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Vaccine Preventable Diseases and Vaccination Coverage in Australia, 2001 to 2002

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Vaccine Preventable Diseases and Vaccination Coverage in Australia 2001 to 2002

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Overview

This, the third biennial report on vaccine preventable diseases and vaccine coverage in Australia, brings together the four most important national sources of routinely collected data about vaccine preventable diseases and vaccination (deaths, notifications, hospitalisations and vaccination coverage) for all age groups between 2001 and 2002. The general trend towards improved control of disease and improved vaccine coverage is evident, particularly in the childhood years. Detailed results are available in 14 individual chapters.

Notifications, hospitalisations and deaths for 11 diseases are summarised in Table 1. Although these data have limitations which are discussed in detail in the body of the report, some clear trends are evident. First, vaccination coverage, estimated using Australian Childhood Immunisation Register (ACIR) data, reached the targets set by the *Immunise Australia* program, with levels of full immunisation exceeding 90 per cent and approaching 95 per cent at 12 months of age and reaching 90 per cent at 24 months of age by the end of 2003.

Second, accompanying this increase in vaccine coverage, notifications for the eight diseases covered by the routine childhood vaccination schedule (diphtheria, *Haemophilus influenzae* type b (Hib) disease, measles, mumps, pertussis, polio, rubella and tetanus) continued to decline, although less sharply than in the previous review period, from an average of 8,046 cases each year in 1997–2000 to 7,806 in 2001–2002. Table 1 highlights the decrease in measles, mumps and rubella which has continued since the national Measles Control Campaign (MCC) was conducted in 1998. However, reductions in these three diseases were offset to some extent by the large number of pertussis notifications arising from the 2001 epidemic, with six deaths in 2001–2002. These pertussis deaths were almost all in very young infants and emphasise the importance of initiatives implemented in the 2003 *Australian Immunisation Handbook* to improve pertussis control.

Third, cases of Hib disease also continued to decline with improved vaccine coverage. Of vaccine preventable diseases not included on the childhood schedule during the review period, the greatest morbidity and mortality at all ages was from influenza (44 deaths), pneumococcal disease (15 deaths), meningococcal disease (44 deaths) and varicella (10 deaths). Deaths from meningococcal disease increased from an average of 31 and influenza decreased from an average of 117, compared with 1997–2000.

Comment

The years 2001 and 2002 have been a period of consolidation in immunisation practice and coverage in Australia, following the implementation of the new vaccination schedule in 2000. Australia, like many other industrialised countries, faces the dual challenges of maintaining high immunisation coverage and public confidence in immunisation while implementing increasingly complex decisions about the introduction of new vaccines for children and adults.

In surveillance, improved control of vaccine preventable diseases means that laboratory confirmation, together with accurate information on the vaccination status and any underlying medical conditions, is essential to evaluate program impact. In vaccination practice, vaccination coverage targets are probably close to their highest achievable levels in children. However, improving control through vaccination of measles and pertussis in young adults and adolescents stands out as a challenge for the next few years.

Important programs across the age spectrum in influenza and pneumococcal disease have commenced or gained momentum during this review period. The impact of these and the campaign to control disease due to serogroup C meningococcal disease should be more apparent in 2003–2004. Careful evaluation of the additional benefits of new programs and continued efforts to maintain current programs will be required to sustain the success of immunisation in Australia over the first decade of the 21st Century.

Table 1. Notifications, hospitalisations and deaths from 11 diseases preventable by vaccination, Australia, 1997 to 2002*

Disease†	Notifications		Hospitalisations		Deaths	
	Average per year 1997–2000	Average per year 2001–2002	Average per year July 1996–June 2000	Average per year July 2000–June 2002	Average per year 1997–2000	Average per year 2001–2002
Diphtheria	0	0.5	1	0.5	0	0
Hib (<5 yr)	22	12	40‡	30‡	0.5‡	0.5‡
Influenza§	NN	2,480‖	4,767	2,905	117	44
Measles	368	86	96	53	0	0
Meningococcal disease	547	677	741	871	31	44
Mumps¶	193	92	53	43	0	0.5
Pertussis	6,749	7,359	708	639	1.5	3
Pneumococcal disease	NN	2,294‖	754	1,055	15	15
Polio	0	0	1.5**	1.5**	0	0
Rubella	710	256	43	27	0	0
Tetanus	6	3	35	27	1.25	0.5
Total	8,593††	8,483†† (13,257)‡‡	7,238††	5,651††	166	107.5

NN = not notifiable.

* Notifications where the month of onset was between January 1997 and December 2002; hospitalisations where the month of separation was between 1 July 1996 and 30 June 2002; deaths where the date of death was recorded between 1997 and 2002.

† See Chapter 3 for case definitions.

‡ Note that hospitalisations and deaths are for *Haemophilus influenzae* disease and, unlike notifications, are not limited to type b.

§ Limitations of notification systems and coding for influenza hospitalisations and deaths limit the representativeness of these data, which grossly underestimate the disease burden due to influenza.

‖ Notifications only complete for 2002 – notifications for 2002 only.

¶ Queensland did not notify mumps in 2000 or for complete calendar years in 1999 or 2001.

** Principal diagnosis only.

†† Average per year for the total does not equal the sum of that for each disease, due to rounding.

‡‡ Total including influenza and pneumococcal notifications.

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The report was reviewed by a representative of the Commonwealth, the Australian Institute of Health and Welfare and each Australian jurisdiction from the Communicable Diseases Network Australia before publication.

Abbreviations

ABS	Australian Bureau of Statistics
ACIR	Australian Childhood Immunisation Register
ADT	Adult diphtheria-tetanus
AFP	Acute flaccid paralysis
AIHW	Australian Institute of Health and Welfare
Anti-HBc	Hepatitis B core antibody
ASVS	Australian Standard Vaccination Schedule
CDT	Combined diphtheria-tetanus
CRS	Congenital rubella syndrome
CSF	Cerebrospinal fluid
cVDPV	Circulating vaccine-derived poliovirus
DT	Diphtheria-tetanus
DTP	Diphtheria-tetanus-pertussis
DTPa	Diphtheria-tetanus-pertussis (acellular)
dTpa	Adult diphtheria-tetanus-pertussis (acellular)
DTPw	Diphtheria-tetanus-pertussis (whole cell)
FAG	Finance assistance grant
HAV	Hepatitis A virus
HBV	Hepatitis B virus
Hep B	Hepatitis B (vaccine abbreviation)
HBsAg	Hepatitis B surface antigen
Hib	*Haemophilus influenzae* (type b)
HFG	Hospital funding grant
HZ	Herpes zoster
ICD	International Classification of Diseases
IgM	Immunoglobulin M
IPD	Invasive pneumococcal disease
IPV	Inactivated poliomyelitis vaccine
LOS	Length of stay
MCC	Measles Control Campaign
MenCCV	Meningococcal C conjugate vaccine
MMR	Mumps-measles-rubella
NCIRS	National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases
NHMRC	National Health and Medical Research Council
NIS	National Immunisation Strategy
NNDSS	National Notifiable Diseases Surveillance System
OPV	Oral poliomyelitis vaccine
PCV7	7-valent conjugate pneumococcal vaccine
PHOFA	Public health outcome funding agreement
PPV	Positive predictive value
PRP-OMP	*Haemophilus influenzae* type b polysaccharide conjugated to the outer membrane protein of *Neisseria meningitidis* vaccine
RSV	Respiratory syncytial virus
SSPE	Subacute sclerosing panencephalitis
VAPP	Vaccine-associated paralytic poliomyelitis
VPD	Vaccine preventable disease
VZV	Varicella-zoster virus
WHO	World Health Organization
7vPCV	7-valent conjugate pneumococcal vaccine
23vPPV	23-valent polysaccharide pneumococcal vaccine

1. Introduction

This is the third national report on the morbidity and mortality from vaccine preventable diseases (VPDs) and vaccine coverage in Australia. The first (1993 to 1998) was published in 2000 and the second (1999 to 2000) in 2002.[1,2] The progressive decline in the incidence of all the childhood VPDs continues, with the exception of pertussis. Even more striking has been the 99 per cent decline in the number of deaths from these diseases since the prevaccination era, despite the Australian population increasing almost threefold (Table 2), and the close association this decline has had with the introduction of specific vaccination programs.[3]

The past decade has seen the introduction of a number of major surveillance and vaccination initiatives in Australia:

- a national disease notification system in 1991;
- the Australian Childhood Immunisation Register (ACIR) in 1996;[4]
- the *Seven Point Plan* in 1997 (this included the Measles Control Program in the later part of 1998);[5]
- the General Practice Immunisation Initiative in 1998; and
- several new vaccination programs for children, the elderly and Indigenous people.

Although specific evaluations are important, much can be learned from examining routinely collected data, especially for trends over time. This third report uses similar methods to the first two, bringing together data sources available at the national level relevant to VPDs and vaccination (deaths, notifications, hospitalisations and vaccination coverage) for all age groups. The diseases covered in this report include those for which vaccines were funded nationally for children during the review period (diphtheria, *Haemophilus influenzae* type b (Hib) disease, hepatitis B, measles, mumps, pertussis, poliomyelitis, rubella and tetanus), those for which vaccines were available but only funded or recommended for specific risk groups (hepatitis A, invasive pneumococcal disease, influenza) and varicella and meningococcal disease (for which new vaccines became available in 2000 and late 2001, respectively). The report does not cover some other diseases which are at least partially preventable by vaccination, such as tuberculosis, for which reports can be found elsewhere.[6]

This and the previous two reports, both from the National Centre for Immunisation Research and Surveillance (NCIRS), provide evidence of the impact of changes in vaccination policy over the past decade, as detailed in Appendix 4. These reports provide baselines against which further initiatives can be evaluated.

Table 2. Number of deaths from diseases commonly vaccinated against, by decade, Australia 1926 to 1995 and 1996 to 2002*

Period	Diphtheria	Pertussis	Tetanus	Poliomyelitis	Measles†	Population estimate (yearly average)
1926–1935	4,073	2,808	879	430	1,102	6,600,000
1936–1945	2,791	1,693	655	618	822	7,200,000
1946–1955	624	429	625	1,013	495	8,600,000
1956–1965	44	58	280	123	210	11,000,000
1966–1975	11	22	82	2	146	13,750,000
1976–1985	2	14	31	2	62	14,900,000
1986–1995	2	9	21	0	32	17,300,000
1996–2002	0	15	6	0	0	18,900,000

* Sources: Feery B. One hundred years of vaccination. *Public Health Bulletin* 1997; 8:61–63; Feery B. Impact of immunization on disease patterns in Australia. *Med J Aust* 1981;2:172–176. Deaths recorded for 1966–1975 and 1996–2002 updated with data provided by AIHW Mortality Database.

† Excludes deaths from subacute sclerosing panencephalitits.

Indicates decade in which community vaccination started for the disease.

2. Methods

Vaccine preventable diseases data

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

Notifications

The NNDSS database was established in its current form in 1991, and includes information about cases of vaccine preventable diseases reported by laboratories and health workers to State/Territory authorities under their current public health legislation. State/Territory notification criteria are based on the National Health and Medical Research Council (NHMRC) surveillance case definitions.[7] However, application of these definitions and even the definitions themselves may differ between jurisdictions. In 2001 invasive pneumococcal disease and laboratory-confirmed influenza became notifiable to the NNDSS. Varicella and zoster are not notifiable to the NNDSS.

Data extracted from the NNDSS as at 18 December 2003 was examined. Note that these data are later versions than those used for the 2001 and 2002 Australia's Notifiable Disease Status reports and the AIHW publication *Australia's Health 2004*.[8–10] Disease notification data for cases with an onset between 1 January 2001 and 31 December 2002 (2 years) are included in this report. Notification data are presented and reported by date of onset. Date of onset is collected from the clinical history where available, or the specimen collection date for laboratory-confirmed cases. Those with onset dates between 1 January 1993 and 31 December 2000 were reported previously.[1,2] However, to reflect the ongoing process of improving and cleaning of the NNDSS database since the publication of the previous report, all historical notification data included within this report has been updated. The variables extracted for analysis were disease, date of disease onset, age at onset, sex and State/Territory of residence. The fields for laboratory confirmation, vaccination status and Aboriginality were too incomplete to warrant analysis. Data from each State/Territory were included when calculating rates only when that jurisdiction had been reporting for a complete year (see Appendix 2, *Notifications by State/Territory and year*, for the years in which States/Territories were reporting). Differences in surveillance systems between jurisdictions may have accounted for some of the differences in notification rates. Where there were known differences that were likely to differentially affect notification rates, these have been described under the disease of interest.

Hospitalisations

The AIHW National Hospital Morbidity Database has received administrative, demographic and clinical information about patients admitted to public and private hospitals in Australia since 1993. Data are received by financial year of separation (discharge), and the two most recent years for which data are available (2000/2001 and 2001/2002) were examined. Note, however, that receipt of the 2000/2001 and 2001/2002 hospitalisation data was delayed due to concerns over possible coding problems with New South Wales data. Due to production deadlines, this report uses the finalised data for 2001/2002 and the provisional data (possibly with some minor errors for New South Wales data) for 2000/2001. Cases with separation dates between 1 July 1993 and 30 June 2000 (7 years) were reported previously.[1,2] In the current report all hospitalisations with a separation date in 2000/2001 or 2001/2002 were included. Hospitalisation data are presented and reported by date of admission. Data were extracted based on the International Statistical Classification of Diseases and Related Health Problems, 10th Revision, Australian Modification, 1st Edition (ICD-10-AM). Eligible separations were those with the code of interest listed in the principal diagnosis (the diagnosis chiefly responsible for the admission of the patient to hospital) or in any other diagnoses. The proportion of separations for which the diseases were coded as the principal diagnosis is reported for each disease. For hepatitis B, only principal diagnoses were included. Where the ICD-10-AM code for a disease specifies a severe manifestation (e.g. measles encephalitis) the number and type of these were reported as complications. The variables extracted for analysis were date of admission, financial year of separation, age at admission, sex, State/Territory of residence, length of stay (LOS), and diagnosis (principal and other diagnoses—up to 31 diagnoses were recorded for each admission) coded using ICD-10-AM. The mode of separation (whether a patient died while in hospital) and hours of mechanical ventilation (as a measure of critical care) were not included in this report due to concerns over the accuracy of these data. The only exception to this is deaths in hospital due

to meningococcal disease, as for this disease this measure was felt to be of importance. Where State of residence was missing in hospitalisation data this variable was replaced with State of hospitalisation, affecting 0.3 per cent of records in 2000/2001 and 2001/2002.

Deaths

Death data were obtained from the AIHW Mortality Database. These data are supplied annually to the AIHW from the Registrars of Births Deaths and Marriages in each State and Territory via the Australian Bureau of Statistics (ABS). Deaths include those in Australian waters as well as on Australian soil, whereas ABS published data exclude deaths in Australian waters. Since 1997, the International Classification of Disease, 10th Revision (1992), ICD-10 has been used to identify the cause of death. Although multiple causes of death have been recorded since 1997, only those where the underlying cause of death was the disease of interest are used in this report. Deaths analysed in this report were those registered in 2001 to 2002 (two years). The variables extracted for each death were: underlying cause, age, year death was reported, sex, and State/Territory in which death was recorded.

Calculations

All rates were calculated using finalised ABS mid-year estimated resident populations, and are presented as annual rates or average annual rates per 100,000 total population or population in age, sex or geographical subgroups, as appropriate. Average annual rates were calculated by dividing the total number of cases for the period of investigation by the sum of each year's population for the same period. For hospitalisation data, the mid-year population estimate for the first half of the financial year was used as the denominator—e.g. the 2000 mid-year population estimate was used to calculate rates for 2000/2001. For notification data, the denominator population for each year included only jurisdictions notifying cases for that entire year. Averages were calculated for rates of notifications and hospitalisations and for bed days per year. Medians and ranges, rather than averages, were used to describe the distribution of notifications and hospitalisations per month, and length of stay per admission, as these data were not normally distributed.

Report structure for individual diseases

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

- secular trends — the pattern of notifications and hospitalisations over time, with reference to seasonality and outbreaks;
- severe morbidity and mortality — hospital bed days, length of stay, principal diagnosis, complications and mortality by age group in standard categories;
- age and sex distribution — data by age and sex groups as relevant for each particular disease;
- geographical distribution — case numbers and rates by State/Territory, as shown in Appendices 2 and 3;
- comment — discussion of the data presented.

Vaccination coverage data

During the review period of this report there was one source of data about national vaccination coverage: the Australian Childhood Immunisation Register (ACIR). The ACIR commenced in January 1996 and is administered by the Health Insurance Commission for the Australian Government Department of Health and Ageing. The ACIR records details, as supplied by vaccination providers, about the vaccination status of children aged less than seven years. Vaccination coverage estimates derived from ACIR data have been reported in *Communicable Diseases Intelligence* since early 1998. A complete description of the method for calculating coverage estimates by age cohorts is given elsewhere.[11] In this report we have described trends in ACIR vaccination coverage estimates for all vaccines on the current childhood schedule.

Notes on interpreting data

Vaccine preventable diseases data

Comparisons between the notification, hospitalisation and death databases should be made with caution as they differ in their purposes, reporting mechanisms and accuracy. To provide the most recent information available and to account for the varied reporting formats, different time periods have been reviewed for each data set. As there were no unique identifying codes to link records for the same individual across databases and because of differences in the accuracy of each database, it was not possible to analyse deaths and hospitalisations as a subset of notifications.

The rates presented here are crude rates and may be confounded by differences in the population structure (e.g. age, ethnicity and population density) between jurisdictions. An exploratory analysis of 2002 pneumococcal and incident hepatitis B notification rates for the Northern Territory found that directly age-standardising the rates to the 2001 Australian population did not change the rates significantly (pneumococcal crude rate 20.2 per 100,000 *vs* 20.5 per 100,000 age-standardised; hepatitis B crude rate 6.8 per 100,000 *vs* 5.7 age-standardised.) Therefore, given that the Northern Territory is the jurisdiction with the most different age structure, we have elected to use crude rates. It is also important to note that jurisdictions with small populations may have high rates even with low absolute numbers of cases, so that a small change in numbers results in a large change in rates.

Notification data

A major limitation of the notification data is that they represent only a proportion of the total cases occurring in the community. This proportion may vary between diseases, with infections diagnosed by a laboratory test more likely to be notified. Data accuracy may also vary between States/Territories due to the use of different case definitions for surveillance and varying reporting requirements by medical practitioners, laboratories and hospitals. For example, during this reporting period notifications of rubella and mumps in New South Wales were purely laboratory based, whereas in other jurisdictions clinical or epidemiologically linked cases were notifiable by medical practitioners. In addition, data accuracy may change over time as new diagnostic tests are introduced or surveillance practices change.

Hospitalisation data

Comparisons over time and between jurisdictions should theoretically be more valid for hospitalisation data than for notification data, because methods of collecting hospitalisation data are more uniform. However, some variation in hospital access, admission practices, use of diagnostic tests and record coding may occur between regions and over time. In 1998/1999 most States and Territories began using ICD-10-AM and in 1999/2000 all jurisdictions were using the new classification. This change impacted on the sensitivity and specificity of some diagnostic codes. The most notable impact has been on the number of hospitalisations for acute hepatitis B as, unlike the previously used ICD-9-CM, ICD-10-AM allows differentiation between acute and unspecified infection.

There are also limitations associated with the use of ICD codes to identify cases. Hospital coding errors have been reported to occur more commonly for diseases that the coder was less familiar with (e.g. rare diseases such as tetanus or diphtheria) and for admissions with multiple diagnoses.[12] Assignation of codes is based on information in medical records, as recorded by clinicians, and there are few strict case definitions. As indicated in relevant disease chapters, the short lengths of stay and lack of notification to public health authorities strongly suggest that some cases with hospitalisation codes for diseases such as tetanus and diphtheria are likely to be due to coding errors. For some diseases, such as *Haemophilus influenzae* type b infection, the previously used ICD-9-CM and ICD-10-AM codes lack specificity. This is in contrast to the more stringent case definitions used for notification data. It must also be noted that the hospitalisation database contains a record for each admission, which means that there are separate records for each readmission or inter-hospital transfer. This is unlikely to have a major impact on the numbers reported for most diseases reviewed, as they are acute illnesses. For hospitalisations where the code of interest was not the principal diagnosis, the code of interest will have been recorded as a co-morbidity (additional or secondary diagnosis), the relative importance of which cannot be gauged.

Death data

Mortality data were analysed by year of registration rather than by year of death, as annual reports to AIHW are by year of registration, so not all deaths occurring in a year would be included in that year's data. Approximately six per cent of deaths in a particular calendar year are registered in the subsequent year, with the bulk comprising that calendar year's December deaths.

Only those deaths where the underlying cause of death was the disease of interest are reported here. Hence deaths where the disease of interest was a contributing cause of death are not included.

The problems associated with the accuracy of the ICD codes used for hospital separations may also apply to the mortality data. As noted for hospitalisation data, the move from ICD-9 to ICD-10 codes (which occurred in 1997) may impact on the comparability of some death data and this must be borne in mind when comparing years. This is especially important for numbers of deaths where the underlying cause was recorded as hepatitis B. Prior to the use of ICD-10, acute, chronic and unspecified infections could not be differentiated.

Vaccination coverage data

Limitations of data available from the ACIR must be considered when it is used to estimate vaccination coverage. Vaccine coverage estimates calculated using ACIR data should be considered minimum estimates due to under-reporting.[4,9,10] Another limitation of ACIR data is that records are only held for children up to seven years of age. Also, coverage is calculated only for children registered on Medicare; however, by the age of 12 months it is estimated that over 98 per cent of Australian children have been registered with Medicare.[11,13,14]

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 toxaemia. 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.[15,16] 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.[17] 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.[18] 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.[19] 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.[20] 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.[21] International and Australian (NCIRS, unpublished data) serosurveys have shown that many adults in developed countries are now susceptible to diphtheria.[22,23] 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,[24] 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.[25] 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.[26,27] 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.[27–29] Aboriginal children had a particularly high risk of Hib meningitis with rates among the highest recorded anywhere in the world.[30] 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.[31]

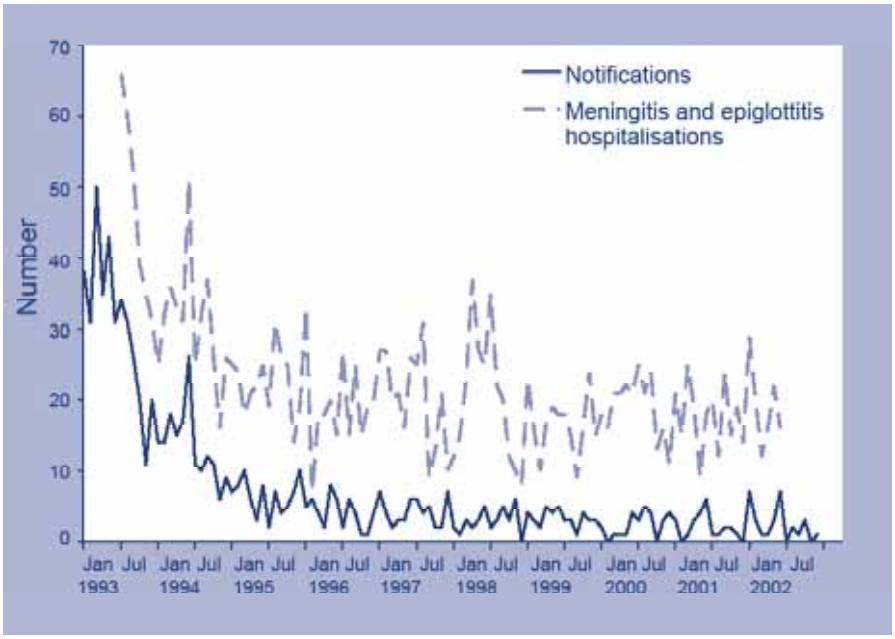
Hospitalisations and deaths

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.

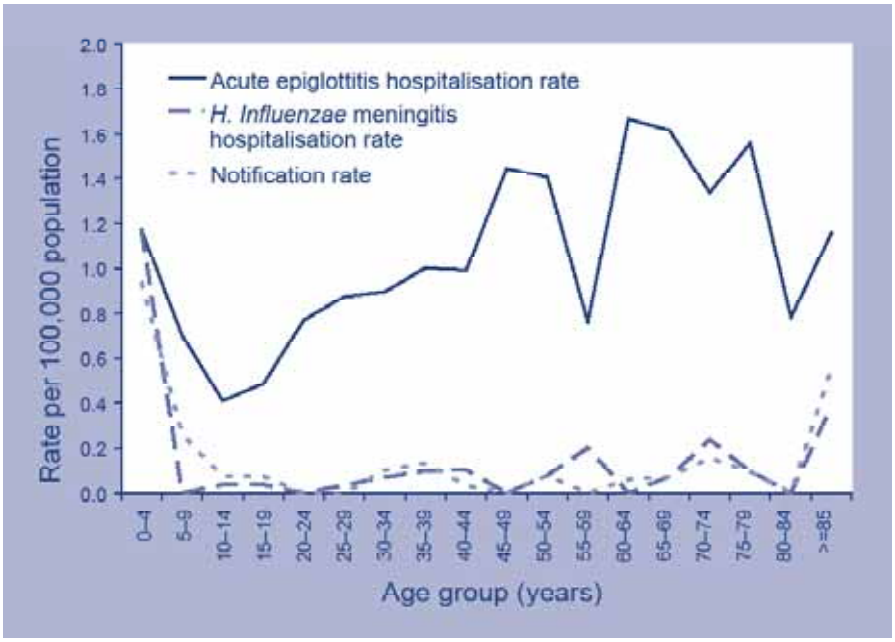
‖ Includes cases with unknown ages.

** 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.

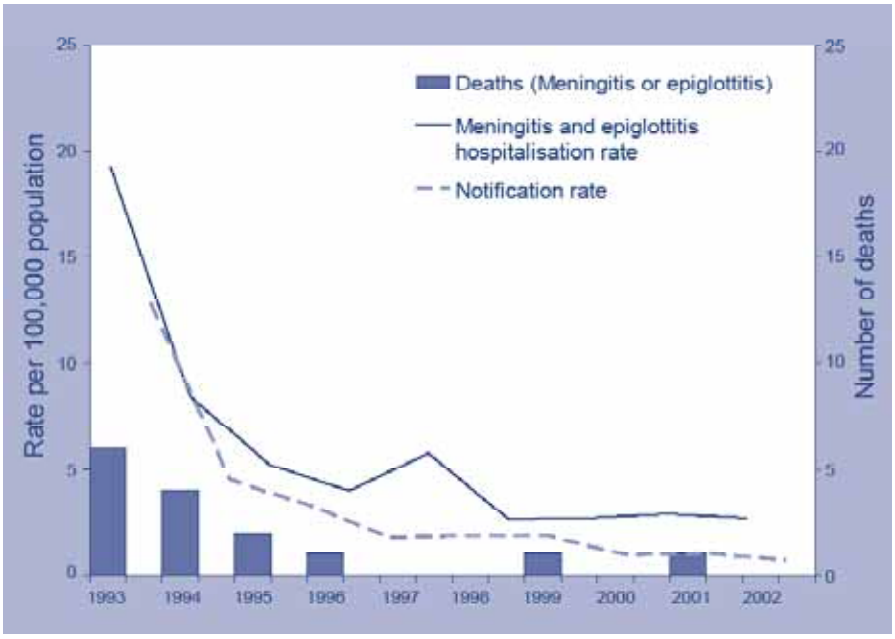
Figure 2. *H. influenzae* type b (Hib) notification and presumed Hib hospitalisation rates, Australia, 2000 to 2002,* by age at admission



* 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.[32] 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.[31] The case definition for national notification has recently been modified to include only those with laboratory definitive evidence as outlined above.[33] 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.[34]

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.[35] 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.[36] However, most cases of epiglottitis in adults have no identifiable cause or may be due to organisms other than *H. influenzae*.[37,38] 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.[39] 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.[40–42] 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.[15,16]

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.

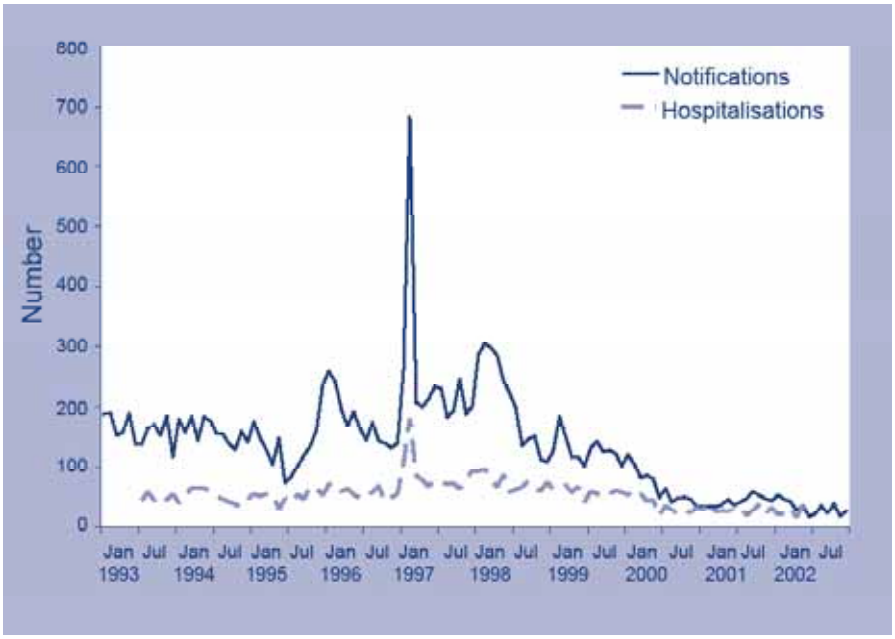
Notification and hospitalisation rates declined in 2001 and again in 2002 compared with previous years (Figure 4). There was no apparent seasonality in notifications or hospitalisations.

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† per admission (days)	Deaths 2 years (2001–2002)	
	n	Rate‡	n	(‖)	Rate‡	(‖)	Median	n	Rate‡
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§	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.

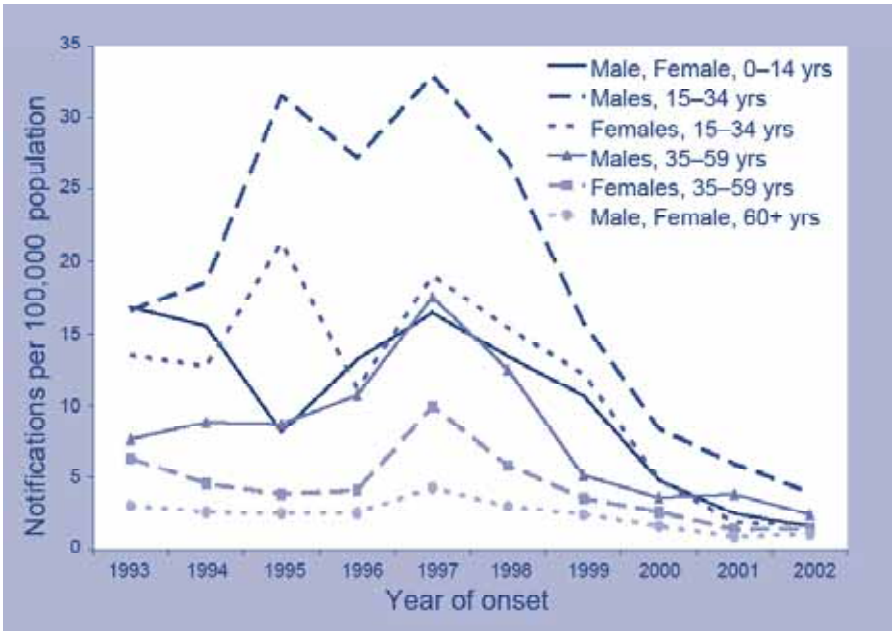
‖ Principal diagnosis.

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[43] and large community-wide epidemics mainly among men who have sex with men and illicit drug users.[44–47] 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.

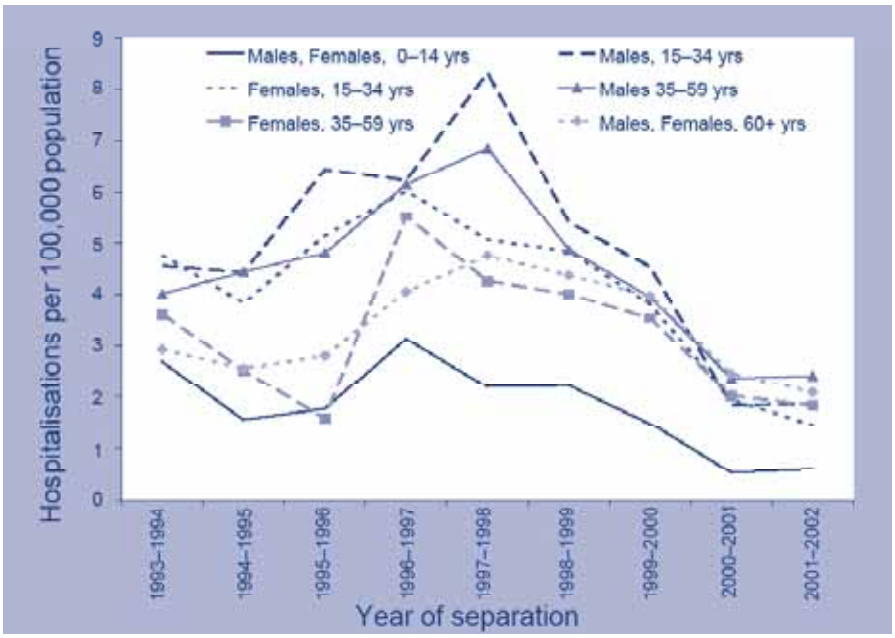
In Australia, the groups most at risk of acquiring and transmitting hepatitis A are travellers to countries where hepatitis A is endemic, children attending child care and preschool, people who use illicit drugs, men who have sex with men, sewage workers, food handlers and Indigenous Australians.[48–50]

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.[46] 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.[50] During 1999–2002 there were three deaths due to hepatitis A among children aged less than five years; all were Indigenous.[45,50]

Hepatitis A vaccines are effective in preventing disease in individuals[49] and in controlling outbreaks in some settings.[49,51] 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.[52] 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.[53] 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.[48]

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.[16,54] 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.[16] Of people chronically infected with HBV, 15–40 per cent develop cirrhosis of the liver and/or hepatocellular carcinoma.[55,56]

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.[16] 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.[16] 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.[57] 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.[56,58,59]

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.[2]

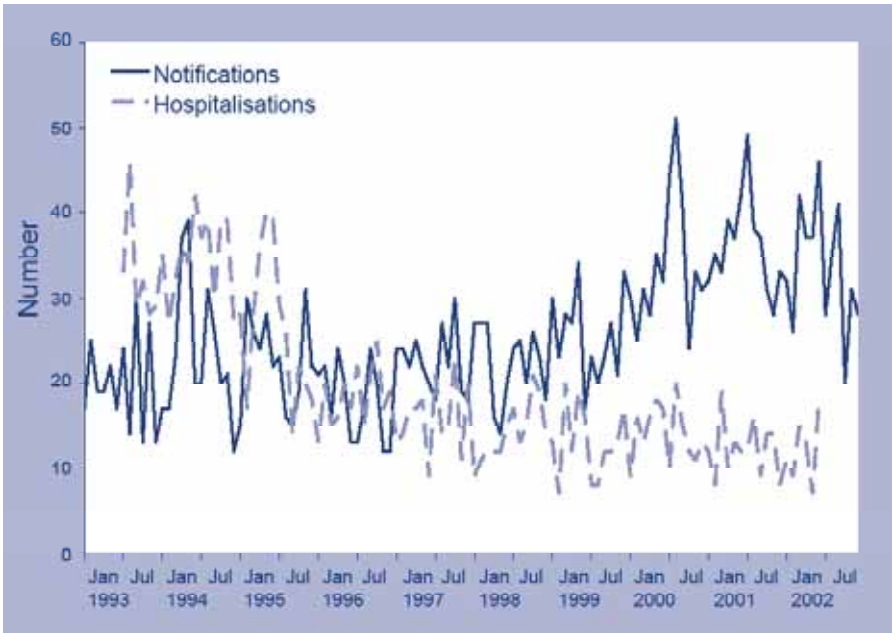
Deaths

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.

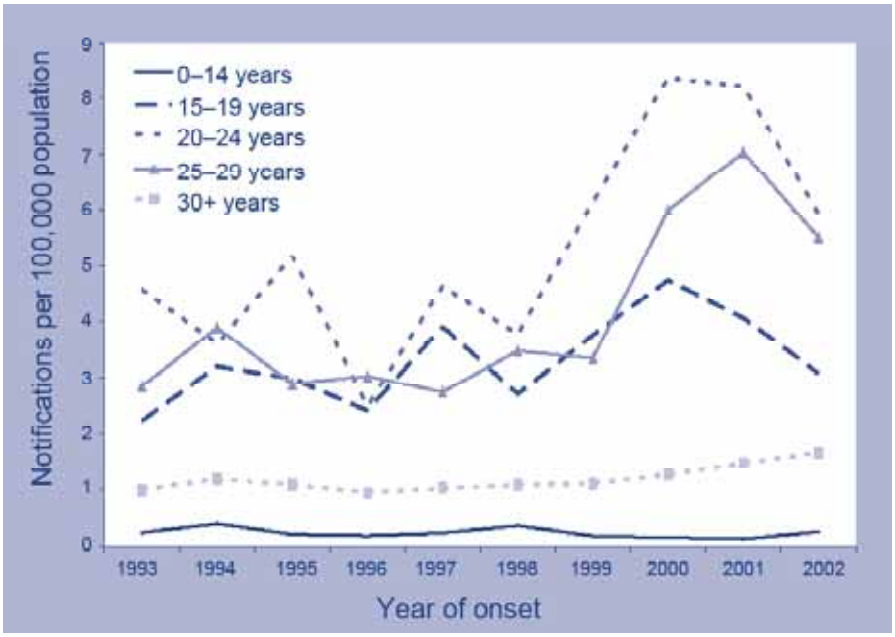
† ICD-10-AM codes B16.0 and B16.2.

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.

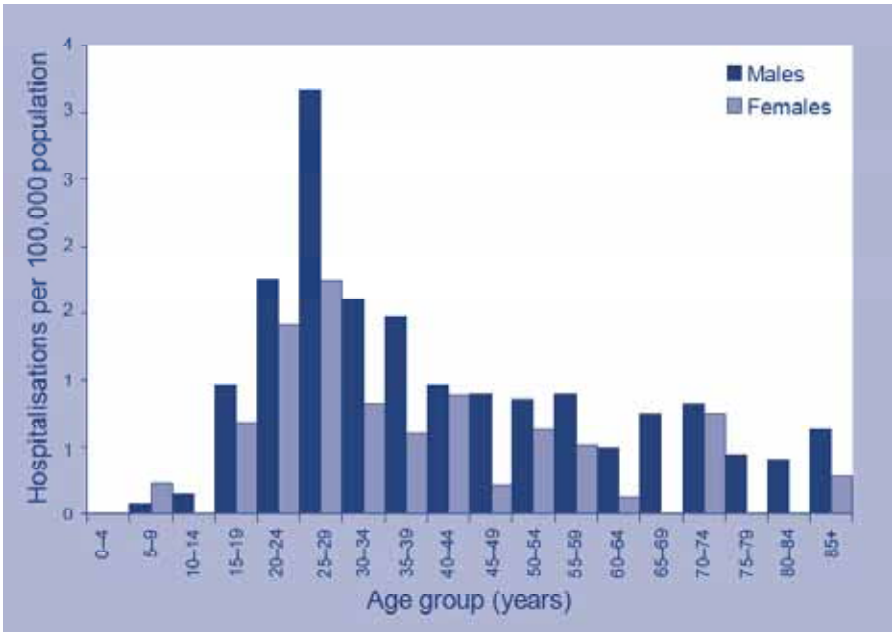
During 2000/2001 and 2001/2002, rates for hospitalisations with a principal diagnosis of acute hepatitis B were the highest in adults aged 25–29 years (2.5 per 100,000) and 20–24 years (1.6 per 100,000) (Figure 9). Like notifications and as in previous years, hospitalisations occurred predominantly in 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.[60]

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.[58,61] 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.[62] 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

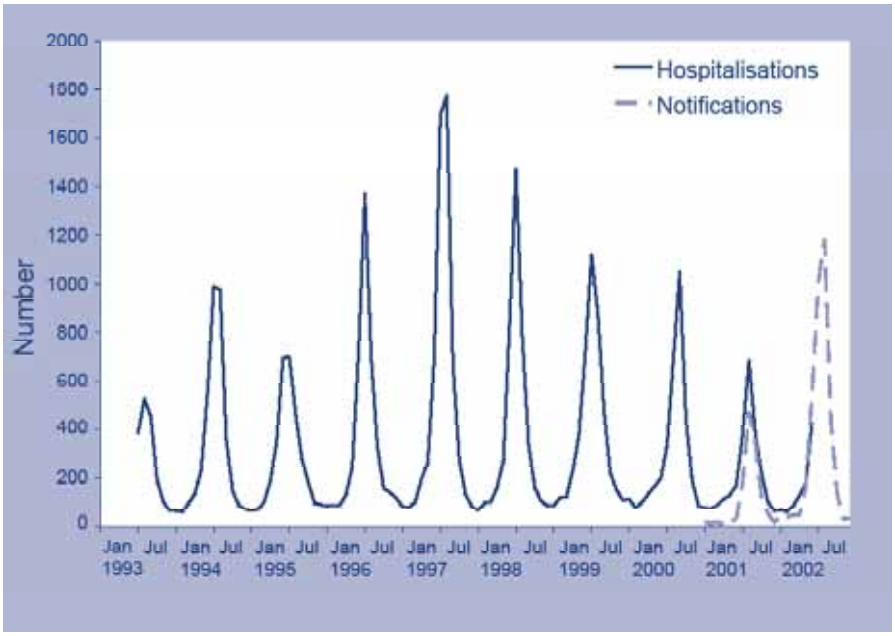
The ICD-10 codes used to identify deaths were: J10 (influenza due to identified influenza virus) and J11 (influenza, virus not identified).

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† per admission (days)	Deaths 2 years (2001–2002)	
	n	(‖)	Rate‡	(‖)	Median	n	Rate‡
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§	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).

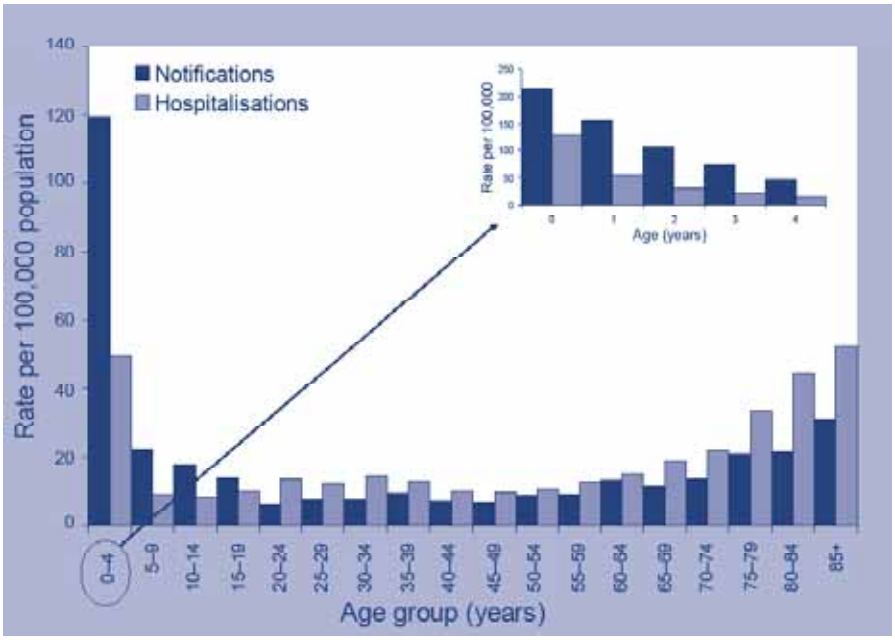
Age and sex distribution

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)[63] or picornavirus.[64,65] 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.[66–68] 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.[69,70] 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.[49] Vaccination uptake in Australians aged 65 and older was estimated at 76.9 per cent in 2001, 2002[71] and 2003.[72] 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.[73] 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,[67,74,75] has also recommended the routine vaccination of healthy American children aged six to 23 months with influenza vaccine.[76,77] 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.[78,79]

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.[62] 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.[80] 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.[81–84]

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.[16]

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 38ºC 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

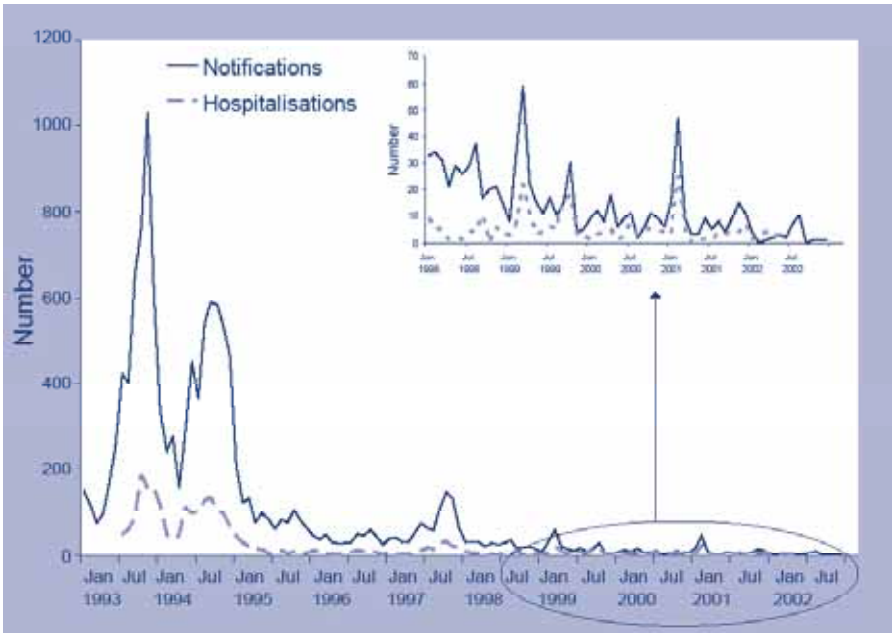
The ICD-10-AM/ICD-10 code B05 (measles) was used to identify hospitalisations and deaths. SSPE was not included in these analyses.

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

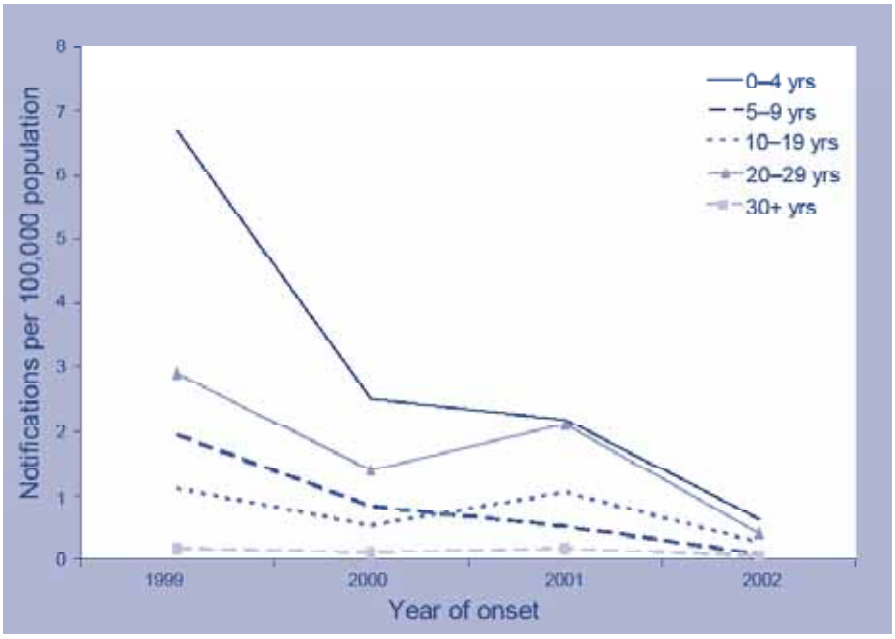
In the two year review period, notification and hospitalisation rates for children under 10 years of age continued a downward trend (Figures 13 and 14). In 2002, the notification rate for the 0–4 year age group (0.6 per 100,000) and 5–9 year age group (0.07 per 100,000) was the lowest on record. Similarly, 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.

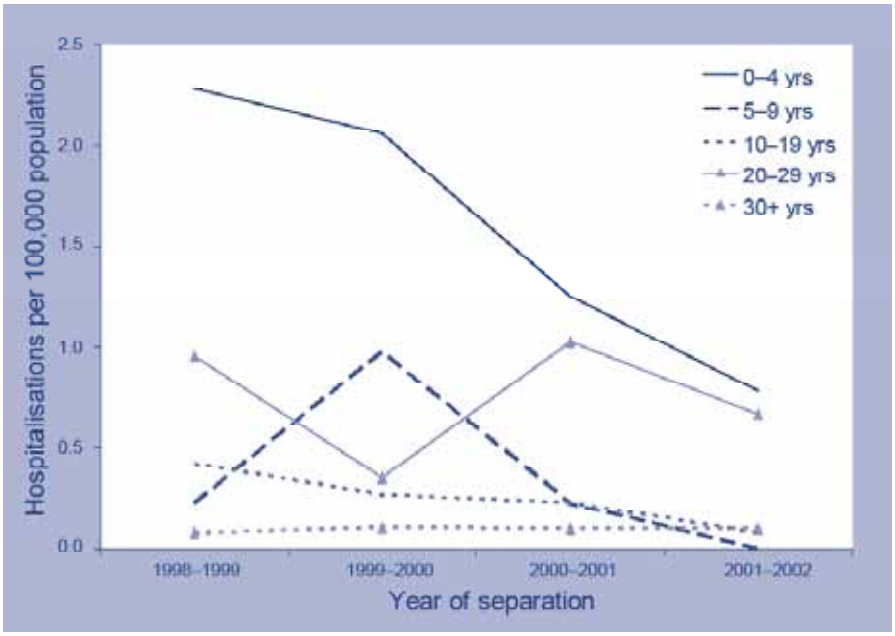
Over the two year review period there were slightly more notifications for females than males (male:female ratio 1:1.1). Conversely, there were more hospitalisations of males than females (male:female ratio 1.4:1).

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).[8,85,86] A smaller

outbreak of 18 cases occurred later between October and December 2001.[8,86] The only other outbreak to be reported for 2001 was in Sydney and involved seven cases.[8] 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.[87]

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.[88,89]

Now that measles is rare, enhanced surveillance including a high level of confirmation is required and recommended by the WHO.[90] 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.[91] 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.[92] Since 1999, over 80 per cent of notified cases have been confirmed with most of the improvement in ages less than 15 years.[93] 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,[94] 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.[95] 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[96] 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.[97] 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.[98]

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

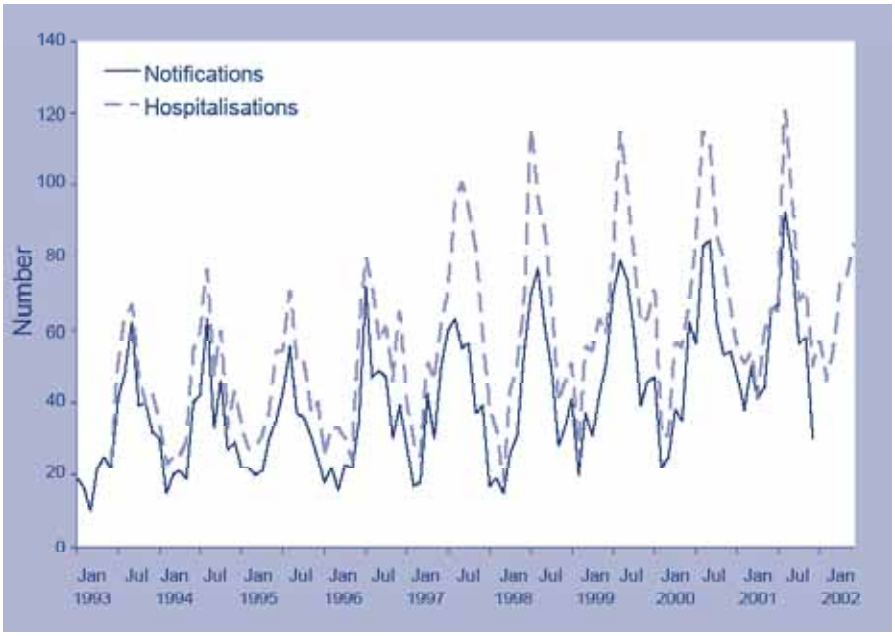
The ICD-10 code used to identify deaths was A39 (meningococcal infection).

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.

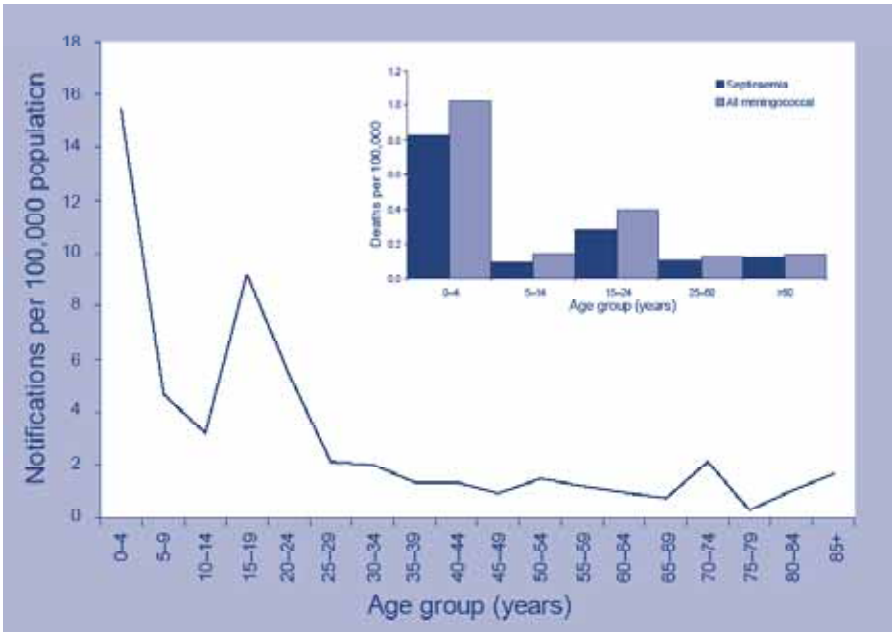
† LOS = length of stay in hospital.

‡ 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

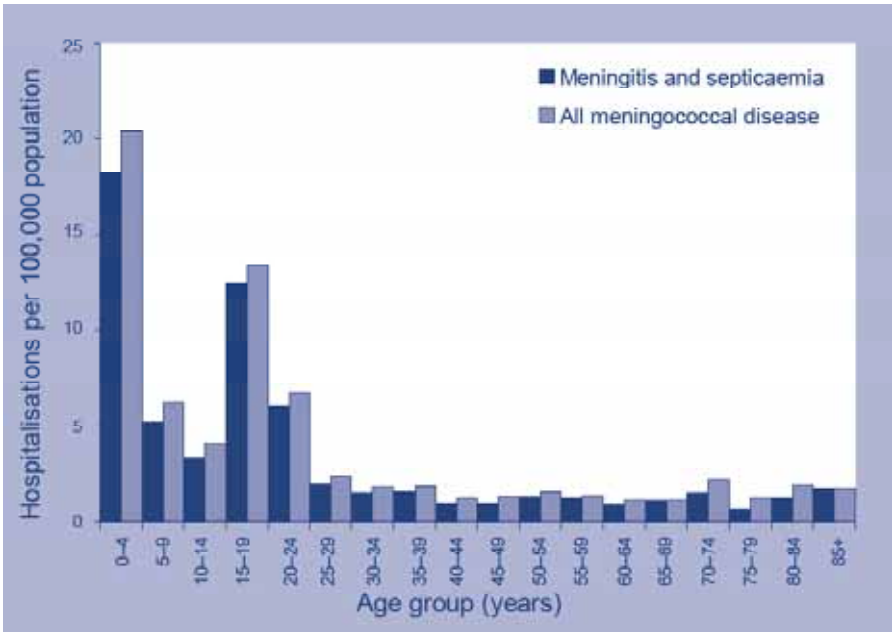


* Notifications where the month of onset was between January 2001 and December 2002, deaths where the date of death was recorded between 2001 and 2002.

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.[99] 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.[100,101] 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.[102] 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.[103,104] Serotype-specific data are important for vaccine policy, as conjugate vaccines against serogroup C are now in widespread use.[105] 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.[103,106–108] 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.[109] 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.[106–108]

As found elsewhere, in Australia serogroup C meningococcal disease is associated with a higher mortality than serogroup B.[103,106–108] 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.[110] 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.[111] 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.[112] 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.[16]

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

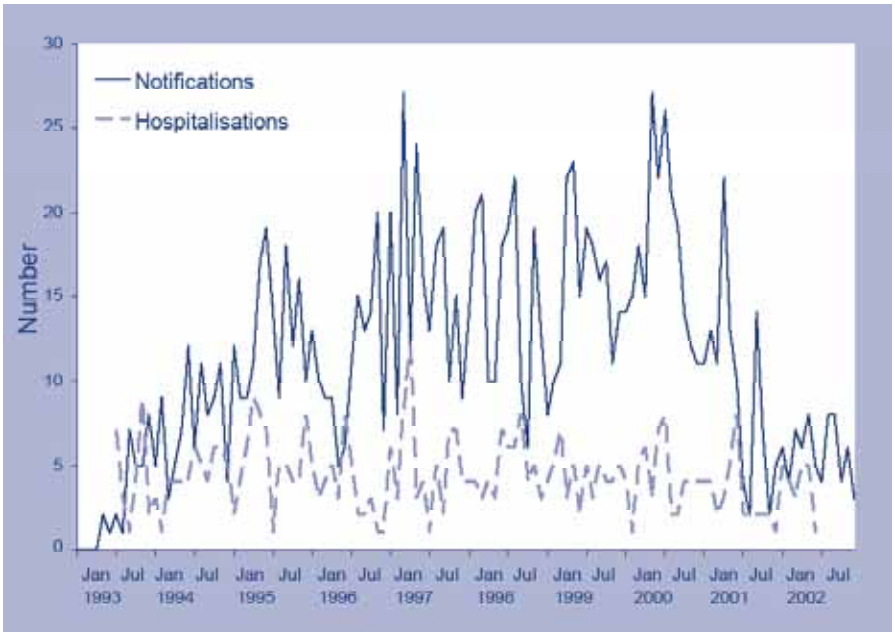
During the two years from 2001 to 2002 there were 180 notifications of mumps (an average annual notification rate of 0.5 per 100,000) (Table 11). Notification rates have shown a twofold decline each year between 2000 and 2002; in 2002 they were the lowest on record (0.4 per 100,000) since all jurisdictions 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.

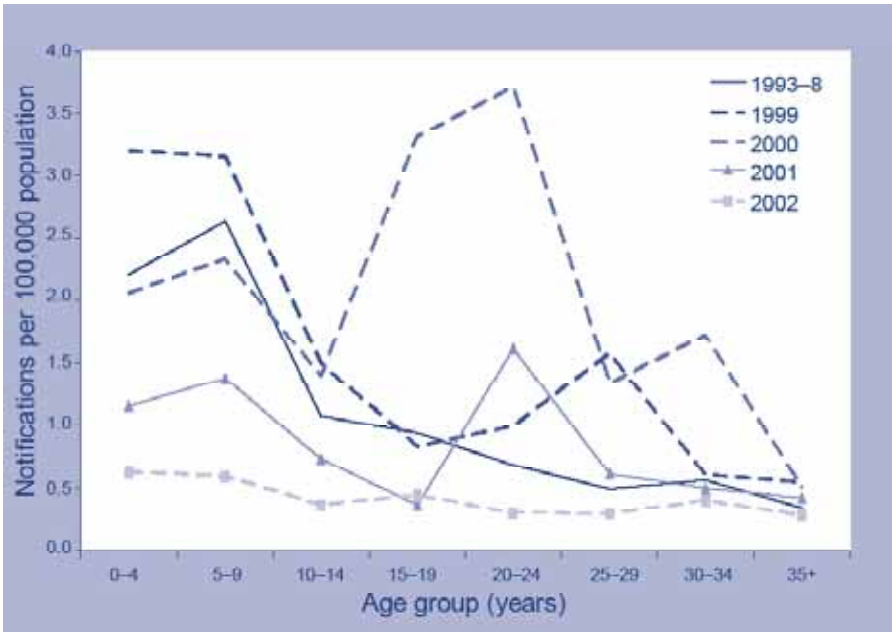
Age and sex distribution

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.

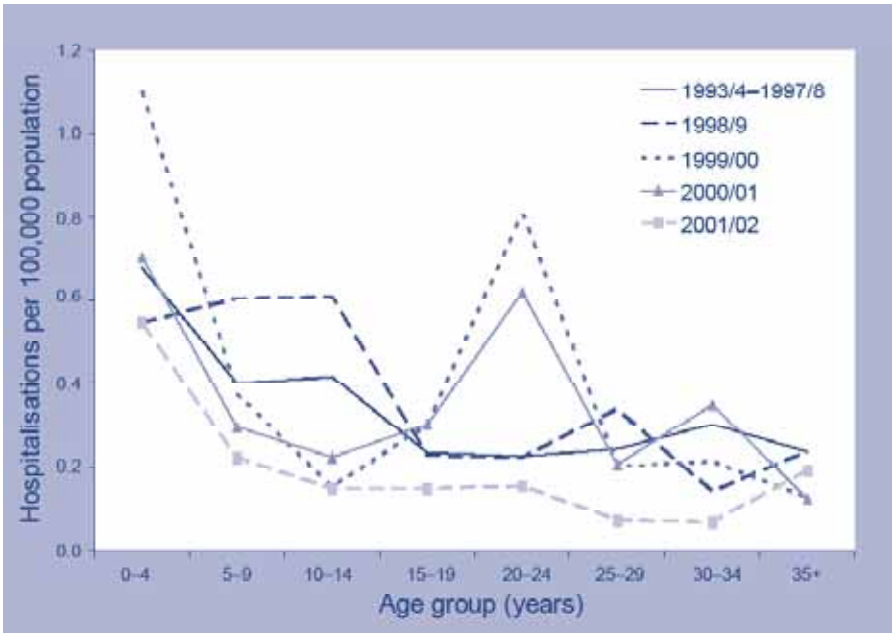
Over the two year review period the male:female ratio was 1.0:1 for notifications and 1.2:1 for hospitalisations. However, this conceals annual differences. In the first year of the review there were more females (M:F ratio 0.9:1 for both notifications and hospitalisations) while in the second year there 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[113] and Finland (where mumps has been eliminated).[89]

The record low rates are probably due to several factors. In July 2001, Victoria introduced a system of enhanced surveillance for mumps.[114–116] 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.[95] However, there is evidence to suggest that the latter campaign did not significantly improve immunity levels[96] 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.[114–116] 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.[117] 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.[16]

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

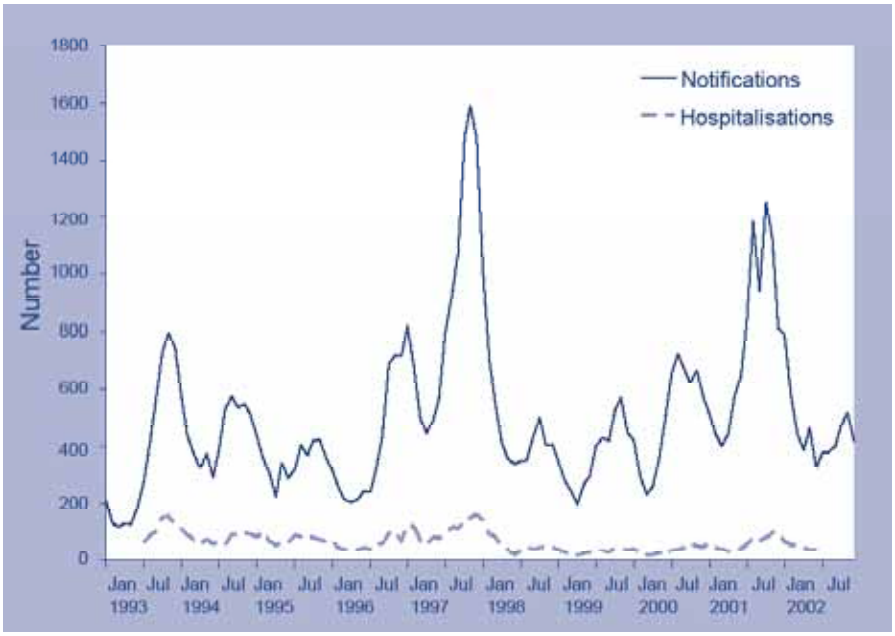
The ICD-10-AM/ICD-10 code A37 (whooping cough) was used to identify hospitalisations and deaths.

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.[2]

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† per admission (days)	Deaths 2 years (2001–2002)	
	n	Rate‡	n	(‖)	Rate‡	(‖)	Median	n	Rate‡
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§	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.

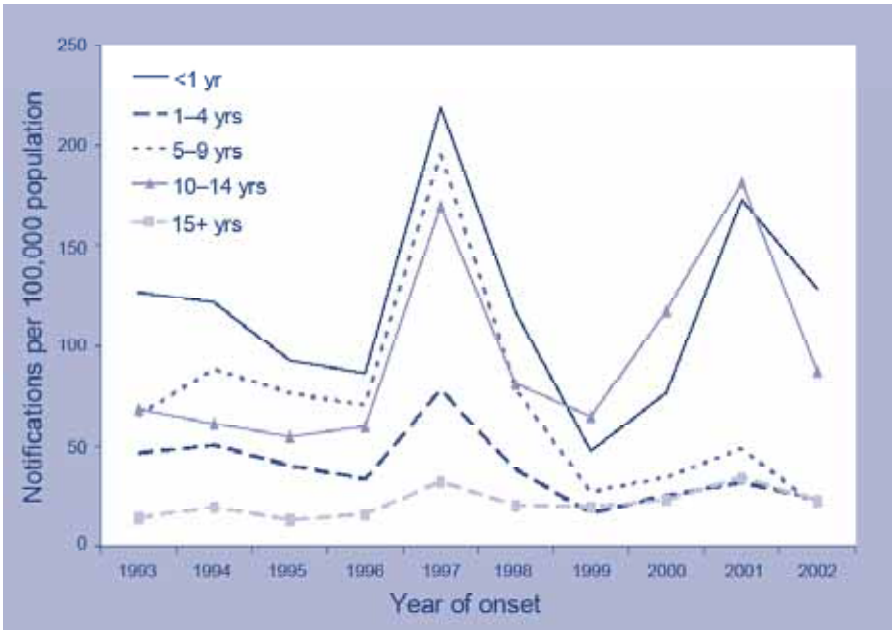
For the two years 2001 to 2002, six deaths were recorded where pertussis was the underlying cause (Table 13). Two occurred in 2001 and four in 2002. All but one, who was 85 years of age, were two months old or younger. The 85-year-old case is rather atypical and could be a coding error. Between 1993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

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).[2]

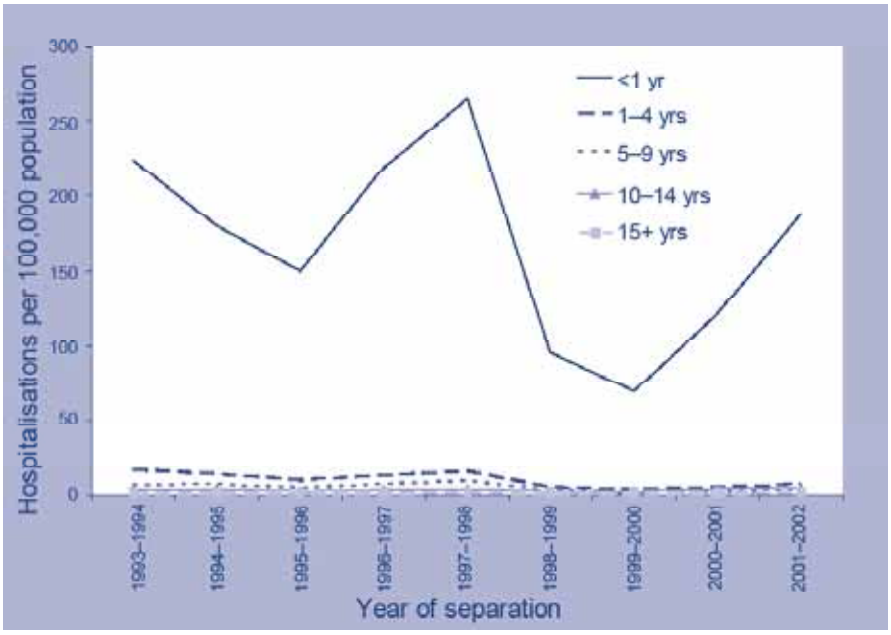
The 10–14 year age group accounted for 25 per cent of pertussis notifications in 2001 and 2002 (n=3,646) and seven per cent of all hospitalisations (n=88; average hospitalisation rate of 3.3 per 100,000, twice the average rate of 1.6 per 100,000 for the period 1998/1999–1999/2000).[2] The 10–14 year 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).[1,2] 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.[1,2] 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.[2,118] In children, hospitalisations coded as whooping cough have been shown to have a high correlation with clinical pertussis.[119] 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.[120]

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,[121] in New South Wales[122] and internationally.[123]

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.[124] To address this problem, and following the approval in 2001 of an acellular adult-formulated vaccine (dTpa),[125] 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.[49] 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.[126] The change should also reduce the frequency of local reactions following the booster dose at four years.[127] 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

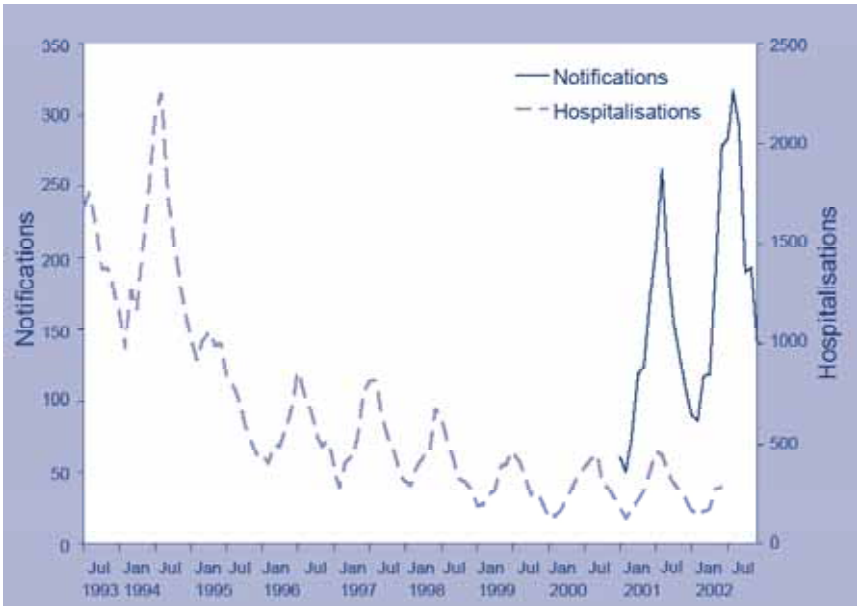
ICD-10 codes G00.1, A40.3 and J13 were used to select deaths from IPD.

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).

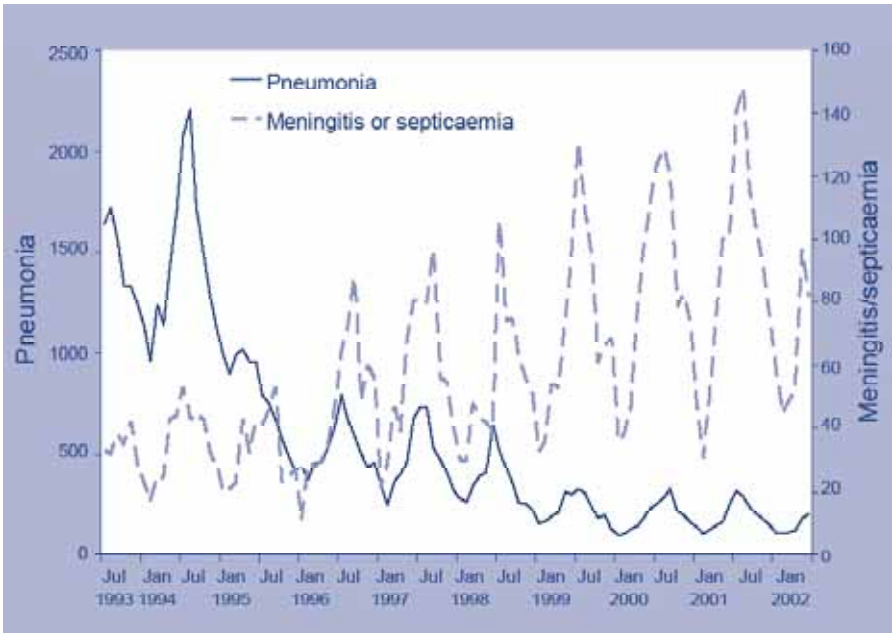
The total number of hospitalisations coded as pneumococcal meningitis, septicaemia or pneumonia for 2001 to 2002 was 6,782, an average annual rate of 17.6 per 100,000 (Table 14). Hospitalisations coded as meningitis or septicaemia accounted for 31 per cent of total episodes giving a hospitalisation rate 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† per admission (days)		Deaths 2 years (2001–2002)			
	n‡	Rate§	nII	(M/S)¶	RateII §	(M/S)¶ §	MedianII	(M/S)¶	nII	(M/S)¶	RateII §	(M/S)¶ §
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.

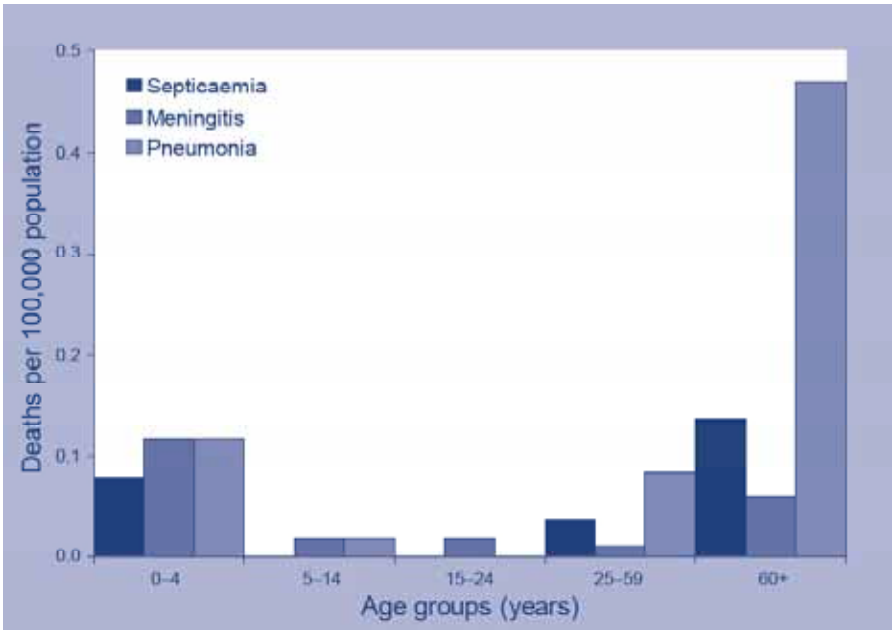
§ Average annual age-specific rate per 100,000 population.

II 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.

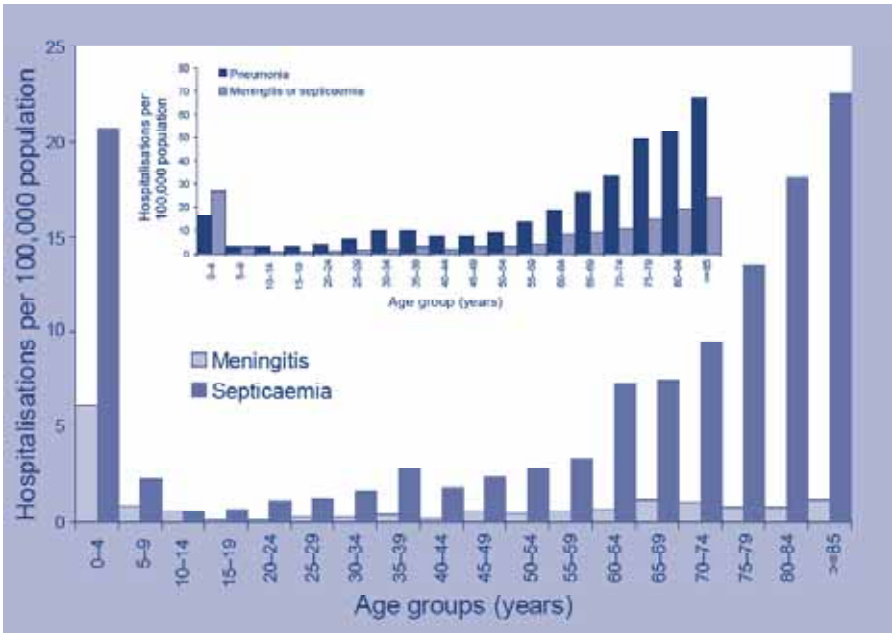
Age and sex distribution

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[128] and 2002.[129] 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.[49] 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.[130] 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,[49] 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.[128] Estimates of overall incidence from laboratory surveillance in industrialised countries comparable to Australia range from 9 to 22 per 100,000 per year.[131] 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.[132] Aboriginal people are also known to have high rates of pneumococcal disease in Western Australia[133] and north Queensland, where the polysaccharide vaccine program targeting Indigenous adults has been associated with significant reductions in IPD.[134] 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,[135] 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.[16]

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.[136] All cases notified since 1972 have been fully investigated with subsequent reclassification as VAPP. The most recent case of VAPP was reported in 1995.[137] 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.[138,139]

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.[140] The region of the Americas was declared polio free in 1994 and the Western Pacific Region in October 2000.[140,141] 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.[140,142]

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).[138] 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.[139] 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).[142]

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[140] 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.[49] 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).[16]

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.[143–145]

Secular trends

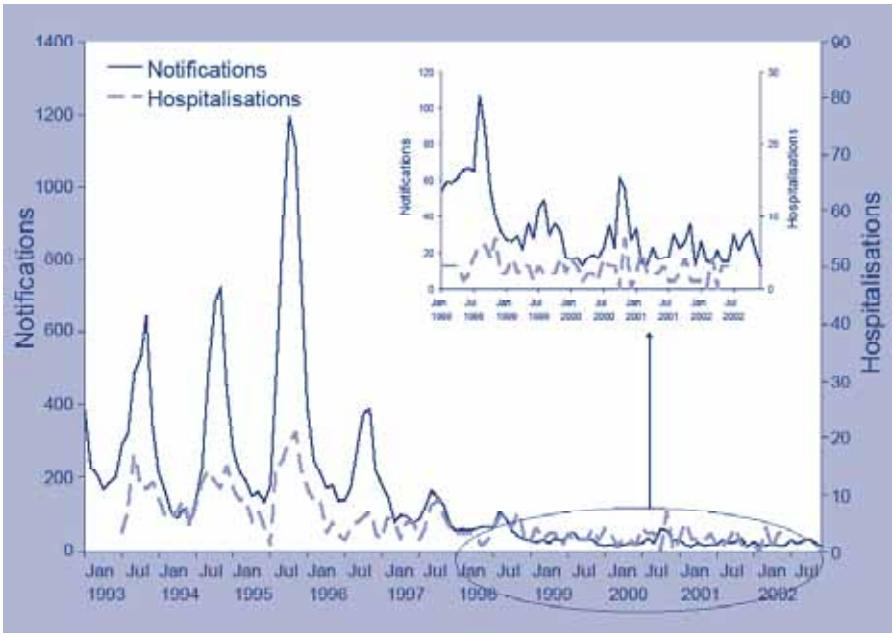
During 2001–2002 there were 511 notified cases of rubella, an average annual notification rate of 1.3 per 100,000 (Table 21). Between July 2000 and June 2002, 54 hospitalisations were coded as being due to rubella (an average annual rate of 0.1 per 100,000). Notification and hospitalisation rates were 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).

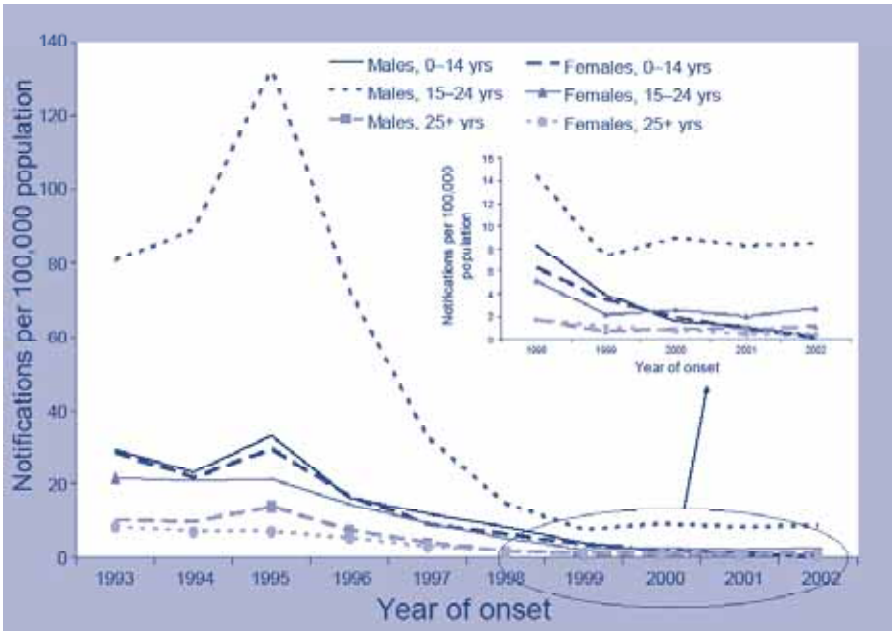
For the two years combined, 2000/2001 and 2001/2002, children aged 0–4 years continued to have the highest hospitalisation rates (average annual rate 0.5 per 100,000). However, as with notifications, hospitalisation rates in children less than 15 years, especially in those aged 0–4 years, have been declining 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.

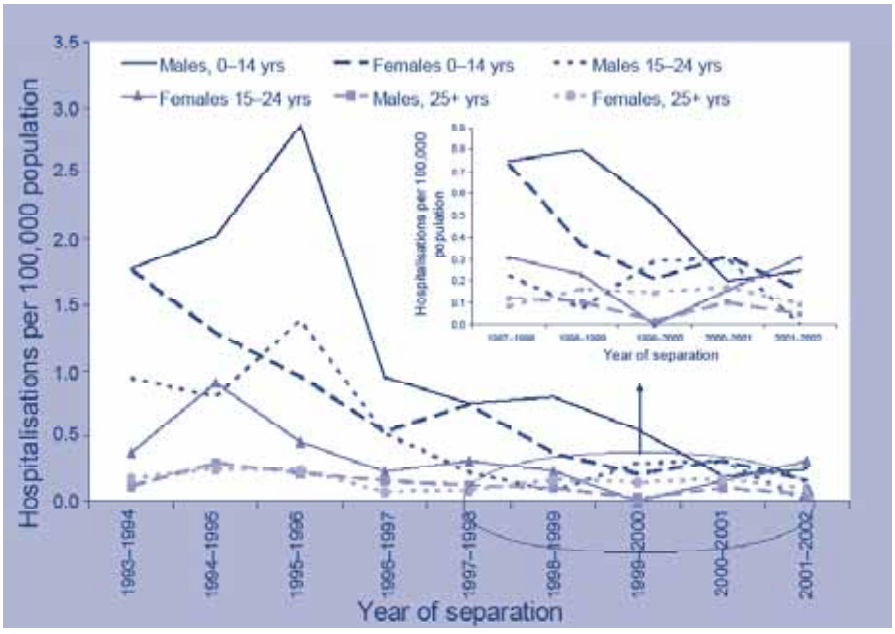
There were 95 notified cases of rubella in women of child bearing age (15–44 years) in 2001 and 2002, an average annual rate 1.1 per 100,000. This rate has been declining each year since the outbreak in 1995 (rate 16.0 per 100,000). However, in the three most recent years, only the 25–44 year age group 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.[96] 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;[94] 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.[114] 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.[146] This trend is of concern, as young adult males (who have also been shown in Australian serosurveys to have a low level of immunity)[147,148] 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.[95] However, there is evidence to suggest that coverage did not improve significantly[96] 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.[149] These were the first locally acquired cases of CRS since 1996.

Rubella and CRS are candidates for eradication after measles and polio.[150] Cuba has already eliminated rubella and CRS with mass vaccination campaigns targeting women aged 18–30 years and children aged 1–14 years.[146] 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.[146] 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.[146] 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.[16]

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.

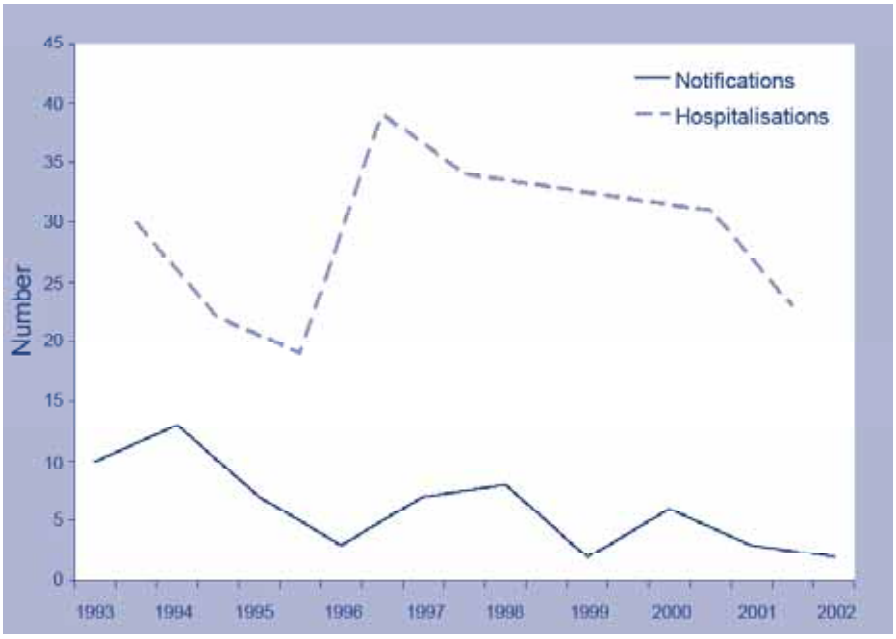
Secular trends

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† per admission (days)	Deaths 2 years (2001–2002)	
	n	Rate‡	n	(‖)	Rate‡	(‖)	Median	n	Rate‡
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§	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).

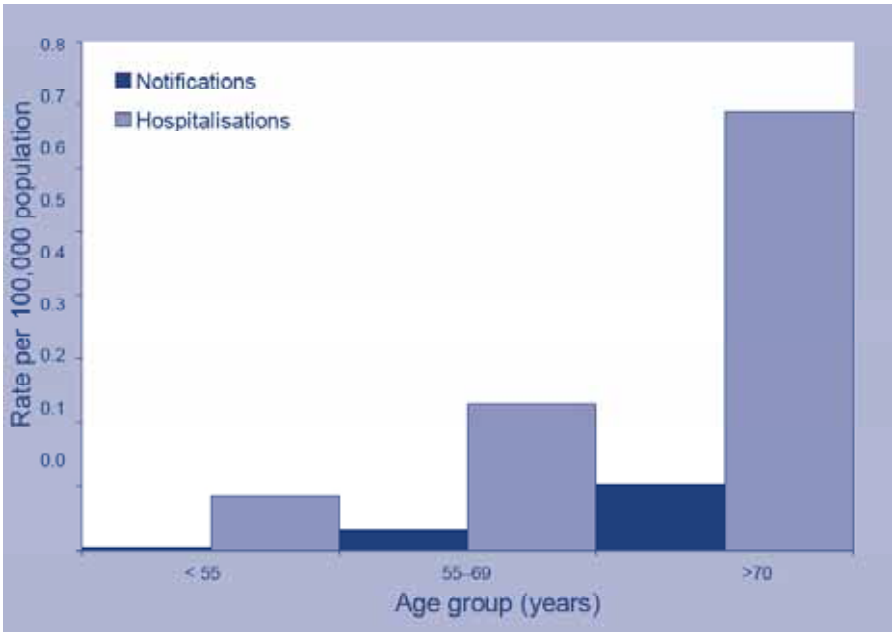
In the review period (2001 to 2002) there was one death in a person aged over 60 years with tetanus recorded as the underlying cause.

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.[151] International serosurveys and the Australian National Serosurvey have shown that immunity to tetanus is poor in older adults, particularly women.[23,152] 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.[86,153] One case was unimmunised and developed tetanus following abrasions sustained during gardening.[154] 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.[113,155,156] A tetanus booster is recommended at the age of 50 unless a booster has been documented within 10 years.[49] 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.[155,157] 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.[16]

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.[158] In Australia, however, seropositivity was 83 per cent by age 10–14 years.[159] Varicella is generally a benign, self-limiting illness in children, but morbidity and mortality rates are higher in adults,[160] at the extremes of ages, and in the immunocompromised.[161]

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.[162] Although herpes zoster can occur at any age, most cases occur after the age of 50 and incidence increases with age.[163] 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.[164–166]

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.

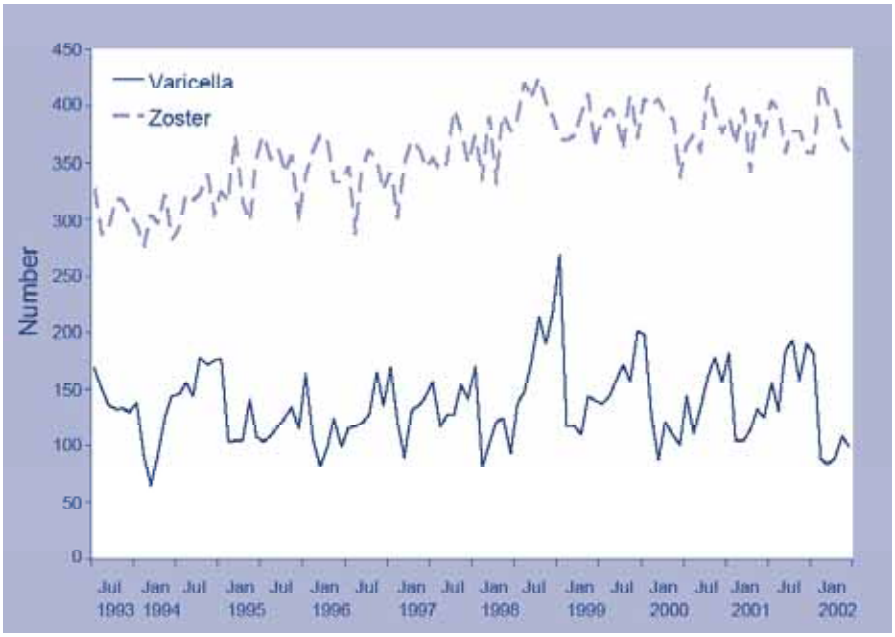
Secular trends in varicella and herpes zoster

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.

Figure 33. Varicella and herpes zoster hospitalisations, Australia, July 1993 to June 2002,* by month of admission



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

Severe morbidity and mortality, varicella

For patients with an ICD-10-AM code for chickenpox 16,057 hospital bed days (average 8,028 per year) were recorded. Of the 3,318 varicella hospitalisations, 2,247 (68%) had a principal diagnosis of varicella (average annual rate 5.8 per 100,000) (Table 18). Complications arising from varicella infection were recorded for 1,109 hospitalisations (33%). Of all varicella hospitalisations, 107 (3.2%) were coded as having encephalitis and 313 (9.4%) were coded as having pneumonitis (Table 19). Although most hospitalisations were in the youngest age group, people 60 years and older had the longest median length of stay. There were 19 deaths recorded with varicella as the underlying cause in the calendar years 2001–2002, 9 (43%) of them for people 60 years and older. The highest death rate was also recorded in people 60 years and older.

Table 18. Varicella hospitalisations and deaths, Australia, 2000 to 2002,* by age group

Age group (years)	Hospitalisations 2 years (July 2000–June 2002)				LOS† per admission (days)	Deaths 2 years (2001–2002)	
	n	(‖)	Rate‡	(‖)	Median	n	Rate‡
0–4	1,383	(951)	54.0	(37.1)	2	1	0.0
5–14	509	(341)	9.4	(6.3)	2	2	0.0
15–24	306	(223)	5.8	(4.2)	2	1	0.0
25–59	912	(629)	4.8	(3.3)	3	6	0.0
60+	208	(103)	3.2	(1.6)	9	9	0.1
All ages§	3,318	(2,247)	8.6	(5.8)	2	19	0.0

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

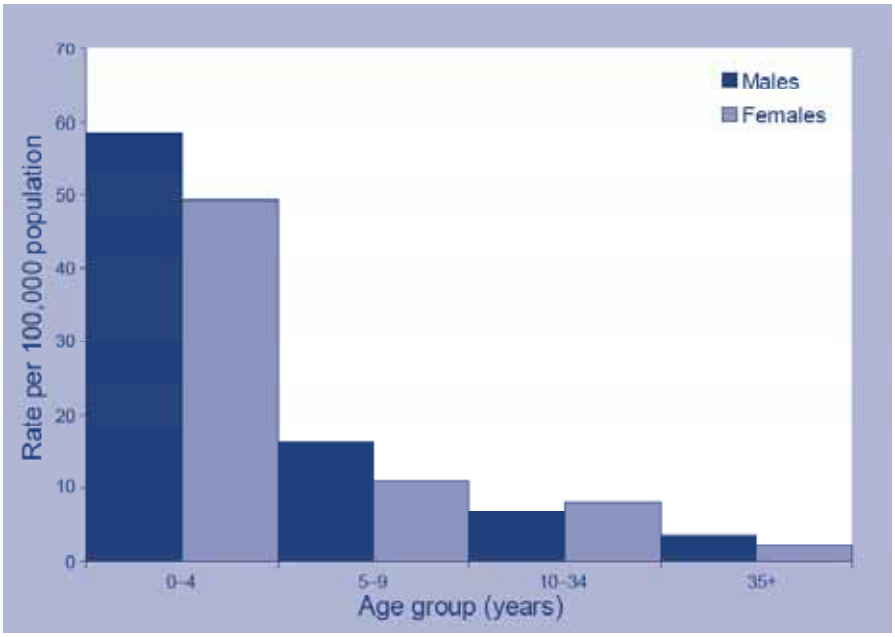
Age group (years)	Varicella encephalitis		Varicella pneumonitis	
	n	% total per age group	n	% total per age group
0–4	38	2.8	56	4.1
5–14	30	5.9	20	3.9
15–24	10	3.3	36	11.8
25–59	17	1.9	179	19.6
60+	12	5.8	22	10.6
All ages	107	3.2	313	9.4

* 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

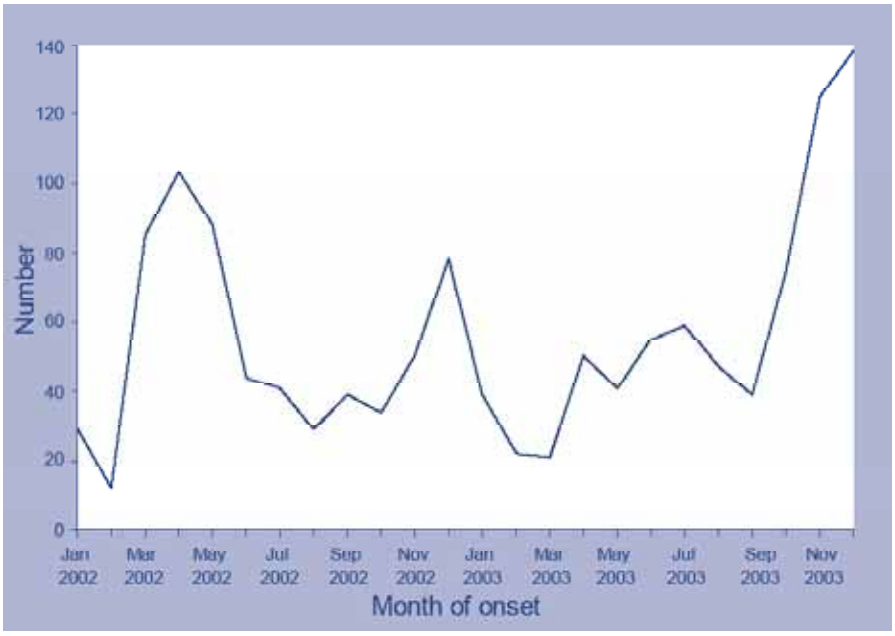
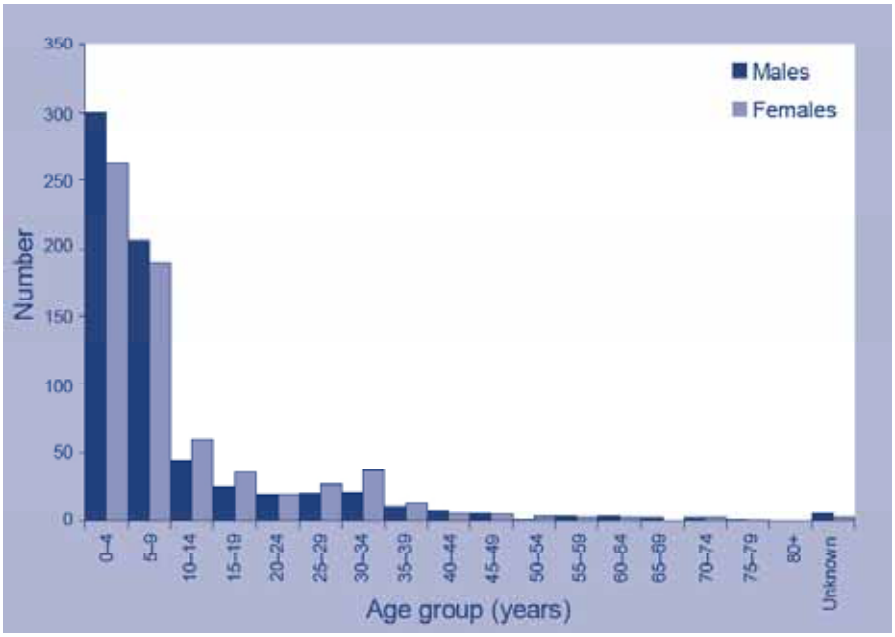


Figure 36. Varicella notifications, South Australia, January 2002 to December 2003 (based on date of onset), by age group and sex



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† per admission (days)	Deaths 2 years (2001–2002)	
	n	(‖)	Rate‡	(‖)	Median	n	Rate‡
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§	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

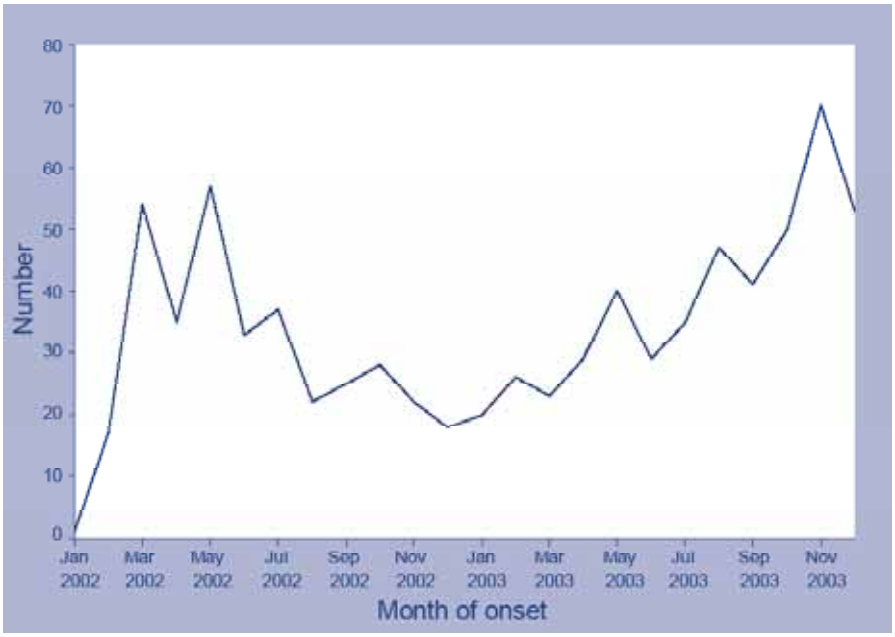
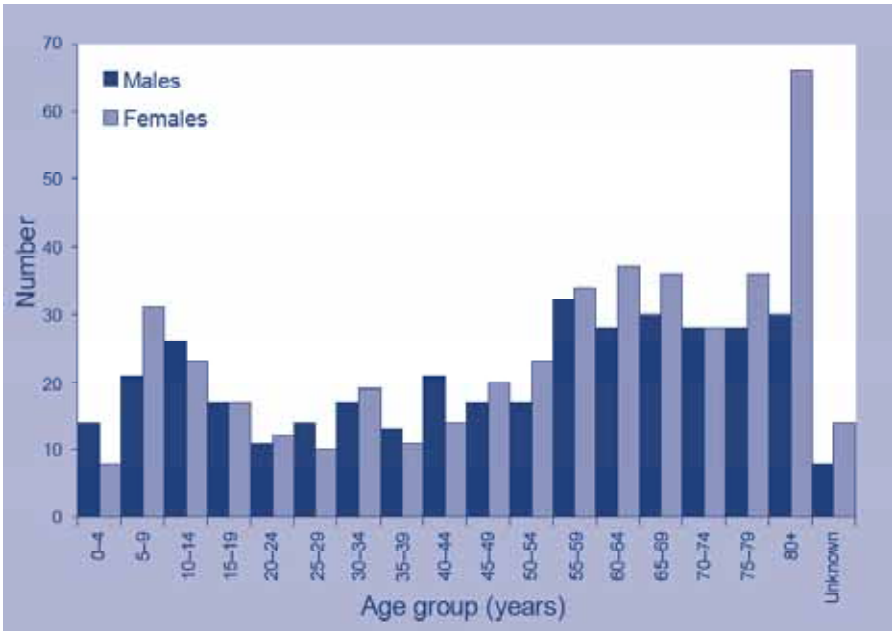


Figure 38. Herpes zoster notifications, South Australia, January 2002 to December 2003 (based on date of onset), by age group and sex



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.[167] In that study, 16 per cent had ophthalmic zoster, which is a serious complication because it threatens vision.[167]

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.[168] 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.[169] 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.[170] 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.[171,172] 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.[170] 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.

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4. Vaccination coverage

Australian Standard Vaccination Schedule 1998 to 2003

The Australian Standard Vaccination Schedule (ASVS) for children aged 0–6 years changed in the second half of 1998 with the second dose of measles-mumps-rubella (MMR) vaccine (previously given at 12–13 years) moved to four years. More changes were made in May 2000 with the introduction of a new ASVS with two distinct paths for children born on or after 1 May 2000.[49] For the immunisations at 2, 4, 6 and 12 months, two options for the use of combination vaccines are recommended. The full schedule and changes to it are outlined in Table 22. Pathway 1 uses hepatitis B (Hep B) vaccine in combination with diphtheria-tetanus-acellular pertussis (DTPa) vaccine, while Pathway 2 uses it in combination with *Haemophilus influenzae* type b (Hib) vaccine. From May 2000, full vaccination at 12 months of age (first milestone) requires three doses of DTPa and oral poliomyelitis (OPV) vaccines, and immunisation against Hib and hepatitis B. Full Hib immunisation at 12 months now requires two doses of PRP-OMP (*Haemophilus influenzae* type b polysaccharide conjugated to the outer membrane protein of *Neisseria meningitidis*). Full hepatitis B immunisation at 12 months requires either three doses of combined DTPa-hepatitis B (Pathway 1) or two doses of combined Hib-hepatitis B vaccine (Pathway 2). The neonatal dose (scheduled for all newborns since May 2000) is not yet accounted for in ACIR coverage estimates.

In the second year of life, a dose of MMR vaccine is scheduled at 12 months of age as well as booster doses of DTPa (at 18 months) and Hib vaccine (at 12 months)—for Pathway 2 this Hib vaccine is given with hepatitis B vaccine. However, the DTPa booster dose at 18 months of age was dropped from the schedule in September 2003. In the sixth year of life, a second dose of MMR vaccine is scheduled as well as booster doses of DTPa and OPV. In September 2003, the schedule was changed to include universal 7-valent conjugate pneumococcal vaccine at 2, 4 and 6 months of age, varicella-zoster vaccine at 18 months, inactivated poliomyelitis vaccine in place of oral polio vaccine and meningococcal C conjugate vaccine at 12 months of age.

Table 22. Australian Standard Vaccination Schedule 1998 to 2003 for children (see footnotes for year of introduction on to schedule)

Age	Vaccine							
Birth	Hep B *							
2 months	Hep B † ‡	DTP † §	Hib ‡ ¶	OPV/IPV **			7vPCV §§	
4 months	Hep B † ‡	DTP † §	Hib ‡ ¶	OPV/IPV			7vPCV	
6 months	Hep B †	DTP † §		OPV/IPV			7vPCV	
12 months	Hep B ‡		Hib ‡ ¶		MMR ††			MenCCV ¶¶
18 months		DTP ‖				VZV ‡‡	23vPPV ‖‖	
4 years		DTP ‖		OPV/IPV	MMR			

* Monovalent hepatitis B vaccine from May 2000.

† Acellular diphtheria-tetanus-pertussis/hepatitis B vaccine from May 2000 (Pathway 1).

‡ Hib PRP-OMP/hep B from May 2000 (Pathway 2).

§ Acellular diphtheria-tetanus-pertussis vaccine from 1999.

‖ Acellular pertussis vaccines were generally used at 18 months and 4 years from 1998. The DTP booster dose at 18 months of age was dropped from the schedule in September 2003.

¶ Hib PRP-OMP (Pathway 1) from May 2000.

** Oral poliomyelitis vaccine, inactivated poliomyelitis vaccine (in combination) from September 2003.

†† Measles-mumps-rubella vaccine.

‡‡ Varicella-zoster vaccine from September 2003.

§§ 7-valent pneumococcal vaccine universal from September 2003 (national program for high-risk children 2001).

‖‖ 23-valent pneumococcal polysaccharide vaccine for Aboriginal and Torres Strait Islander children in high prevalence jurisdictions only, from September 2003.

¶¶ Meningococcal C conjugate vaccine national program announced and added to schedule 2003.

Vaccination coverage estimates from the ACIR 1996 to 2003

The methodology for calculating cohort-based vaccination coverage from the Australian Childhood Immunisation Register (ACIR) was published with the first coverage estimates in 1998.[4] Using this method, a cohort of children is defined by date of birth in three-month groups, the first cohort being born between 1 January 1996 and 31 March 1996.[11] The vaccination status of each cohort is assessed at the three key milestones of 12 months, 24 months and six years of age. Coverage is measured several months after the due date for completion of each milestone, to allow for delayed notification to the ACIR. To minimise duplicate records, the cohort includes only children enrolled with Medicare.[4] It is assumed that notification of receipt of a later vaccine dose implies receipt of earlier doses, even if no earlier vaccination is recorded (third dose assumption).[11]

A child is now defined as 'fully vaccinated' at 12 months of age if he or she has received a third dose of DTPa and poliomyelitis vaccine (oral or inactivated), a second or third dose of Hib vaccine (PRP-OMP), and either a second or third dose of Hep B vaccine, depending on the pathway taken on the new schedule. ACIR coverage estimates (using the third dose assumption) for the first vaccination milestone (the first three scheduled doses of DTPa, OPV, Hib and, recently, two or three doses of Hep B and only two or three doses of Hib) have been reported in *Communicable Diseases Intelligence* since 1998.[173] The coverage for MMR has been reported in *Communicable Diseases Intelligence* since 1998.[173] Coverage for the third vaccination milestone at six years of age has been reported in *Communicable Diseases Intelligence* since 2002.[174]

Trends in vaccination coverage estimates from the ACIR

Vaccines scheduled in the first year of life

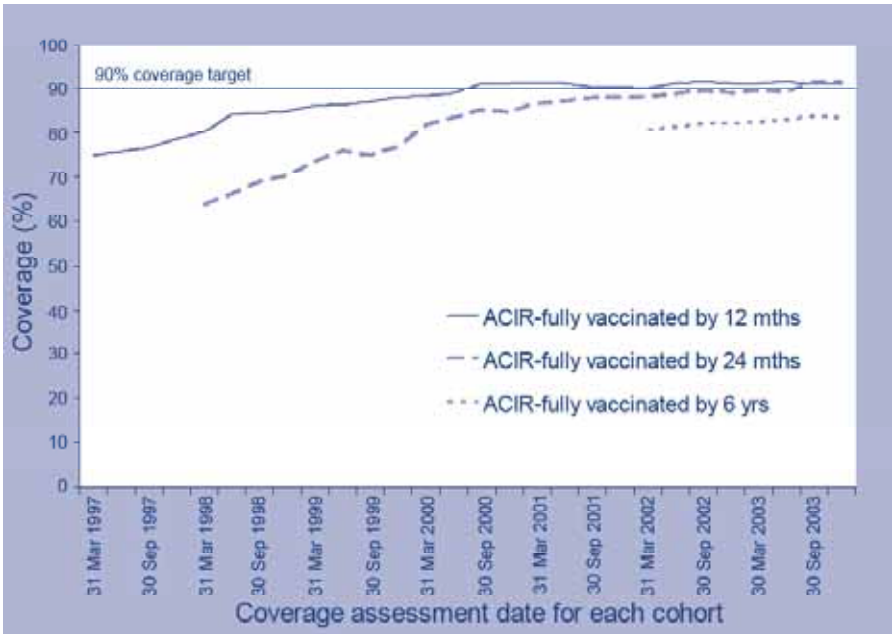
The trends in childhood vaccination coverage in Australia for three doses of DTPa and OPV, and two or three doses of Hib and Hep B assessed at one year, and for three or four doses of DTPa, two or three doses of Hib and Hep B, three doses of OPV, and one dose of MMR assessed at two years of age, and for two doses of MMR, five doses of DTPa, and four doses of OPV assessed at 6 years of age are shown in Figure 39. Coverage was calculated for 28 consecutive three-month cohorts born from 1 January 1996 to 31 December 2002. For all vaccines due by one year of age, coverage estimates increased steadily from 75 per cent for the first cohort, to 9 per cent by the 28th cohort, assessed on 31 December 2003. For all vaccines due by two years of age, coverage estimates also increased steadily from 64 per cent for the first cohort to 91.5 per cent by December 2003. Coverage estimates for all vaccines due by six years of age were first reported in *Communicable Diseases Intelligence* in 2002, and have also increased steadily from 80.6 per cent in early 2002 to 83.5 per cent in late 2003.

Coverage estimates for the 12-month age group have, however, remained steady over the past two years, fluctuating around the 90 per cent level. With up to 3 per cent of Australian parents not immunising their children because they object to, disagree with, or are concerned about immunisation,[175] it will be difficult for coverage estimates to exceed 95 per cent, especially as the reporting of immunisation encounters is still not totally complete.

Differences between estimates of the proportion of children classified as fully vaccinated by State/Territory are shown in Figure 40. Fully vaccinated coverage remained reasonably stable over the three-year assessment period for all jurisdictions with almost all of them reaching the Immunise Australia Program target of 90 per cent coverage for the first milestone vaccines. Coverage in the Northern Territory and the Australian Capital Territory fluctuated noticeably over the whole period. Significant changes in coverage in jurisdictions like the Northern Territory and the Australian Capital Territory, which have relatively small populations, are likely to be the result of small numbers of unimmunised children having large impacts on the coverage percentages.

The trends in childhood vaccination coverage in Australia for individual vaccines (DTPa, OPV, Hib and Hep B assessed at one year) are shown in Figure 41, calculated for 12 consecutive three-month cohorts born from 1 January 2000 to 31 December 2002. Coverage estimates for all vaccines remained stable throughout the 2001 to 2003 period, hovering around the 9 per cent to 95 per cent mark. Coverage for the Hib and Hep B vaccines is greater than for DTPa and OPV due to the change in the immunisation schedule in mid-2000, and the subsequent change in the algorithm used to calculate coverage at 12 months of age, where a record of two or three doses of Hib and Hep B on the ACIR is enough for a child to be considered fully immunised.

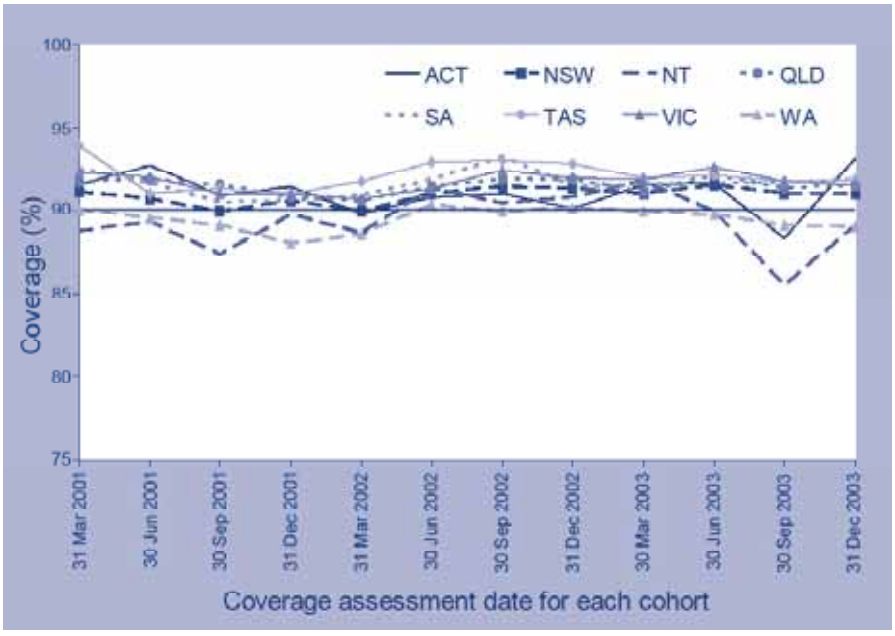
Figure 39. Trends in vaccination coverage estimates from the Australian Childhood Immunisation Register for 1, 2 and 6 year olds*



Source: Australian Childhood Immunisation Register.

* By 3-month birth cohorts born between 1 January 1996 and 31 December 2002. Coverage assessment date was 12 months, 24 months or 6 years after the last birth date of each cohort.

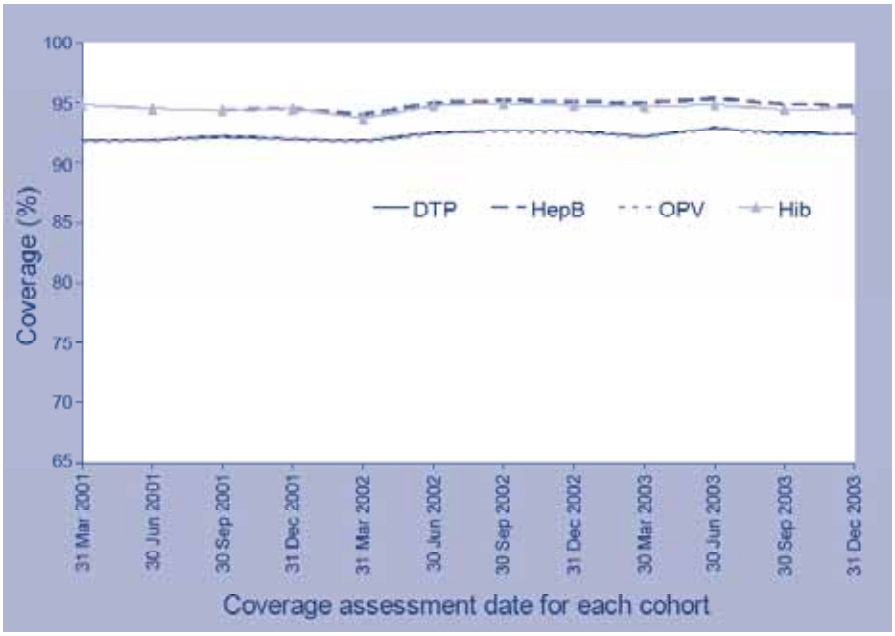
Figure 40. Trends in vaccination coverage estimates by jurisdiction: children 'fully vaccinated' at the age of 1 year*



Source: Australian Childhood Immunisation Register.

* By 3-month birth cohorts born between 1 January 2000 and 31 December 2002. Coverage assessment date was 12 months after the last birth date of each cohort.

Figure 41. Trends in vaccination coverage estimates for individual vaccines: children vaccinated for 3 doses of DTP, OPV, Hib and Hep B at the age of 1 year*

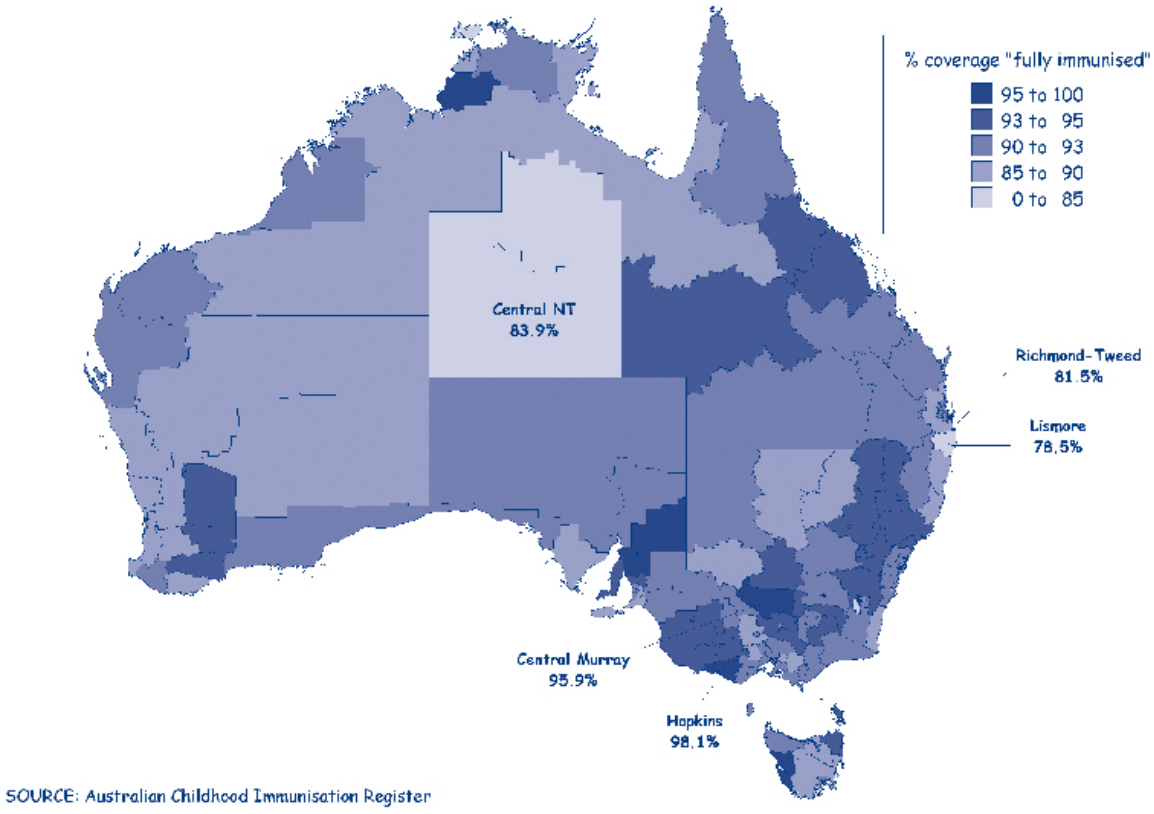


Source: Australian Childhood Immunisation Register.

* By 3-month birth cohorts born between 1 January 2000 and 31 December 2002. Coverage assessment date was 12 months after the last birth date of each cohort.

Figure 42 presents a map of immunisation coverage at 12 months of age in Australia by Australian Bureau of Statistics (ABS) Statistical Subdivision. The map demonstrates that, whilst coverage is greater than 90 per cent in almost all jurisdictions, there exist a significant number of areas within jurisdictions that have low levels of coverage, below 90 per cent, and even below 85 per cent in a few areas such as the Northern Rivers area of New South Wales.

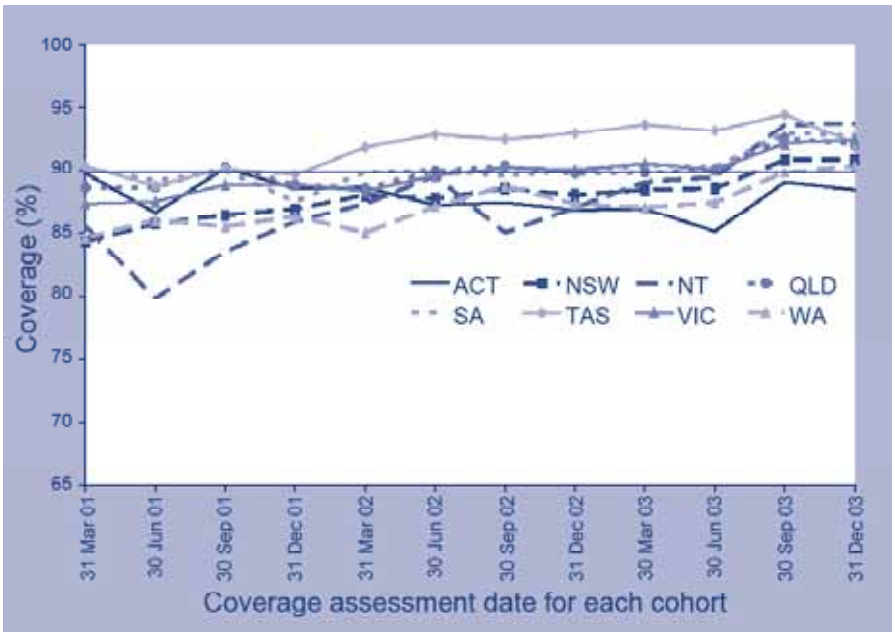
Figure 42. Immunisation coverage for 'fully immunised' at 12 months of age, Australia, December 2003



Vaccines scheduled in the second year of life

Differences between estimates of the proportion of children classified as fully vaccinated at two years of age by State/Territory are shown in Figure 43. Fully vaccinated coverage at two years of age for consecutive cohorts increased steadily over the three-year assessment period for all jurisdictions without significant variation between the jurisdictions. In early 2001, only one jurisdiction had reached the 90 per cent coverage target—Tasmania with 90.3 per cent coverage. But by the end of 2003, coverage at two years of age was greater than 90 per cent in all jurisdictions except for the Australian Capital Territory (88.4%), and almost 94 per cent in the Northern Territory.

Figure 43. Trends in vaccination coverage estimates, by jurisdiction: children 'fully vaccinated' for 4 doses of DTPa and Hib, 3 doses of OPV and 1 dose of MMR at the age of 2 years*

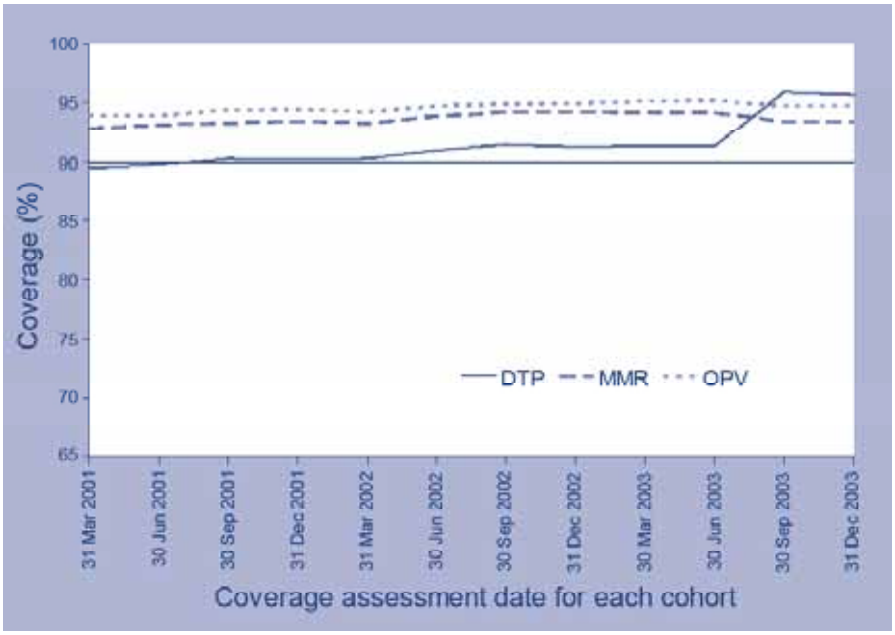


Source: Australian Childhood Immunisation Register.

* By 3-month birth cohorts born between 1 January 1999 and 31 December 2001. Coverage assessment date was 24 months after the last birth date of each cohort.

The trends in childhood vaccination coverage in Australia for individual vaccines (DTPa, OPV and MMR assessed at two years) are shown in Figure 44, calculated for 12 consecutive three-month cohorts born from 1 January 1999 to 31 December 2001. Coverage for MMR and OPV was higher than coverage for DTPa and remained steady across the whole period, hovering around 93 per cent with very little change. However, coverage for DTPa at two years of age increased significantly in mid-2003 due to the removal of the fourth dose of DTPa (due at 18 months) from the immunisation schedule from the September 2003 quarter onwards. The coverage assessment for the 24-month cohort now excludes the requirement for the 18-month dose of DTPa. Coverage for this cohort now looks for a third or a fourth dose of diphtheria, tetanus and pertussis vaccine. Prior to the change, the 24-month cohort assessment looked for the fourth dose only.

Figure 44. Trends in vaccination coverage estimates for individual vaccines: children vaccinated for 4 doses of DTP, 3 doses of OPV and 1 dose of MMR at the age of 2 years*

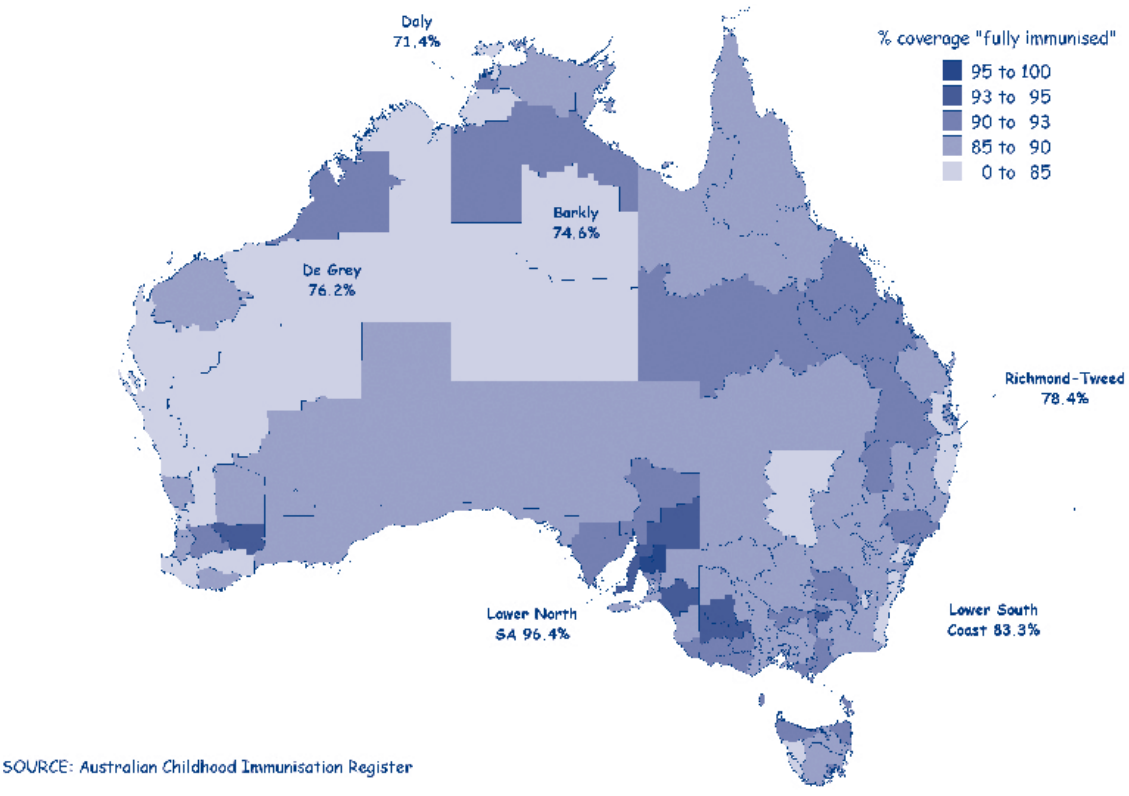


Source: Australian Childhood Immunisation Register.

* By 3-month birth cohorts born between 1 January 1999 and 31 December 2001. Coverage assessment date was 24 months after the last birth date of each cohort.

Figure 45 presents a map of immunisation coverage at 24 months of age in Australia by Australian Bureau of Statistics (ABS) Statistical Subdivision. The map demonstrates (as did Figure 42) that, whilst coverage approaches 90 per cent in most jurisdictions, there exist a large number of smaller areas within jurisdictions that have low levels of coverage, some as low as 70 per cent to 75 per cent, such as regional areas in Western Australia and the Northern Territory.

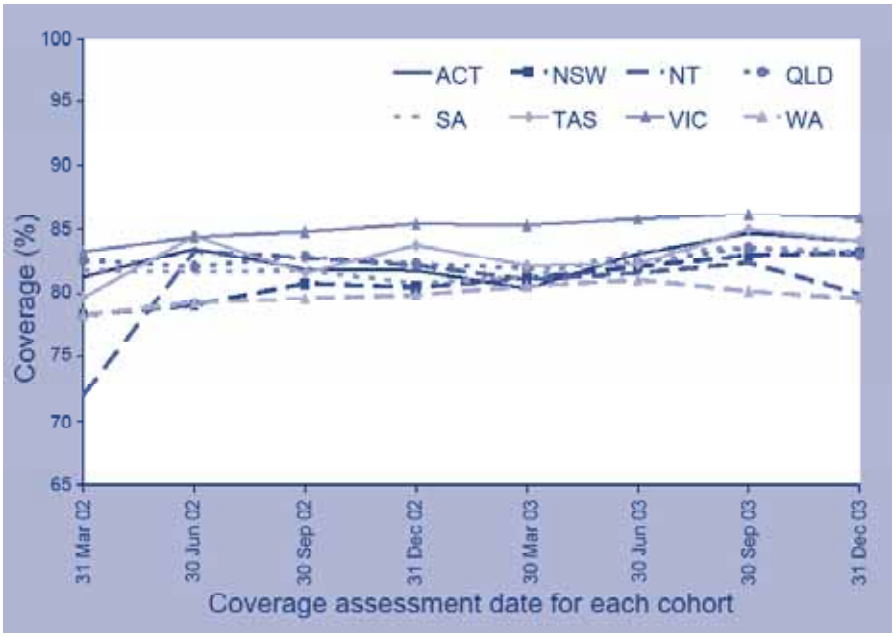
Figure 45. Immunisation coverage for 'fully immunised' at 24 months of age, Australia, December 2003



Vaccines given at 4–5 years of age

Coverage estimates for 4–5 year olds were not reported by the ACIR until early 2002, so there are only two years of coverage data for this age group. Differences between estimates of the proportion of children classified as fully vaccinated by six years of age by State/Territory are shown in Figure 46. Fully vaccinated coverage increased only slightly over the two year assessment period for all jurisdictions, with some jurisdictions experiencing a greater increase than others. However, coverage at six years of age remained below 85 per cent for all jurisdictions except Victoria across the whole time period.

Figure 46. Trends in vaccination coverage estimates, by jurisdiction: children 'fully vaccinated' for 5 doses of DTP, 4 doses of OPV and 2 doses of MMR at the age of 6 years*



Source: Australian Childhood Immunisation Register.

* By 3-month birth cohorts born between 1 January 1996 and 31 December 1997. Coverage assessment date was 72 months after the last birth date of each cohort.

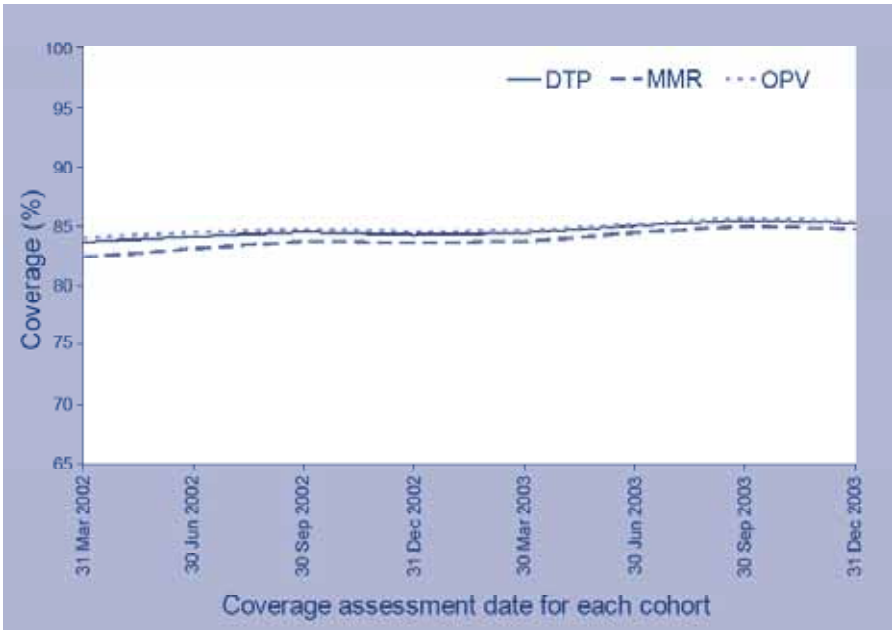
The trends in childhood vaccination coverage in Australia for individual vaccines (DTPa, OPV, and MMR assessed at six years) are shown in Figure 47, calculated for eight consecutive three-month cohorts born from 1 January 1996 to 31 December 1997. Coverage for DTPa and OPV was marginally higher than coverage for MMR, and coverage for all three vaccines remained steady across the whole period hovering around 82 per cent to 85 per cent with very little change.

Comment

Estimates of vaccination coverage in Australia for all jurisdictions have increased steadily since the ACIR commenced in 1996 but in recent years have reached a plateau. There have been increases in coverage for one year olds, two year olds and six year olds, with fully immunised coverage for one and two year olds reaching the Immunise Australia Program target of 90 per cent coverage for the first and second milestone vaccines.

The ACIR is now likely to be close to its maximal achievable performance following the impact of the General Practitioner Immunisation Incentive Scheme, parental incentives and data cleaning initiatives. Mechanisms aimed at further improving notification of immunisations to the ACIR by general practitioners and other providers need to be considered. The functionality of the Medicare database would be enhanced if greater currency of address details could be achieved.

Figure 47. Trends in vaccination coverage estimates for individual vaccines: children vaccinated for 5 doses of DTP, 4 doses of OPV and 2 doses of MMR at the age of 6 years*



Source: Australian Childhood Immunisation Register.

* By 3-month birth cohorts born between 1 January 1996 and 31 December 1997. Coverage assessment date was 72 months after the last birth date of each cohort.

Limitations of the ACIR database, related to reliance on provider notification and the currency of Medicare registration, mean that official estimates of coverage are unlikely to rise significantly above current levels, unless mechanisms are put in place to further improve notification to the ACIR. Increases in actual coverage will also be difficult to achieve from this point, as there are probably 2 per cent to 3 per cent of parents who are opposed to immunisation.

To maintain the current high levels and to achieve further increases in coverage, efforts need to be directed at improving reporting by providers (and subsequent data cleaning), and at immunisation of the small group of children now not up to date with their immunisations. The latter will require carefully targeted initiatives, which may include efforts to further improve access to services for disadvantaged groups as well as specific educational initiatives for those parents and providers concerned about contraindications to immunisation.

In addition, there are several national, publicly funded, targeted immunisation programs for which systematically collected data on vaccine coverage are not currently available. These include hepatitis B vaccine for adolescents, dTpa vaccine for adolescents, MMR vaccine for 18–30 year olds, influenza and pneumococcal vaccines for Aboriginal and Torres Strait Islander persons over 50 years of age, and influenza vaccine for persons over the age of 64 years. While data are available from surveys in local subpopulations[60] or national special purpose surveys[176] for three of these programs, lack of widely applicable data inhibits planning and evaluation at the regional and national levels. As the number and scope of immunisation programs increases, extension of the ACIR to collect data for some or all of the other age groups targeted by vaccines merits active consideration.

5. Discussion

Changes in vaccination practice

The years 2001 to 2002 have been a period of consolidation in immunisation practice and coverage in Australia, following the implementation of the new vaccination schedule in mid-2000. There has been continuing evidence of the effectiveness of immunisation strategies undertaken in the late 1990s, such as the Measles Control Campaign in 1998 and the introduction of a fourth dose of pertussis-containing vaccine in 1995, as well as improvements in vaccination coverage. In December 2000 the Australian Childhood Immunisation Register (ACIR) documented the fact that the 90 per cent coverage target for immunisation of 12 month olds had been achieved for the first time. This has been maintained and exceeded following the introduction of the new schedule through to the end of 2003.

These vaccine policy and program changes represent a large investment in public health, which is set to further increase in coming years, with increasingly expensive new vaccines and the increasing cost of all the supporting pillars of immunisation in Australia. Like other industrialised countries, Australia faces the dual challenges of maintaining both high immunisation coverage and public confidence in immunisation, while implementing increasingly complex decisions about the introduction of new vaccines for both children and adults. Although the full evaluation of the impact of current programs, and prioritisation and planning for future programs, require more detailed and precise data, the multiple data sources (notifications, hospitalisations and mortality) contained in this report provide an ongoing picture of progress across the spectrum of Australian immunisation activity.

Current and comparative morbidity from vaccine preventable diseases

A summary of the relative morbidity and mortality due to the diseases covered in the four years prior to the current report (1996 to 2000) is shown in Table 23 and for the two years 2001 to 2002 in Table 24. While the limitations of notification, hospitalisation and death data should be borne in mind (see Chapter 2), and may be especially evident for rare diseases or diseases which lack a specific diagnostic test, together these data provide an informative overview of trends in the burden of vaccine preventable diseases in Australia over the past several years.

In children under five years of age (the main target of the current childhood program), ongoing reductions in relative disease burden have continued in 2001 and 2002. Among diseases currently targeted by immunisation, hospitalisations due to measles, rubella, mumps and Hib disease have all decreased substantially. Hospitalisations due to pertussis continue to be a significant burden in young infants. Influenza, pertussis, varicella, pneumococcal disease and meningococcal disease accounted for the largest numbers of hospitalisations in those under the age of five years. Outside this age group, the three most common causes of hospitalisation also included influenza and varicella, but zoster was the third most common recorded cause. By contrast with the hospitalisation data, the most prominent causes of death in all age groups were influenza and meningococcal disease. The implications of these data are discussed below, first with respect to vaccines included in the Australian Standard Vaccination Schedule (ASVS) during the review period and second with respect to vaccines available in Australia but not included in the ASVS up to the end of 2002.

Table 23. Average annual morbidity and mortality from vaccine preventable diseases in Australia for 4 years 1996/1997–1999/2000*

Disease†	Notifications 1997–2000 (average n)		Notification rate/100,000 (average rate)		Hospitalisations 1996/97–1999/00 (average n)		Hospitalisation rate/100,000 (average rate)		Deaths‡ 1997–2000 (average n)		Death rate /100,000 (average rate)	
	0–4 yr	All ages	0–4 yr	All ages	0–4 yr	All ages	0–4 yr	All ages	0–4 yr	All ages	0–4 yr	All ages
Diphtheria	0	0	–	–	0.3	0.8	0.02	0.00	0	0	–	–
Hib§	21.8	26.5	1.69	0.68	39.5	77.5	3.06	1.97	0.5	1.3	0.04	0.03
Hepatitis A	118.5	1977.0	9.21	10.50	29.0	824.0	2.25	4.43	0.5	3.0	0.04	0.02
Hepatitis B‡‖	1.8	312.3	0.14	1.66	1.5	181.8	0.12	0.98	0	22.5‖	–	0.12‖
Influenza	NN	NN	NN	NN	880.3	4766.5	68.14	25.60	3.3	117.3	0.25	0.62
Measles	166.8	367.8	12.95	1.95	43.0	95.5	3.33	0.51	0	0	–	–
Meningococcal disease	202.0	546.8	15.69	2.90	283.8	740.5	21.97	3.98	9.5	31.3	0.74	0.17
Mumps¶	31.3	192.8	2.43	1.02	8.3	53.3	0.64	0.29	0	0.5	–	<0.01
Pertussis	694.5	6748.5	53.95	35.84	486.5	708.3	37.66	3.80	1.3	1.5	0.10	0.01
Pneumococcal (invasive)**	NN	NN	NN	NN	252.0	754.0	19.50	4.05	4.8	14.8	0.37	0.08
Poliomyelitis‡	0	0	–	–	0	1.5	–	0.01	0	0	–	–
Rubella	130.5	709.8	10.14	3.77	19.5	43.0	1.51	0.23	0	0	–	–
Tetanus	0.3	5.8	0.02	0.03	0.3	34.5	0.02	0.19	0	1.3	–	0.01
Varicella	NN	NN	NN	NN	726.5	1701.8	56.24	9.14	1.3	8.3	0.10	0.04
Zoster††	NN	NN	NN	NN	28.8	4469.0	2.23	24.01	0.3††	19.3††	0.03††	0.10††

NN = Not Notifiable.

* Notification data, National Notifiable Diseases Surveillance System, January 1997–December 2000; hospitalisation data, Australian Institute of Health and Welfare (AIHW) National Morbidity database, July 1996–June 2000; death data, AIHW National Mortality database, January 1997–December 2000.

† See Chapter 3 for case definitions.

‡ Principal diagnosis only for hospitalisations, and for deaths only cases with disease classified as underlying cause of death.

§ Data for *Haemophilus influenzae* disease include only cases aged 0–14 years of age. For hospitalisations and deaths only includes meningitis or epiglottis cases.

‖ Includes deaths from acute and chronic hepatitis B infection up to 1998, acute deaths only from 1999, due to change from ICD9 to ICD10 codes; includes only acute hepatitis B notifications and hospitalisations.

¶ In Queensland mumps not notifiable in 2000 or for complete year in 1999.

** Includes pneumococcal meningitis and septicaemia only.

†† Includes zoster deaths for 1998–2000 only.

Table 24. Average annual morbidity and mortality from vaccine preventable diseases in Australia for 2 years 2000/2001–2001/2002*

Disease†	Notifications 2001–2002 (average n)		Notification rate/100,000 (average rate)		Hospitalisations (average n)		Hospitalisation rate/100,000 (average rate)		Deaths‡ 2001–2002 (average n)		Death rate/100,000 (average rate)	
	0–4 yr	All ages	0–4 yr	All ages	0–4 yr	All ages	0–4 yr	All ages	0–4 yr	All ages	0–4 yr	All ages
Diphtheria	0	0.5	–	0.00	0	0.5	–	0.00	0	0	–	–
Hib‖	12.0	16.5	0.94	0.41	30.0	45.5	2.34	1.14	0.5	1.5	0.04	0.04
Hepatitis A	28.0	454.5	2.19	2.33	9.0	335.5	0.70	1.74	0.5	1.0	0.04	0.01
Hepatitis B‡§	1.0	418.5	0.08	2.14	0	152.5	–	0.79	0	10.0	–	0.05
Influenza††	962.5	2479.5	119.13	18.72	615.5	2905.0	48.06	15.06	1.5	43.5	0.12	0.22
Measles	18.0	85.5	1.41	0.44	13.0	52.5	1.02	0.27	0	0	–	–
Meningococcal disease	196.5	677.5	15.39	3.47	261.0	871.5	20.38	4.52	13.0	44.0	1.02	0.23
Mumps¶	10.5	91.5	0.82	0.47	8.0	42.5	0.62	0.22	0	0.5	–	<0.01
Pertussis	657.0	7358.5	51.46	37.68	443.5	638.5	34.63	3.31	2.5	3.0	0.20	0.02
Pneumococcal (invasive) **††	676.0	1975.5	59.01	11.68	344.0	1055.5	26.86	5.47	2.5	14.5	0.20	0.07
Polio‡	0	0	–	–	0	1.5	–	0.01	0	0	–	–
Rubella	15.5	255.5	1.21	1.31	6.5	27.0	0.51	0.14	0	0	–	–
Tetanus	0	2.5	–	0.01	0.5	27.0	0.04	0.14	0	0.5	–	<0.01
Varicella	NN	NN	NN	NN	691.5	1659.0	54.00	8.60	0.5	9.5	0.04	0.05
Zoster	NN	NN	NN	NN	34.5	4580.5	2.69	23.75	0	15.0	–	0.08

NN = Not Notifiable

* Notification data, National Notifiable Diseases Surveillance System, January 2001–December 2002; Hospitalisation data, Australian Institute of Health and Welfare (AIHW), July 2000–June 2002; death data, AIHW National Morbidity database, January 2001–December 2002.

† See Chapter 3 for case definitions.

‡ Principal diagnosis only for hospitalisations and for deaths only cases with disease classified as underlying cause of death.

§ Includes only acute hepatitis B notifications and hospitalisations, and deaths from acute hepatitis B.

‖ Data for *Haemophilus influenzae* disease include only cases aged 0–14 years of age. For hospitalisations and deaths only includes meningitis or epiglottis cases.

¶ Mumps not notifiable in Queensland for first half of 2001.

** Includes pneumococcal meningitis and septicaemia only.

†† Notifications only complete for 2002—notification rate for 2002 only.

Diseases on the Australian Standard Vaccination Schedule in 2001–2002

Measles

During 2001–2002 measles notifications and hospitalisations continued their decline to record lows, reflecting the success of the Measles Control Campaign and ongoing high childhood vaccine coverage with measles-mumps-rubella (MMR) vaccine. Continued high childhood vaccine coverage for two doses of MMR vaccine will be required to maintain this status. Evaluation of the young adult MMR vaccination campaign of 2001 will help decide whether further initiatives targeting this relatively susceptible group are required. In the face of declining public familiarity with measles, due to its successful control through immunisation, Australia needs to maintain good communication strategies to emphasise first the benefits of measles control and second the lack of evidence to support concerns about adverse effects. The most prominent of the latter is the purported link between MMR vaccine and neurodevelopmental disorders which, despite now well-defined evidence to counter it,[177] continues to cause suboptimal uptake of MMR vaccine in England.[178]

Rubella and mumps

Like measles, rubella and mumps notifications and hospitalisations have continued to decline to record lows in 2001–2002, reflecting improvements in coverage with MMR vaccine in Australia. Laboratory confirmation of cases is increasingly important as these diseases become rare. Vigilance for cases in young adults, and particularly in ensuring rubella immunity in women of childbearing age (especially those not born in Australia), is required. Although mass adult vaccination campaigns have been successfully implemented in some countries, any additional initiative in Australia will be difficult to implement and will require innovative approaches.[146]

Hib disease

The virtual disappearance of invasive Hib disease among children less than five years old has been the greatest success story for vaccination in the past decade. In the two years covered by this review period, there has been a further 20–25 per cent reduction in the average annual notification and hospitalisation rates of Hib disease in 0–4 year olds, with invasive Hib disease and deaths becoming a rarity. Laboratory confirmation is increasingly important, now that Hib disease is rare, as the relative incidence of non-type b invasive *Haemophilus influenzae* becomes greater. The recent resurgence of Hib disease in both children and adults in the United Kingdom exemplifies this, and most likely reflects their different childhood schedule, with no booster dose after six months of age and progressive decrease of herd immunity.[179,180]

Pertussis

Of the diseases with well-established vaccination programs, pertussis again stands out, as in the previous review period, as causing the greatest morbidity in 2001–2002, when epidemics occurred in several regions of Australia. Most infection occurs in those too young to be immunised[120] and in adolescents and adults whose vaccine or infection induced immunity has waned over time. There is good epidemiological evidence of the benefit of the addition of the preschool booster dose in late 1994, with a subsequent decline in national notifications in older children.[121] The recent availability of pertussis vaccines suitable for adolescents and adults promises to improve our ability to control pertussis. Immunisation should reduce the spread of pertussis by adolescents (who currently have the highest rates of notification) and provide protection against pertussis to adults who have contact with infants too young to be immunised. In September 2003, a booster dose of dTpa vaccine was substituted for DT vaccine in the ASVS at age 15–17 years and the 18-month booster was discontinued. Current recommendations also encourage dTpa vaccination for prospective and recent parents and for adults working with young children.[49] These recommendations will require careful implementation strategies in order to achieve reasonable uptake: in the interim, research into neonatal pertussis vaccination strategies will continue.

Influenza

Inactivated influenza vaccines have been provided free of charge annually to all people 65 years of age and over since 1999, except in Victoria where funding was made available in 1998. This makes influenza vaccine a large, recurrent and therefore costly part of the overall immunisation program. The data presented in this report, although minimal estimates of influenza cases, indicate that the disease burden from influenza is also large, with the highest number of hospitalisations and bed days, both for children under five years of age and

for older age groups. Influenza notifications commenced nationally in 2001 for laboratory-confirmed cases and, although a gross underestimate of disease burden, will provide useful information about relative size of influenza seasons, circulating influenza strains and changes in age distribution consequent upon vaccination. However, caution in interpretation of these data is required due to differential rates of testing among jurisdictions and age groups. Influenza was the underlying cause of death more frequently than any other disease under review, especially in the elderly, with 84 per cent of deaths attributed to influenza in people over 60 years of age. The high disease burden from influenza among young children (Tables 23 and 24) is similar to that described in the United States of America,[67] where universal childhood influenza vaccination has recently been recommended for infants aged 6–23 months.

Hepatitis B

Although vaccines against hepatitis B first became available in 1982, and have been used consistently in high-risk groups since then, they were not included in the ASVS until 2000, with the exception of the Northern Territory which has had routine vaccination since 1990. The long incubation of hepatitis B infection means that the impact of infant immunisation takes many years to become evident. The national notification rate of acute hepatitis B infection peaked in 2001. During the review period notification rates were highest in those aged 15–24 years and hospitalisation rates were highest in those aged 25–29 years. A national serosurvey of samples from 1996–1999 of 1–59 year olds found that only 29 per cent of subjects had evidence of vaccine-induced immunity to hepatitis B.[56] In addition to childhood vaccination, there is an ongoing need to promote hepatitis B immunisation for groups at high risk of acquisition of infection, including prisoners, men who have sex with men and injecting drug users.

Rare vaccine preventable diseases (tetanus, diphtheria and poliomyelitis)

Cases of tetanus continue to occur, despite tetanus toxoid being available for more than 60 years. However, tetanus has become a disease of older adults, reinforcing the need for vigilance in assessing tetanus immunity when older adults present for treatment of tetanus-prone injuries and for ensuring implementation of the ADT booster dose recommended at 50 years of age. In 2001 a cutaneous case of diphtheria was notified, the first case since 1992, but acquired overseas. There is an ongoing risk of the importation of diphtheria into Australia from regions where diphtheria is not well controlled, reinforcing the need for ensuring adequate immunisation across all age groups, especially amongst travellers. A travel-related acquisition of diphtheria was recently reported in an unimmunised child in New Zealand.[181] Australia and the Western Pacific region have been declared polio free,[141] but high vaccination coverage and continued active surveillance for acute flaccid paralysis will be required until global certification is achieved.

Vaccine preventable diseases not on the Australian Standard Vaccination Schedule in 2001–2002

Varicella zoster

Currently, only South Australia has made varicella zoster a notifiable disease. Zoster hospitalisation data are presented for the first time in this report and demonstrate that, as measured by hospitalisation codes, the burden of zoster is higher than varicella in Australia. For varicella, the very young were most commonly hospitalised while the elderly had the longest length of stay. Should the 2003 recommendation for the use of varicella vaccine in Australian infants at the age of 18 months be fully funded and achieve coverage comparable to other vaccines on the ACIR, significant improvements to routinely available surveillance of these two conditions will be required.

Pneumococcal disease

Notification of invasive pneumococcal disease (IPD) was instituted nationally in 2001, and implemented in full in 2002, together with the introduction of conjugate pneumococcal vaccine for high-risk children. Two national reports on IPD have been published, for 2001[182] and 2002,[183] and these notification data illustrate the underestimation of IPD from hospitalisation data, with the exception of pneumococcal meningitis. Thus, for bacteraemic disease, these ongoing national reports, with serotype data, will be crucial in evaluating the impact of pneumococcal vaccine programs in children and in Indigenous and elderly adults, as both the conjugate and polysaccharide vaccines cover only a proportion of the serotypes associated with pneumococcal disease. The success of the conjugate pneumococcal program for children under the age of two years in

the United States of America, with significant reductions in diseases among older age groups due to herd immunity, have given an important lead for the impact to be expected in Australia when universal conjugate pneumococcal vaccination commences in January, 2005.[184]

Meningococcal disease

Meningococcal disease ranked behind zoster, invasive pneumococcal disease, influenza and varicella in terms of total hospital bed days in 2001–2002. It accounted for the highest number of childhood and adult deaths (although the deaths attributable to influenza in adults are grossly underestimated from death certificate coding.) However, only a small proportion of meningococcal disease (that due to serogroups A, C, W135 and Y) is vaccine-preventable, with a range from 10 per cent to 40 per cent depending on age group and region.[106–108] A protein conjugate vaccine protective against serogroup C meningococcal disease has been available in Australia since the beginning of 2002. It was funded for all children aged 1–18 years in 2002, with a dose at 12 months of age becoming part of the National Immunisation Program from 2003. These vaccines have been used in a universal program for children two months to 18 years of age in the United Kingdom, with dramatic reductions in cases and deaths due to serogroup C, compared with age groups not targeted for vaccination.[105] Australia has an incidence of meningococcal disease (overall and due to type C meningococci) which is intermediate between the high rates seen in the United Kingdom[105] and the rates of 1 per 100,000 population or less generally seen in North America.[185] Vaccines effective against serogroup B meningococci are under development but must be tailored to specific subtypes. The subtype of serogroup B meningococcal disease currently causing a prolonged epidemic in Auckland, New Zealand,[186] although present in Australia, is a much lower proportion of B serotypes than in New Zealand and has not been responsible for any disease outbreaks to date.

Hepatitis A

There was a decline in hepatitis A rates in 2001–2002 following peaks in total hepatitis cases during the 1990s. The epidemiology of hepatitis A differs significantly for the Indigenous population, where it is endemic and is acquired primarily in early childhood, compared with the non-Indigenous population. An immunisation program targeting Indigenous children aged 18 months to six years living in north Queensland commenced in 1999, following cases of severe disease due to hepatitis A and day-care outbreaks. Data indicate that this program has had a significant impact on reducing hepatitis A in both Indigenous and non-Indigenous people in North Queensland.[52]

Vaccine preventable disease notification rates compared with other industrialised countries

The most recent notification rates for the five most frequently occurring vaccine preventable diseases compared with the rates in New Zealand, the USA, Canada and England, are shown in Table 25. Notifications of invasive Hib disease were low in all countries, reflecting the excellent results of Hib vaccination programs, although starting to show an increase in cases in the United Kingdom which now has a rate higher than Australia. Australia has moved closer to the situation in North America with respect to measles eradication, with notification rates decreasing from 1.7 in 1998 to 0.2 per 100,000 in 2002. Pertussis notification rates in Australia remain higher than in the other countries shown in Table 25. Comparisons with other countries are difficult because of differences in notification case definitions and particularly the ready availability of serology and compulsory laboratory notification in Australia. Nevertheless, it is likely that Australia still has a comparatively high pertussis disease burden, as reflected in hospitalisations.

Future surveillance priorities

For this biennial report, access to and the scope of the data available from the Australian Institute of Health and Welfare (AIHW) National Hospital Morbidity Database and Causes of Death Collection have been enhanced by NCIRS' relationship with the AIHW as a collaborating centre. On the National Notifiable Diseases Surveillance System (NNDSS) database some additional important fields, such as laboratory confirmation and immunisation status, are becoming available through enhanced surveillance initiatives. The Communicable Diseases Network Australia has recently revised case definitions, including those for vaccine preventable diseases, which will provide increasing consistency to notification data. Increasing requirements for the laboratory confirmation of diseases that have become rare due to the success of immunisation (e.g. Hib disease, measles, mumps and rubella) should provide increasing confidence in notification data. The recent additions of varicella, meningococcal C and pneumococcal conjugate vaccines to the ASVS in 2003 make close moni-

Table 25. Most recent* notification rates per 100,000 population for frequently notified vaccine preventable diseases, by country of residence

Disease	Australia	New Zealand[187]	USA[113]	Canada[188]	England/ Wales[189]
Hib disease	0.2	0.2	0.6	0.1	0.5
Measles	0.2	1.8	<0.05	0.7	6.1 (0.6)†
Mumps	0.4	1.3	0.1	0.3	3.8 (0.9)‡
Pertussis	28.3	24.3	3.5	16.1	1.7
Rubella	1.3	0.8	<0.05	0.1	3.2 (0.1)§

* Australia 2002; New Zealand 2004; USA 2002; Canada 2000; England/Wales 2002.

† Incidence corrected for proportion serologically confirmed = 10%.

‡ Incidence corrected for proportion serologically confirmed = 25%.

§ Incidence corrected for proportion serologically confirmed = 4%.

toring of the impact of these vaccination programs critical. Although enhanced laboratory surveillance is in place for meningococcal and pneumococcal disease, additional mechanisms at minimum increased sentinel surveillance sites for both varicella and zoster will be needed should varicella vaccination increase above the present low rate. The United Kingdom experience with Hib disease resurgence highlights the importance of investing in high quality surveillance in the long term.

Future vaccination priorities

Table 24 provides a number of measures of morbidity for comparison of disease burden relevant to current general or targeted programs as well as potential future vaccination programs. For most vaccine preventable diseases, the notification and hospitalisation rates are highest in children under five years of age. Immunisation programs targeting this age group are probably nearing their highest practically achievable targets, as measured by the Australian Childhood Immunisation Register and supported by a range of parent and provider incentives.[14,175,190,191] For other vaccine preventable diseases there is either a greater disease burden in older age groups, such as hepatitis A and B, pertussis (although rates in infants remain high) and tetanus, or important secondary age peaks such as 20–29 years for measles and mumps and in young adults for meningococcal disease.

With respect to immunisation programs targeting diseases currently included in the ASVS, measles and pertussis in young adults and adolescents, respectively, stand out as priorities. Australia has so far not invested significantly in vaccination programs in older adolescents and young adults, other than the relatively passive approach adopted for the promotion of MMR vaccine to 18–30 year olds. However, school-based delivery of conjugate meningococcal C vaccine is now in place nationally, so the potential for ongoing programs has been established. Delivery of vaccines such as MMR to the 18–30 year old age group is difficult to implement and it is likely that this age group will represent an ongoing challenge for measles, mumps and rubella control over the next five years. Approaches to adolescent pertussis must have the twin focus of morbidity in adolescents themselves and projected impact on disease transmission to infants. The 2003 addition to the ASVS of dTpa for adolescents in Australia has the potential to provide an international lead in pertussis control, as long as high coverage can be achieved. Australia, along with other industrialised countries, is now entering an era when the increasing array of new vaccines will have less easily defined benefits and greater costs than programs to date. In the near future it is likely that vaccines against diseases such as human papillomavirus and rotavirus will become available. Careful evaluation of the additional benefits of new programs as well as continued efforts to maintain current programs will be required to sustain the success of immunisation in Australia over the first decade of the 21st Century.

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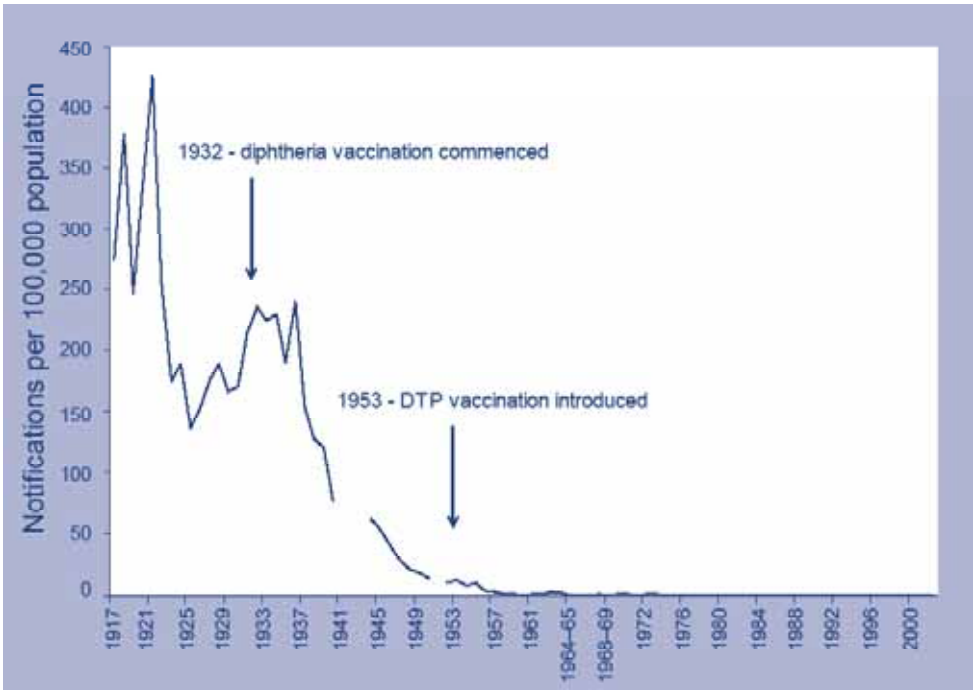
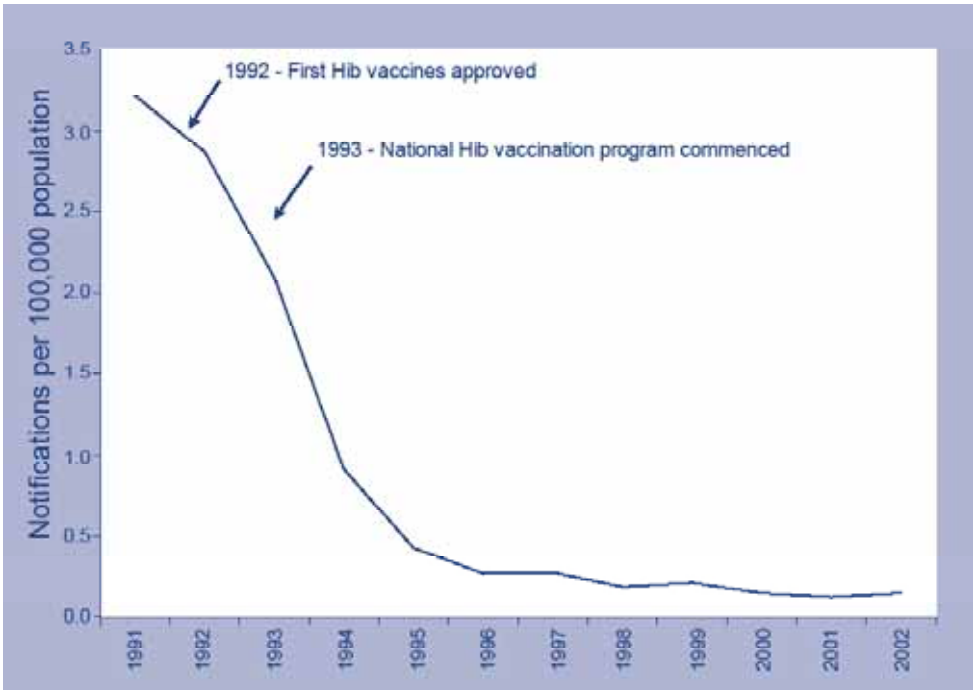
Appendix 1.

Historical charts of notifications of vaccine preventable diseases

Diphtheria, 1917–2002

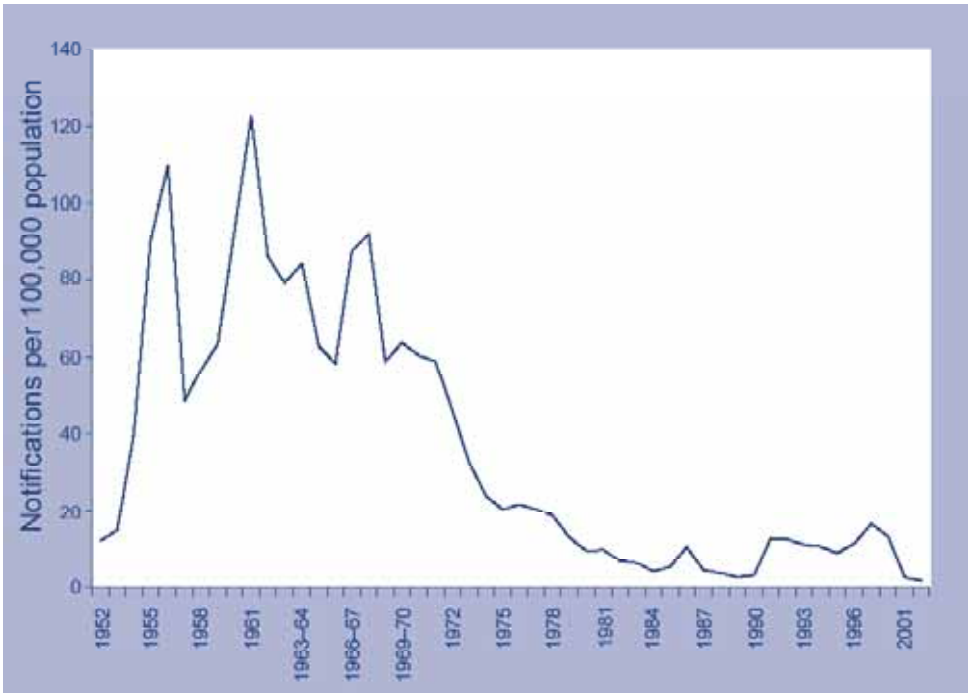


Haemophilus influenzae type b disease, 1991–2002

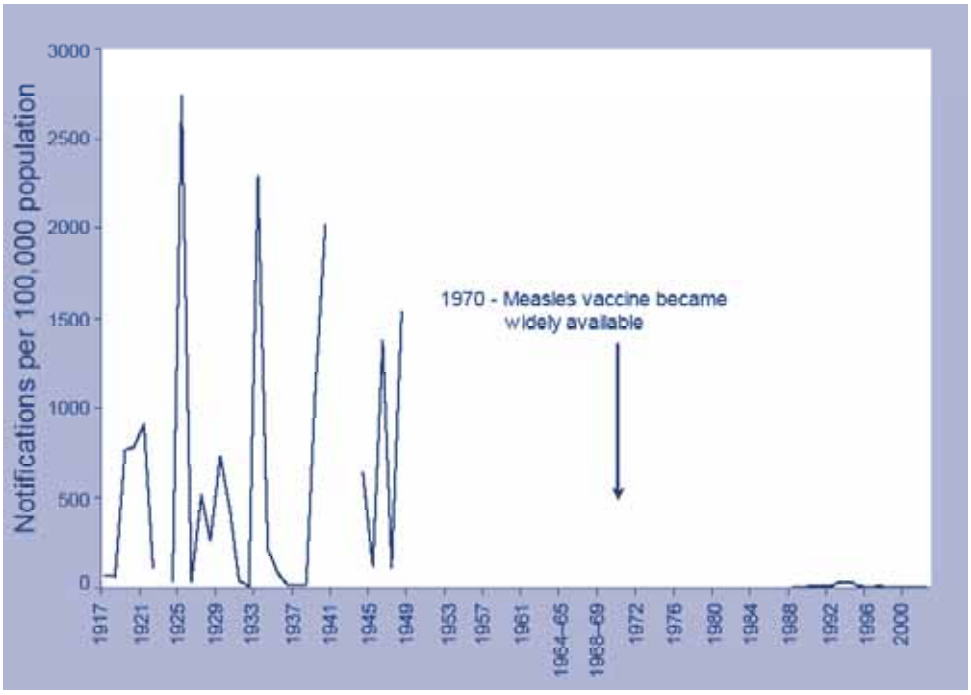


↓ Indicates major change in vaccination policy. 1993; Source: Hall R. Notifiable disease surveillance, 1917 to 1991. *Commun Dis Intell* 17:226–36. Updated with NNDSS data 1992–2002.

Hepatitis A, 1952–2002

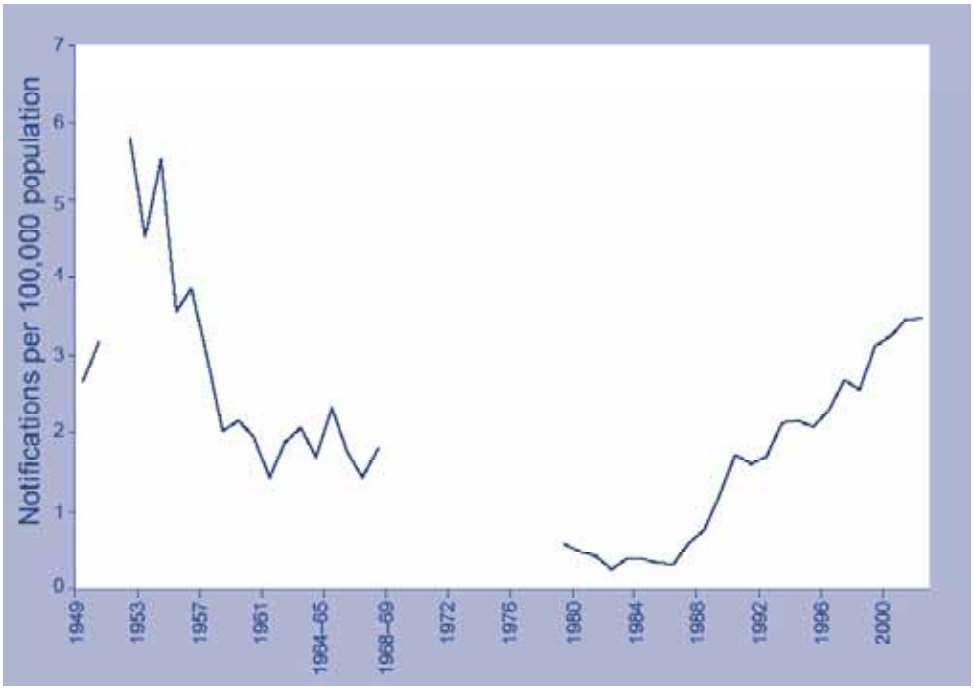


Measles, 1917–2002

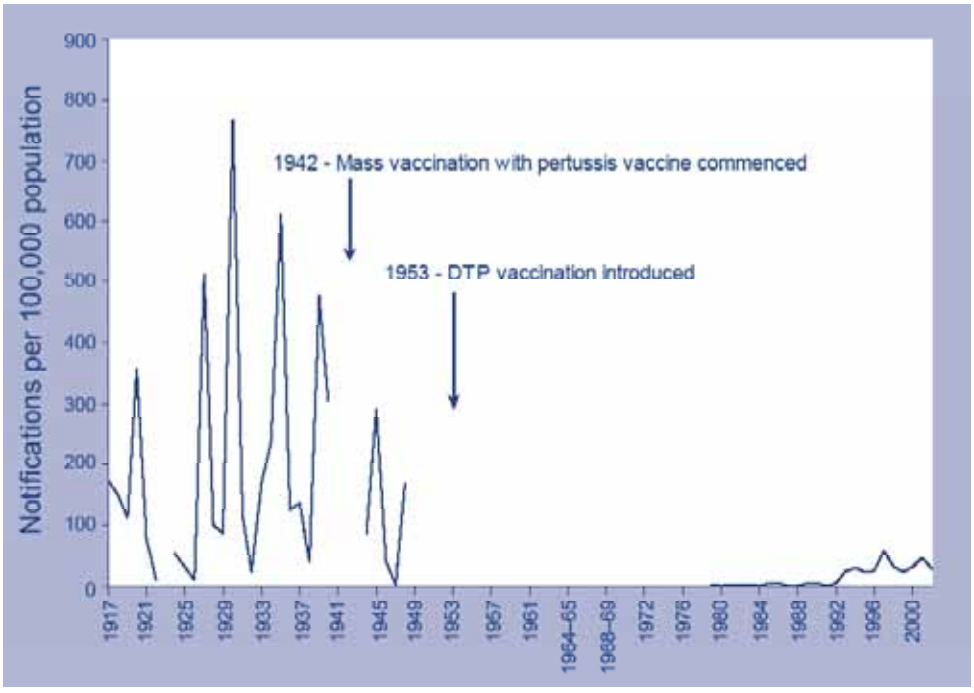


↓ Indicates major change in vaccination policy. 1993; Source: Hall R. Notifiable disease surveillance, 1917 to 1991. *Commun Dis Intell* 17:226–36. Updated with NNDSS data 1992–2002.

Meningococcal disease (invasive), 1949–2002

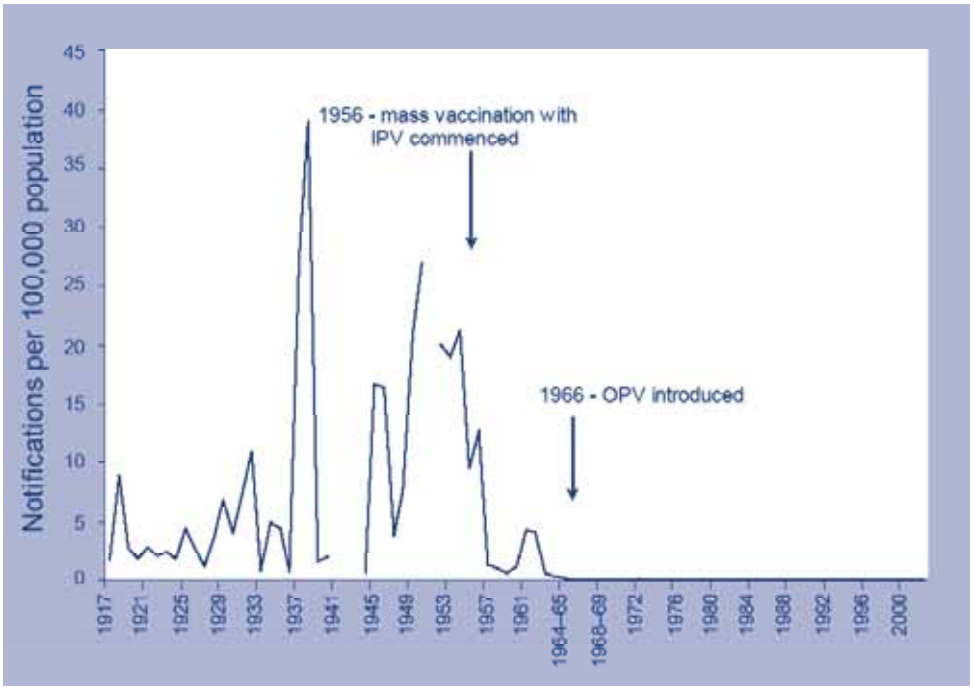


Pertussis, 1917–2002

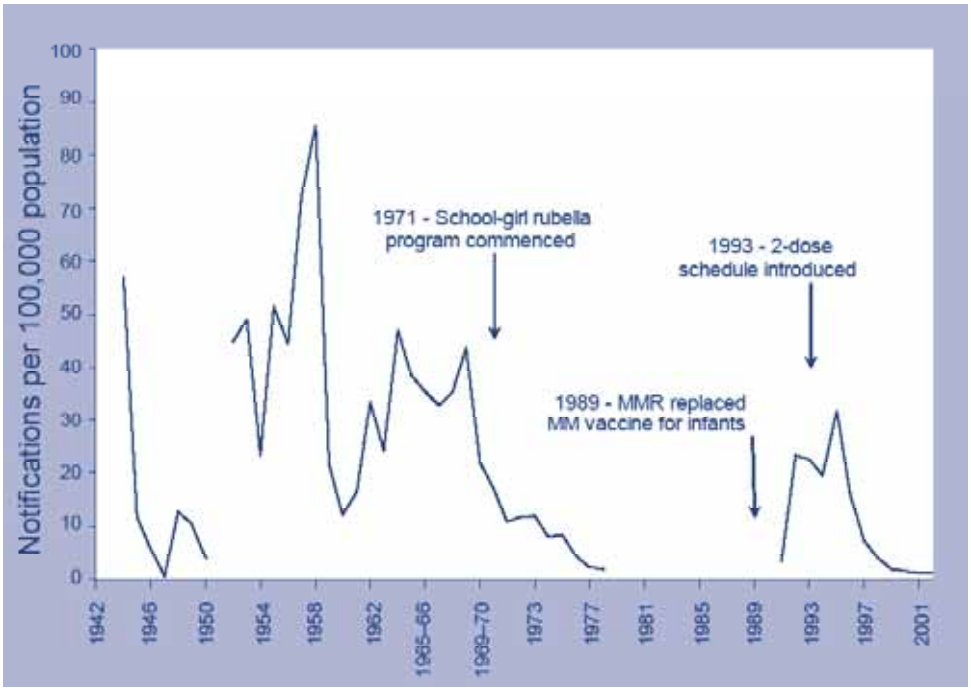


↓ Indicates major change in vaccination policy. 1993; Source: Hall R. Notifiable disease surveillance, 1917 to 1991. *Commun Dis Intell* 17:226–36. Updated with NNDSS data 1992–2002.

Poliomyelitis, 1917–2002

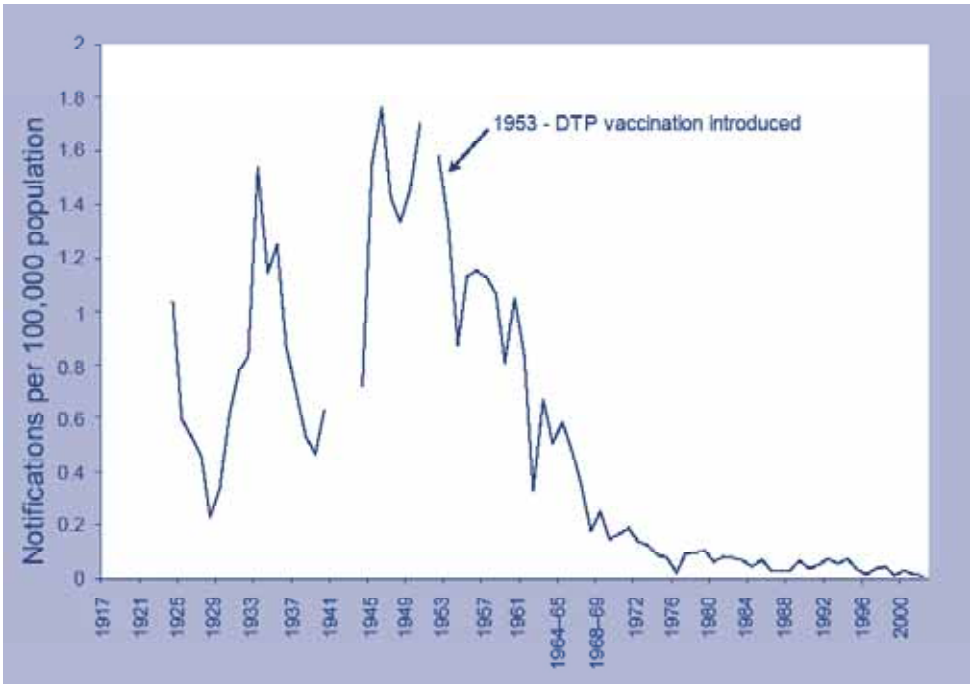


Rubella, 1917–2002



↓ Indicates major change in vaccination policy. 1993; Source: Hall R. Notifiable disease surveillance, 1917 to 1991. *Commun Dis Intell* 17:226–36. Updated with NNDSS data 1992–2002.

Tetanus, 1917–2002



↓ Indicates major change in vaccination policy. 1993; Source: Hall R. Notifiable disease surveillance, 1917 to 1991. *Commun Dis Intell* 17:226–36. Updated with NNDSS data 1992–2002.

Appendix 2.

Notifications by State/Territory and year (January 1997–December 2002)

Table 26. Notifications by State/Territory and year (January 1997–December 2002)

Disease*	Year	Number of notifications									Notification rate per 100,000 population								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Diphtheria	1997	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
	1998	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
	1999	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
	2000	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
	2001	0	0	1	0	0	0	0	0	1	–	–	0.5	–	–	–	–	–	0.0
	2002	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
	Total†	0	0	1	0	0	0	0	0	1	–	–	0.1	–	–	–	–	–	0.0
Haemophilus influenzae type b disease (<15 yr only)	1997	0	11	3	12	2	2	7	3	40	–	0.8	6.0	1.6	0.7	1.9	0.7	0.8	1.0
	1998	0	11	0	6	1	2	2	5	27	–	0.8	–	0.8	0.3	2.0	0.2	1.3	0.7
	1999	1	8	2	6	2	0	3	3	25	1.5	0.6	4.0	0.8	0.7	–	0.3	0.8	0.6
	2000	0	4	0	7	1	0	2	0	14	–	0.3	–	0.9	0.3	–	0.2	–	0.4
	2001	0	6	3	3	2	0	2	1	17	–	0.4	5.9	0.4	0.7	–	0.2	0.2	0.4
	2002	0	4	2	1	2	0	1	6	16	–	0.3	3.9	0.1	0.7	–	0.1	1.5	0.4
	Total†	1	44	10	35	10	4	17	18	139	0.3	0.6	3.3	0.8	0.6	0.7	0.3	0.8	0.6
Hepatitis A	1997	52	1,427	95	917	92	3	341	117	3,044	16.8	22.7	50.8	27.0	6.2	0.6	7.4	6.5	16.4
	1998	53	927	45	1,050	97	8	171	146	2,497	17.1	14.6	23.7	30.5	6.5	1.7	3.7	8.0	13.3
	1999	8	407	89	360	121	5	269	295	1,554	2.6	6.3	46.2	10.3	8.1	1.1	5.7	15.9	8.2
	2000	5	199	45	133	54	3	193	181	813	1.6	3.1	23.0	3.7	3.6	0.6	4.1	9.7	4.2
	2001	14	195	37	115	20	3	97	37	518	4.4	3.0	18.7	3.2	1.3	0.6	2.0	1.9	2.7
	2002	4	149	47	65	16	4	74	32	391	1.2	2.2	23.7	1.8	1.1	0.8	1.5	1.7	2.0
	Total†	136	3,304	358	2,640	400	26	1,145	808	8,817	7.2	8.5	30.8	12.4	4.4	0.9	4.0	7.2	7.7

NN = not notifiable.

* Refer to Chapter 3 for case definitions.

† Total cases for 6-year period and average annual rate per 100,000 population.

‡ Disease not notifiable for complete year for some jurisdictions.

§ National rate only includes jurisdictions where disease was notifiable for complete year.

Table 26. Notifications by State/Territory and year (January 1997–December 2002) (continued)

Disease*		Number of notifications									Notification rate per 100,000 population								
	Year	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Hepatitis B (acute)	1997	2	52	20	42	16	1	119	19	271	0.6	0.8	10.7	1.2	1.1	0.2	2.6	1.1	1.5
	1998	1	55	18	47	18	6	91	31	267	0.3	0.9	9.5	1.4	1.2	1.3	2.0	1.7	1.4
	1999	3	65	20	54	19	5	95	45	306	1.0	1.0	10.4	1.5	1.3	1.1	2.0	2.4	1.6
	2000	3	96	6	56	30	18	121	75	405	1.0	1.5	3.1	1.6	2.0	3.8	2.6	4.0	2.1
	2001	2	91	3	47	23	21	208	39	434	0.6	1.4	1.5	1.3	1.5	4.5	4.3	2.1	2.2
	2002	0	86	22	53	11	19	175	37	403	–	1.3	11.1	1.4	0.7	4.0	3.6	1.9	2.1
	Total†	11	445	89	299	117	70	809	246	2,086	0.6	1.1	7.7	1.4	1.3	2.5	2.9	2.2	1.8
Influenza	2001	14	241	(91) ‡	(392) ‡	135	(0) ‡	(176) ‡	234	(1,283) ‡	4.4	3.7	NA	NA	8.9	NA	NA	12.3	6.1§
	2002	19	1011	56	1152	291	5	598	544	3,676	5.9	15.2	28.3	31.1	19.1	1.1	12.3	28.2	18.7
	Total	33	1,252	(147) ‡	(1,544) ‡	426	(5) ‡	(774) ‡	778	(4,959) ‡	5.1	9.5	37.1	21.0	14.1	0.5	8.0	20.3	14.4§
Measles	1997	44	273	8	268	28	41	93	83	838	14.2	4.3	4.3	7.9	1.9	8.7	2.0	4.6	4.5
	1998	8	118	1	35	6	36	34	50	288	2.6	1.9	0.5	1.0	0.4	7.6	0.7	2.7	1.5
	1999	5	32	22	33	5	11	110	20	238	1.6	0.5	11.4	0.9	0.3	2.3	2.3	1.1	1.3
	2000	3	35	0	26	11	1	21	10	107	1.0	0.5	–	0.7	0.7	0.2	0.4	0.5	0.6
	2001	0	30	0	10	2	2	82	13	139	–	0.5	–	0.3	0.1	0.4	1.7	0.7	0.7
	2002	0	8	0	8	2	0	14	0	32	–	0.1	–	0.2	0.1	–	0.3	–	0.2
	Total†	60	496	31	380	54	91	354	176	1,642	3.2	1.3	2.7	1.8	0.6	3.2	1.2	1.6	1.4
Meningococcal disease	1997	9	219	15	72	22	9	100	49	495	2.9	3.5	8.0	2.1	1.5	1.9	2.2	2.7	2.7
	1998	2	186	14	108	26	14	59	71	480	0.6	2.9	7.4	3.1	1.7	3.0	1.3	3.9	2.6
	1999	5	221	8	92	27	13	138	86	590	1.6	3.4	4.2	2.6	1.8	2.8	2.9	4.6	3.1
	2000	5	254	9	59	32	15	162	86	622	1.6	3.9	4.6	1.7	2.1	3.2	3.4	4.6	3.2
	2001	6	230	13	120	39	23	164	76	671	1.9	3.5	6.6	3.3	2.6	4.9	3.4	4.0	3.5
	2002	6	215	9	120	31	26	210	67	684	1.9	3.2	4.5	3.2	2.0	5.5	4.3	3.5	3.5
	Total†	33	1,325	68	571	177	100	833	435	3,542	1.7	3.4	5.9	2.7	2.0	3.5	2.9	3.9	3.1

NN = not notifiable.

* Refer to Chapter 3 for case definitions.

† Total cases for 6-year period and average annual rate per 100,000 population.

‡ Disease not notifiable for complete year for some jurisdictions.

§ National rate only includes jurisdictions where disease was notifiable for complete year.

Table 26. Notifications by State/Territory and year (January 1997–December 2002) (continued)

Disease*	Year	Number of notifications									Notification rate per 100,000 population								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Mumps	1997	7	29	10	16	26	3	64	36	191	2.3	0.5	5.4	0.5	1.8	0.6	1.4	2.0	1.0
	1998	5	39	5	31	8	3	53	38	182	1.6	0.6	2.6	0.9	0.5	0.6	1.1	2.1	1.0
	1999	8	33	3	(12) ‡	12	4	73	39	(184) ‡	2.6	0.5	1.6	NA	0.8	0.8	1.6	2.1	1.1§
	2000	17	94	4	NN	15	2	43	39	(214) ‡	5.4	1.4	2.0	NA	1.0	0.4	0.9	2.1	1.4§
	2001	1	28	1	(3) ‡	12	2	38	29	(114) ‡	0.3	0.4	0.5	NA	0.8	0.4	0.8	1.5	0.7§
	2002	0	29	1	6	10	0	10	13	69	–	0.4	0.5	0.2	0.7	–	0.2	0.7	0.4
	Total†	38	252	24	(68) ‡	83	14	281	194	(954) ‡	2.0	0.7	2.1	0.3	0.9	0.5	1.0	1.7	0.9§
Pertussis	1997	106	4,249	24	1,902	1,639	120	1,584	1,204	10,828	34.3	67.7	12.8	56.0	110.6	25.3	34.5	67.1	58.5
	1998	100	2,308	24	1,393	549	55	1,078	284	5,791	32.3	36.4	12.6	40.4	36.9	11.7	23.2	15.6	30.9
	1999	83	1,414	2	963	227	635	997	96	4,417	26.6	22.1	1.0	27.5	15.2	134.7	21.3	5.2	23.3
	2000	208	3,693	9	527	588	142	699	92	5,958	66.0	56.9	4.6	14.8	39.1	30.1	14.7	4.9	31.1
	2001	86	4,240	143	1,507	2,010	103	851	227	9,167	26.9	64.5	72.3	41.5	133.0	21.8	17.7	11.9	47.2
	2002	55	2,011	37	1,820	473	41	884	229	5,550	17.1	30.3	18.6	49.0	31.1	8.7	18.2	11.9	28.3
	Total†	638	17,915	239	8,112	5,486	1,096	6,093	2,132	41,711	33.8	46.3	20.6	38.2	60.9	38.7	21.5	19.1	36.5
(<5 yr only)	Total†	39	1,741	59	604	462	47	623	517	4,092	30.7	66.5	55.7	41.0	82.1	24.6	33.5	67.9	53.1
Pneumococcal disease (invasive)	2001	18	434	93	407	(114) ‡	61	(325) ‡	205	(1,657) ‡	5.6	6.6	47.0	11.2	NA	12.9	NA	10.8	9.3§
	2002	33	861	65	428	180	63	453	211	2,294	10.3	13.0	32.8	11.5	11.8	13.3	9.3	10.9	11.7
	Total	51	1,295	158	835	(294) ‡	124	(778) ‡	416	(3,951) ‡	8.0	9.8	39.9	11.4	9.7	13.1	8.0	10.9	10.7§
Poliomyelitis	1997	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
	1998	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
	1999	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
	2000	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
	2001	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
	2002	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
	Total†	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–

NN = not notifiable.

* Refer to Chapter 3 for case definitions.

† Total cases for 6-year period and average annual rate per 100,000 population.

‡ Disease not notifiable for complete year for some jurisdictions.

§ National rate only includes jurisdictions where disease was notifiable for complete year.

Table 26. Notifications by State/Territory and year (January 1997–December 2002) (continued)

Disease*	Year	Number of notifications								Notification rate per 100,000 population								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Rubella	1997	32	153	7	539	183	18	371	84	1,387	10.4	2.4	3.7	15.9	12.4	3.8	8.1	4.7	7.5
	1998	22	78	5	372	16	14	181	65	753	7.1	1.2	2.6	10.8	1.1	3.0	3.9	3.6	4.0
	1999	17	45	4	157	4	7	121	21	376	5.4	0.7	2.1	4.5	0.3	1.5	2.6	1.1	2.0
	2000	4	192	0	46	7	1	67	6	323	1.3	3.0	–	1.3	0.5	0.2	1.4	0.3	1.7
	2001	1	58	0	131	5	2	60	3	260	0.3	0.9	–	3.6	0.3	0.4	1.2	0.2	1.3
	2002	3	35	1	187	5	1	16	3	251	0.9	0.5	0.5	5.0	0.3	0.2	0.3	0.2	1.3
	Total†	79	561	17	1,432	220	43	816	182	3,350	4.2	1.4	1.5	6.7	2.4	1.5	2.9	1.6	2.9
Tetanus	1997	0	3	0	2	0	1	1	0	7	–	0.0	–	0.1	–	0.2	0.0	–	0.0
	1998	0	3	0	1	1	1	1	1	8	–	0.0	–	0.0	0.1	0.2	0.0	0.1	0.0
	1999	0	1	0	1	0	0	0	0	2	–	0.0	–	0.0	–	–	–	–	0.0
	2000	0	2	0	0	3	0	1	0	6	–	0.0	–	–	0.2	–	0.0	–	0.0
	2001	0	0	0	0	1	1	1	0	3	–	–	–	–	0.1	0.2	0.0	–	0.0
	2002	0	0	0	1	0	0	0	1	2	–	–	–	0.0	–	–	–	0.1	0.0
	Total†	0	9	0	5	5	3	4	2	28	–	0.0	–	0.0	0.1	0.1	0.0	0.0	0.0

NN = not notifiable.

* Refer to Chapter 3 for case definitions.

† Total cases for 6-year period and average annual rate per 100,000 population.

‡ Disease not notifiable for complete year for some jurisdictions.

§ National rate only includes jurisdictions where disease was notifiable for complete year.

Appendix 3.

Hospitalisations by State/Territory and financial year (July 1997–June 2002)

Table 27. Hospitalisations by State/Territory and financial year (July 1997–June 2002)

Disease*	Year	Number of hospitalisations									Hospitalisation rate per 100,000 population								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Diphtheria‡	97/98	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
	98/99	0	1	0	0	0	0	0	0	1	–	0.0	–	–	–	–	–	–	0.0
	99/00	0	0	0	0	0	0	1	0	1	–	–	–	–	–	–	0.0	–	0.0
	00/01	0	1	0	0	0	0	0	0	1	–	0.0	–	–	–	–	–	–	0.0
	01/02	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
	Total†	0	2	0	0	0	0	1	0	3	–	0.0	–	–	–	–	0.0	–	0.0
Haemophilus influenzae type b disease (<15 yr only)	97/98	1	34	3	18	4	2	13	43	118	1.5	2.6	6.0	2.4	1.3	1.9	1.4	10.9	3.0
	98/99	1	17	1	15	6	2	8	10	60	1.5	1.3	2.0	2.0	2.0	2.0	0.8	2.5	1.5
	99/00	1	9	1	18	3	1	11	5	49	1.5	0.7	2.0	2.4	1.0	1.0	1.2	1.3	1.2
	00/01	0	4	1	23	5	4	8	2	47	0.0	0.3	2.0	3.0	1.7	4.0	0.8	0.5	1.2
	01/02	0	17	3	9	4	0	6	5	44	0.0	1.3	5.9	1.2	1.4	0.0	0.6	1.2	1.1
	Total†	3	81	9	83	22	9	46	65	318	0.9	1.2	3.6	2.2	1.5	1.8	1.0	3.3	1.6
Hepatitis A	97/98	11	393	20	291	53	0	96	74	938	3.6	6.3	10.7	8.6	3.6	0.0	2.1	4.1	5.1
	98/99	5	256	11	252	64	4	110	74	781	1.6	4.0	5.8	7.3	4.3	0.8	2.4	4.1	4.2
	99/00	1	181	20	122	57	6	157	100	649	0.3	2.8	10.4	3.5	3.8	1.3	3.4	5.4	3.4
	00/01	5	120	13	66	28	7	75	32	346	1.6	1.9	6.6	1.9	1.9	1.5	1.6	1.7	1.8
	01/02	5	145	14	60	28	3	54	16	325	1.6	2.2	7.1	1.7	1.9	0.6	1.1	0.8	1.7
	Total†	27	1,095	78	791	230	20	492	296	3,039	1.7	3.4	8.1	4.5	3.1	0.8	2.1	3.2	3.2
Hepatitis B (acute) (principal diagnosis only)	97/98	3	54	1	23	13	0	66	15	175	1.0	0.9	0.5	0.7	0.9	–	1.4	0.8	0.9
	98/99	2	45	3	31	12	0	70	20	187	0.6	0.7	1.6	0.9	0.8	–	1.5	1.1	1.0
	99/00	1	46	3	19	11	1	53	21	158	0.3	0.7	1.6	0.5	0.7	0.2	1.1	1.1	0.8
	00/01	0	36	1	14	10	10	65	22	158	–	0.6	0.5	0.4	0.7	2.1	1.4	1.2	0.8
	01/02	0	46	6	13	4	1	61	16	147	–	0.7	3.0	0.4	0.3	0.2	1.3	0.8	0.8
	Total†	6	227	14	100	50	12	315	94	825	0.4	0.7	1.5	0.6	0.7	0.5	1.3	1.0	0.9

* Refer to Chapter 3 for case definitions.

† Total cases for 5-year period and average annual rate per 100,000 population.

‡ Hospitalisations for rare diseases such as diphtheria, poliomyelitis and tetanus should be interpreted with extreme caution due to possible coding errors. For NSW, polio, tetanus and diphtheria occurrences are considered inaccurate, given no notifications of these diseases have been identified to NSW Health for the period 2001–2002. Caution should be exercised in analysing these data and users should consult the disease notifications as published in the *New South Wales Public Health Bulletin*.

Table 27. Hospitalisations by State/Territory and financial year (July 1997–June 2002)

Disease*	Year	Number of hospitalisations									Hospitalisation rate per 100,000 population								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Influenza	97/98	37	1,698	32	1,058	559	68	1,591	1,090	6,133	12.0	27.1	17.1	31.2	37.7	14.4	34.6	60.7	33.1
	98/99	20	1,494	46	1,145	442	36	836	573	4,592	6.5	23.6	24.2	33.2	29.7	7.6	18.0	31.4	24.5
	99/00	10	1,154	25	858	282	41	781	847	3,998	3.2	18.0	13.0	24.5	18.8	8.7	16.7	45.8	21.1
	00/01	17	1,190	38	700	331	54	602	536	3,468	5.4	18.3	19.4	19.7	22.0	11.5	12.7	28.6	18.1
	01/02	11	817	24	631	293	36	493	502	2,807	3.4	12.4	12.1	17.4	19.4	7.6	10.3	26.4	14.5
	Total†	95	6,353	165	4,392	1,907	235	4,303	3,548	20,998	6.1	19.8	17.1	25.0	25.5	10.0	18.3	38.4	22.2
Measles	97/98	11	63	1	59	4	3	12	3	156	3.6	1.0	0.5	1.7	0.3	0.6	0.3	0.2	0.8
	98/99	0	17	0	13	6	3	30	8	78	–	0.3	–	0.4	0.4	0.6	0.6	0.4	0.4
	99/00	1	9	14	10	3	1	15	7	67	0.3	0.1	7.3	0.3	0.2	0.2	0.3	0.4	0.4
	00/01	2	13	0	6	4	0	36	3	64	0.6	0.2	–	0.2	0.3	–	0.8	0.2	0.3
	01/02	0	10	0	2	1	2	18	8	41	–	0.2	–	0.1	0.1	0.4	0.4	0.4	0.2
	Total†	14	112	15	90	18	9	111	29	406	0.9	0.3	1.6	0.5	0.2	0.4	0.5	0.3	0.4
Meningococcal disease	97/98	6	335	17	179	33	14	102	75	761	1.9	5.3	9.1	5.3	2.2	3.0	2.2	4.2	4.1
	98/99	2	284	15	155	39	23	105	124	749	0.6	4.5	7.9	4.5	2.6	4.9	2.3	6.8	4.0
	99/00	5	297	13	108	51	18	210	107	817	1.6	4.6	6.7	3.1	3.4	3.8	4.5	5.8	4.3
	00/01	5	320	10	140	51	15	230	96	867	1.6	4.9	5.1	3.9	3.4	3.2	4.9	5.1	4.5
	01/02	9	240	15	178	60	44	253	77	876	2.8	3.7	7.6	4.9	4.0	9.3	5.3	4.1	4.5
	Total†	27	1,476	70	760	324	114	900	479	4,070	1.7	4.6	7.3	4.3	3.1	4.8	3.8	5.2	4.3
Mumps	97/98	2	18	1	10	3	1	8	8	51	0.6	0.3	0.5	0.3	0.2	0.2	0.2	0.4	0.3
	98/99	1	35	0	9	6	1	4	2	85	0.3	0.6	–	0.3	0.4	0.2	0.1	0.1	0.3
	99/00	2	22	0	14	2	0	7	7	54	0.6	0.3	–	0.4	0.1	–	0.1	0.4	0.3
	00/01	3	15	0	10	4	0	12	4	48	1.0	0.2	–	0.3	0.3	–	0.3	0.2	0.3
	01/02	0	18	0	5	4	1	7	2	37	–	0.3	–	0.1	0.3	0.2	0.1	0.1	0.2
	Total†	8	108	1	48	19	3	38	23	248	0.5	0.3	0.1	0.3	0.3	0.1	0.2	0.2	0.3
Pertussis	97/98	9	439	8	213	123	11	125	237	1,165	2.9	7.0	4.3	6.3	8.3	2.3	2.7	13.2	6.3
	98/99	2	171	2	87	25	1	79	27	396	0.6	2.7	1.1	2.5	1.7	0.2	1.7	1.5	2.1
	99/00	8	115	2	53	13	21	118	17	349	2.6	1.8	1.0	1.5	0.9	4.5	2.5	0.9	1.8
	00/01	8	269	10	55	78	6	62	19	507	2.5	4.1	5.1	1.5	5.2	1.3	1.3	1.0	2.6
	01/02	4	260	44	156	134	6	98	68	770	1.3	4.0	22.2	4.3	8.9	1.3	2.0	3.6	4.0
	Total†	31	1,254	66	564	373	45	482	368	3,187	2.0	3.9	6.9	3.2	5.0	1.9	2.1	4.0	3.4
(<5yr only)	Total†	22	884	57	362	213	25	341	295	2,200	20.7	40.4	64.6	29.6	45.1	15.6	21.9	46.3	34.2

* Refer to Chapter 3 for case definitions.

† Total cases for 5-year period and average annual rate per 100,000 population.

Table 27. Hospitalisations by State/Territory and financial year (July 1997–June 2002) (continued)

Disease*	Year	Number of hospitalisations									Hospitalisation rate per 100,000 population								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Pneumococcal disease (invasive) ‡	97/98	6	260	32	71	63	12	143	54	641	1.9	4.1	17.1	2.1	4.3	2.5	3.1	3.0	3.5
	98/99	9	292	54	88	74	16	173	44	753	2.9	4.6	28.4	2.6	5.0	3.4	3.7	2.4	4.0
	99/00	27	333	43	116	82	23	200	118	949	8.6	5.2	22.3	3.3	5.5	4.9	4.3	6.4	5.0
	00/01	14	373	58	170	65	13	218	99	1,010	4.4	5.8	29.7	4.8	4.3	2.8	4.6	5.3	5.3
	01/02	20	365	46	189	104	25	254	98	1,101	6.3	5.6	23.3	5.2	6.9	5.3	5.3	5.2	5.7
	Total†	76	1,623	233	634	388	89	988	413	4,454	4.9	5.1	24.2	3.6	5.2	3.8	4.2	4.5	4.7
Poliomyelitis§	97/98	0	31	0	19	6	1	14	0	71	–	0.5	–	0.6	0.4	0.2	0.3	–	0.4
	98/99	0	34	0	11	1	2	20	1	69	–	0.5	–	0.3	0.1	0.4	0.4	0.1	0.4
	99/00	0	1	0	0	4	0	16	0	21	–	0.0	–	–	0.3	–	0.3	–	0.1
	00/01	0	15	1	0	3	0	1	0	20	–	0.2	0.5	–	0.2	–	0.0	–	0.1
	01/02	1	6	3	0	2	2	0	0	14	0.3	0.1	1.5	–	0.1	0.4	–	–	0.1
	Total†	1	87	4	30	16	5	51	1	195	0.1	0.3	0.4	0.2	0.2	0.2	0.2	0.0	0.2
(Principal diagnosis only)	Total†	0	3	0	2	1	0	2	0	8	–	0.0	–	0.0	0.0	–	0.0	–	0.0
Rubella	97/98	1	22	0	12	5	0	3	5	48	0.3	0.4	–	0.4	0.3	–	0.1	0.3	0.3
	98/99	0	16	0	9	3	1	11	3	43	–	0.3	–	0.3	0.2	0.2	0.2	0.2	0.2
	99/00	0	8	0	8	3	0	10	0	29	–	0.1	–	0.2	0.2	–	0.2	–	0.2
	00/01	0	24	0	3	1	0	4	1	33	–	0.4	–	0.1	0.1	–	0.1	0.1	0.2
	01/02	0	15	0	1	1	1	3	0	21	–	0.2	–	0.0	0.1	0.2	0.1	–	0.1
	Total†	1	85	0	33	13	2	31	9	174	0.1	0.3	–	0.2	0.2	0.1	0.1	0.1	0.2

* Refer to Chapter 3 for case definitions.

† Total cases for 5-year period and average annual rate per 100,000 population.

‡ Pneumococcal meningitis and septicaemia.

§ Hospitalisations for rare diseases such as diphtheria, poliomyelitis and tetanus should be interpreted with extreme caution due to possible coding errors. For NSW, polio, tetanus and diphtheria occurrences are considered inaccurate, given no notifications of these diseases have been identified to NSW Health for the period 2001–2002. Caution should be exercised in analysing these data and users should consult the disease notifications as published in the *New South Wales Public Health Bulletin.*

Table 27. Hospitalisations by State/Territory and financial year (July 1997–June 2002) (continued)

Disease*	Year	Number of hospitalisations									Hospitalisation rate per 100,000 population								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Tetanus‡	97/98	0	20	0	2	3	1	4	4	34	–	0.3	–	0.1	0.2	0.2	0.1	0.2	0.2
	98/99	0	15	0	6	0	1	4	7	33	–	0.2	–	0.2	–	0.2	0.1	0.4	0.2
	99/00	0	8	0	8	6	2	5	2	32	–	0.1	–	0.2	0.4	0.4	0.1	0.1	0.2
	00/01	0	17	0	6	3	1	3	1	31	–	0.3	–	0.2	0.2	0.2	0.1	0.1	0.2
	01/02	0	6	0	4	3	0	4	6	23	–	0.1	–	0.1	0.2	0.0	0.1	0.3	0.1
	Total†	0	66	0	26	15	5	20	20	153	–	0.2	–	0.1	0.2	0.2	0.1	0.2	0.2
Varicella	97/98	27	440	18	300	126	25	365	214	1,515	8.7	7.0	9.6	8.8	8.5	5.3	7.9	11.9	8.2
	98/99	22	677	33	427	188	36	399	199	1,991	7.1	10.7	17.4	12.4	12.6	7.6	8.6	10.9	10.6
	99/00	30	580	32	332	93	48	418	190	1,734	9.6	9.0	16.6	9.5	6.2	10.2	8.9	10.3	9.2
	00/01	22	544	14	335	152	30	344	198	1,639	7.0	8.4	7.2	9.4	10.1	6.4	7.3	10.6	8.6
	01/02	22	570	21	308	185	32	344	197	1,679	6.9	8.7	10.6	8.5	12.2	6.8	7.2	10.4	8.6
	Total†	123	2,811	118	1,702	744	171	1,870	998	8,558	7.9	8.8	12.3	9.7	9.9	7.2	8.0	10.8	9.0
Zoster	97/98	37	1,496	22	776	486	115	1,042	421	4,395	12.0	23.8	11.8	22.9	32.8	24.3	22.7	23.5	23.7
	98/99	33	1,717	21	808	508	123	1,074	422	4,718	10.6	27.1	11.1	23.4	34.1	26.1	23.2	23.2	25.2
	99/00	35	1,574	26	853	519	147	1,103	406	4,673	11.2	24.6	13.5	24.4	34.7	31.2	23.5	21.9	24.7
	00/01	44	1,499	15	838	444	135	1,142	416	4,533	14.0	23.1	7.7	23.5	29.5	28.6	24.1	22.2	23.7
	01/02	56	1,453	21	891	514	138	1,175	380	4,628	17.5	22.1	10.6	24.6	34.0	29.2	24.5	20.0	23.8
	Total†	205	7,739	105	4,166	2,471	658	5,536	2,045	22,947	13.1	24.1	10.9	23.8	33.0	27.9	23.6	22.1	24.2

* Refer to Chapter 3 for case definitions.

† Total cases for 5-year period and average annual rate per 100,000 population.

‡ Hospitalisations for rare diseases such as diphtheria, poliomyelitis and tetanus should be interpreted with extreme caution due to possible coding errors. For NSW, polio, tetanus and diphtheria occurrences are considered inaccurate given no notifications of these diseases have been identified to NSW Health for the period 2001–2002. Caution should be exercised in analysing these data and users should consult the disease notifications as published in the *New South Wales Public Health Bulletin*.

Appendix 4.

*Changes to the Australian Standard Vaccination Schedule (1992–2002)**

Table 28. Diphtheria, tetanus and pertussis (DTP) vaccination practice in Australia, 1992 to 2002

Date	Intervention
1994	5th dose of DTP at 4–5 years added to the recommended vaccination schedule (replacing CDT vaccine) Active ADT school vaccination programs commenced in some States for 15–19 year olds
1996	Diphtheria-tetanus-acellular pertussis vaccine (DTPa) licensed in Australia
1997	DTPa recommended for 4th and 5th doses of DTP vaccination (due at 18 months and 4–5 years)
1999	DTPa recommended for all 5 childhood DTP doses Combined DTPa-hepatitis B vaccine approved
2000	Second booster dose of DTPa recommended at 4 years instead of 4–5yrs NHMRC recommended 10-yearly booster doses of ADT be replaced with a routine booster dose at 50 yrs of age unless a booster dose has been documented within last 10 years DTPa-hepB vaccine included on childhood schedule (used in Qld, NSW, ACT, SA, and NT) Adult/adolescent formulation (dTpa) available for boosting adolescents and adults against pertussis

Table 29. *Haemophilus influenzae* type b vaccination practice in Australia, 1992 to 2002

Date	Intervention
1992	1st Hib vaccines (PRP-D, ProHIBit) licensed in Australia for vaccinating infants aged at least 18 months
1993	Hib vaccine recommended as part of the childhood vaccination schedule Hib vaccines: HBOC (HibTITER), PRP-T (Act-HIB), and PRP-OMP (PedvaxHIB) licensed for infants aged <18 months PRP-OMP recommended at 2, 4 and 12 months, HBOC and PRP-T at 2, 4, 6 and 18 months
2000	Combined Hib(PRP-OMP)-hepatitis B vaccine approved PRP-OMP recommended for all infants (administered separately or in combination with hepatitis B vaccine)

* See also Gidding HF, Burgess MA, Kempe AE. A brief history of vaccination and childhood vaccination practices in Australia. *Medical Journal of Australia* 2001;174:37–40.
Detailed historical tables are available at: http://www.ncirs.usyd.edu.au/publ/publ-79-tbls.html

Table 30. Hepatitis B vaccination practice in Australia, 1992 to 2002

Date	Intervention
1997	Vaccination recommended for adolescents aged 10–16 years
1997	Interim recommendation for universal vaccination of infants at birth
1998	School-based programs commenced for 10–16 year olds in Victoria. A 'catch up' campaign was conducted in the NT for children 6–16 years of age
1999	SA commenced year 8 immunisation program provided by councils Combined DTPa-hepatitis B vaccine approved
2000	Thiomersal-free paediatric hepatitis B vaccine approved Combined PRP-OMP-hepB vaccine approved May: Universal infant vaccination included in childhood schedule with a birth dose of monovalent paediatric hepatitis B vaccine, followed by 3 doses as part of a combination vaccine schedule DTPa-hepB vaccine included on childhood schedule (used in Qld, NSW, ACT, SA, and NT) PRP-OMP-hepB vaccine included in childhood schedule (used in Tas, Vic, WA) Preadolescent vaccination recommended at 10–13 years rather than 10–16 years of age Booster doses no longer recommended by NHMRC

Table 31. Influenza vaccination practice in Australia, 1992 to 2002

Date	Intervention
1997	In Victoria, influenza vaccine funded for all adults aged 65 years and over
1999	Funding provided for both the national Older Australian Flu program and the National Indigenous Pneumococcal and Influenza Immunisation (NIPII) program

Table 32. Measles, mumps and rubella vaccination practice in Australia, 1992 to 2002

Date	Intervention
1992 (Nov)	NHMRC recommended 2nd dose of MMR vaccine for both sexes to replace schoolgirl rubella vaccination program
1993 (Nov)	Childhood vaccination schedule updated to include second dose of MMR vaccine for 10–16 year olds (replacing schoolgirl rubella vaccination)
1998	Recommended age for 1st dose of MMR vaccine for Aboriginal children in the Northern Territory increased to 12 months of age (in line with non-Aboriginal infants) July: Recommended age for 2nd MMR vaccine dose lowered to 4–5 years July–December: Implementation of Measles Control Campaign (involving mass vaccination of primary school aged children with MMR vaccine)
2000	Recommended age for second MMR dose lowered to 4 years not 4–5 years MMR rather than rubella vaccine recommended for non-immune women of child-bearing age

Table 33. Meningococcal C vaccination practice in Australia, 1992 to 2002

Date	Intervention
2001	Meningococcal C conjugate vaccine (Meningitec) available for purchase in private market
2002	Meningococcal C conjugate vaccines (NeisVac-C, Menjugate) available for purchase in private market
2002	Funding announced for National Meningococcal C Vaccination Program commencing 2003

Table 34. Pneumococcal vaccination practice in Australia, 1992 to 2002

Date	Intervention
1994	Vaccination recommended for Aboriginal and Torres Strait Islanders living in high risk communities aged over 50 years
1997	Vaccination recommended for all persons aged over 65 years Vaccination recommended for all Aboriginal and Torres Strait Islanders aged over 50 years
1998	In Victoria, pneumococcal vaccine funded for all adults aged 65 years and over and all Aboriginal and Torres Strait Islanders aged 50 years and over
1999	Vaccination recommended for Aboriginal and Torres Strait Islanders aged 15–50 years with any of the high risk underlying conditions 23-valent polysaccharide vaccine funded by the Commonwealth (under the National Indigenous Pneumococcal and Influenza Immunisation program–NIPII) for all Aboriginal and Torres Strait Islanders aged at least 50 years and those aged 15–50 years with any of the high risk underlying conditions
2000	Vaccination recommendation for Aboriginal and Torres Strait Islanders changed from >50 to ≥50 years Vaccination recommendation for all persons changed from >65 to ≥65 years NT recommended 23-valent vaccine for all Aboriginal and Torres Strait Islander people aged 15 years and over 7-valent conjugate pneumococcal vaccine approved
2001	Funding made available for the at-risk conjugate pneumococcal vaccination program (all Aboriginal and Torres Strait Islander infants; all Australian children with underlying predisposing medical conditions; non-Indigenous children residing in central Australia up to the second birthday, as ‘catch-up’ vaccination)

Table 35. Polio vaccination practice in Australia, 1992 to 2002

Date	Intervention
1994	Recommendation for reinforcing dose of OPV to 15 year old adolescents
2002	Fifth dose of OPV at 15–17 years of age no longer recommended

Appendix 5.

Vaccination funding in Australia

Vaccination funding in Australia

Prior to 1988, the Commonwealth provided childhood vaccines to States/Territories for distribution to providers in the public sector. During the same time, live attenuated vaccines such as oral polio vaccine (OPV) and measles vaccine were provided to private practitioners, although it is not certain that this occurred in all States/Territories. Private practitioners who provided vaccination services were required to issue prescriptions for the supply of inactivated vaccines, such as DTPw, by a pharmacist.

In July 1988, the Commonwealth made a decision to withdraw from the direct provision of funding to purchase childhood vaccines, and instead increased funding provided to States/Territories as part of the Finance Assistance Grants (FAGs) and Hospital Funding Grants (HFGs). The increase in funding was equivalent to the level of immunisation activity in each jurisdiction in 1988.

The level of funding provided via the FAGs/HFGs was in dispute by States/Territories from a very early stage, as increases in vaccination activity above the 1988 level began to put pressure on the resources provided. Details of the funding arrangements were also interpreted differently by the Commonwealth and each State/Territory, leading to variations in implementation of immunisation programs and uncoordinated and fragmented service delivery.

In April 1993, the National Health and Medical Research Council (NHMRC) reported on Australia's immunisation programs and made recommendations concerning a National Immunisation Strategy (NIS). The NHMRC Report identified a number of factors contributing to poor immunisation coverage and the rising incidence of vaccine preventable diseases in Australian children. These were the lack of a coordinated scheme for the provision of vaccines, and the wide variation in prices which the States/Territories paid for vaccines, with the smaller jurisdictions paying higher prices. The Strategy recommended that vaccine purchase be coordinated centrally and funding occur directly to States/Territories, based on population size.

In 1992, the first *Haemophilus influenzae* type b (Hib) vaccine was approved for use in children aged 18 months and older. In January 1993, vaccines approved for use in younger children became available. As these were new vaccines, no funding was available within existing arrangements for purchase by States/Territories. In July 1993, the Commonwealth provided funds to States/Territories, so Hib vaccines became the first to be funded via the mechanism recommended in the NIS.

In 1994, the Commonwealth Government decided to fund the purchase of a number of childhood vaccines (DTP, MMR, OPV) via Specific Purpose Payments to States/Territories. Commonwealth funding was conditional on vaccines being provided to all public and private practitioners and was formalised in bilateral agreements with each State/Territory.

From 1997–1998 funds for vaccination were included in the Public Health Outcome Funding Agreements (PHOFAs). However, a number of vaccines continued to be funded via Finance Assistance Grants (OPV doses 1, 2, 3 and 4 and MMR dose 1) and Hospital Funding Grants (ADT).

In 1997, the NHMRC recommended that the diphtheria-tetanus-acellular pertussis vaccine (DTPa) be used for the fourth and fifth doses of DTP vaccination. These became funded nationally in September 1997.

The 1998–1999 Commonwealth Budget included an initiative to streamline all childhood vaccine funding as from 1999 to 2000, resulting in funding for all childhood vaccines on the Australian Standard Vaccination Schedule (ASVS) (up to 15 years of age) being included in the PHOFAs. In the same financial year, pneumococcal vaccine for Indigenous Australians and influenza vaccine for those aged over 65 years were also funded. Existing vaccine funding allocations via FAGs and HFGs were not adjusted, thereby freeing up State/Territory resources to purchase non-Commonwealth funded vaccines.

Federal funding to use DTPa for all five infant vaccinations began in February 1999, immediately after the NHMRC recommended the schedule change.

In 1999 to 2000, PHOFA funding to purchase enough vaccine for 105 per cent of the eligible cohort for each vaccine (with the current exception of influenza vaccine) was made available. Funding for vaccines is approved by the Federal Minister for Health and Aged Care as a 'special appropriation' under the provisions of Section 9B of the *National Health Act 1953*. Based on interpretation of this provision, funds appropriated are for the sole purpose of vaccine purchase.

From May 2000, universal infant vaccination with hepatitis B vaccine was recommended and funded. In 2001, the 7-valent pneumococcal conjugate vaccine was made available free of charge for the following categories of children. First, all Aboriginal and Torres Strait Islander children aged up to two years; second, in the Central Australian region, Indigenous children aged up to five years and non-Indigenous children aged up to two years; and third, all children under five years with medical risk factors predisposing them to a high incidence or severity of pneumococcal infection.

Table 36 summarises the dates when vaccines became free of charge in the public and private sectors as outlined above.

Table 36. Dates when childhood vaccines became available in Australia free of charge* in the public and private sectors, up to December 2002

Vaccine	Public sector		Private sector†	
	Australia	Exceptions	Australia	Exceptions
OPV	1966		1994	Qld (? 1998) NSW 1966 Tas 1966
DTPw	1953		1994	WA 1988
Rubella (adolescent girls)	1971			
MMR (infant dose)	1989		1994	NSW 1989 Qld 1989
MMR (adolescent dose)	1994	SA 1996	1994	WA 1993 SA 1996
ADT	1982		1994	WA 1988
CDT	1975		1994	WA 1988
Hib vaccines (infants born from Feb 1993)	1993 April		1993 April	
Hib vaccines (all infants aged <5 years)	1993 July	WA 1993 Jan NT 1993 April	1993 July	WA 1993 Jan NT 1994
DTPa boosters (infants aged 18 months and 4–5 years)	1997 Sept	Tas 1997 Oct Qld 1997 Dec	1997 Sept	Tas 1997 Oct Qld 1997 Dec
DTPa (infants aged 2, 4 and 6 months)	1999 Feb	NT 1997 Aug SA 1997 Aug Tas 1999 Feb Qld 1999 April	1999 Feb	NT 1997 Aug SA 1997 Aug Tas 1999 Feb Qld 1999 April
Hep B (at-risk infants)	1987	NT 1988 Jan SA 1996	Not funded by the C'wealth	NSW 1987
Hep B (adolescent dose)	1998 Jan	Qld 1998 March Tas 1998 March NT 1998 April NSW 1999 SA 1999	?1998	Qld 1998 March Tas 1998 March NT 1998 April NSW 1999
Hep B (universal infant dose)	2000 May	NT 1990 Aug	2000 May	NT 1994
7vPCV (at-risk children)	2001		2001	

* Vaccines on the current Australian Standard Childhood Vaccination schedule became free of charge in the public and private sector in all jurisdictions in 1999/2000.

† All scheduled childhood vaccines became free in the private sector in the Australian Capital Territory in 1993 (except for MMR vaccine which became free in the private sector in 1994) and in the Northern Territory in 1994.