

11. Other communicable diseases

This chapter presents information on other communicable diseases in children. This is a selective coverage of certain communicable diseases which are thought to be important to monitor in children. These include meningococcal disease, invasive pneumococcal disease, varicella (chickenpox), rotavirus, respiratory syncytial virus, hepatitis A and rheumatic fever. Of these diseases, hepatitis A and meningococcal disease have been notifiable in Australia since 1991, and invasive pneumococcal disease since 2001. Although vaccines are available for most of these diseases, they are not currently included in the recommended childhood immunisation schedule. This is partly related to a combination of factors, including the lack of effectiveness of certain vaccines to warrant their universal use, or where cost-benefit analysis indicates that it is more cost effective if the vaccines are indicated only for those at high risk of the disease. However, intervention against a number of diseases through publicly funded programmes is currently being considered. These diseases include varicella, meningococcal disease and invasive pneumococcal disease (DHA, pers. comm., April 2002).

Information on notifiable communicable diseases is derived from the National Notifiable Diseases Surveillance System (NNDSS) maintained by the Commonwealth Department of Health and Ageing. Information on deaths and hospitalisations is derived from the AIHW Mortality Database and the AIHW National Hospital Morbidity Database.

Meningococcal disease

Meningococcal infections are caused by the bacterium *Neisseria meningitidis* (meningococcus). Strains of this bacterium can be divided into 13 distinct groups (serogroups), with serogroups A, B and C accounting for over 90% of cases of meningococcal disease. Meningococci are a common cause of bacterial meningitis in Australia. Meningococcal disease mainly affects children under 5 years and adolescents. Transmission occurs between people through infected droplets and respiratory secretions spread by coughing, sneezing, kissing, and sharing utensils or food. The case fatality rate from invasive meningococcal diseases is about 10% (NHMRC 2000).

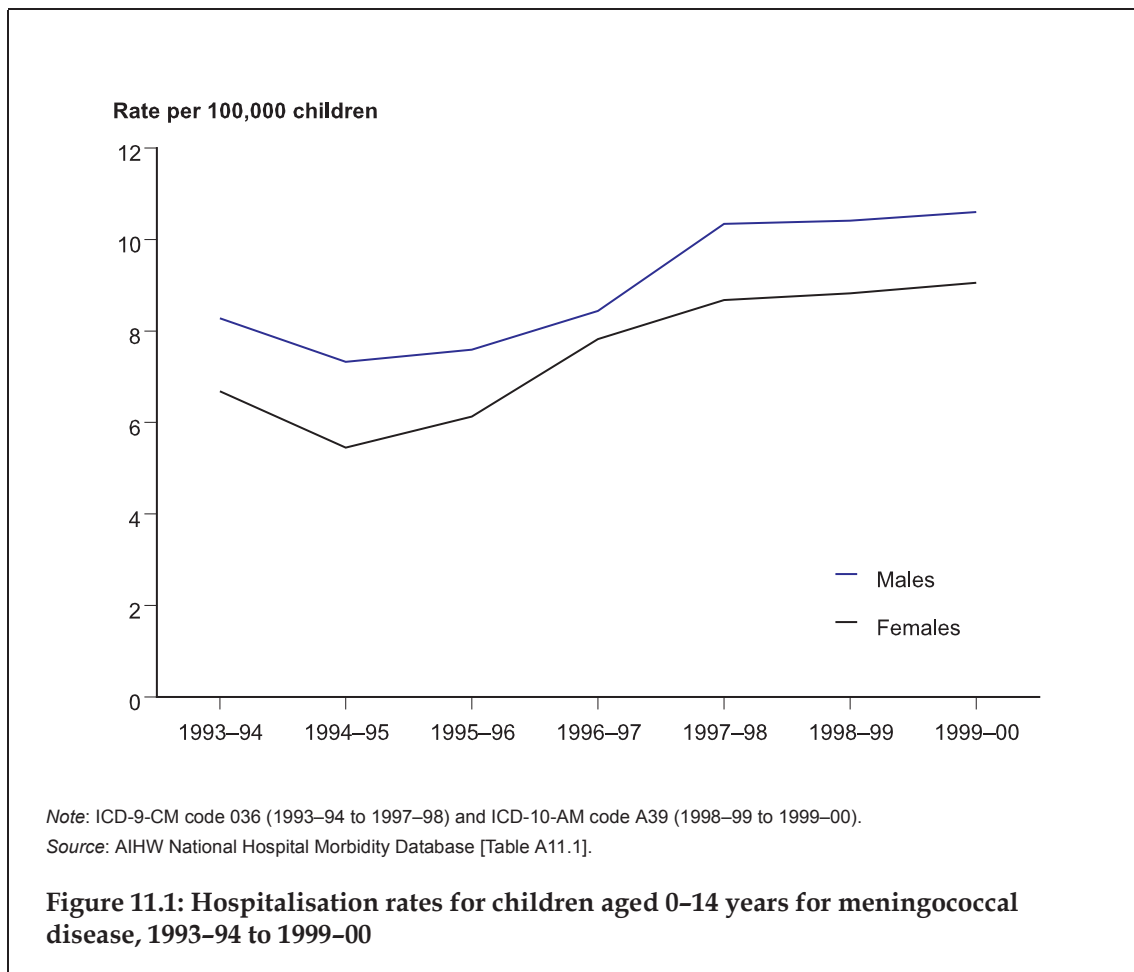
In Australia, the incidence of meningococcal disease and the frequency of outbreaks have been rising over the last decade. Most of the notifiable cases in 2000 (93%) were due to serogroups B and C. Serogroup B caused the majority of meningococcal disease nationally (56%) and in all States and Territories, except in Victoria where serogroup C disease predominated (54%). Serogroup C is associated with a significantly higher case fatality rate (12%) than serogroup B (5.9%) (Tapsall 2001). Between 1993 and 2000 there were 1,818 notifications for meningococcal disease in children aged 0–14 years. Notification rates were highest for children aged 0–4 years (NNDSS, unpublished data).

No one vaccine is effective against all types of meningococcus. One available vaccine is effective against some types (A, C, Y and W135), but not against type B, which is responsible for most of the disease. Also, this vaccine is effective in older children and adults, but less effective in children under 2 years (Patel et al. 1997). Therefore, routine vaccination of children with the available vaccine is not recommended at the present time (NHMRC 2000). School or community-based vaccination programs, however, have been used in Australia to manage clusters of outbreaks of the disease. In the UK, a conjugate meningococcal group C vaccine introduced among people aged 0–18 years resulted in a dramatic decline in the incidence and death due to serogroup C in this age group (Miller et al. 2001). The use of this vaccine in Australian children and adolescents is currently being considered (DHA, pers. comm., April 2002).

As effective management of an individual with meningococcal disease relies on the accuracy of the diagnosis (which in some cases can be difficult), it is recommended that

when meningococcal disease is suspected, immediate intravenous antibiotic therapy be used. It has also been recommended that antibiotics against the other common causes of meningitis, which include *Streptococcus pneumoniae* and *Haemophilus influenzae* type b, be used.

Hospitalisations



- There were 2,224 hospitalisations of children aged 0-14 years due to meningococcal disease between 1993-94 and 1999-00. Of all hospitalisations, 56% were of boys and 44% girls.
- The overall hospitalisation rate rose from 7.5 per 100,000 children aged 0-14 years to 9.8 per 100,000, an increase of 31%. The rate was consistently higher for boys than for girls. Although rates rose for both boys and girls, the increase was greater for girls. Hospitalisation rates for girls rose from 6.7 to 9.1 (a 36% increase), while those for boys rose from 8.3 to 10.6 (a 28% increase).

Deaths

Table 11.1: Meningococcal disease deaths of children aged 0–14 years, 1991–00

	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
Number	15	12	18	12	11	14	9	15	14	12
Rate per 100,000 children	0.4	0.3	0.5	0.3	0.3	0.4	0.2	0.4	0.4	0.3

Note: ICD-9 code 036 (1991 to 1996) and ICD-10 code A39 (1997 to 2000).

Source: AIHW Mortality Database.

- Between 1991 and 2000, 132 children aged 0–14 years died from meningococcal disease. Over half of these deaths (55%) were of boys. The majority (82%) were children under 5 years. Of children who died from meningococcal disease, 8% (11) were Aboriginal and Torres Strait Islander children.
- In 2000, the death rate was 0.4 per 100,000 boys and 0.3 per 100,000 girls. Of the 12 children who died in 2000, the majority (83%) were under 10 years of age.

Invasive pneumococcal disease

Invasive pneumococcal disease (IPD) is caused by the bacterium *Streptococcus pneumoniae* (the pneumococcus) which usually inhabits the upper respiratory tract. It can spread from the nasopharynx to cause infection in other parts of the respiratory tract. The major clinical conditions of invasive pneumococcal disease are pneumonia, bacteraemia, meningitis and otitis media – with pneumonia being the most common clinical disease. Although up to 90 types (serogroups) have been identified (each inducing a specific immune response), only a limited number are responsible for causing most of the disease. In a study carried out in Western Australia, 18 types were responsible for causing disease in 92% of cases (NHMRC 2000).

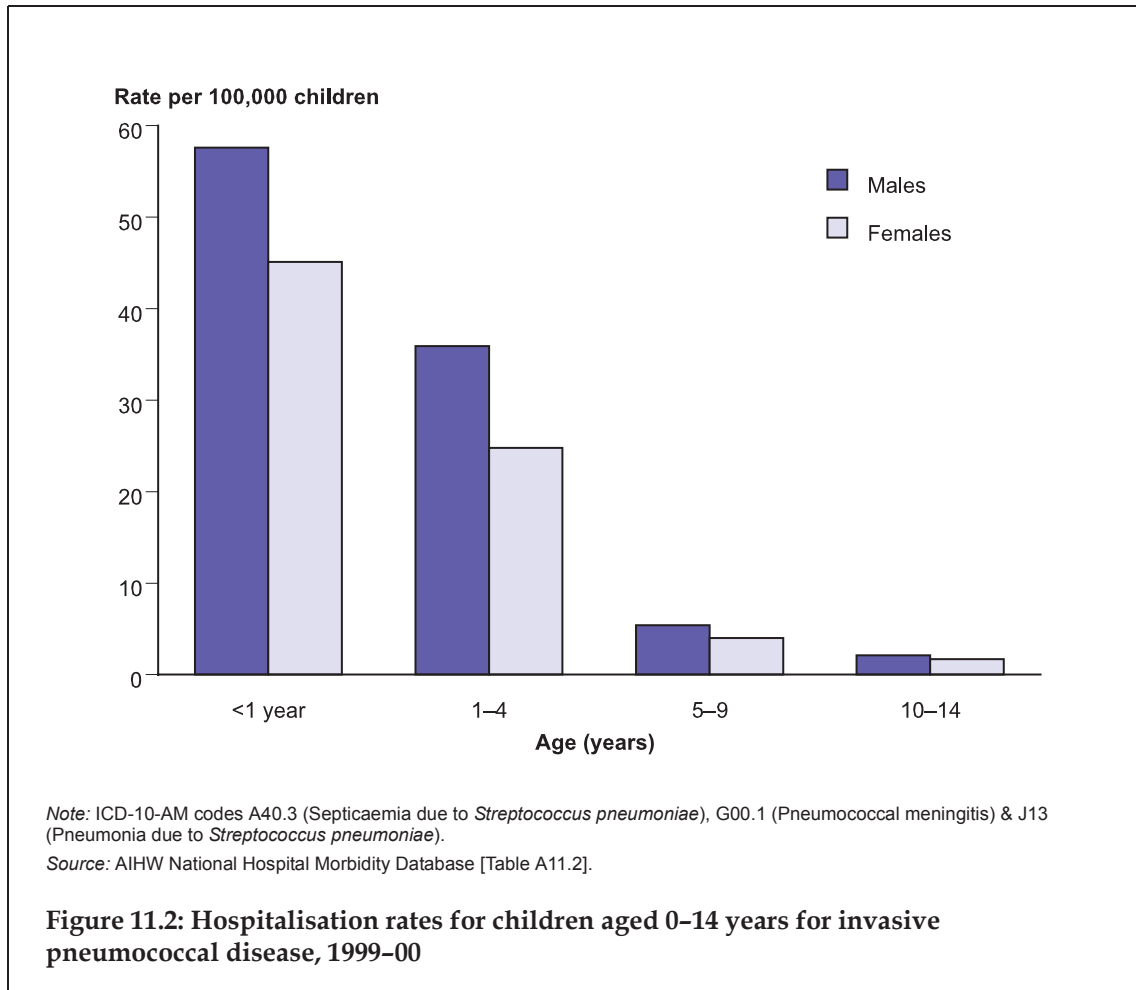
Management of IPD relies on the use of antibiotics. The increasing rates of antimicrobial resistance in pneumococcus, however, represent major problems for therapy for IPD, especially for meningitis. Resistance to penicillin rose from 1% in 1989 to 7% in 1994, and to more than 20% in 1997 (Turnidge et al. 1999). This increased resistance has implications for life-threatening as well as non-life-threatening infections with pneumococcus.

In Australia, the incidence of IPD is highest among Aboriginal and Torres Strait Islander people, and this incidence is as high as anywhere in the world (Torzillo et al. 1995). The incidence was estimated to be 76 per 100,000 Indigenous people, compared with 6 per 100,000 other Australians (NHMRC 2000). In a survey carried out in central Australia, there were 185 episodes of IPD; of these, 88% were in Indigenous people. More than half of these cases were in children under 5 years. Indigenous children are 11 times more likely to suffer IPD than other Australian children (Torzillo et al. 1995). Among Indigenous children under 2 years, the incidence was 2,053 per 100,000 children. The main diagnoses in Indigenous children were pneumonia, meningitis and septicemia. The case fatality rate for Indigenous children aged 1–4 years was 2%.

The currently available vaccine contains components derived from the 23 most frequent types causing pneumonia infections in the USA. These are similar to the types causing the disease in Australia. Although most adults appear to respond to the vaccine, the response among children under 2 years is poor (NHMRC 2000). Pneumococcal vaccines are funded by the Commonwealth Government for all Aboriginal and Torres Strait Islander adults aged 50 years and over, and for other age groups where the person is at an increased risk of complications from pneumococcal disease. In 2001, a conjugate

pneumococcal vaccine was introduced for children at high risk: Indigenous children under 2 years of age, and children with predisposing medical conditions (DHA, pers. comm., April 2002).

Hospitalisations



- In 1999-00, there were 528 hospitalisations of children aged 0-14 years for pneumococcal disease. Of all hospitalisations, 59% were of boys and 41% girls.
- The rates were highest for infants, followed by children aged 1-4 years: 51.5 per 100,000 infants, compared with 30.5 per 100,000 children aged 1-4 years.

Deaths

Between 1997 and 2000, 21 children aged 0-14 years died from invasive pneumococcal disease. Of these, 2 were Aboriginal and Torres Strait Islander children.

Varicella (Chickenpox)

Chickenpox is a highly contagious infection caused by the varicella-zoster virus. In healthy children, chickenpox is usually a mild disease of short duration, with an average incubation (time between infection and the appearance of symptoms) of about 2 weeks. The majority of children (75%) will have had chickenpox by the age of 12.

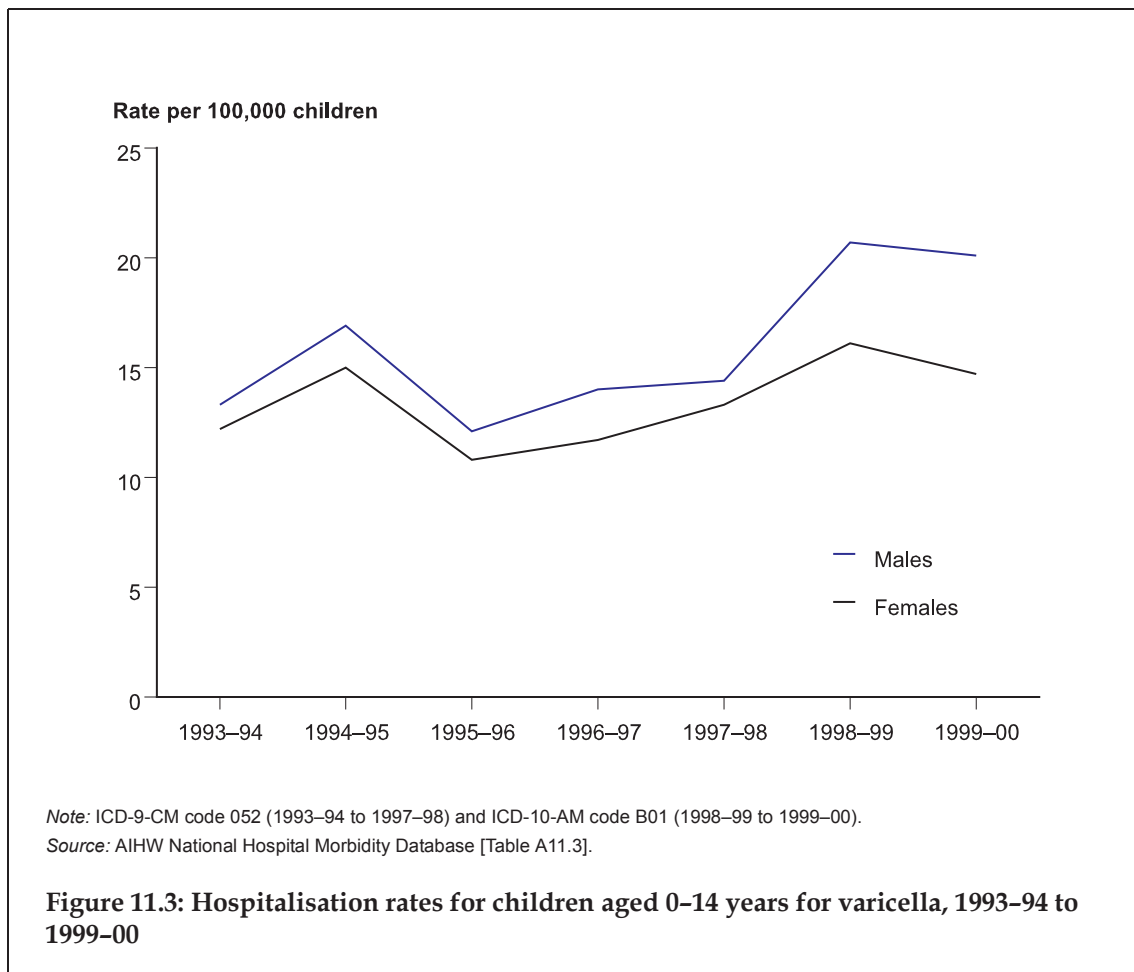
Chickenpox is more severe and can cause serious and fatal illness at any age if the immune system is weakened or suppressed. For children with weakened immune systems, the case fatality rate is estimated to be between 7% and 10%, compared with between 0.1% and 0.4% among healthy children (NHMRC 2000).

Chickenpox is more serious in adults, and although only about 2% of the cases occur in adults, they account for 25% of all varicella-zoster viral-related deaths (Joseph & Noah 1988). Varicella-zoster can cause severe illness in pregnant women, the foetus and the newborn baby. The impact of this viral infection during pregnancy for the mother and the foetus varies with the period of gestation. For the mother, the risk of adverse effects is greatest in the third trimester, whereas for the foetus the risk is greatest in the first and second trimesters (Heuchan & Isaacs 2001).

Chickenpox during pregnancy may result in foetal varicella, which is a mild and self-limiting disease. Occasionally, however, it produces a characteristic pattern of abnormality known as congenital varicella syndrome (CVS), which includes congenital malformations such as skin scars, limb underdevelopment and eye damage. The incidence of CVS in Australia is estimated to be 1 in 107,000 pregnancies (NHMRC 2000).

There are two vaccines available for varicella in Australia which have been approved by the NHMRC for use in children from 12 months of age. These vaccines, however, are not currently included in the standard childhood immunisation schedule. They have been recommended for adults in high-risk occupations (workers in day care centres, teachers and health workers) and for non-immune women prior to pregnancy. The Commonwealth Department of Health and Ageing is currently considering the benefits of including the varicella vaccine on the NHMRC vaccination schedule (DHA, pers. comm., April 2002).

Hospitalisations



- Between 1993-94 and 1999-00, there were 3,962 hospitalisations of children aged 0-14 years due to varicella: 56% were of boys and 44% girls.
- The hospitalisation rate increased from 12.8 to 17.5 per 100,000 children. The rate was consistently higher for boys than for girls; although rates rose for both, the increase was higher for boys. Hospitalisation rates for boys rose from 13.3 to 20.1, a 51% increase, while those for girls rose by 20%, from 12.2 to 14.7.

In 1999-00, hospitalisation rates were highest for infants, followed by those aged 1-4 years: 49.5 per 100,000 infants, compared with 34.3 per 100,000 children aged 1-4 years. The rate was substantially lower for children aged 10-14 years at 3.9 per 100,000.

The greatest difference in hospitalisation rates between boys and girls was for infants, where rates were 1.7 times higher for boys.

Deaths

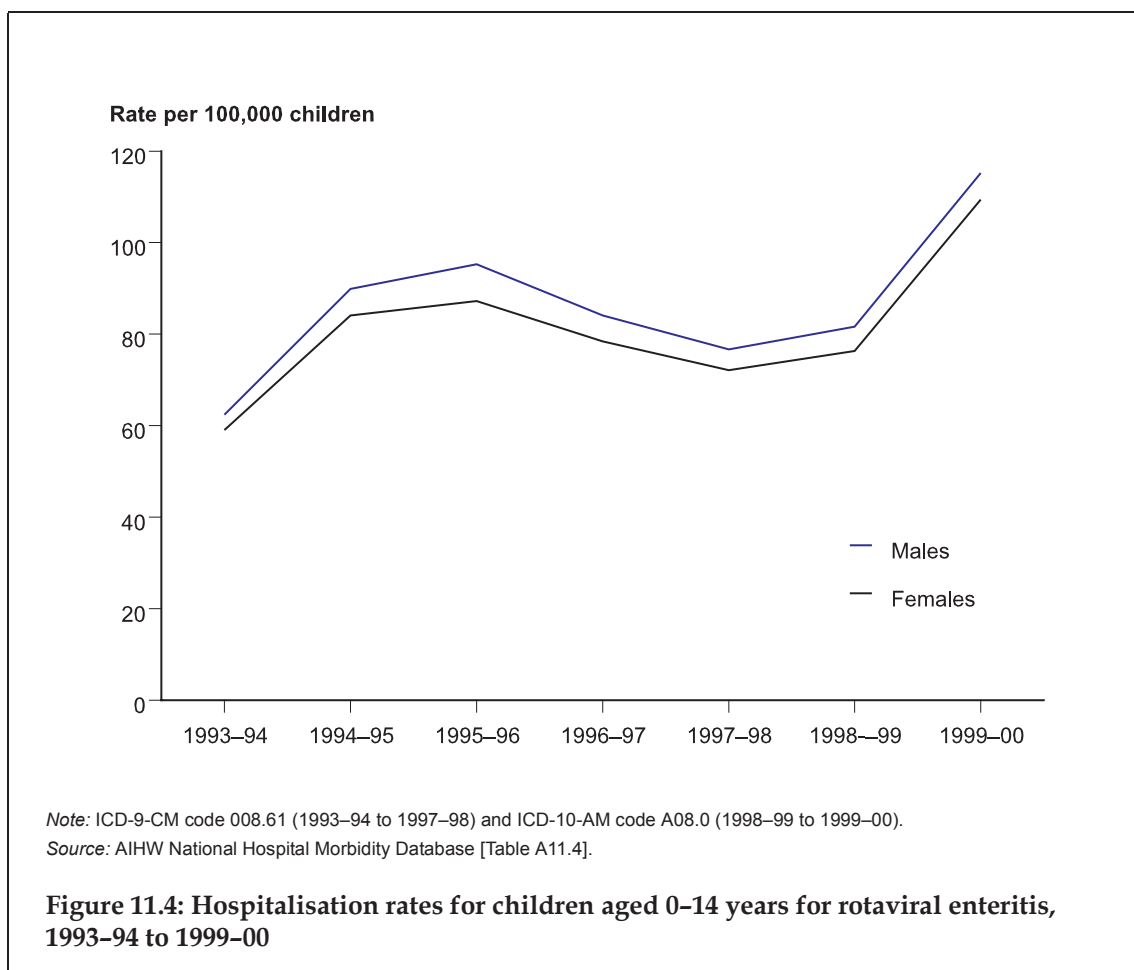
Death from chickenpox among children is not common. Between 1991 and 2000, there were 20 deaths of children aged 0–14 years attributed to chickenpox.

Rotavirus

Rotavirus is a major cause of gastroenteritis, affecting infants and young children worldwide. In some children the illness is mild with only some watery diarrhoea, while in others it can be severe, with fever, vomiting and diarrhoea leading to dehydration. In Australia, it has been estimated that between 50% and 60% of hospitalisations of children with acute gastroenteritis can be attributed to rotavirus (Ferson 1996; Carlin et al. 1998).

Because of the public health benefits, preventing rotavirus gastroenteritis through vaccination has an obvious appeal. Currently, vaccination trials are in progress to determine the effectiveness of different vaccines. However, as an interim measure, it is recommended that efforts should be directed towards improving the management of acute cases of gastroenteritis in children presenting in emergency departments and in community settings, where evidence indicates that management is not optimal (Ferson & Henry 1998).

Hospitalisations



- Between 1993–94 and 1999–00, there were 22,450 hospitalisations of children aged 0–14 years due to rotavirus. Throughout this period, the hospitalisation rate was always higher for boys than for girls.
- The rate rose from 60.8 per 100,000 children in 1993–94 to 112.4 in 1999–00, an increase of 85%.
- In 1999–00, the rate was highest for infants and for children aged 1–4 years: 406.4 per 100,000 infants, compared with 297.4 per 100,000 children aged 1–4 years.
- Among infants, the hospitalisation rate was higher for boys (450.6 per 100,000) than for girls (359.9).

Deaths

Between 1997 and 2000, 1 male infant died from rotavirus (in 1999). It is difficult to estimate deaths prior to 1997 because ICD-9 codes are not specific enough to obtain these data.

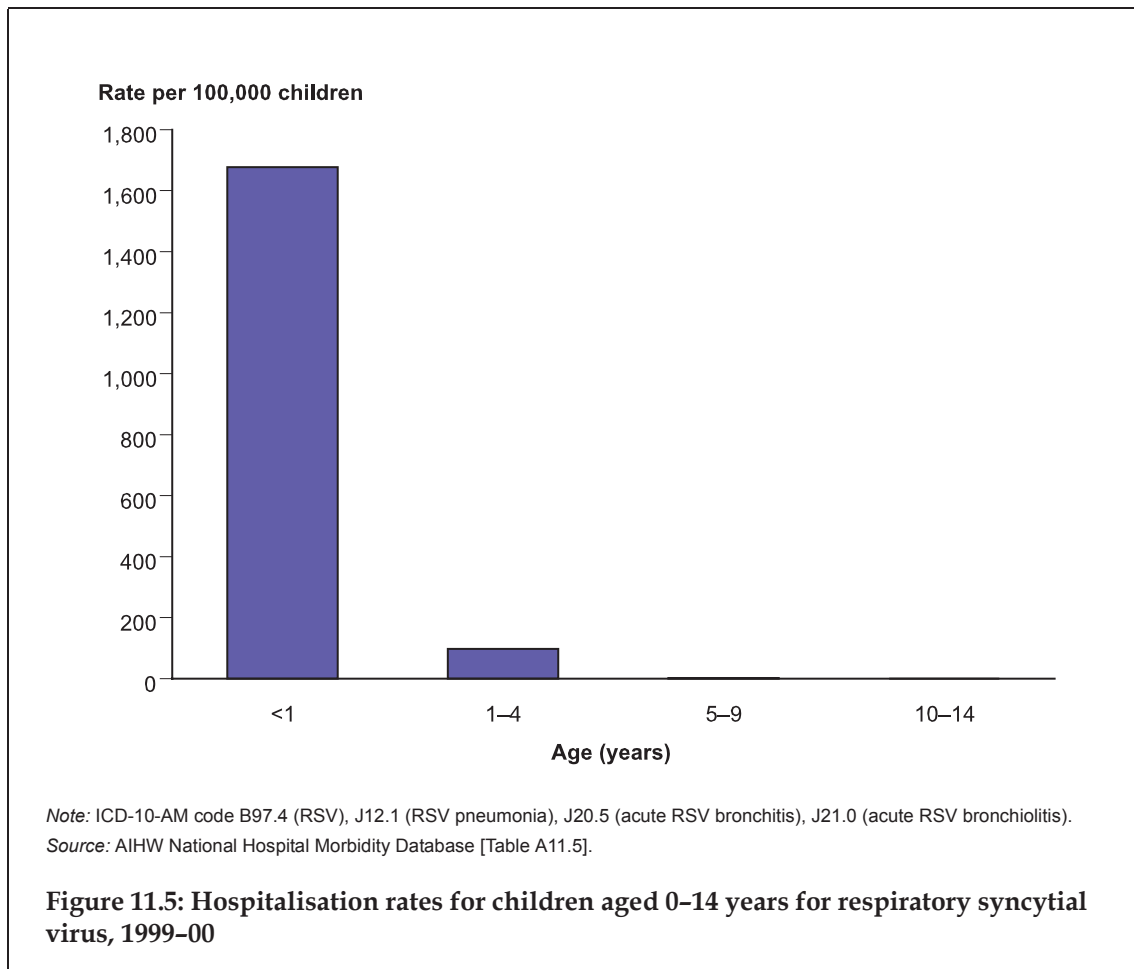
Respiratory syncytial virus

Respiratory syncytial virus is the major cause of lower respiratory infections in infants. The virus is highly infectious, and is transmitted by close contact, coughing or sneezing. Annual outbreaks cause many hospital admissions, putting a high strain on the hospital system. The peak incidence of the disease is between 1 and 6 months of age (NHMRC 2000). It is estimated that around 3% of each year's birth cohort are admitted to hospital with bronchiolitis every winter in Europe, Australasia and North America (Allport et al. 1997). In infants, it was estimated that 25–40% of infections with respiratory syncytial virus lead to pneumonia and bronchiolitis (NHMRC 2000). Traditionally, certain groups of infants are considered to be at a high risk of developing severe respiratory syncytial virus bronchiolitis. These include infants born prematurely, and those with chronic lung disease due to prematurity, other cardio-respiratory disease or those with some form of immunodeficiency (Sharland & Bedford-Russell 1999).

There is currently no available vaccine for respiratory syncytial virus. Treatment includes the use of bronchodilators, anti-inflammatory drugs and antibodies to neutralise the virus. One type of antibody has been registered in Australia to prevent serious lower respiratory tract disease caused in children at high risk of respiratory syncytial virus infection (NHMRC 2000). Results from initial clinical trials in the United Kingdom with this product showed a relative reduction in hospital admissions related to respiratory syncytial virus of 55% among infants born prematurely (Sharland & Bedford-Russell 1999).

Hospitalisations

As the disease classification code prior to 1997–98 (ICD-9-CM) was only specific to respiratory syncytial virus pneumonia, data on hospitalisations are only presented for the most recent year 1999–00.



- In 1999–00, there were 5,224 hospitalisations of children aged 0–14 years for respiratory syncytial virus (pneumonia, acute bronchitis, acute bronchiolitis): 58% boys and 42% girls.
- The overall hospitalisation rate was 142 per 100,000. Rates were highest among infants, followed by children aged 1–4 years: 1,677 per 100,000 infants, compared with 98 per 100,000 children aged 1–4 years.

Deaths

Between 1997 and 2000, 1 female infant died from respiratory syncytial virus (in 1999). As the disease classification code prior to 1997 (ICD-9) was only specific to respiratory syncytial virus pneumonia, data on deaths are only presented for the most recent years.

Hepatitis A

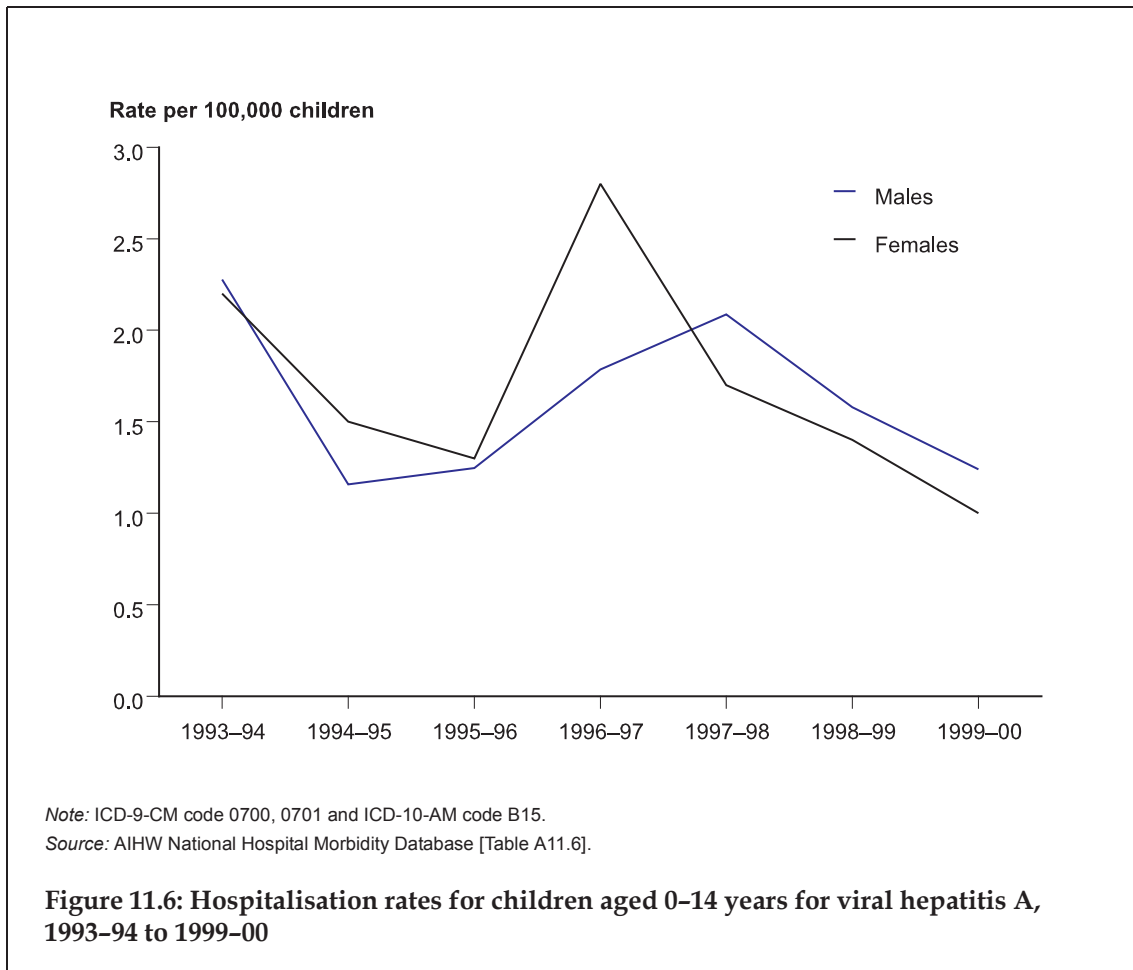
Hepatitis A is an acute infection of the liver caused by the hepatitis A virus. The virus is transmitted by a faecal oral route, and the incubation period ranges from 14 to 50 days. Infected individuals excrete the virus in their faeces for 2 weeks prior to illness, and for 1 week after the appearance of symptoms.

Fulminant (severe, rapidly developing) hepatitis A is rare in children in industrialised countries. Occasional cases tend to be among children who travelled to hepatitis A endemic countries or who live in conditions of considerable socioeconomic disadvantage and poverty. Hepatitis A is readily transmitted in environments with inadequate sanitation and water supply, low hygiene and overcrowding.

The incidence of hepatitis A viral infection (HAV) in Aboriginal and Torres Strait Islander children is higher than in other Australian children. In north Queensland, the incidence in 1996–97 was found to be up to 6 times higher among Indigenous people than among other Australian people. Indigenous people accounted for 29% of all HAV infections, but accounted for only 8% of the population. The notification rate for hepatitis A among children aged less than 5 years was 264 per 100,000 children, compared with 10 per 100,000 other Australian children in that age group (Merritt et al. 1999).

It has been argued that an effective hepatitis A control program requires effective vaccines as well as more general programs to improve Aboriginal and Torres Strait Islander health (McCaughan & Torzillo 2000). Vaccination of preschool aged Indigenous children could provide a means of preventing cases of fulminant hepatitis A in Indigenous communities, as well as reducing the extent of future outbreaks in these communities (Hanna et al. 2000).

Hospitalisations



- Between 1993-94 and 1999-00, there were 455 hospitalisations for hepatitis A of children aged 0-14 years. The hospitalisation rate for children aged 0-14 years was highest in 1996-97 (2.3 per 100,000 children) and lowest in 1999-00 (1.1 per 100,000 children).
- The rate declined from 2.2 in 1993-94 to 1.1 per 100,000 in 1999-00, although the rate for girls peaked in 1996-97 at 2.8. While rates were higher for girls than for boys during the early part of the review period, the opposite was true during the later part.
- With the exception of 1996-97, the highest hospitalisation rates were for children aged 5-9 years.

Deaths

Death from hepatitis A is uncommon. Since 1991, there have been 3 deaths, all of children aged under 5 years. Two were of girls aged 4 years, and both died in 1998. The third child was a boy aged 2 years who died in 1994. All deaths were of Aboriginal and Torres Strait Islander children.

Rheumatic fever and rheumatic heart disease

Rheumatic fever is a recurrent disease, caused by group A streptococcus bacteria associated with infections of the throat and the skin. Rheumatic fever occurs mainly in children and young adults and is characterised by fever, inflammation, pain and swelling in and around the joints, and inflammatory involvement of the pericardium and valves of the heart. Repeated attacks of acute rheumatic fever lead to cumulative damage to the heart valves and muscles, leading to rheumatic heart disease.

Rheumatic fever used to be a common disease in paediatric hospitals in Australia in the early part of the 20th century (Carapetis & Currie 1998). Its decline occurred as a result of economic development and improved living conditions. Rheumatic fever continues, however, to be common among socially and economically disadvantaged populations worldwide and in disadvantaged population groups living in affluent countries, such as Aboriginal and Torres Strait Islander people. Aboriginal people living in the Top End of the Northern Territory have one of the highest reported incidences of acute rheumatic fever in the world (Carapetis et al. 1996). In this population, the annual incidence between 1989 and 1993 was estimated to be between 2 and 7 cases per 1,000 children aged 5–14 years. Up to 3% of some Aboriginal communities have rheumatic heart disease, while, in contrast, prevalence of the disease in non-Aboriginal populations is 0.014% (Carapetis & Currie 1998).

The causes of the high rates of rheumatic fever among Aboriginal and Torres Strait Islander people are thought to be overcrowding, poor living conditions and sanitation, which expose children to group A streptococci, and limited access to medical care.

In the short term, prevention strategies which rely on accurate diagnosis of rheumatic fever and timely treatment with penicillin can be used. However, in the long term, improving the living standards of Indigenous Australians, especially those living in remote communities, is the only cost-effective viable objective. This strategy will impact not only on rheumatic fever levels but also on a number of other communicable diseases which continue to afflict Aboriginal and Torres Strait Islander children.

Incidence of rheumatic fever

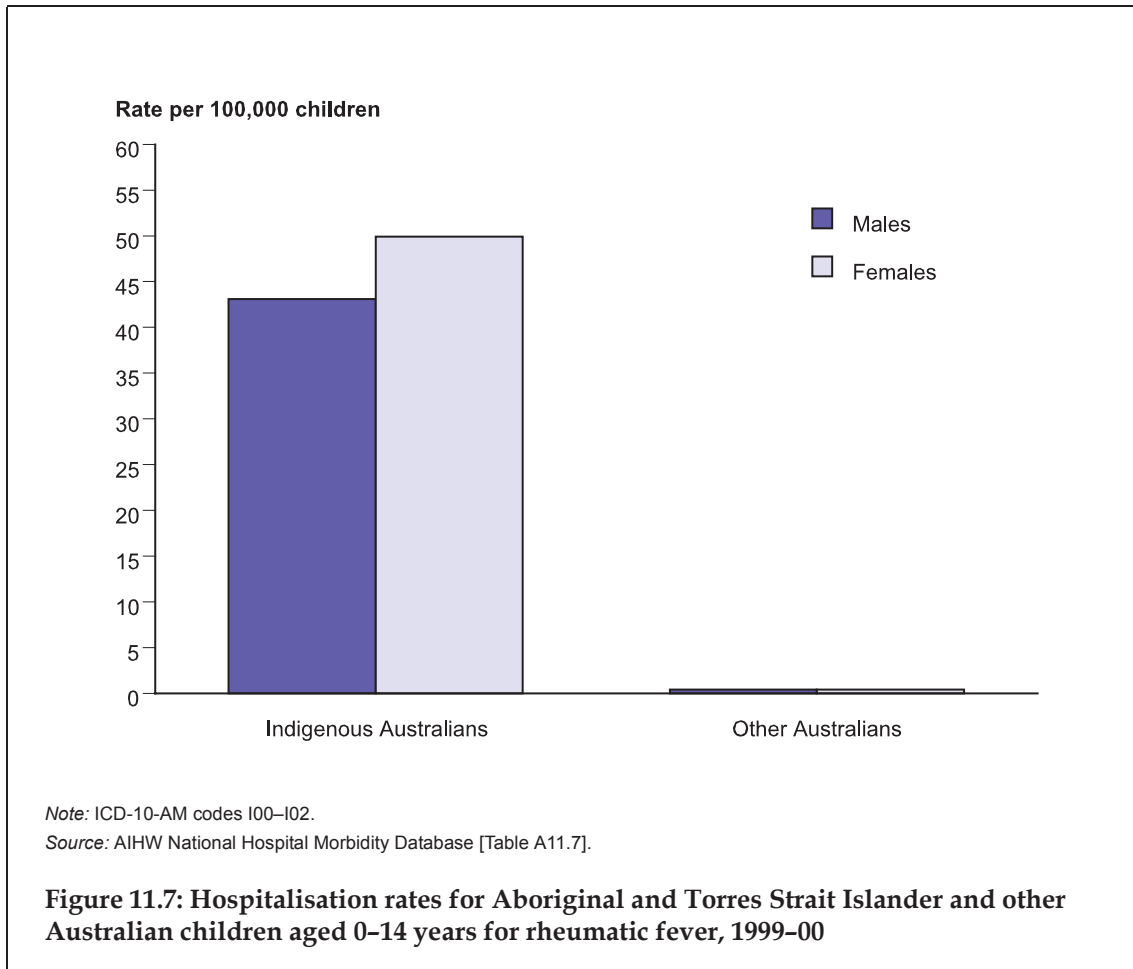
Table 11.2: Rheumatic fever among Aboriginal and Torres Strait Islander children aged 5–14 years in the Top End of the Northern Territory, 1994–01

Year	Number	Rate per 100,000 children
1994	18	204
1995	13	148
1996	21	238
1997	14	159
1998	24	270
1999	17	191
2000	14	154
2001	13	143

Sources: AIHW 2001b (1994 to 1999); Northern Territory Rheumatic Heart Disease Register, unpublished data (2000 to 2001).

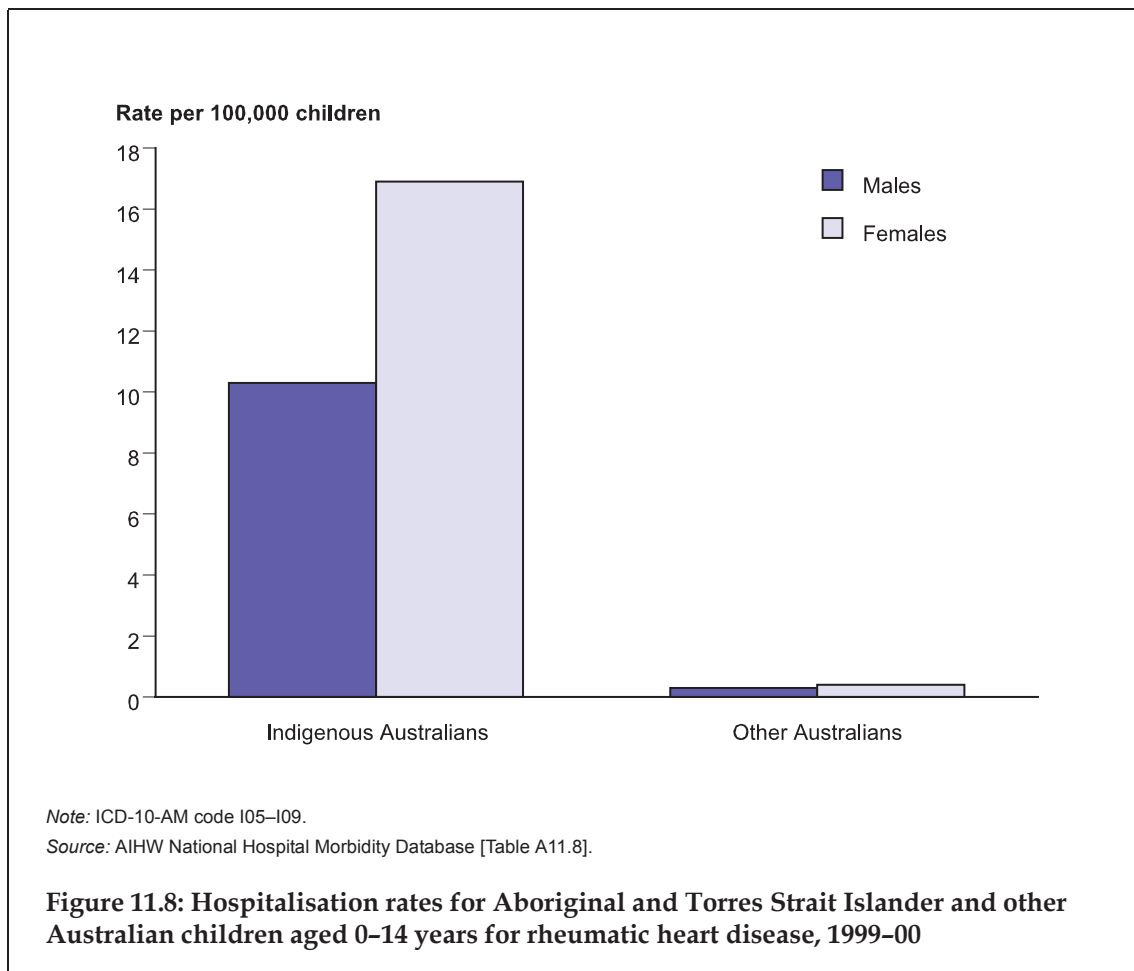
- Between 1994 and 2001, there were 134 cases of rheumatic fever in Aboriginal and Torres Strait Islander children aged 5–14 years.
- The rate of acute rheumatic fever among Indigenous children in 2001 was the lowest over this period.

Hospitalisations



- In 1999–00, there were 88 hospitalisations for acute rheumatic fever in children aged 0–14 years. Of these, 82% were Aboriginal and Torres Strait Islander children.
- The rate of hospitalisation of Indigenous children was 46.4 per 100,000, compared with 0.4 per 100,000 for other Australian children.
- Among Indigenous children, hospitalisations were higher for girls (53%, with a rate of 49.9) than for boys (47%, with a rate of 43.1).

Repeated attacks of rheumatic fever can lead to rheumatic heart disease. Hospitalisation rates for children aged 0–14 years for rheumatic heart disease in 1999–00 are shown in Figure 11.8.



- In 1999–00, there were 33 hospitalisations for rheumatic heart disease in children aged 0–14 years. Of these, 64% were of Indigenous children and 36% of other Australian children. This represents a rate of 13.5 per 100,000 for Indigenous children, compared with just 0.3 for other Australian children.
- Among Indigenous children, the hospitalisation rate was 1.6 times higher for girls than for boys (16.9 compared with 10.3).

Deaths

Between 1991 and 2000, 3 children died from rheumatic fever; none was identified as Aboriginal and Torres Strait Islander. Over the same period, 6 children died from rheumatic heart disease; all but one were Indigenous.