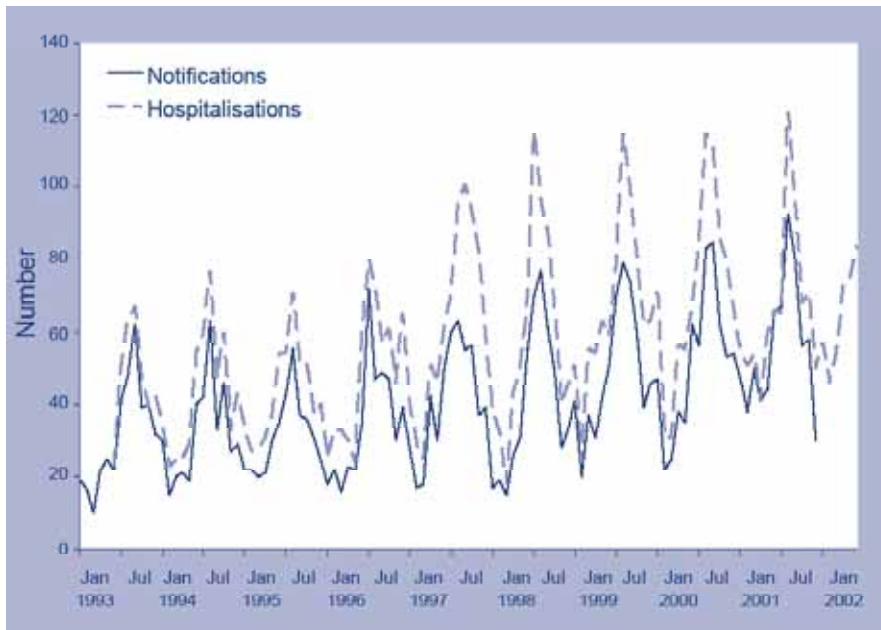
The ICD-10 code used to identify deaths was A39 (meningococcal infection).

## Secular trends

There were 1,355 notifications of meningococcal disease in the two years 2001 to 2002, an average annual notification rate of 3.5 per 100,000 (Table 10). A median of 55.5 cases was notified each month, with a range of 30 to 93 cases. There were 1,743 hospital admissions recorded as ICD code A39 (average annual rate 4.5 per 100,000), and a median of 67 cases (range 41–121) per month.

Notifications and hospitalisations were similar in 2001 and 2002, and higher than in previous years. A clear seasonal pattern was apparent, with the highest number of notifications and hospitalisations occurring between June and September each year (Figure 15).

**Figure 15. Meningococcal notifications and hospitalisations, Australia, 1993 to 2002,\* by year of onset or admission**



\* Notifications where the year of onset was between 1993 and 2002; hospitalisations where the month of separation was between 1 July 1993 and 30 June 2002.

### Severe morbidity and mortality

Over the two year review period, 13,309 hospital bed days were recorded for patients with an ICD-10-AM code A39, of which 86.3 per cent were coded as meningococcal meningitis (A39.0). For all categories of meningococcal disease, the hospitalisation and notification rates were greatest among 0–4 and 15–24 year olds, who accounted for 60 per cent of cases. In 0–4 year olds, the hospitalisation rate for meningococcal meningitis was 10.0 per 100,000, and 20.4 per 100,000 when all meningococcal disease categories were considered (Table 10). The proportion where a meningococcal disease code was the principal diagnosis varied from 97 per cent of diagnoses among 0–14 year olds to 80 per cent of cases for those aged 15–59 years, but was only 62 per cent for those aged 60 years and over. Meningococcal meningitis was the first mentioned diagnosis in 704 (40%) of hospitalised meningococcal cases overall, slightly higher in 0–4 year olds (245, 47%) and notably lower in those over 60 years (19, 20%). For all hospitalisations for meningococcal infection, length of stay increased with age.

There were 88 deaths with meningococcal disease recorded as the underlying cause of death over the two years 2001 to 2002. The death rate was highest among those under five years of age (1.0 per 100,000), followed by those aged 15–24 years (0.4 per 100,000), and most deaths (77%) were coded as septicaemia without meningitis. Of the total of 1,743 hospitalisations over a different two year time period (Table 10), 74 (4.2%) were recorded as dying before hospital discharge. The proportion of hospitalisations dying before discharge increased steadily with age from approximately three per cent of those 0–24 years to 6.7 per cent for 25–59 year olds and 15.6 per cent of hospitalisations for meningococcal infection in those aged over 60 years.

### Age and sex distribution

Overall there was a predominance of male cases (male:female ratio 1.2:1). However, among adults 60 years and over, there were more females (male:female ratio 0.8:1). Among children under five years of age, those under one year of age had the highest rates of notification (34.6 per 100,000) and hospitalisation (40.6 per 100,000). There was a second peak in both notification (Figure 16) and hospitalisation rates (Figure 17) among 15–19 year olds (9.2 and 13.4 per 100,000, respectively), with rates in 20–24 year olds remaining elevated, comparable to 5–14 year olds, before falling to levels appreciably lower than those in childhood over 25 years of age and remaining relatively constant thereafter. Nevertheless, persons over 25 years still accounted for 26 per cent of total notifications (Table 10).

**Table 10. Meningococcal notifications, hospitalisations and deaths, Australia, 2000 to 2002\* by age group**

Age group (years)	Notifications 2 years (2001–2002)		Hospitalisations 2 years (July 2000–June 2002)				LOS† per admission (days)	Deaths 2 years (2001–2002)	
	n	Rate‡	n	(  )	Rate‡	(  )	Median	n	Rate‡
0–4	393	15.4	522	(504)	20.4	(19.7)	5	26	1.0
5–14	213	3.9	278	(267)	5.4	(5.0)	5	8	0.1
15–24	391	7.3	533	(422)	10.1	(8.0)	6	21	0.4
25–59	286	1.5	314	(253)	1.7	(1.3)	7	24	0.1
60+	72	1.1	96	(59)	1.5	(0.9)	8	9	0.1
All ages§	1,355	3.5	1,743	(1,505)	4.5	(3.9)	6	88	0.2

\* Notifications where the month of onset was between January 2001 and December 2002; hospitalisations where the month of separation was between 1 July 2000 and 30 June 2002; deaths where the date of death was recorded between 2001 and 2002.

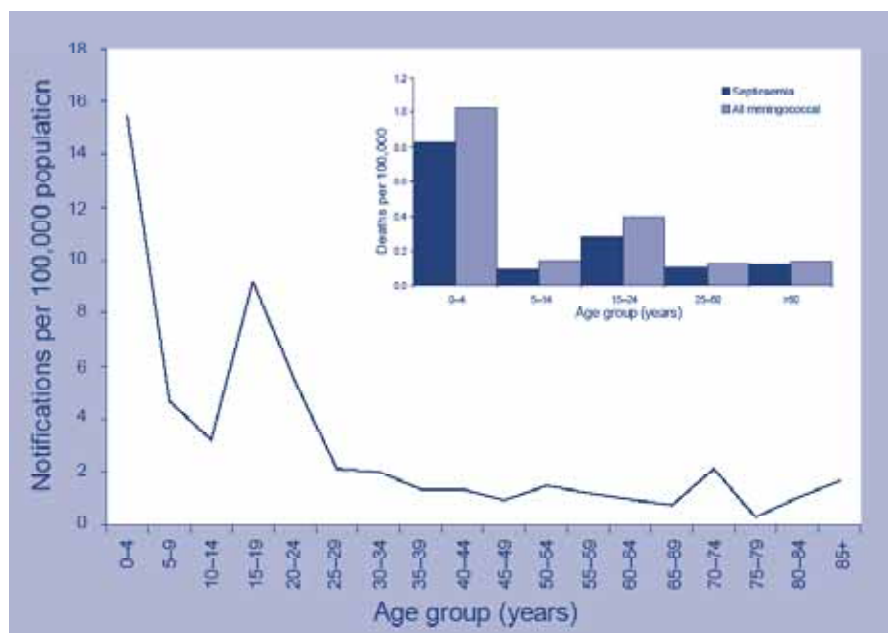
† LOS = length of stay in hospital.

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

§ Includes cases with unknown ages.

|| Principal diagnosis (hospitalisations)

**Figure 16. Meningococcal disease notification and death rates, Australia, 2001 to 2002,\* by age group**

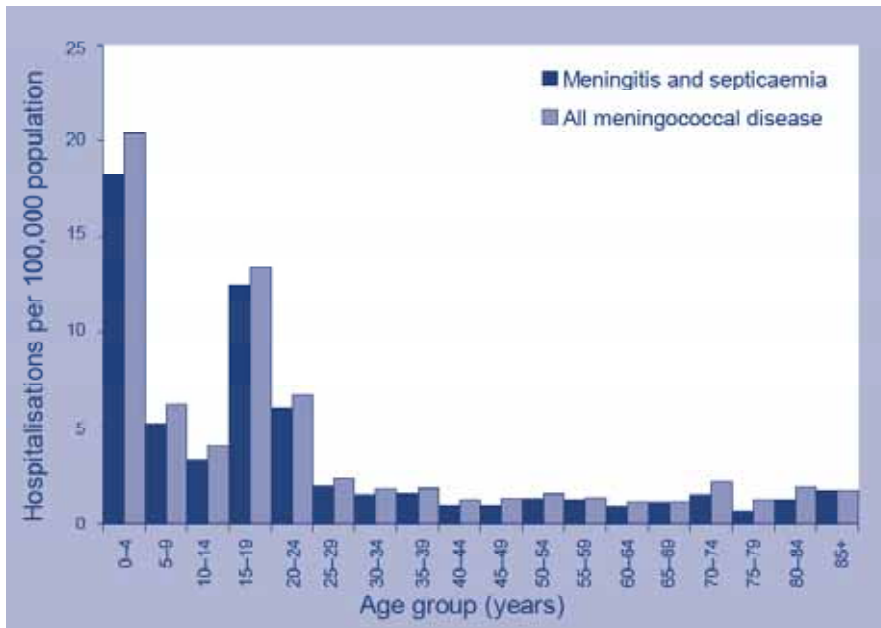


\* Notifications where the month of onset was between January 2001 and December 2002, deaths where the date of death was recorded between 2001 and 2002.

### Geographical distribution

The pattern of notification and hospitalisation rates varied across the country, with the Northern Territory having the highest average annual notification (5.6 per 100,000) and hospitalisation (6.4 per 100,000) rates, followed by Tasmania (5.2 and 6.3 per 100,000) and Victoria (3.9 and 5.1 per 100,000) (Appendices 2 and 3). The Australian Capital Territory had the lowest rates of both notifications and hospitalisations (1.9 and 2.2 per 100,000, respectively).

Figure 17. Meningococcal disease hospitalisation rates, Australia, 2000 to 2002,\* by age group



\* Hospitalisations where the month of separation was between 1 July 2000 and 30 June 2002.

## Comment

The incidence of meningococcal disease in Australia, based on notifications, has increased steadily from 1.6 per 100,000 in 1991 to 3.5 per 100,000, a doubling over the past decade.<sup>99</sup> The Northern Territory consistently had the highest overall notification rate over the past five years (5.9, range 4.2–8.0 per 100,000), followed by Western Australia (3.9, range 2.7–4.6 per 100,000). As these two jurisdictions have a relatively high proportion of Aboriginal and Torres Strait Islander people, it is likely that the higher incidence is related to disproportionate rates in this group, as recently reported from enhanced surveillance in Queensland.<sup>100,101</sup> In New South Wales, enhanced surveillance reports for 1991–2002 found that the notification rate among Indigenous people was 7.5 compared with 2.6 among non-Indigenous people, although the case-fatality rate was the same.<sup>102</sup> In Tasmania and Victoria, there were noticeable increases in notification rates in 2001 and 2002, so that both these jurisdictions had rates higher than Western Australia and similar to the Northern Territory in the most recent period.

There is considerable heterogeneity across the country in the incidence and serogroup distribution of meningococcal disease.<sup>103,104</sup> Serotype-specific data are important for vaccine policy, as conjugate vaccines against serogroup C are now in widespread use.<sup>105</sup> The National Neisseria Network has published reports on serotype-specific data since 1994, showing that the proportion of serogroup C varies widely by jurisdiction and age group.<sup>103,106–108</sup> Serogroup C emerged as the predominant serogroup among older children and adolescents in Victoria in 1999 to 2000 and in Tasmania in 2001 and 2002, when more than 70 per cent of isolates were of this serogroup.<sup>109</sup> However, serogroup B predominates among children under five years in all jurisdictions and among all age groups in jurisdictions other than Victoria, Tasmania and New South Wales.<sup>106–108</sup>

As found elsewhere, in Australia serogroup C meningococcal disease is associated with a higher mortality than serogroup B.<sup>103,106–108</sup> The United Kingdom was the first country to conduct a program to provide conjugate C vaccine for a wide age cohort (all 0–18 year olds). Similar programs are now in place in The Netherlands, Belgium, the Republic of Ireland and regions of Spain. The campaign in England, Scotland and Wales was followed by a dramatic decrease in cases and deaths due to serogroup C in the target age group.<sup>110</sup> There was no evidence of a compensatory rise in other serogroups, but there was evidence of decrease in age groups not targeted by the campaign through presumed herd immunity effects.<sup>111</sup> Conjugate meningococcal serogroup C vaccines were approved for use in Australia in 2001 and a national campaign targeting children 1–18 years was announced in 2003.<sup>112</sup> Early indications of the impact on serogroup C disease should be possible by the end of 2004, especially in Victoria and Tasmania where serogroup C predominated.

## Mumps

Mumps is an acute viral disease caused by a paramyxovirus. The disease is characterised by fever, swelling and tenderness of one or more salivary glands, most commonly the parotid glands. The central nervous system is frequently involved, usually without sequelae.<sup>16</sup>

### *Case definitions*

#### **Notifications**

a) Isolation of mumps virus from a clinical specimen

**or**

b) Significant rise in mumps antibody level by any standard serological assay, except following vaccination

**or**

c) A clinically compatible illness (unilateral or bilateral swelling of the parotid or other salivary glands lasting two days or more without other apparent cause).

**Notes:** In New South Wales only laboratory confirmed cases [(a) or (b)] are notifiable. Mumps was not notifiable in Queensland between July 1999 and June 2001. From July 2001, notifications based on a clinical case definition alone [(c)] were no longer notifiable in Victoria.

#### **Hospitalisations and deaths**

The ICD-10-AM/ICD-10 code B26 (mumps) was used to identify hospitalisations and deaths.

## Secular trends

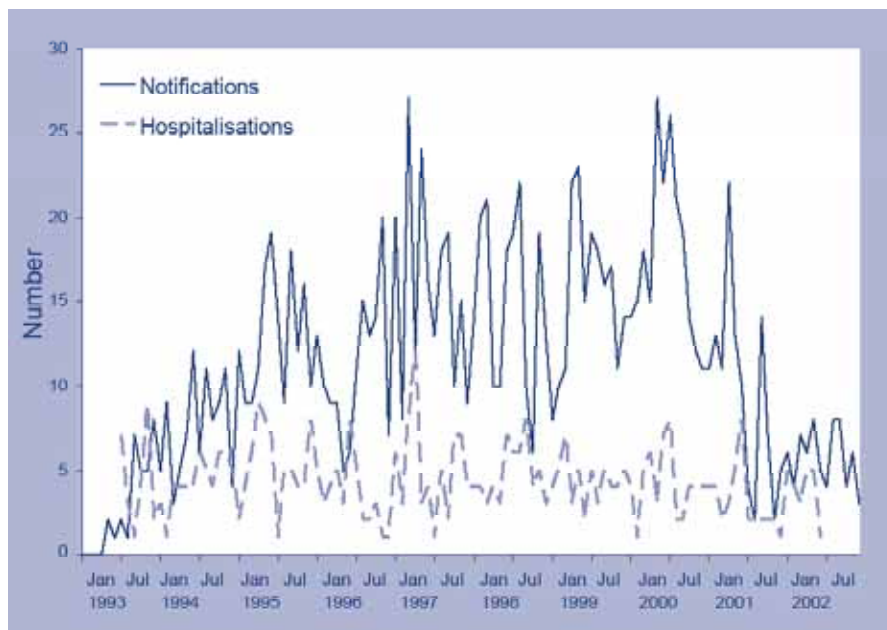
During the two years from 2001 to 2002 there were 180 notifications of mumps (an average annual notification rate of 0.5 per 100,000) (Table 11). Notification rates have shown a twofold decline each year between 2000 and 2002; in 2002 they were the lowest on record (0.4 per 100,000) since all jurisdictions began notifying the disease in 1996. Monthly numbers of notifications varied considerably, with a median of 6.5 (range 2–22) notifications per month. Notifications peaked in April and again in September 2001 and declined to eight or fewer notifications per month in 2002.

From July 2000 to June 2002 there were 85 hospitalisations coded as due to mumps (average annual rate of 0.2 per 100,000; Table 11) with one to eight admissions each month (median 3.5 per month). In previous years (1993/1994–1999/2000) hospitalisation rates remained fairly constant despite changing notification rates. However, in this review period, hospitalisations showed a similar trend to notifications, declining to record low levels in 2001/2002 (Appendix 3).

## Severe morbidity and mortality

There were 385 hospital bed days (average 193 per year) recorded for patients with the ICD-10-AM code for mumps (Table 11). Of the 85 hospitalisations, 67 (79%) had mumps recorded as the principal diagnosis (average annual rate 0.2 per 100,000). Complications arising from mumps infection were recorded for 12 hospitalisations (14%). As in the past, the most commonly reported complication was orchitis. There were seven (8%) hospitalised cases coded with orchitis; five of whom were between 15 and 59 years of age (Table 12). There were no hospitalisations coded as neurological (encephalitis or meningitis) or multiple complications. The median length of stay (LOS) in hospital was three days, but adults aged 25 years and older had a longer median LOS compared with younger age groups (Table 11). Children aged 0–4 years had the highest hospitalisation rate and accounted for 19 per cent of the hospitalisations. However, adults aged 15 years and over accounted for 67 per cent of the total hospitalisations, all except one of the hospitalisations with a mumps-related complication, and 86 per cent of the hospital bed days. Mumps was recorded as the underlying cause of death in one adult (aged over 80 years) in 2001.

**Figure 18. Mumps notifications and hospitalisations, Australia, 1993 to 2002,\* by month of onset or admission†**



\* Notifications where the month of onset was between January 1993 and December 2002; hospitalisations where the month of separation was between 1 July 1993 and 30 June 2002.

† Note that the number of jurisdictions notifying mumps increased over the review period until July 1996 when mumps became notifiable in all States and Territories. From July 1999 until June 2001 mumps was not notifiable in Queensland. Only the Australian Capital Territory, New South Wales and Victoria notified for the entire review period.

**Table 11. Mumps notifications, hospitalisations and deaths, Australia, 2000 to 2002,\* by age group**

Age group (years)	Notifications 2 years (2001–2002)		Hospitalisations 2 years (July 2000–June 2002)				LOS† per admission (days)	Deaths 2 years (2001–2002)	
	n	Rate‡	n	(  )	Rate‡	(  )	Median	n	Rate‡
0–4	20	0.9	16	(15)	0.6	(0.6)	2	0	–
5–14	36	0.7	12	(11)	0.2	(0.2)	1	0	–
15–24	31	0.6	16	(13)	0.3	(0.2)	2.5	0	–
25–59	78	0.4	30	(22)	0.2	(0.1)	4	0	–
60+	15	0.2	11	(6)	0.2	(0.1)	5	1	0.0
All ages§	180	0.5	85	(67)	0.2	(0.2)	3	1	0.0

\* Notifications where the month of onset was between January 2001 and December 2002; hospitalisations where the month of separation was between 1 July 2000 and 30 June 2002; deaths where the date of death was recorded between 2001 and 2002.

† LOS = length of stay in hospital.

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

§ Includes cases with unknown ages.

|| Principal diagnosis (hospitalisations).

**Table 12. Indicators of severe morbidity and mortality for hospitalised cases of mumps, Australia, 2000 to 2002,\* by age group**

Age group (years)	Mumps meningitis or encephalitis		Mumps orchitis		Mumps pancreatitis		Mumps with other complications	
	n	% total	n	% total	n	% total	n	% total
0-4	0	0.0	0	0.0	0	0.0	0	0.0
5-14	0	0.0	1	8.3	0	0.0	0	0.0
15-24	0	0.0	2	12.5	0	0.0	0	0.0
25-59	0	0.0	3	10.0	1	3.3	4	13.3
60+	0	0.0	1	9.1	0	0.0	0	0.0
All ages	0	0.0	7	8.2	1	1.2	4	4.7

\* Measured using National Hospital Morbidity data where the month of hospital separation was between 1 July 2000 and 30 June 2002.

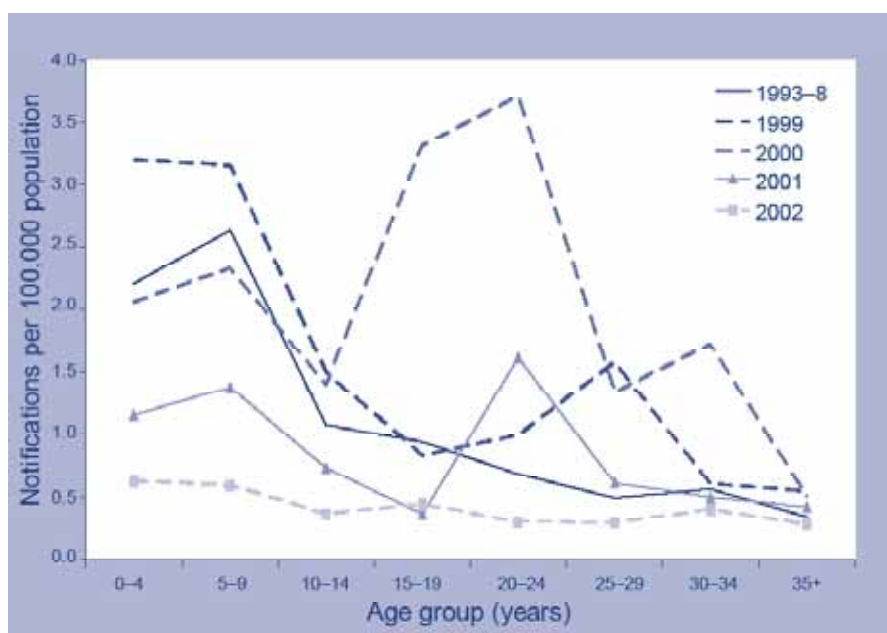
### Age and sex distribution

The pattern of notifications in 2001 and hospitalisations in 2000/2001 was similar to that seen in the previous year (Figures 19 and 20). Notification rates were highest in 20-24 year olds (1.6 per 100,000) and hospitalisation rates were highest in the 0-4 year age group (0.7 per 100,000) followed by the 20-24 year age group (0.6 per 100,000). However, for most age groups, rates were lower than in the previous year.

In the most recent year reviewed, notification and hospitalisation rates continued to decline, especially in the 0-4, 5-9 and 20-24 year age groups. As in years prior to 2000, notifications rates were highest in 0-4 and 5-9 year olds (0.6 per 100,000 for both groups). However, rates were fairly similar across all age groups and were the lowest on record for all ages except 15-19 year olds. The 0-4 year age group continued to have the highest hospitalisation rates, but rates were considerably lower than in the past two years. All other ages reported uniformly low numbers of hospitalisations.

Over the two year review period the male:female ratio was 1.0:1 for notifications and 1.2:1 for hospitalisations. However, this conceals annual differences. In the first year of the review there were more females (M:F ratio 0.9:1 for both notifications and hospitalisations) while in the second year there were more males (M:F ratio 1.5:1 for notifications and 1.7:1 for hospitalisations).

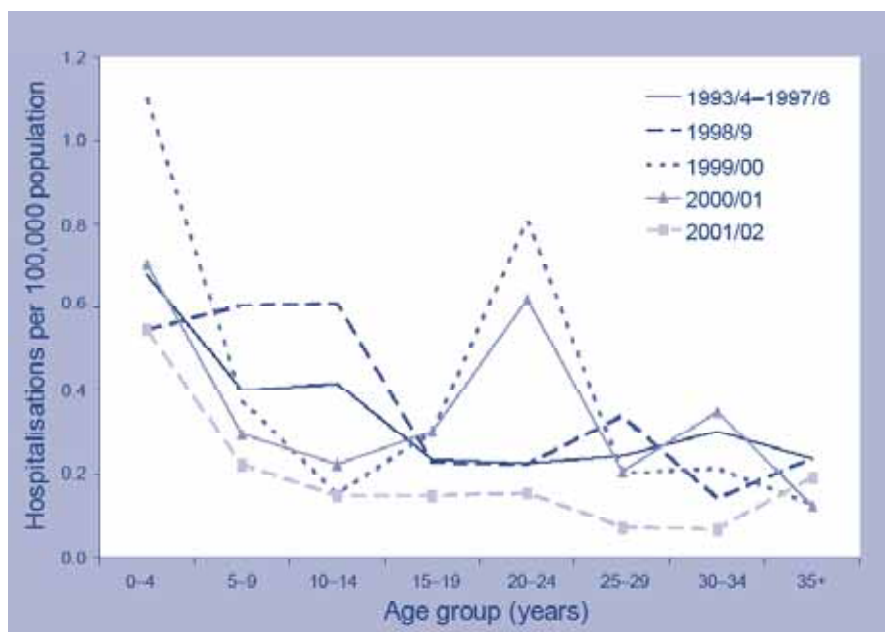
**Figure 19. Mumps notification rates, Australia, 1993 to 2002,\* by age group and year of onset**



\* Notifications where the month of onset was between January 1993 and December 2002.



**Figure 20. Mumps hospitalisation rates, Australia, 1993 to 2002,\* by age group and year of separation**



\* Hospitalisations where the month of separation was between 1 July 1993 and 30 June 2002.

### Geographical distribution

New South Wales accounted for most of the decline in notifications between 2000 and 2001, although all States showed lower numbers. In 2002, numbers continued to decline in each State except New South Wales, where numbers were similar to those for 2001. The greatest decreases between 2001 and 2002 were in Victoria and Western Australia. Average annual notification rates for the two year review period were below 1.0 per 100,000 in all States except Western Australia (1.1 per 100,000).

As with notifications, most of the decrease in hospitalisations between 1999/2000 and 2000/2001 was in New South Wales while Victoria, Western Australia and Queensland all showed declines between 2000/2001 and 2001/2002. Average annual hospitalisation rates for the two year review period were below 0.5 per 100,000 in all States.

### Comment

Mumps notification and hospitalisation rates declined during this review period, in contrast to previous years. In 2002, the notification rate was 0.4 per 100,000, the lowest on record and well under the World Health Organization elimination target of less than 1 per 100,000. The downward trend is similar to that seen for measles and rubella in Australia and to the recent epidemiology of mumps in the United States of America<sup>113</sup> and Finland (where mumps has been eliminated).<sup>89</sup>

The record low rates are probably due to several factors. In July 2001, Victoria introduced a system of enhanced surveillance for mumps.<sup>114-116</sup> The new case definition excluded cases based on clinical criteria alone, and this led to a dramatic reduction in notifications; in 2002 only nine of the original 60 reported mumps cases met the new case definition for notification. Although this can explain some of the decline in notifications, it is unlikely to be the sole cause, as a downward trend was also noted for hospitalisations, which are unaffected by notification criteria. In children, declining notification and hospitalisation rates are most likely due to improved coverage with a two-dose measles-mumps-rubella (MMR) vaccine prior to school entry and the ongoing impact of the Measles Control Campaign (which involved the mass vaccination of primary school aged children in 1998). In adults, the lower rates may be due to the impact of the young adult MMR vaccination campaign, which targeted susceptible 18-30 year olds in 2001.<sup>95</sup> However, there is evidence to suggest that the latter campaign did not significantly improve immunity levels<sup>96</sup> and further studies are under way to measure vaccine uptake using serosurveillance data.

The surveillance data presented in this report suggest that mumps is now a rare disease. It is therefore important to confirm all cases, as clinical criteria alone are now insufficient for diagnosis. The results from Victoria's enhanced surveillance system support this recommendation.<sup>114-116</sup> To reflect the changing pattern of mumps, the case definition for national notification has recently been modified to only include those with laboratory evidence of mumps or epidemiological linkage to a confirmed case.<sup>117</sup> This new case definition is being implemented during 2004 and should lead to more accurate notification rates in the future, provided that increased efforts are made to follow up clinical notifications and perform laboratory tests.

## Pertussis

Pertussis (whooping cough) is an acute illness, caused by the *Bordetella pertussis* bacterium, involving the respiratory tract. The illness begins with an irritating cough that gradually becomes paroxysmal and lasts for one to two months or longer. Paroxysms are characterised by repeated violent coughs and are followed by a characteristic crowing or high-pitched inspiratory whoop. Infants less than six months old, adolescents and adults often have fewer classical symptoms without paroxysms or whoop.<sup>16</sup>

### Case definitions

#### Notifications

a) Isolation of *B. pertussis* from a clinical specimen

**or**

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

**or**

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

- paroxysms of coughing, *or*
- inspiratory whoop without other apparent causes, *or*
- post-tussive vomiting

**or**

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

#### Hospitalisations and deaths

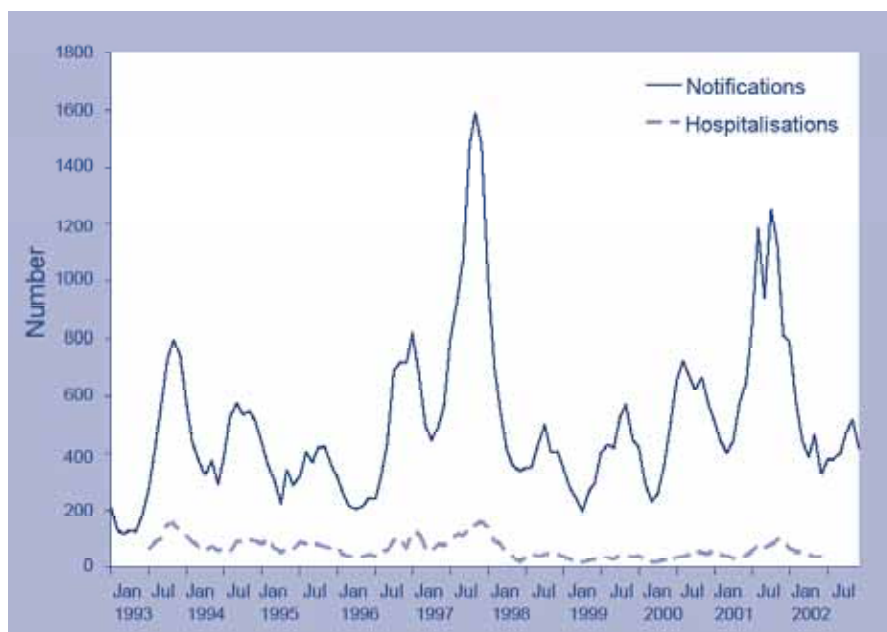
The ICD-10-AM/ICD-10 code A37 (whooping cough) was used to identify hospitalisations and deaths.

## Secular trends

There were 14,717 notifications of pertussis received by the National Notifiable Diseases Surveillance System (NNDSS) with dates of onset in 2001 or 2002 (average annual rate 37.7 per 100,000) (Table 13). A median of 490 cases was notified each month (range 325–1,253). There was an epidemic year in 2001, with nearly two-thirds of the cases ( $n=9,167$ ; 62%), compared with 5,958 and 5,550 notifications for 2000 and 2002 respectively. Epidemic peaks have occurred every three to four years since national notifications were available in 1991. The national notification rate was 47.2 per 100,000 in 2001 compared with 31.1 and 28.2 per 100,000 for 2000 and 2002 respectively. This was the second highest national rate recorded since 1993, after the 1997 national rate of 58.9 per 100,000 with 10,828 notified cases. A clear seasonal pattern remained apparent, with the highest number of notifications in the spring and summer months (between August and February) each year between 1993 and 2002 (Figure 21).

Hospitalisations followed a similar pattern to notifications. There were 1,277 hospital separations coded as pertussis during the review period, 507 in 2000/2001 and 770 in 2001/2002 (Table 13 and Appendix 3). The median number of pertussis hospitalisations per month was 47 (range 29–98). The average annual national hospitalisation rate was 3.3 per 100,000 for this reporting period, compared with 2.0 per 100,000 for the previous two years 1998/1999 to 1999/2000.<sup>2</sup>

**Figure 21. Pertussis notifications and hospitalisations, Australia, 1993 to 2002,\* by month of onset or admission**



\* Notifications where the month of onset was between January 1993 and December 2002; hospitalisations where the month of admission was between 1 July 1993 and 30 June 2002.

**Table 13. Pertussis notifications, hospitalisations and deaths, Australia, 2000 to 2002,\* by age group**

Age group (years)	Notifications 2 years (2001–2002)		Hospitalisations 2 years (July 2000–June 2002)				LOS <sup>†</sup> per admission (days) Median	Deaths 2 years (2001–2002)	
	n	Rate <sup>‡</sup>	n	(  )	Rate <sup>‡</sup>	(  )		n	Rate <sup>‡</sup>
0–4	1,314	51.5	887	794	34.6	31.0	3	5	0.2
5–14	4,569	84.3	138	107	2.6	2.0	2	0	–
15–24	2,112	39.5	34	17	0.6	0.3	1.5	0	–
25–59	5,591	29.2	152	99	0.8	0.5	4	0	–
60+	1,125	17.1	66	37	1.0	0.6	5	1	0.0
All ages <sup>§</sup>	14,717	37.7	1,277	1,054	3.3	2.7	3	6	0.0

\* Notifications where the month of onset was between January 2001 and December 2002; hospitalisations where the month of separation was between 1 July 2000 and 30 June 2002; deaths where the date of death was recorded between 2001 and 2002.

† LOS = length of stay in hospital.

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

§ Includes cases with unknown ages.

|| Principal diagnosis hospitalisations.

### Severe morbidity and mortality

There were 7,087 hospital bed days recorded with an ICD-10-AM code for pertussis between July 2000 and June 2002 (2,508 for 2000/2001 and 4,579 for 2001/2002). The median length of stay per admission was three days (Table 13). Of the 1,277 hospitalisations, 1,054 (83%) had a principal diagnosis of pertussis (average annual rate 2.7 per 100,000). The discharge diagnosis code A37.0 (*B. pertussis*) was recorded for 474 (37%) hospitalisations and was the principal diagnosis for 397 (84%) of these. *Bordetella parapertussis* (A37.1) was recorded for 11 hospitalisations, and other *Bordetella* species (A37.8) for nine hospitalisations. The remaining 783 (61%) hospitalisations were coded as whooping cough (organism unspecified – A37.9), and this was the principal diagnosis for 640 (82%) of these.

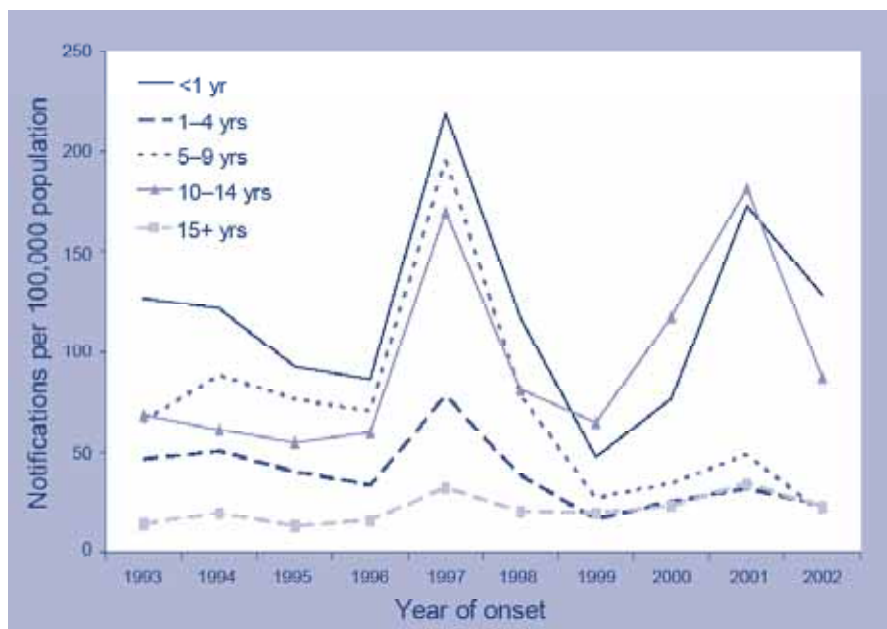
For the two years 2001 to 2002, six deaths were recorded where pertussis was the underlying cause (Table 13). Two occurred in 2001 and four in 2002. All but one, who was 85 years of age, were two months old or younger. The 85-year-old case is rather atypical and could be a coding error. Between 1993 and 2000 there were 10 deaths attributed to pertussis: all were younger than 12 months of age; six occurred in 1997.<sup>1,2</sup>

### Age and sex distribution

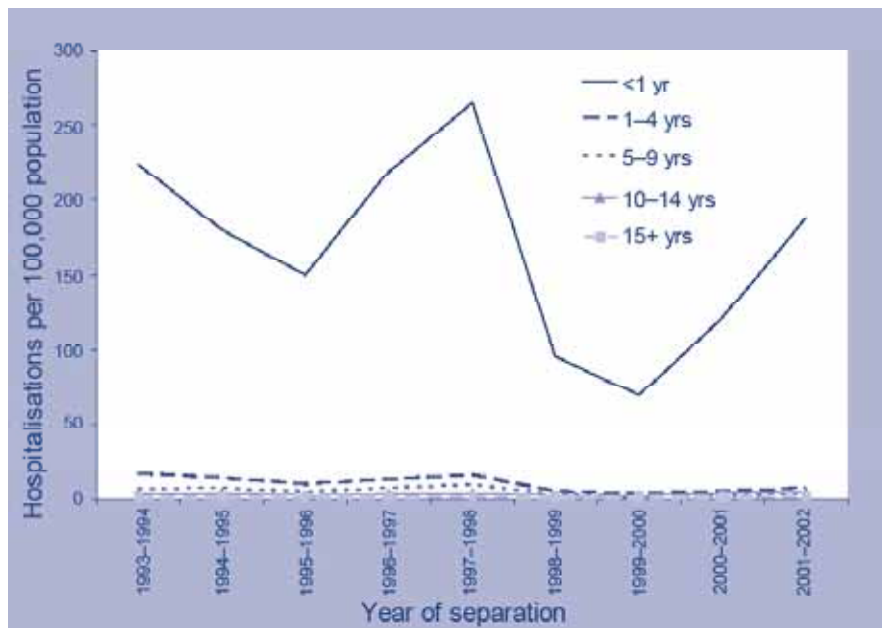
The highest notification rates were seen in infants aged less than one year and adolescents 10–14 years of age (Figure 22) with annual average rates of 172.7 and 181.1 per 100,000, respectively. In the two year review period, infants aged less than one year accounted for five per cent of all notifications (n=752) but 61 per cent of hospitalisations (n=778). The average hospitalisation rate for infants was 154.1 per 100,000 in this reporting period compared with 82.0 per 100,000 for the previous two years, 1998/1999–1999/2000) (Figure 23).<sup>2</sup>

The 10–14 year age group accounted for 25 per cent of pertussis notifications in 2001 and 2002 (n=3,646) and seven per cent of all hospitalisations (n=88; average hospitalisation rate of 3.3 per 100,000, twice the average rate of 1.6 per 100,000 for the period 1998/1999–1999/2000).<sup>2</sup> The 10–14 year age group had higher notification rates than any other 5-year age group for each year 2001 and 2002, at about three to four times those of the 5–9 year age group. This contrasts with 1994 and 1995, when the rates for 5–9 year olds were approximately 40 per cent higher than the rates for 10–14 year olds (Figure 22).

**Figure 22. Pertussis notification rates, Australia, 1993 to 2002,\* by age group**



\* Notifications where onset was between 1 January 1993 and 31 December 2002.

**Figure 23. Pertussis hospitalisation rates, Australia, 1993 to 2002,\* by age group**

\* Hospitalisations where separation was between 1 July 1993 and 30 June 2002.

People aged 15 years or more (adults) accounted for 60 per cent of notifications in 2001 and 2002 ( $n=8828$ ) and 20 per cent of hospitalisations ( $n=252$ , with an average annual hospitalisation rate of 0.8 per 100,000 compared with 0.6 per 100,000 for the previous two years 1998–2000).<sup>1,2</sup> While the percentage of notifications for this age group is comparable to that in the previous two years, 1999 and 2000 (62%), it has increased since 1993–1998 where people aged 15 years and over accounted for only 46 per cent of notifications.<sup>1,2</sup> The median age of pertussis notifications increased from 13–15 years in 1993–1998 to 23 years in 2002. This could be partly related to the increased use, especially in adults, of serology as a diagnostic tool. Hospitalisations in adults are most likely to be related to complications, but could also be falsely inflated because of coding errors.

The overall male:female ratio was 1:1.2 for notifications and 1:1.1 for hospitalisations. Higher rates among females were apparent in most age groups for both notifications and hospitalisations.

### Geographical distribution

There was a large variation in notification (Appendix 2) and hospitalisation rates (Appendix 3) between regions and years. As already mentioned, Australia experienced a pertussis epidemic in 2001, reflected by significant increases in notification rates in all jurisdictions except the Australian Capital Territory and Tasmania. The highest notification rate in 2001 occurred in South Australia at 133.0 per 100,000 population, followed by the Northern Territory (72.3 per 100,000) and New South Wales (64.5 per 100,000). Rates for other jurisdictions ranged from 41.5 per 100,000 in Queensland to 11.9 per 100,000 in Western Australia. In 2002, the highest notification rates were in Queensland, South Australia and New South Wales (49.0, 31.1 and 30.3 per 100,000, respectively) with the lowest rates in Western Australia and Tasmania (11.9 and 8.7 per 100,000, respectively).

### Comment

Since 1993, pertussis has caused the greatest morbidity of any disease preventable by vaccines recommended for children on the Australian Standard Vaccination Schedule (ASVS). The highest numbers of pertussis notifications were seen in 1997, with most jurisdictions experiencing an epidemic in that year, followed by 2001. Notification rates are known to underestimate incidence; this is illustrated by the finding that hospitalisations in infants aged less than one year have exceeded notifications for the two year period 2001–2002, as for the previous two year review 1999–2000.<sup>2,118</sup> In children, hospitalisations coded as whooping cough have been shown to have a high correlation with clinical pertussis.<sup>119</sup> The high proportion (greater than 50%) of hospital-

ised cases aged less than one year is consistently observed over the years and demonstrates the increased morbidity of pertussis in this age group. Mortality is also highest in infants, with five of the six deaths recorded for 2001–2002 aged less than three months old.<sup>120</sup>

Nationally, the highest notification rates up to 1998 inclusive were among children aged less than one year, followed closely by children aged 5–9 and 10–14 years (Figure 22). Since 1999, notification rates have fallen significantly among 5–9 year olds, reflecting the impact of the fifth dose of pertussis vaccine, introduced since 1994 for four year olds because of waning immunity over time. Incidence rates among 10–14 year olds were highest until 2001 and have been experiencing a downward trend since 2002; this is also likely to be related to the impact of the fifth dose of pertussis vaccine reaching this older cohort. In 2002 and 2003, as for the years 1993 to 1998, infants under one year of age again had the highest incidence. This moving cohort effect has been recently described nationally,<sup>121</sup> in New South Wales<sup>122</sup> and internationally.<sup>123</sup>

In essence, pertussis is now a problem in two broad age groups: infants with the highest notification and hospitalisation rates, particularly those under six months who are too young to have received two or more doses of DTPa, and people aged 15 years and over, who account for 60 per cent of pertussis notifications. The latter is explained by a combination of low historical coverage (whole-cell vaccine safety concerns in the 1970s and 1980s), and waning immunity (cohort not eligible for school entry booster dose) as well as improved diagnosis and reporting in this age group.<sup>124</sup> To address this problem, and following the approval in 2001 of an acellular adult-formulated vaccine (dTpa),<sup>125</sup> the Australian Standard Vaccination Schedule (ASVS) was changed in September 2003, with the 18-month booster no longer recommended and the dTpa booster replacing adult diphtheria-tetanus (ADT) at 15–17 years of age.<sup>49</sup> The 18-month DTPa booster was removed from the ASVS because of evidence that three doses of acellular pertussis vaccine in the first year of life provide good (>80%) protection until the age of six years.<sup>126</sup> The change should also reduce the frequency of local reactions following the booster dose at four years.<sup>127</sup> A booster dose of dTpa is now also recommended for new parents or parents planning a pregnancy and for health care workers, which may in time have an impact on neonatal cases.

## Pneumococcal disease

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

### Case definitions

#### Notifications

Invasive pneumococcal disease has been notifiable in Queensland and the Northern Territory since 1997. From January 2001, invasive pneumococcal disease became notifiable Australia wide with cases identified by:

a) Isolation of *Streptococcus pneumoniae* by culture from a normally sterile site

or

b) Detection of *Streptococcus pneumoniae* from a normally sterile site by nucleic acid testing.

#### Hospitalisations

The ICD-10-AM codes used to identify hospitalisations were: G00.1, pneumococcal meningitis; A40.3, pneumococcal septicaemia (together considered to be a proxy for invasive pneumococcal disease) and J13, pneumococcal pneumonia. To avoid double counting, cases were identified in a hierarchical fashion. First, all those with code G00.1 were classified as meningitis, then those without G00.1 but with A40.3 were classified as septicaemia without meningitis and then those with neither of these codes but with code J13 were counted as pneumococcal pneumonia.

#### Deaths

ICD-10 codes G00.1, A40.3 and J13 were used to select deaths from IPD.

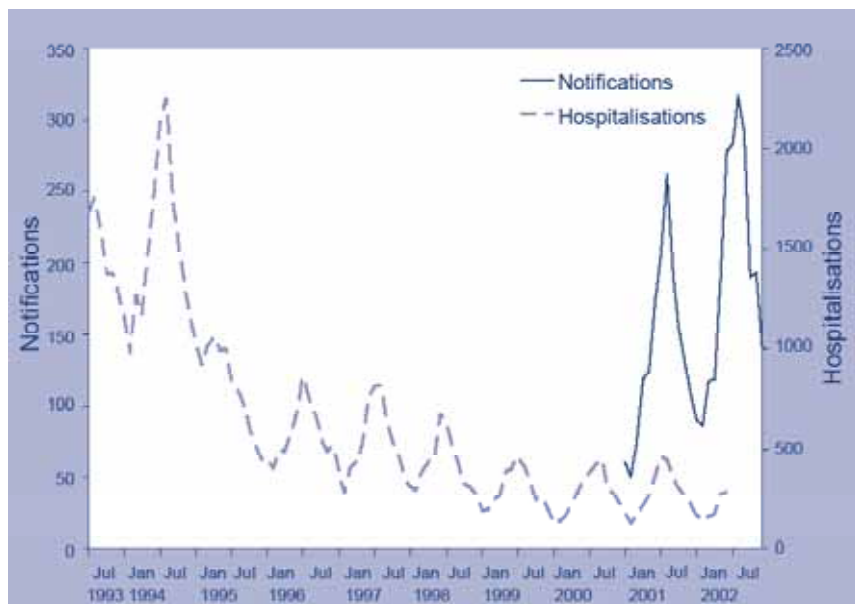
## Secular trends

Although only some States and Territories were notifying invasive pneumococcal disease for the entire period in 2001, a total of 3,951 notifications was received for the two year period (1,657 in 2001 and 2,294 in 2002). If only States and Territories which notified for the entire 2001 calendar year are included (all jurisdictions except Victoria and South Australia), there were 3,512 notified cases of invasive pneumococcal disease (IPD) with dates of onset in 2001 and 2002, an average annual notification rate of 10.7 per 100,000 (Table 14). In both years, there was a winter peak in pneumococcal notifications in August (Figure 24).

The total number of hospitalisations coded as pneumococcal meningitis, septicaemia or pneumonia for 2001 to 2002 was 6,782, an average annual rate of 17.6 per 100,000 (Table 14). Hospitalisations coded as meningitis or septicaemia accounted for 31 per cent of total episodes giving a hospitalisation rate of 5.5 per 100,000. The median number of hospitalisations per month was 82.5 for meningitis or septicaemia (predominantly septicaemia) and ranged from 31 to 148. For pneumococcal pneumonia the median number of hospitalisations per month was 178 and ranged from 103 to 326. Meningitis and septicaemia showed a clear winter peak each year, which was also present but less evident for pneumonia (Figure 25).

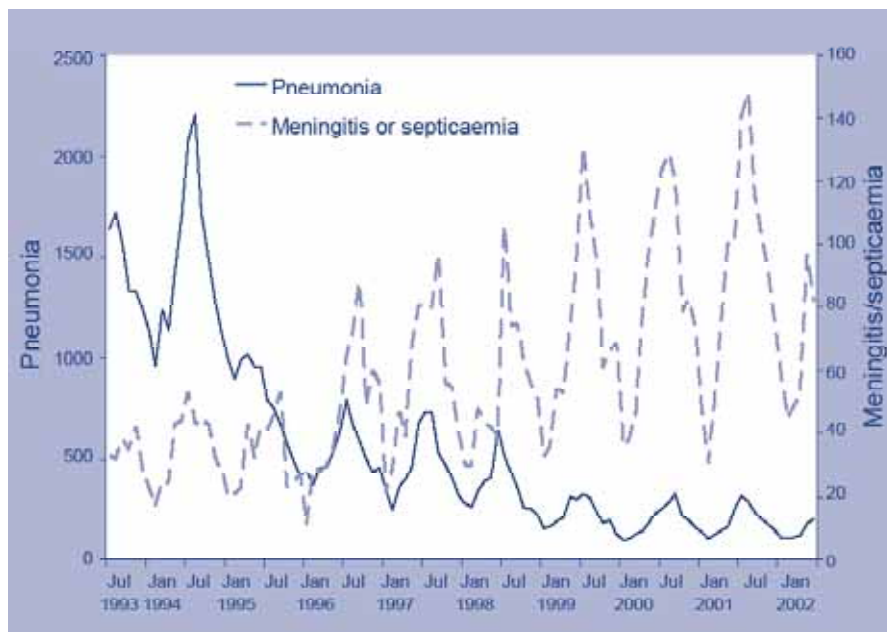


**Figure 24. Pneumococcal disease notifications and hospitalisations, Australia, January 1993 to December 2002,\* by month of onset or admission**



\* Notifications where the month of onset was between January 2001 and December 2002; hospitalisations where the month of admission was between 1 January 1993 and 30 June 2002. Hospitalisations include pneumonia, meningitis and septicaemia.

**Figure 25. Pneumococcal disease hospitalisations, Australia, July 1993 to June 2002,\* by month of admission**



Note: varying scales between pneumonia and meningitis/septicaemia hospitalisations.

\* Hospitalisations where the month of admission was between 1 July 1993 and 30 June 2002.

**Severe morbidity and mortality**

A total of 72,317 hospital bed days (average 36,159 days per year) was recorded for hospital separations with an ICD-10-AM code corresponding to pneumococcal meningitis, septicaemia or pneumonia. Length of stay increased with age in all categories of infection (Table 14). The average length of stay for pneumococcal meningitis was 10 days in all age groups (data not shown), more than double that for septicaemia or pneumonia in younger age groups.

The mortality rate for meningitis and septicaemia from death certificate data (Table 14) was low across all age groups, but when pneumococcal pneumonia was included, the mortality rate was higher in people over 60 years (Table 14, Figure 26).

**Table 14. Pneumococcal disease notifications, hospitalisations and deaths, Australia, 2000 to 2002,\* by age group**

Age group (years)	Notifications 2 years (2001–2002)		Hospitalisations 2 years (July 2000–June 2002)				LOS <sup>†</sup> per admission (days)		Deaths 2 years (2001–2002)			
	n <sup>‡</sup>	Rate <sup>§</sup>	n <sup>  </sup>	(M/S) <sup>¶</sup>	Rate <sup>  §</sup>	(M/S) <sup>¶§</sup>	Median <sup>  </sup>	(M/S) <sup>¶</sup>	n <sup>  </sup>	(M/S) <sup>¶</sup>	Rate <sup>  §</sup>	(M/S) <sup>¶§</sup>
0–4	1,173	54.5	1,092	(688)	42.6	(26.9)	3	(3)	8	(5)	0.3	(0.1)
5–14	184	4.0	271	(109)	5.0	(2.0)	3	(3)	2	(1)	0.0	(0.0)
15–24	133	3.0	230	(53)	4.4	(1.0)	4	(5)	1	(1)	0.0	(0.0)
25–59	969	6.0	2,150	(490)	11.4	(2.6)	5	(7)	25	(9)	0.1	(0.0)
60+	1,053	19.2	3,039	(771)	47.2	(12.0)	8	(10)	44	(13)	0.7	(0.1)
All ages**	3,512	10.7	6,782	(2,111)	17.6	(5.5)	6	(6)	80	(29)	0.2	(0.0)

\* Notifications where the month of onset was between January 2001 and December 2002; hospitalisations where the month of separation was between 1 July 2000 and 30 June 2002; deaths where the date of death was recorded between 2001 and 2002.

† LOS = length of stay in hospital.

‡ Victoria and South Australia not included in 2001.

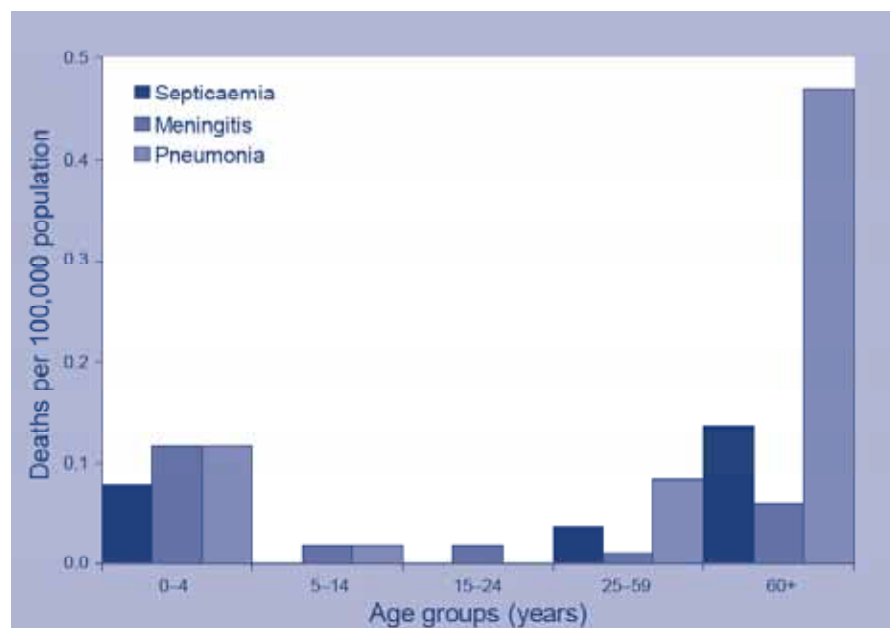
§ Average annual age-specific rate per 100,000 population.

|| All pneumococcal disease.

¶ (M/S) = meningitis and septicaemia.

\*\* Includes cases with unknown ages.

**Figure 26. Pneumococcal meningitis, septicaemia and pneumonia death rates, Australia, 2001 to 2002,\* by age group**



\* Measured using AIHW Mortality data where the date of death was recorded between 2001 and 2002.

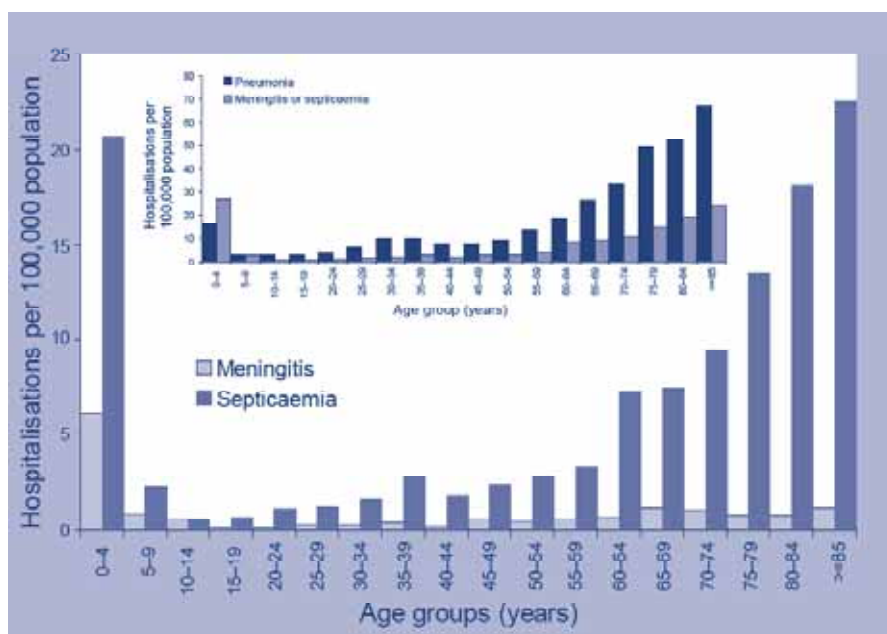
### Age and sex distribution

The hospitalisation rate for each age group varied with the focus of infection (Figure 27). For meningitis, children aged 0–4 years had the highest hospitalisation rate (6.1 per 100,000), with those less than one year of age having an incidence almost four times higher (15.3 per 100,000) than those 1–4 years of age (3.9 per 100,000). The annual hospitalisation rate for meningitis was lower among 5–9 year olds (1 per 100,000) and did not increase to this level again until over 65 years of age. By contrast, the incidence of hospitalisation for septicaemia without meningitis increased dramatically from the age of 60 years, so that the total incidence of septicaemia and meningitis was highest in those over 80 years.

Overall, 28.5 per cent of hospitalisations coded as pneumococcal septicaemia were also coded as pneumonia. The proportion varied with age, with 8.5 per cent of hospitalisations coded as septicaemia without meningitis also coded as pneumonia among 0–4 year olds, rising to 43.1 per cent among 25–59 year olds.

When total hospitalisations (meningitis, septicaemia and pneumonia) were considered, adults aged 60 years or more had the highest total rate of hospitalisation (47.2 per 100,000, Table 14). The male:female ratio varied with age. There was a strong predominance of male cases coded as meningitis or septicaemia for ages 40–49 years, but among those 65 years and over the male:female ratio was lower. However, when calculated as rates, males over the age of 65 years had a hospitalisation rate of 17.3 per 100,000, compared with 14.1 per 100,000 among females.

**Figure 27. Pneumococcal meningitis, septicaemia and pneumonia hospitalisation rates,\* Australia, 2000 to 2002, by age group**



\* Hospitalisations where the month of separation was between 1 July 2000 and 30 June 2002

### Geographical distribution

The average annual notification rate was 10.7 per 100,000 for Australia, increasing from 9.3 to 11.7 at a national level between 2001 and 2002 and ranging from 8.0 to 13.1 except in the Northern Territory where it was 39.9. The average annual hospitalisation rate for meningitis or septicaemia (26.4 per 100,000) in the Northern Territory was more than fourfold higher than in any other jurisdiction (Appendix 3). The average annual hospitalisation rate for other States and Territories ranged from 4.0 to 5.7 per 100,000.

### Comment

Invasive pneumococcal disease has become notifiable in all jurisdictions from the beginning of 2001, with national notification data published in *Communicable Disease Intelligence* annually for 2001<sup>128</sup> and 2002.<sup>129</sup> Recommendations for the use of pneumococcal vaccines and the funding arrangements for pneumococcal vaccines vary by vaccine type, age group and ethnicity. One significant change occurred in 2001, when a publicly funded 7-valent conjugate pneumococcal vaccine (7vPCV) program commenced for children at high risk, defined as Aboriginal and Torres Strait Islander children under two years and children with predisposing medical conditions under five years of age.<sup>49</sup> The 23-valent polysaccharide pneumococcal vaccine (23vPPV) has been recommended and funded for Aboriginal and Torres Strait Islander people over the age of 50 years since 1997. It is also recommended for non-Aboriginal people 65 years and older, but is funded only in Victoria.<sup>130</sup> The recommendation for universal 7vPCV vaccination in children under two years of age and universal 23vPPV for adults over 65 years of age in September 2003,<sup>49</sup> which received public funding to commence at the beginning of 2005, will heighten the need for close scrutiny of the impact of pneumococcal vaccines on disease over the coming years.

The hospitalisation rates reported here are based on a narrow case definition. While underestimating the incidence of invasive pneumococcal disease, the hospitalisation rates for cases coded as either meningitis or septicaemia have risen from a mean of 2.2 per 100,000 for 1993–1998 to 4.5 for 1999–2000 and 5.5 per 100,000 in the current review period. This increase in hospitalisations so coded is likely to reflect changes in diagnostic and/or coding practices. The increase in notification rates seen in all jurisdictions except the Northern Territory is also likely to be due to greater completeness of notification rather than any real increase. The substantial decrease in the Northern Territory has been attributed to unusually high notification rates in 2001, out of keeping with previous years.<sup>128</sup> Estimates of overall incidence from laboratory surveillance in industrialised countries comparable to Australia range from 9 to 22 per 100,000 per year.<sup>131</sup> The dramatic fall in the number of hospitalisations coded as pneumococcal pneumonia without septicaemia, since 1993 (Figure 25), is also likely to be attributable to coding practices and has remained relatively constant since 1999. Additional evidence for a change in coding comes from the fact that all-cause pneumonia hospitalisations did not change over this period (data not shown).

The death rate per 100,000 population from meningitis, based on death certificate data, was substantially lower for 2001–2002 (0.1) than in 1998–2000 (0.6) and similar to that seen in persons over 60 years of age (Table 14). It is highest in children under the age of five years and especially in infants. As expected, the death rate for all categories of pneumococcal infection, including pneumonia, was highest in those over 60 years. The Northern Territory had the highest rate of notification for IPD as well as the highest rate of hospitalisation for codes corresponding to presumed IPD, shown by data from enhanced surveillance to be almost entirely due to a high incidence among Aboriginal and Torres Strait Islander people.<sup>132</sup> Aboriginal people are also known to have high rates of pneumococcal disease in Western Australia<sup>133</sup> and north Queensland, where the polysaccharide vaccine program targeting Indigenous adults has been associated with significant reductions in IPD.<sup>134</sup> As evaluations of the impact of 23vPPV in Indigenous populations who live in arid areas, such as the Navajo in South-Western USA, have not shown such a high degree of effectiveness,<sup>135</sup> it will be important to examine whether this is also the case in the Northern Territory, especially in Central Australia. The introduction of universal programs for pneumococcal vaccination in certain age groups nationally from 2005 will make careful surveillance of IPD even more important in the future, both from the point of view of possible herd immunity effects in age groups not targeted for vaccination and the potential for serotype replacement.

## Poliomyelitis

Poliomyelitis is caused by an enterovirus, poliovirus. Infection involves the gastrointestinal tract, and may progress to the nervous system, resulting in paralysis. Acute flaccid paralysis (AFP) occurs in less than 1 per cent of infections. More than 90 per cent of 'asymptomatic' cases are characterised by a mild febrile illness. The maximum extent of paralysis is usually reached within 3–4 days of disease onset. Any paralysis still present after 60 days is likely to be permanent.<sup>16</sup>

Vaccine-associated paralytic poliomyelitis (VAPP) is acute flaccid paralysis due to a Sabin-like poliovirus (i.e. a virus similar to that used in the live oral Sabin vaccine).

### *Case definitions*

#### **Notifications**

Acute-onset flaccid paralysis of one or more limbs with decreased or absent tendon reflexes in the affected limbs without apparent cause, and without sensory or cognitive loss.

#### **Hospitalisations**

The ICD-10-AM code A80 (acute poliomyelitis) was used to identify hospitalisations.

**Note:** This code includes VAPP and specific codes for indigenous and imported wild-type polio virus infection.

#### **Deaths**

The ICD-10 code A80 (acute poliomyelitis) was used to identify deaths.

## Notifications, hospitalisations and deaths

No notifications or deaths were recorded for poliomyelitis in 2001 or 2002. From July 2000 to June 2002 there were 34 hospitalisations with a diagnosis of acute poliomyelitis (Appendix 3). Of these, one was coded as VAPP and one as acute non-paralytic poliomyelitis. The remaining hospitalisations were coded as acute unspecified poliomyelitis. Only three hospitalisations were recorded as having a principal diagnosis of poliomyelitis.

## Comment

It is unclear exactly when the last case of locally acquired poliomyelitis occurred in Australia. The last laboratory-confirmed case was in 1967. Three clinically compatible cases were notified in 1972: however, no additional information is currently available.<sup>136</sup> All cases notified since 1972 have been fully investigated with subsequent reclassification as VAPP. The most recent case of VAPP was reported in 1995.<sup>137</sup> In the two year review period 2001–2002 the Australian National Poliovirus Reference Laboratory isolated a Sabin-like poliovirus from two children presenting with AFP. In both cases the virus was determined to be an incidental finding.<sup>138,139</sup>

As there have been no reports of indigenous wild-type poliovirus transmission in Australia for at least 30 years, the hospitalised cases reported here are almost certainly not missed notifications of acute wild-type polio infection. Some hospitalisations could represent cases of AFP where poliomyelitis could not be excluded, but most are likely to be adults with late effects of poliomyelitis rather than acute cases.

It is worth noting that the number of hospitalisations coded with acute poliovirus in this review period is lower than the 90 admissions coded during the previous two year review period. This may be related to the change from ICD-9-CM to ICD-10-AM, or to improved coding practices, although coding standards remained unchanged over this period (Sue Walker, National Centre for Classification in Health, personal communica-

tion). However, the apparent discrepancy with the absence of polio notifications for the two year review period and the hospitalisation case coded as VAPP would be worthwhile investigating, although it could be a coding error.

Global efforts to eradicate poliomyelitis have been considerable. In September 2002, the European region of the World Health Organization (WHO) was the third of six regions to be declared free of indigenous wild poliovirus.<sup>140</sup> The region of the Americas was declared polio free in 1994 and the Western Pacific Region in October 2000.<sup>140,141</sup> Endemic transmission of wild-type poliovirus is now constrained to seven countries, with Nigeria, Pakistan and northern India accounting for most of the disease burden.<sup>140,142</sup>

Although Australia has been declared polio free, high vaccination coverage and improved active surveillance of acute flaccid paralysis are required. For the first time in 2000 and 2001, Australia met the WHO target for surveillance of AFP (one notified case of AFP per 100,000 children aged less than 15 years).<sup>138</sup> However, in 2002 the rate was only 0.83 per 100,000 and the WHO target for laboratory testing of AFP cases has never been achieved.<sup>139</sup> High quality acute flaccid paralysis surveillance is required to detect any imported cases of wild-type polio infection, cases of VAPP, and outbreaks of circulating vaccine-derived polioviruses (cVDPV). Outbreaks have now been reported in Egypt (1988–1993), the Dominican Republic and Haiti (2000), the Philippines (2001), and most recently in Madagascar (2002).<sup>142</sup>

One way to prevent VAPP and cVDPVs from emerging is to use inactivated poliovirus vaccine (IPV) rather than the live OPV. Recently WHO issued a position paper on changing from OPV to IPV<sup>140</sup> and many developed countries have already made the switch because the risk of vaccine-associated poliomyelitis was considered to outweigh that from natural infection. In 2003, the Australian Government recommended, but did not fund, the use of IPV in place of OPV.<sup>49</sup> IPV is safe and effective, but requires an injection and is currently more costly than the oral vaccine, OPV. Therefore consideration of the public's risk perception and the cost-effectiveness of competing alternatives are required to guide future funding arrangements in Australia.

## Rubella

Rubella is caused by the rubella virus (family togaviridae). It is usually a mild febrile viral disease with a rash sometimes resembling that of measles or scarlet fever. More severe disease manifestations, such as arthritis and encephalitis, also occur. Rubella is important because of its ability to produce abnormalities in the developing fetus (congenital rubella syndrome).<sup>16</sup>

### *Case definitions*

#### **Notifications**

A generalised maculopapular rash, fever, and one or more of arthralgia/arthritis or lymphadenopathy or conjunctivitis, and an epidemiological link to a confirmed case

**or**

Demonstration of rubella-specific IgM antibody, except following vaccination

**or**

A fourfold or greater rise in rubella antibody titre between acute and convalescent phase sera obtained at least two weeks apart

**or**

Isolation of rubella virus from a clinical specimen.

#### **Hospitalisations and deaths**

The ICD-10-AM/ICD-10 code B06 (rubella [German measles]) was used to identify hospitalisations and deaths.

Congenital rubella cases were not included in this report. Reviews of congenital rubella cases recorded by the Australian Paediatric Surveillance Unit between 1993 and 2001 are available elsewhere.<sup>143–145</sup>

## Secular trends

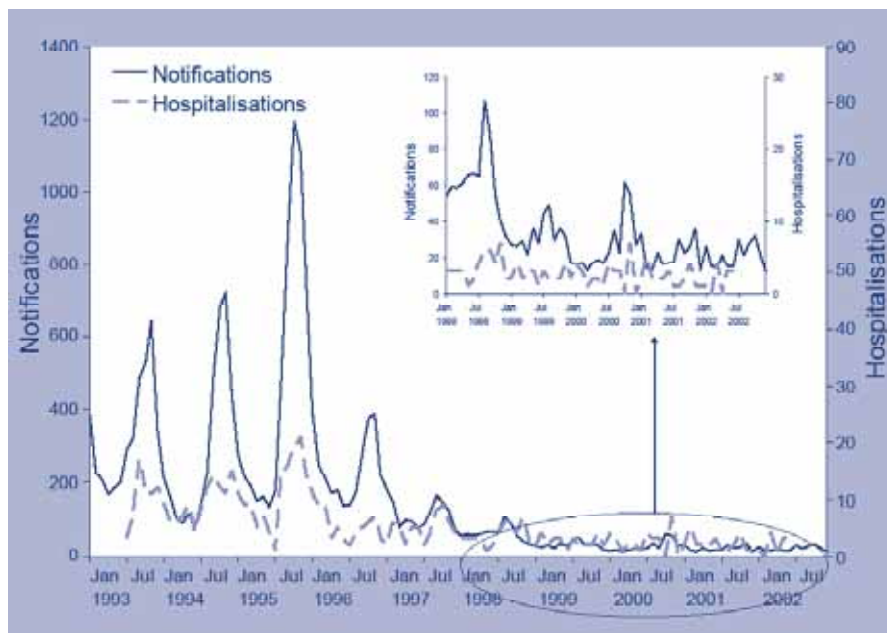
During 2001–2002 there were 511 notified cases of rubella, an average annual notification rate of 1.3 per 100,000 (Table 21). Between July 2000 and June 2002, 54 hospitalisations were coded as being due to rubella (an average annual rate of 0.1 per 100,000). Notification and hospitalisation rates were the lowest on record in the most recent review year, continuing the downward trend from a peak seen in the Spring of 1995 (Figure 28, Appendices 2 and 3). Activity was still highest during the spring months, but the peaks were less pronounced.

## Severe morbidity and mortality

One hundred and forty-two hospital bed days (average 71 per year) were recorded for patients with an ICD-10-AM code for rubella. Of the 54 hospital separations, 28 (52%) had a principal diagnosis of rubella (average annual rate 0.1 per 100,000). The median length of stay in hospital was one day, but varied with age (Table 15). In 2001 to 2002, there were no deaths with rubella recorded as the underlying cause.

Complications arising from rubella infection were recorded for 18 (33%) hospitalisations (Table 16). There were no recorded complications for children aged less than 15 years. The 25–59 year age group accounted for 83 per cent of the complications but only 32 per cent of the notifications and 39 per cent of the hospitalisations.

Figure 28. Rubella notifications and hospitalisations, Australia, 1993 to 2002,\* by month of onset or admission



Note: varying scales between notifications and hospitalisations.

\* Notifications where the month of onset was between January 1993 and December 2002; hospitalisations where the month of admission was between 1 July 1993 and 30 June 2002.

Table 15. Rubella notifications, hospitalisations and deaths, Australia, 2000 to 2002,\* by age group

Age group (years)	Notifications 2 years (2001–2002)		Hospitalisations 2 years (July 2000–June 2002)				LOS† per admission (days)	Deaths 2 years (2001–2002)	
	n	Rate‡	n	(  )	Rate‡	(  )	Median	n	Rate‡
0–4	31	1.2	13	(4)	0.5	(0.2)	2	0	–
5–14	15	0.3	5	(4)	0.1	(0.1)	1	0	–
15–24	289	5.4	10	(6)	0.2	(0.1)	2.5	0	–
25–59	165	0.9	21	(12)	0.1	(0.1)	1	0	–
60+	11	0.2	5	(2)	0.1	(0.0)	3	0	–
All ages§	511	1.3	54	(28)	0.1	(0.1)	1	0	–

\* Notifications where the month of onset was between January 2001 and December 2002; hospitalisations where the month of separation was between 1 July 2000 and 30 June 2002; deaths where the date of death was recorded between 2001 and 2002.

† LOS = length of stay in hospital.

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

§ Includes cases with unknown ages.

|| Principal diagnosis (hospitalisations).



**Table 16. Indicators of severe morbidity for hospitalised cases of rubella, Australia, 2000 to 2002,\* by age group**

Age group (years)	Complication neurological		Complication other	
	n	% total	n	% total
0–4	0	0.0	0	0.0
5–14	0	0.0	0	0.0
15–24	1	10.0	1	10.0
25–59	11	52.4	4	19.0
60+	0	0.0	1	20.0
All ages	12	22.2	6	11.1

\* Measured using National Hospital Morbidity data where the month of hospital separation was between 1 July 2000 and 30 June 2002.

### Age and sex distribution

For the two year review period, notification rates were highest in the 20–24 year age group (average annual rate 7.5 per 100,000—data not shown). However, it is notable that the corresponding rate in this age group was 12.0 per 100,000 for males, compared with 2.9 per 100,000 for females (data not shown). Notifications for 20–24 year olds have been increasing each year since 1999, and although lower than pre-1999 levels, were higher in 2002 than in any year since 1998, almost entirely attributable to notifications in males. In contrast, those aged less than 20 years had in 2002 the lowest notification rates on record (data not shown). The most notable declines have been in children aged less than 10 years. In 2002 there were no notifications from the 5–9 year age group and only six from the 0–4 year olds—a 96 per cent reduction from numbers in 1998. In contrast, in 2002, 82 per cent of notified cases were aged 1–34 years compared with only 48 per cent in 1998 (data not shown).

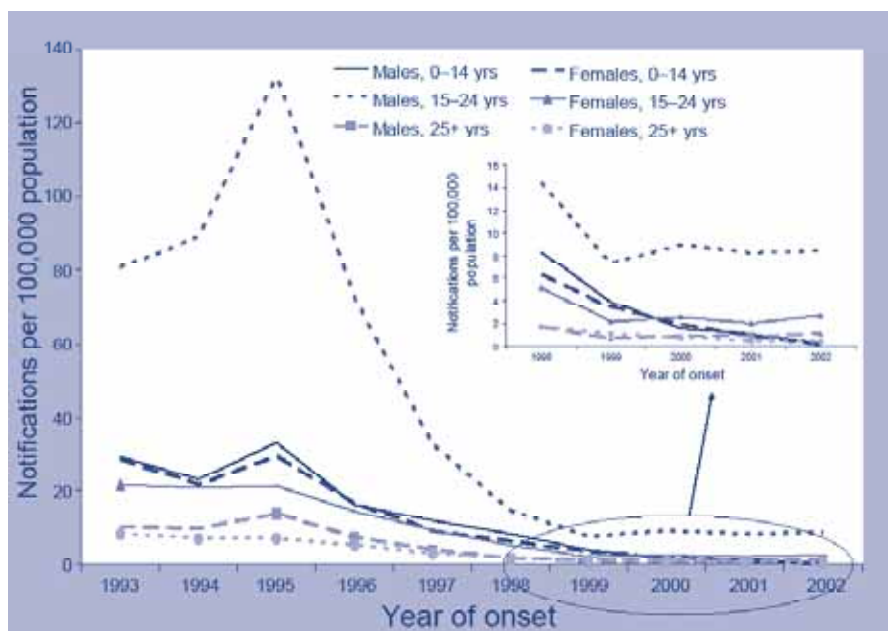
For the two years combined, 2000/2001 and 2001/2002, children aged 0–4 years continued to have the highest hospitalisation rates (average annual rate 0.5 per 100,000). However, as with notifications, hospitalisation rates in children less than 15 years, especially in those aged 0–4 years, have been declining since 1993 (data not shown). In 2001/2002 hospitalisation rates for 0–4 year olds were the lowest on record (rate of 0.5 per 10,000—data not shown). Although rates for other ages remained low during the review period, proportionally more hospitalised cases were from the 15–34 year age group than in past years. In 1998/1999, 26 per cent were aged 15–34 years and 42 per cent were aged 0–4 years, while in 2001/2002 48 per cent were aged 15–34 years and only 29 per cent were in the 0–4 year age group (data not shown).

As with measles, the declining rates of rubella in children and higher proportions in adults have led to an increase in the median age of both notified and hospitalised cases since the Measles Control Campaign (MCC) in 1998. In the most recent year reviewed, the median age for notified cases was 22 years, up from 18 years in 1998. Similarly, the median age of hospitalised cases in this two year review period was 24 years compared with a median of 6 years for the period 1993/1994 to 1997/1998 (data not shown).

In 2001–2002, the male to female ratio for notifications was 2.8:1; it has been increasing each year since 1999 when it was 1.4:1. The ratio was highest in young adults aged 20–24 (M:F ratio, 4.4:1) and 25–29 years (M:F ratio, 6.1:1)—age groups that also had amongst the highest notification rates. In contrast to the trend for notifications, the male to female ratio for hospitalisations has been declining, and for the first time there were more hospitalisations for females than males (M:F ratio 1:1.5). The sex ratio was equal for hospitalised cases aged less than 15 years but for older age groups there were more females.

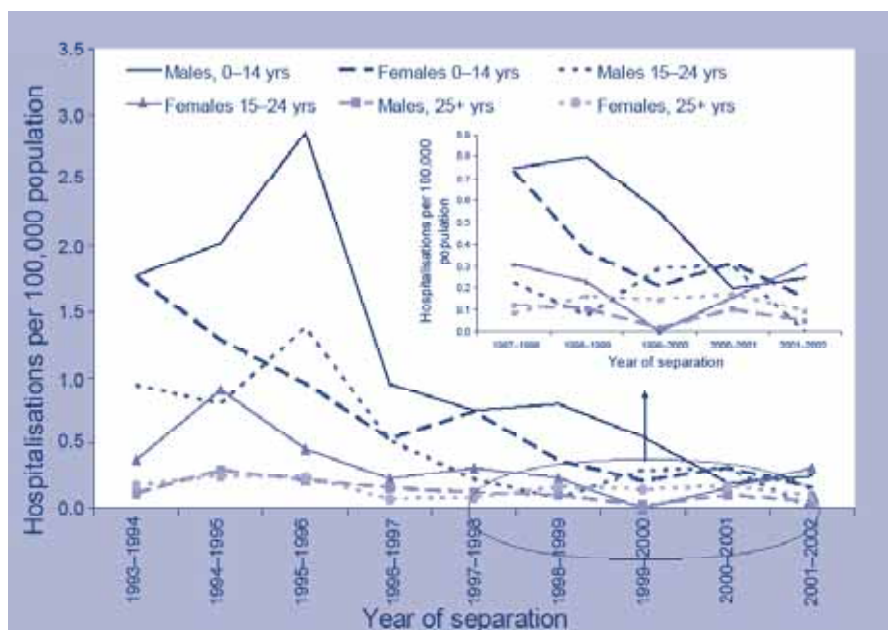
There were 95 notified cases of rubella in women of child bearing age (15–44 years) in 2001 and 2002, an average annual rate 1.1 per 100,000. This rate has been declining each year since the outbreak in 1995 (rate 16.0 per 100,000). However, in the three most recent years, only the 25–44 year age group has shown a decline. In 2002, rates for females in the 20–24 (2.9 per 100,000) and 15–19 year age group (2.4 per 100,000) were the highest of any female age group, and showed an increase compared with the rates in 2001.

Figure 29. Rubella notification rates, Australia, 1993 to 2002,\* by age group, sex and year of onset



\* Notifications where onset was between 1 January 1993 and 31 December 2002.

Figure 30. Rubella hospitalisation rates, Australia, 1993/1994 to 2001/2002,\* by age group, sex and year of separation



\* Hospitalisations where separation date was between 1 July 1993 and 30 June 2002.

## Geographical distribution

In the two year review period, notification rates were the lowest on record in all jurisdictions except Queensland (Appendix 2). From mid-2001 until the end of 2002 there was a sustained increase in notifications from Queensland.<sup>96</sup> In these two years, Queensland accounted for 62 per cent of the notifications and had by far the highest rate of any jurisdiction (average annual rate 4.3 per 100,000).

In stark contrast to notification trends, in the two year review period New South Wales had the highest proportion (72%) and rate of hospitalisation (average annual rate, 0.3 per 100,000; Appendix 3). New South Wales experienced an increased number of notifications in the second half of 2000, which might explain the high proportion of hospitalisations in 2000/2001. However, between July 2001 and June 2002, Queensland accounted for 67 per cent of the notifications but only 5 per cent of the hospitalisations.

## Comment

Rubella notification and hospitalisation rates continue to decline and in the most recent year reviewed were the lowest on record. The downward trend is due to considerable rate reductions in the 0–14 year age group, especially those aged 0–4 years, as a result of several recent vaccination initiatives. First, the mass vaccination of primary school aged children as part of the MCC;<sup>94</sup> second, lowering of the age for the second dose of measles-mumps-rubella (MMR) vaccine from age 10–16 years to age 4–5 years (and later four years); and finally, continued improvement in coverage with the first dose of MMR vaccine. The introduction of enhanced surveillance for rubella in Victoria in mid-2001 may also help to explain the lower rates—in the first year of enhanced surveillance, 67 per cent (67/100) of the notified cases with sera collected were laboratory-rejected as rubella.<sup>114</sup> Most of those rejected were vaccinated children, so the true incidence of rubella in children may be even lower than was reported nationally.

Notification rates have been the highest over the past two to three years in young adults aged 15–29 years, especially males aged 20–24 years. Young adult males may have missed being vaccinated as part of previous young adult or school-girl only programs, and many are too old to have been vaccinated as infants. Countries in the Americas that have had similar vaccination strategies to Australia have shown the same trend.<sup>146</sup> This trend is of concern, as young adult males (who have also been shown in Australian serosurveys to have a low level of immunity)<sup>147,148</sup> could act as a reservoir of infection for females of childbearing age. To improve immunity in both young adult males and females, the young adult MMR vaccination campaign was conducted during 2001.<sup>95</sup> However, there is evidence to suggest that coverage did not improve significantly<sup>96</sup> and that transmission to young adult females may have actually increased following the campaign—notification rates in females aged 15–24 years were higher in 2002 than in 2001 and in both years, for the first time, were the highest of any female age group (data not shown). In fact, in 2002 two females, aged 18 and 21 years, were infected when pregnant and gave birth to children with the congenital rubella syndrome (CRS) in 2003.<sup>149</sup> These were the first locally acquired cases of CRS since 1996.

Rubella and CRS are candidates for eradication after measles and polio.<sup>150</sup> Cuba has already eliminated rubella and CRS with mass vaccination campaigns targeting women aged 18–30 years and children aged 1–14 years.<sup>146</sup> Several other countries in the Americas have conducted similar mass vaccination campaigns either targeting adult women or both adult men and women, to achieve accelerated control.<sup>146</sup> In Costa Rica, a mass vaccination campaign in May 2001 targeting men and women aged 15–39 years achieved a national coverage above 95 per cent, and there have been no reported cases of rubella since August 2001.<sup>146</sup> Although Australia has already attempted to improve immunity in young adults by conducting the young adult MMR campaign, another mass adult vaccination campaign, such as those conducted in the Americas, may be needed to reduce susceptibility in young adults and enhance progress towards elimination in the near future. Such a campaign would be in addition to enhancing nationwide surveillance, maintaining high coverage in children, and continuing vaccination programs for susceptible females before pregnancy and after delivery.

## Tetanus

Tetanus is a disease induced by an exotoxin of the *Clostridium tetani* bacterium, which grows anaerobically at the site of an injury. The disease is characterised by painful muscle contractions, primarily of the masseter and neck muscles, secondarily of the trunk muscles. The case-fatality rate ranges from 10 per cent to 90 per cent, with the highest rates in infants and the elderly.<sup>16</sup>

### Case definitions

#### Notifications

A clinically compatible illness without other apparent cause, with or without a history of injury, and with or without laboratory evidence of the organism or its toxin.

#### Hospitalisations and deaths

The ICD-10-AM/ICD-10 code A033 (tetanus) was used to identify hospitalisations and deaths.

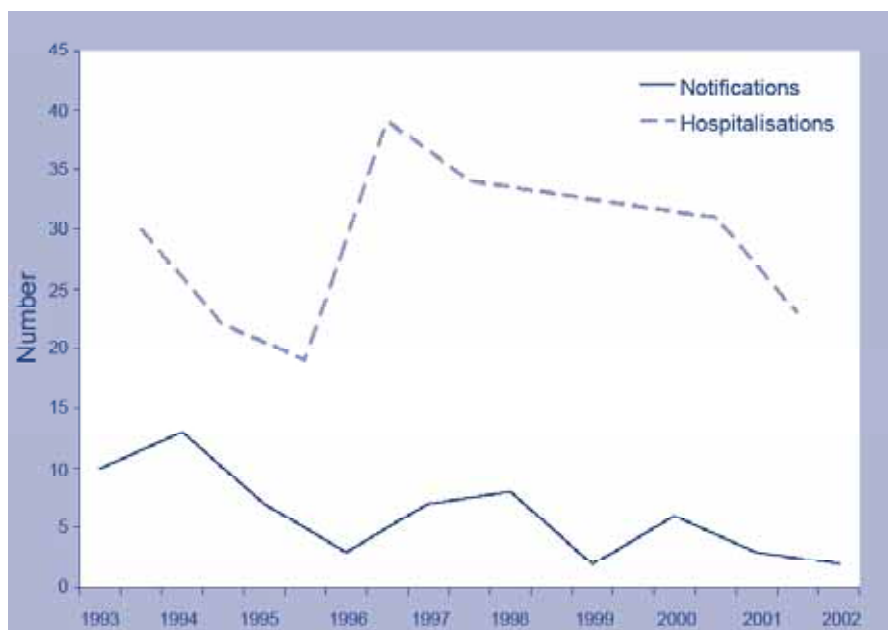
## Secular trends

There were five notifications of tetanus in the 2001 to 2002 review period (an average annual notification rate of 0.01 per 100,000). However, in the period July 2000 to June 2002, there were 54 hospitalisations coded as tetanus (an average annual rate of 0.14 per 100,000). Both notifications and hospitalisations for tetanus have been declining in the last two years (Figure 31).

## Severe morbidity and mortality

A total of 734 hospital bed days were recorded for patients with an ICD-10-AM code for tetanus. Of the 54 separations, 38 (70%) had tetanus recorded as the principal diagnosis. The median length of stay in hospital was three days and varied depending on age. Adults aged at least 60 years had longer median lengths of stay and accounted for the majority of hospitalisations (48%) (Table 17).

**Figure 31. Tetanus notifications and hospitalisations, Australia, 1993 to 2002,\* by year of onset or admission**



\* Notifications where the year of onset was between January 1993 and December 2002; hospitalisations where the month of admission was between 1 July 1993 and 30 June 2002.

**Table 17. Tetanus notifications, hospitalisations and deaths, Australia, 2000 to 2002,\* by age group**

Age group (years)	Notifications 2 years (2001 to 2002)		Hospitalisations 2 years (July 2000–June 2002)				LOS <sup>†</sup> per admission (days)	Deaths 2 years (2001–2002)	
	n	Rate <sup>‡</sup>	n	(  )	Rate <sup>‡</sup>	(  )	Median	n	Rate <sup>‡</sup>
0–4	0	–	1	1	0.0	0.0	16	0	–
5–14	0	–	1	0	0.0	–	1	0	–
15–24	0	–	8	6	0.2	0.1	1	0	–
25–59	1	0.0	18	13	0.1	0.1	3	0	–
60+	4	0.1	26	18	0.4	0.3	9.5	1	0.0
All ages <sup>§</sup>	5	0.0	54	38	0.1	0.1	3	1	0.0

\* Notifications where the month of onset was between January 2001 and December 2002; hospitalisations where the month of separation was between 1 July 2000 and 30 June 2002; deaths where the date of death was recorded between 2001 and 2002.

† LOS = length of stay in hospital.

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

§ Includes cases with unknown ages.

|| Principal diagnosis (hospitalisations).

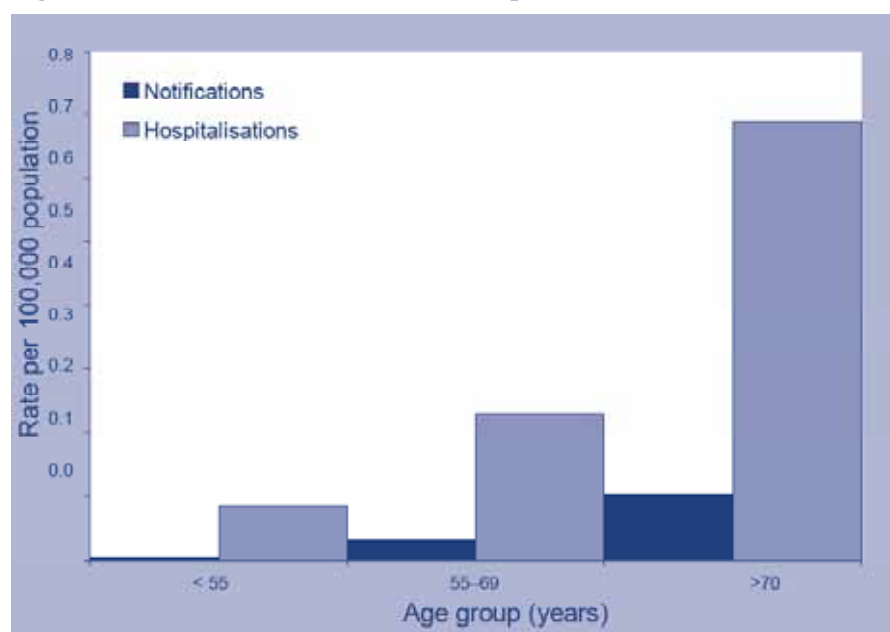
In the review period (2001 to 2002) there was one death in a person aged over 60 years with tetanus recorded as the underlying cause.

### Age and sex distribution

Most notified (4/5, 80%) and hospitalised (26/54, 48%) cases were aged at least 60 years. The youngest person notified was in his 40s, but the youngest person hospitalised was a child aged 0–4 years. There were two females notified with tetanus compared with three males, but more female hospitalisations, with a male:female ratio of 1:1.4. In the age group 70 years and over, 68 per cent of the hospitalised cases (13/19) were females.

For both notifications and hospitalisations, rates increased with increasing age (Figure 32). Females aged at least 70 years had the highest average annual hospitalisation rate (0.65 per 100,000).

**Figure 32. Tetanus notification and hospitalisation rates, Australia, 1998 to 2002,\* by age group**



\* Notifications where the month of onset was between January 1999 and December 2002; hospitalisations where the month of separation was between 1 July 1998 and 30 June 2002.

## **Geographical distribution**

Notification and hospitalisation rates varied over time and between States/Territories (Appendices 2 and 3). However, there were too few cases in each jurisdiction to identify any trends.

## **Comment**

There has been a downward trend in tetanus notification and hospitalisation rates over the last decade. Hospitalisation rates were higher than notification rates. This discrepancy could be due to under-reporting of cases, multiple admissions for the same case and coding errors. Coding errors may have resulted from misclassification of other conditions as tetanus, especially where tetanus was not the principal diagnosis. This cause is supported by the very short lengths of stay in hospital for most patients. Equally, notifications for tetanus rely heavily on clinicians rather than laboratories—since laboratory confirmation of the diagnosis is rarely possible—so under-notification is likely.

Tetanus has become a disease of older adults. Booster doses of tetanus are thought to be poorly implemented in adults, and are mainly given after an injury has occurred.<sup>151</sup> International serosurveys and the Australian National Serosurvey have shown that immunity to tetanus is poor in older adults, particularly women.<sup>23,152</sup> Available documentation demonstrates that two of the five notified cases received a single dose of adult diphtheria-tetanus (ADT) vaccine following minor injuries, but did not receive tetanus immunoglobulin, despite having no previous documented history of tetanus immunisation.<sup>86,153</sup> One case was unimmunised and developed tetanus following abrasions sustained during gardening.<sup>154</sup> Although the tetanus organism is ubiquitous in the environment, and the vaccine only provides individual level protection against the toxin, tetanus vaccination programs have clearly had a significant impact upon the disease burden in Australia. The current tetanus notification rate in Australia is similar to that achieved in other developed countries.<sup>113,155,156</sup> A tetanus booster is recommended at the age of 50 unless a booster has been documented within 10 years.<sup>49</sup> The data presented in this report suggest that this is an appropriate recommendation. Young and middle-aged people have been the focus of a recent tetanus outbreak amongst intravenous drug users in the United Kingdom and also comprise an increasing proportion of notifications in the United States of America.<sup>155,157</sup> Therefore maintenance of immunity in young adults, through the scheduled booster dose at age 15–17 years, is also important.

## Varicella-zoster virus infection

Varicella (chickenpox) is a highly contagious infection caused by the varicella-zoster virus (VZV). The average incubation period is 14–15 days, and is followed by the appearance of a rash. About 5 per cent of cases are subclinical. Acute varicella may be complicated by cerebellitis, aseptic meningitis, transverse myelitis, thrombocytopenia and pneumonia.<sup>16</sup>

In unvaccinated populations, varicella is primarily a childhood illness with more than 90 per cent of the population in temperate countries developing clinical or serological infection by adolescence.<sup>158</sup> In Australia, however, seropositivity was 83 per cent by age 10–14 years.<sup>159</sup> Varicella is generally a benign, self-limiting illness in children, but morbidity and mortality rates are higher in adults,<sup>160</sup> at the extremes of ages, and in the immunocompromised.<sup>161</sup>

Herpes zoster (HZ) or shingles is a sporadic disease, caused by reactivation of latent VZV. It is usually self-limiting and is characterised by severe dermatomal pain, often followed by post-herpetic neuralgia, which can be chronic and debilitating in the elderly.<sup>162</sup> Although herpes zoster can occur at any age, most cases occur after the age of 50 and incidence increases with age.<sup>163</sup> However, children infected *in utero* or those who acquire varicella before the age of one year, and patients on immunosuppressive drugs or infected with human immunodeficiency virus, are also at increased risk of herpes zoster.<sup>164–166</sup>

### Case definitions

#### Notifications

Varicella is not a nationally notifiable disease. Varicella and herpes zoster became notifiable in South Australia in 2002.

#### Hospitalisations and deaths

The ICD-10-AM/ICD-10 code B01 (varicella [chickenpox]) was used to identify varicella hospitalisations and deaths. The ICD-10-AM/ICD-10 code B02 (zoster [shingles]) was used to identify herpes zoster hospitalisations and deaths.

#### South Australian surveillance data

South Australian notification data were included in this report. Varicella and herpes zoster have been notifiable diseases in South Australia since 2002. Clinical diagnoses of chickenpox or herpes zoster, and laboratory diagnoses of varicella-zoster virus infection are considered confirmed cases for the purposes of surveillance.

## Secular trends in varicella and herpes zoster

Figure 33 shows that there are significantly more hospitalisations for herpes zoster than varicella. There were 3,318 hospitalisations (average annual hospitalisation rate 8.7 per 100,000) for varicella between 1 July 2000 and 30 June 2002 (Table 18). A median of 133 cases of varicella (range 84–193) was hospitalised per month (Figure 33).

There were 9,161 hospitalisations (average annual hospitalisation rate 24 per 100,000 for all herpes zoster and 10 per 100,000 for herpes zoster as a principal diagnosis) between 1 July 2000 and 30 June 2002 (Table 18). A median of 376 cases of herpes zoster (range 341–421) were hospitalised per month (Figure 33).

There was a definite indication of seasonality, with hospitalisations for varicella peaking in January and dropping between February and March.





**Table 19. Indicators of severe morbidity\* for hospitalised cases of varicella, Australia, 2000 to 2002,\* by age group**

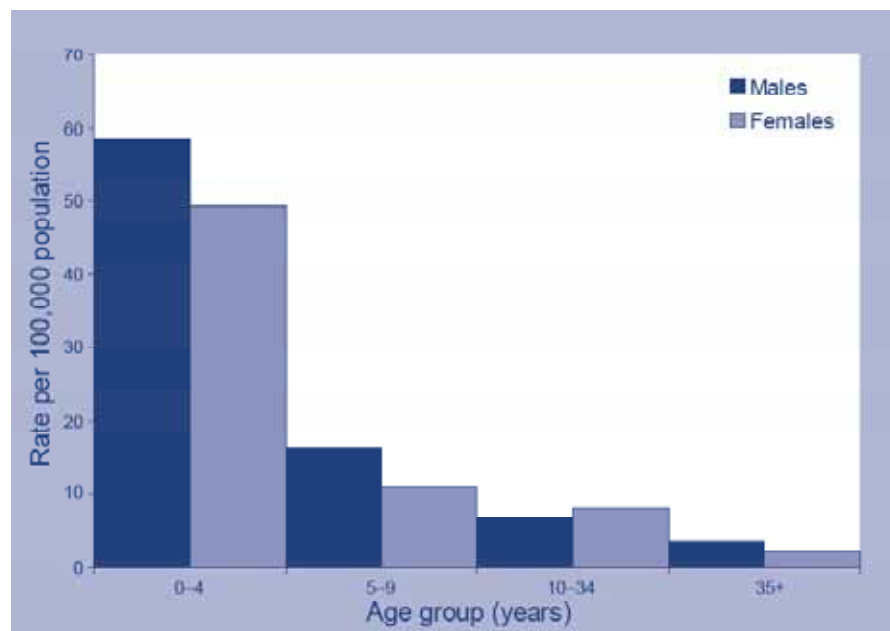
Age group (years)	Varicella encephalitis		Varicella pneumonitis	
	n	% total per age group	n	% total per age group
0–4	38	2.8	56	4.1
5–14	30	5.9	20	3.9
15–24	10	3.3	36	11.8
25–59	17	1.9	179	19.6
60+	12	5.8	22	10.6
All ages	107	3.2	313	9.4

\* Measured using National Hospital Morbidity data where the month of hospital separation was between 1 July 2000 and 30 June 2002.

### Age and sex distribution, varicella

The highest number and rate of varicella hospitalisations occurred in the youngest age groups, especially the 0–4 years age group (Table 18, Figure 34). The overall male:female ratio of hospitalisations was 1.2:1. However, this varied by age group, with males predominant in the younger and older age groups and females predominant in the 20–34 year age group. The male:female ratio for deaths due to varicella was 1:0.46.

**Figure 34. Varicella hospitalisation rates, Australia, July 2000 to June 2002, by age group and sex**



\* Hospitalisations where the month of admission was between 1 July 2000 and 30 June 2002.

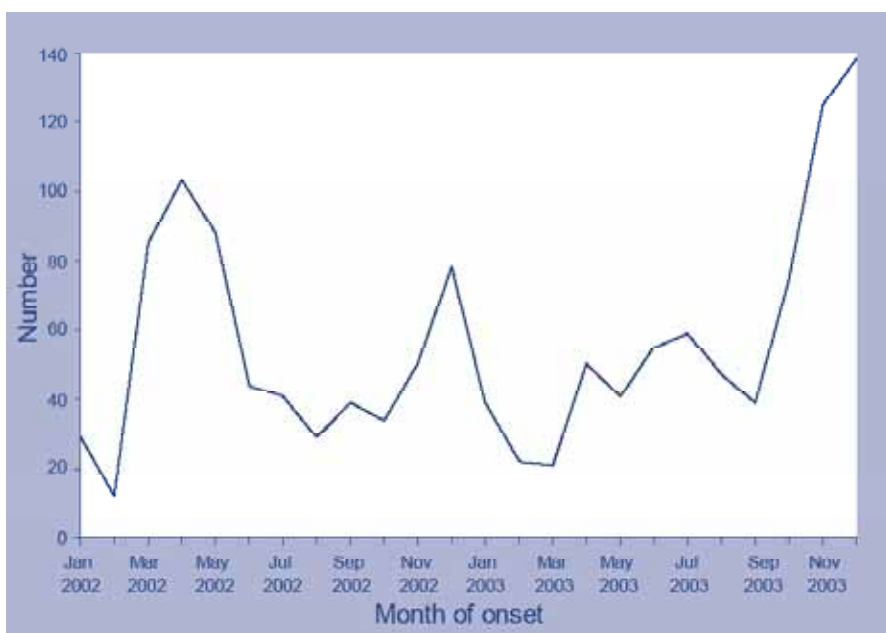
### Geographical distribution, varicella

For the years 2000/2001–2001/2002 South Australia had the highest average annual hospitalisation rate (Appendix 3).

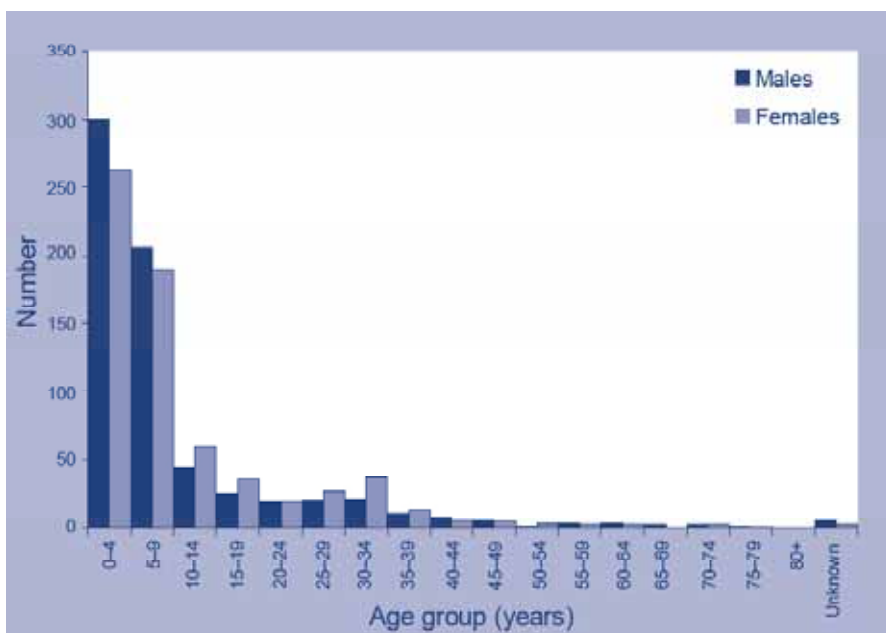
### South Australian surveillance data, varicella

Figure 35 shows the notifications of varicella by month from January 2002 to December 2003. There is a clear seasonality in the reported incidence of varicella. A total of 1,342 cases were notified. Figure 36 shows the notifications by gender. In the age group 0–9 years, males were slightly over-represented compared with females.

**Figure 35. Varicella notifications, South Australia, January 2002 to December 2003, by month of onset**



**Figure 36. Varicella notifications, South Australia, January 2002 to December 2003 (based on date of onset), by age group and sex**



### Severe morbidity and mortality, herpes zoster

For patients with an ICD-10-AM code for herpes zoster 108,686 hospital bed days (average 54,343 per year) were recorded. Of the 9,161 herpes zoster hospitalisations, 3,874 (42%) had a principal diagnosis of HZ (average annual rate 10 per 100,000) (Table 20). Complications arising from HZ infection were recorded for 4,383 hospitalisations (48%). Of all HZ hospitalisations, 80 (0.9%) were coded as having disseminated HZ and 122 (1.3%) were coded as having multiple complications (Table 21). By far the greatest number of hospitalisations were in the oldest age group, who also had the longest median length of stay. There were 30 deaths recorded with herpes zoster as the underlying cause in the calendar years 2001–2002, 28 (93%) of them for people 60 years and older. The highest death rate was also recorded in people 60 years and older.

### Age and sex distribution, herpes zoster

The highest number and rate of herpes zoster hospitalisations occurred in the oldest age groups, especially in the over 60 years age group (Table 20). The overall male:female ratio for hospitalisations was 1:1.25. The male:female ratio for deaths due to herpes zoster was 1:1.5.

**Table 20. Herpes zoster hospitalisations and deaths, Australia, 2000 to 2002,\* by age group**

Age group (years)	Hospitalisations 2 years (July 2000–June 2002)				LOS <sup>†</sup> per admission (days)	Deaths 2 years (2001–2002)	
	n	(  )	Rate <sup>‡</sup>	(  )	Median	n	Rate <sup>‡</sup>
0–4	69	(47)	2.7	(1.8)	4	0	–
5–14	175	(132)	3.3	(2.5)	3	0	–
15–24	117	(60)	2.2	(1.1)	3	0	–
25–59	1,689	(765)	8.9	(4.1)	4	2	0.0
60+	7,111	(2,870)	110.5	(44.6)	7	28	0.4
All ages <sup>§</sup>	9,161	(3,874)	23.8	(10.0)	7	30	0.1

\* Hospitalisations where the month of separation was between 1 July 2000 and 30 June 2002; deaths where the date of death was recorded between 2001 and 2002.

† LOS = length of stay in hospital.

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

§ Includes cases with unknown ages.

|| Principal diagnosis (hospitalisations).

**Table 21. Indicators of severe morbidity for hospitalised cases of herpes zoster, Australia, 2000 to 2002,\* by age group**

Age group (years)	Disseminated zoster		Multiple complications of herpes zoster	
	n	% total	n	% total
0–4	2	2.9	0	0.0
5–14	0	0.0	0	0.0
15–24	2	1.7	0	0.0
25–59	29	1.7	28	1.7
60+	47	0.7	94	1.3
All ages	80	0.9	122	1.3

\* Measured using National Hospital Morbidity data where the month of hospital separation was between 1 July 2000 and 30 June 2002.

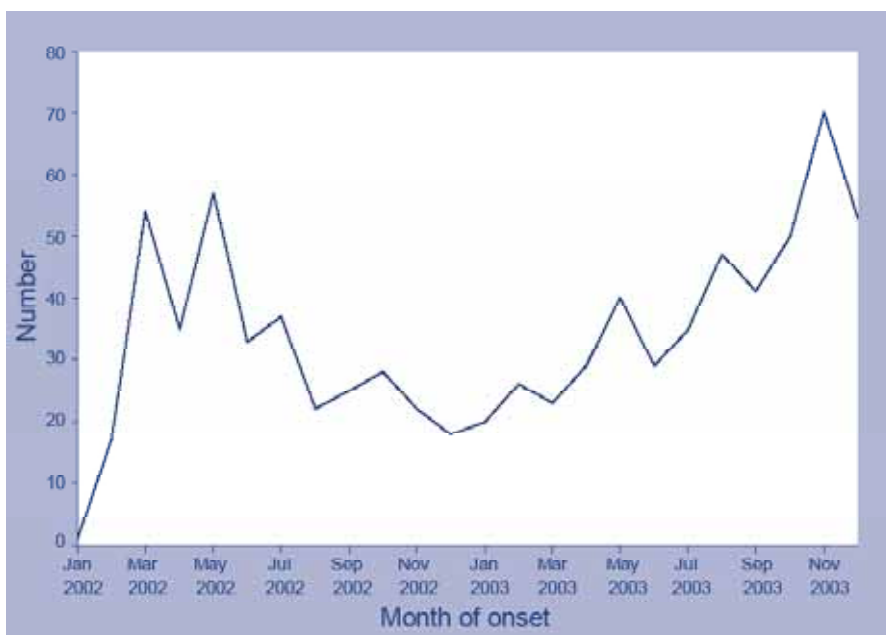
**Geographical distribution, herpes zoster**

For the years 2001–2002, South Australia had the highest crude average annual hospitalisation rate for herpes zoster, followed by Tasmania (Appendix 3).

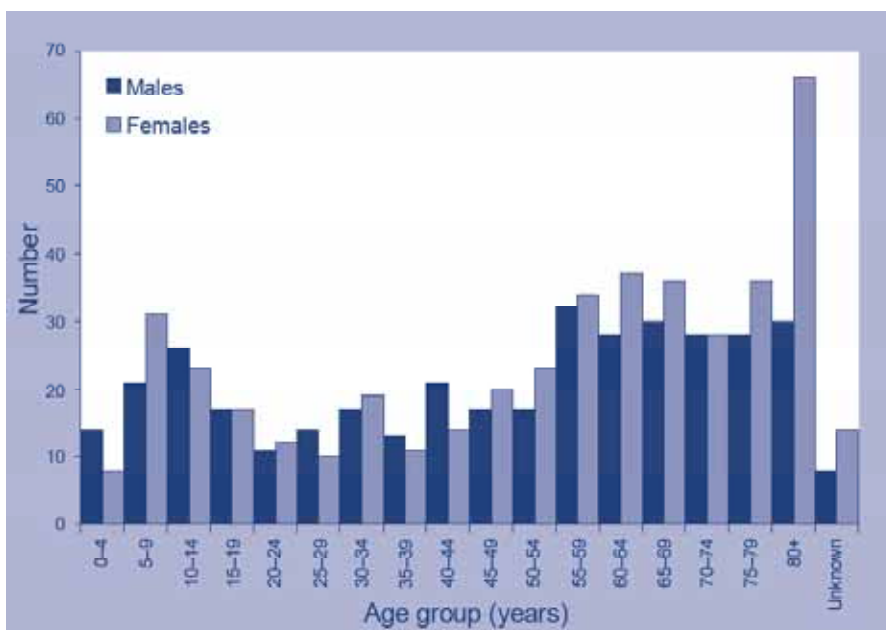
**South Australian surveillance data, herpes zoster**

Figure 37 shows the notifications of herpes zoster by month from January 2002 to December 2003. A total of 812 cases were notified. Figure 38 shows the notifications by gender. From the age of 55 onward, there were more female than male notifications. Females also predominated in the 5–9 year age group.

**Figure 37. Herpes zoster notifications, South Australia, January 2002 to December 2003, by month of onset**



**Figure 38. Herpes zoster notifications, South Australia, January 2002 to December 2003 (based on date of onset), by age group and sex**



## Comment

Hospitalisations for herpes zoster are more common than for varicella, even if only the principal diagnosis is considered. In addition, the average length of stay for herpes zoster is five days longer than for varicella, so that the burden of disease caused by severe herpes zoster is greater than that caused by severe varicella.

For varicella, the very young were most commonly hospitalised while the elderly had the longest length of stay. In our data, 33 per cent of hospitalised cases had a recorded complication. A more detailed study found that over 50 per cent of herpes zoster hospital episodes had a documented complication, the majority of which were neurological.<sup>167</sup> In that study, 16 per cent had ophthalmic zoster, which is a serious complication because it threatens vision.<sup>167</sup>

Varicella vaccine is included in the routine childhood vaccination schedule in Canada and the USA. In regions of the USA where an active immunisation program for varicella is delivered and there is active disease surveillance, the incidence of varicella has been noted to decline. This is evident in all age groups, and is most marked among those aged 1–4 years.<sup>168</sup> Universal VZV vaccination was recommended at 18 months of age in Australia in September 2003, making it important to have a good understanding of the local epidemiology of disease at baseline.

Currently, VZV vaccine is recommended but not funded, so uptake remains low. If the vaccine is funded, uptake will rise to high levels, in keeping with other funded vaccines. In 1952, Hope Simpson proposed the hypothesis that exposure to varicella may boost immunity against HZ.<sup>169</sup> This question has not been addressed in research studies again until recently, when its importance in relation to universal varicella vaccination has become apparent. If exposure to wild varicella provides boosting and protection against activation of HZ, universal infant varicella vaccination and the subsequent decline in wild varicella may result in an increase in HZ incidence.<sup>170</sup> There is increasing evidence that exposure to wild VZV does boost immunity to HZ, with two recent observational studies showing lower rates of HZ in groups who are exposed to varicella.<sup>171,172</sup> Mathematical modelling suggests that widespread infant VZV vaccination might result in a significant increase in the incidence of HZ, affecting more than 50 per cent of people aged 10–50 years at the time of the introduction of vaccination, with the increase in HZ predicted to persist for over 40 years.<sup>170</sup> These predictions might not be correct, particularly if vaccine efficacy is less than that suggested by clinical trial data. However, they indicate the importance of baseline data and ongoing surveillance for herpes zoster in Australia, so that any adverse trends can be detected early.

Without notification data, information about hospitalised cases is our only indicator of varicella and zoster morbidity. The South Australian initiative to make VZV notifiable in 2001 is an important and useful step towards developing adequate community surveillance for VZV. This allows us to look at the epidemiology of disease in the community, rather than relying only on hospitalisation data. The South Australian data show no apparent impact on the transmission of varicella by the availability of VZV vaccine on the private market. The gender-specific and age-specific data from South Australia show a similar epidemiology to the hospitalised cases, which indicates that if varicella-zoster remains a non-notifiable disease in other jurisdictions, hospitalisation data will still provide a useful measure of trends in varicella and herpes zoster.

## Acknowledgements

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