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Vaccine Preventable Diseases and Vaccination Coverage in Australia, 2003 to 2005





National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

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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.

Executive summary

Overview

This, the fourth report on vaccine preventable diseases and vaccination 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 January 2003 and December 2005. Most recently available hospitalisation and death data cover the period up to 30 June 2005 and 31 December 2004, respectively. The general trend towards improved control of disease and improved vaccination coverage is evident, particularly in the childhood years. Detailed results are available in 16 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.

Compared to the previous review period (2001–2002), there are continuing declines in the overall disease burden, as indicated by these routinely collected data, that are driven by improving control of measles, rubella, Hib and pneumococcal disease, and a dramatic fall in meningococcal disease in 2004/2005.

Table 1. Notifications, hospitalisations and deaths from 11 diseases preventable by vaccination, Australia, 2000 to 2005*

Disease†	Notifications		Hospitalisations		Deaths	
	Average per year 2001–2002	Average per year 2003–2005	Average per year July 2000–June 2002	Average per year July 2002–June 2005	Average per year 2001–2002	Average per year 2003–2004
Diphtheria	0.5	0	0.5‡	0‡	0	0
Hib (<5 yr)§	12	8	15	12	0	0
Influenza‖	3,671¶	3,395	3,139	3,039	44	51
Measles	87	49	53	31	0	0
Meningococcal disease	687	452	873	712	44	23
Mumps**	92	140	43	46	0.5	0.5
Pertussis	7,521	8,345	638	440	3	1
Pneumococcal disease††	2,466	2,101	1,057	1,038	15	19
Polio‡‡	0	0	2‡‡	2‡‡	0	0
Rubella	261	39	27	15	0	0
Tetanus	4	4	27	22	0.5	0
Total§§	14,800	14,532	5,873	5,356	106	94

* Notifications where the month of diagnosis was between January 2001 and December 2005; hospitalisations where the month of separation was between 1 July 2000 and 30 June 2005; deaths where the date of death was recorded between 2001 and 2004.

† See Chapter 3 for case definitions.

‡ Only includes hospitalisations coded as pharyngeal, nasopharyngeal or laryngeal diphtheria as per included codes for previous review period.

§ Note that hospitalisations and deaths are for *Haemophilus influenzae* meningitis only 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 for the complete calendar year in 2001.

†† Pneumococcal hospitalisations and deaths for sepsis or meningitis only.

‡‡ Principal diagnosis only.

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

There is an ongoing absence of disease due to polio and diphtheria and a continuing low incidence of tetanus. There have been continuing declines in hepatitis A and hepatitis B incidence. Following the introduction of the National Q Fever Management Program, the incidence of Q fever has also declined. Mumps and pertussis notifications have increased, whereas hospitalisations and deaths for mumps are stable and for pertussis have declined. Influenza and pneumococcal disease continue to contribute the greatest burden of serious disease as indicated by hospitalisation and death data.

Comment

The years 2003 to 2005 have been a period of continuing gains in the control of vaccine preventable diseases and a time of expansion with the implementation of new vaccination programs against meningococcal C disease, varicella-zoster, and pneumococcal disease. Although there has been an increase in the number of diseases against which children are routinely vaccinated, vaccination coverage has been maintained at high levels and such coverage is likely to be facilitated by the now routine use of combination vaccines. 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, the introduction and implementation in 2004 of new national definitions for diseases notified to the National Notifiable Diseases Database was an important step in improving the consistency of notifications reported by different jurisdictions. Further challenges remain in working towards improved consistency and completion in the reporting of fields such as vaccination status. In vaccination practice, vaccination coverage targets are probably close to their highest achievable levels in children. However, improving control of mumps and pertussis through vaccination of young adults stands out as a challenge for the next few years.

Careful evaluation of the additional benefits of new programs, such as those for meningococcal C, pneumococcal, and varicella-zoster disease, will be important to convince stakeholders, including the public and professionals, of the value of vaccination and to ensure the success of immunisation in Australia into the future.

1. Introduction

This is the fourth national report on the morbidity and mortality from vaccine preventable diseases (VPDs) and vaccination coverage in Australia. The first (1993 to 1998) was published in 2000, the second (1999 to 2000) in 2002 and the third (2001 to 2002) in 2004.[1–3] The progressive decline in the incidence of all the childhood VPDs continues, with the possible exceptions of pertussis and mumps where notifications have increased although this is not reflected in hospitalisations. Most striking has been the 99.75% decline in the numbers of deaths from these diseases since the prevaccination era, despite the Australian population increasing almost threefold (Table 2), and the close associations of declines in individual disease mortality with the introduction of specific vaccination programs.[4] Deaths due to pertussis in infants too young to be vaccinated remain an important challenge.

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

- a national disease notification system (NNDSS) in 1991;
- the Australian Childhood Immunisation Register (ACIR) in 1996;[5]
- the *Seven Point Plan* in 1997 (this included the Measles Control Campaign in the later part of 1998);[6]
- the General Practice Immunisation Initiative in 1998;
- implementation of new national notifiable diseases definitions, daily data updates to NNDSS and on-line data publication in 2004; and
- new vaccination programs for children (against hepatitis B, *Haemophilus influenzae* type b, meningococcal C disease, pneumococcal disease, varicella-zoster virus), the elderly (influenza and pneumococcal disease) and Indigenous people (influenza, pneumococcal disease, hepatitis A).

Although specific enhanced evaluations are important, much can be learned from examining routinely collected data, especially for trends over time. This fourth report uses similar methods to the first three, 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, invasive pneumococcal disease, measles, meningococcal C disease, mumps, pertussis, poliomyelitis, rubella, tetanus and varicella), those for which vaccines were available but only funded or recommended for specific risk groups (hepatitis A, influenza and Q fever) and for rotavirus, for which new vaccines became available in 2006. The report does not cover some other diseases which are at least partially preventable by vaccination, such as tuberculosis, on which reports can be found elsewhere.[7–9]

This and the previous three reports, all from the National Centre for Immunisation Research and Surveillance (NCIRS), provide evidence of the impact of changes in vaccination policy over the past fifteen years, 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 2004*

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
1996–2004	0	17	6	0	0	19,200,000

* Sources: Feery B. One hundred years of vaccination. *Public Health Bulletin* 1997;8:61–63; Feery B. Impact of immunisation on disease patterns in Australia. *Medical Journal of Australia* 1981;2:172–176. Deaths recorded for 1966–1975 and 1996–2004 updated with data provided by AIHW Mortality Database.

† Excludes deaths from subacute sclerosing panencephalitis.

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 and territory authorities under their current public health legislation. State and territory notification criteria are based on the National Health and Medical Research Council (NHMRC) surveillance case definitions.[10] However, historically, application of these definitions has differed between jurisdictions, with some using the 1994 NHMRC case definitions as written (e.g. South Australia and Western Australia) and others using their own definitions (e.g. New South Wales and Victoria) (see Appendix 6 for case definitions in use prior to 2004). In September 2003, new national case definitions for notifications reported to NNDSS were endorsed by the Communicable Diseases Network Australia,[11] with nearly all jurisdictions implementing the new definitions in January 2004 (New South Wales commenced August 2004). In Queensland, most notifications are from laboratory-confirmed cases and the notification process is highly automated. There are some minor differences between the laboratory criteria for notification in Queensland and the national case definitions for some diseases (e.g. the criteria for laboratory notification of Hib, pertussis and Q fever). However, the public heath protocols for notification in Queensland require cases to meet the national case definitions for notification. In 2001, invasive pneumococcal disease and laboratory-confirmed influenza became notifiable to the NNDSS. Varicella and herpes zoster were made notifiable in all jurisdictions (except New South Wales) from 2006. Rotavirus infection is not notifiable to NNDSS.

Data extracted from the NNDSS as at 4 June 2006 were examined. Data were checked and cleaned where apparent errors were detected through consultation with appropriate surveillance staff in states and territories. Note that these data are later versions than those used for the 2003 and 2004 Australia's Notifiable Disease Status reports and the AIHW publication *Australia's Health 2006*.[12–14] Disease notification data for cases with a date of diagnosis between 1 January 2003 and 31 December 2005 (three years) are included in this report. Notification data are presented and reported by date of diagnosis. Previous reports analysed notifications by date of onset where date of onset was collected from the clinical history where available, or the specimen collection date for laboratory-confirmed cases. As of mid 2005, a date of diagnosis field was generated for all NNDSS records. Date of diagnosis is completed using an algorithm whereby the earliest date in the fields date of onset, date of specimen, date of notification and date notification received (the only compulsory date field) is selected. This applies for all diseases except hepatitis B unspecified and hepatitis C unspecified (not included in this report) where onset date is not used in calculation of the date of diagnosis. Notifications with onset dates between 1 January 1993 and 31 December 2002 were reported previously.[1–3] Historical notification data included within this report have been updated and are now presented by date of diagnosis. Q fever notifications are reported for the first time in this report.

The variables extracted for analysis for every disease were disease, date of diagnosis, age at onset, sex, and state or territory of residence. For the first time in this report, the fields for laboratory confirmation and vaccination status have been examined where relevant to that disease. This reflects the ongoing improvement in the completion rate of these fields. For a recent analysis of vaccine preventable diseases data by Indigenous status, please refer to the NCIRS report 'Vaccine preventable diseases and vaccination coverage in Aboriginal and Torres Strait Islander people, Australia, 1999 to 2002'.[15] Data from each state and territory were included when calculating rates only when that jurisdiction had been reporting for a complete year (see Appendix 2, *Notifications by state or territory and year*, for the years in which states and 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 chapter 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 three most recent years for which data are available (2002/2003, 2003/2004 and 2004/2005) were examined. Cases with separation dates between 1 July 1993 and 30 June 2002 (9 years) were reported previously.[1–3] This report presents previously analysed historical data for years prior to and including 1997/1998 and updated data for all years from 1998/1999 onwards. Where hospitalisation data is analysed by month, reflecting seasonal trends, data are presented and reported by date of admission. Otherwise, as hospitalisation data for the most recent period (2002/2003–2004/2005) is defined by date of separation, analysis by other variables such as age and sex is grouped by year of separation. Data were extracted based on the International Statistical Classification of Diseases and Related Health Problems, 10th Revision, Australian Modification (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 acute 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 or 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 in the relevant edition for the collection period. In addition, the mode of separation (whether the patient died in hospital) was analysed for hospitalisations 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 0.5% of records in 2002/2003, 2003/2004 and 2004/2005.

Appendix 3 presents hospitalisation data by disease, year and state or territory. All jurisdictions except New South Wales, Queensland and South Australia required data suppression for cells containing less than five hospitalisations. The figures less than five (but non-zero) in this table for the jurisdictions that requested suppression have been replaced with the symbol <5. Calculation of the suppressed figures (by subtraction from totals) has been prevented by the suppression of another figure with less than five in the same disease category but a different year and of another state's data where less than five (for the same row/year). Where there was not another figure in the same row or column that was less than five, a cell has been suppressed using the symbol n.p. (not published) to denote that the number was greater than five. In some cases, this has been achieved by suppressing the total for the disease/year or for the five year total by disease/state. The corresponding hospitalisation rates for suppressions are also designated with the symbol n.p.

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 Diseases, 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 2003 to 2004 (two years). The variables extracted for each death were: underlying cause, age, year death was reported, sex, and state or territory in which death was recorded.

Calculations

All rates were calculated using finalised ABS mid-year estimated resident populations as at June 2006. Rates 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 2002 mid-year population estimate was used to calculate rates for 2002/2003. 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. Where there were small cell sizes for national hospitalisation data (three or less hospitalisations recorded per age/disease category), median length of stay estimates were not published (denoted by n.p.).

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 age 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 or territory, as shown in Appendices 2 and 3. For hospitalisations, some jurisdictions required suppression of hospitalisation data for those cells where there were less than five cases;
- vaccination status of notified cases and laboratory typing, as relevant;
- 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 Medicare Australia (formerly 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.[16] In this report, we have described trends in ACIR vaccination coverage estimates for all vaccines on the current childhood schedule except meningococcal C vaccine, pneumococcal vaccines and varicella vaccine.

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.) The Northern Territory is the jurisdiction with the most different age structure and we have elected to continue using crude rates as per previous reports. It is also important to note that jurisdictions with small populations (e.g. the Australian Capital Territory, Tasmania, the Northern Territory) 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 and over time, with infections diagnosed by a laboratory test more likely to be notified. Data accuracy may also vary between states and territories due to the use of different case definitions for surveillance and varying reporting requirements by medical practitioners, laboratories and hospitals. Under-reporting of notifiable diseases by doctors and from hospitals has been documented in Australia.[17–19] There are eight different Public Health Acts

in operation and no legislative requirement to report to NNDSS although all jurisdictions do so, with daily updates entering the system as of 2004.[20] Data constraints are applied to uploaded fields to ensure validity. This is important given that each jurisdiction has its own reporting system with different fields and coding systems in use. A recent evaluation of NNDSS found that this was a major factor limiting data quality and completeness.[20] Assessing the sensitivity and positive predictive value of the system was beyond the scope of the evaluation. The review noted that the main use of NNDSS for public health action has been in the area of vaccine preventable diseases.

Hospitalisation data

The AIHW publishes regular overviews of Australian hospitalisation statistics, including details of the number of hospitals reporting and any documented data problems. In the periods covered by this report (2002/2003, 2003/2004, 2004/2005), in each financial year there were over 6.6 million, 6.8 million and 7 million separations, respectively.[21–23] Almost all public and private hospitals were included in each of these periods.[21–23] The AIHW performs logical validations on the ICD-10-AM coded data; for example, for sex and age specific diagnoses. Coding audits are also variously performed at hospital level or state and territory level using software such as PICQ (Performance Indicators for Coding Quality) developed by the National Centre for Classification in Health (NCCH).[24]

Some variation in hospital access, admission practices and record coding may occur between regions and over time and impact upon the use of hospitalisation data for monitoring disease trends over time and between jurisdictions. It is likely that the quality of coding in Australia has improved over time due to increasing levels of training amongst coders[25] and the use of coding audits (M Cumerlato, NCCH, personal communication). The National Clinical Coder Workforce Survey of over 1,000 Australian coders in 2002 found that, whilst just over half held tertiary qualifications,10% had no formal coding education. About two thirds of coders reported undertaking regular quality assurance activities in relation to coding.[25]

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 relevant to this report. 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. The NCCH updates the ICD-10-AM every two years, under the guidance of the Australian Coding Standards Advisory Committee.[26,27]

There are also limitations associated with the use of ICD codes to identify cases. Errors that cause the ICD code to differ from the true disease include both random and systematic measurement error and may either occur along the patient pathway (e.g. level of detail documented in medical records, clinician experience) or along the paper trial (e.g. transcribing errors, coder errors such as miss-specification, unbundling (assigning codes for all the separate parts of a diagnosis rather than the overall diagnosis) and upcoding (using reimbursement values to determine the order of coding)).[28] A study of pertussis in children's hospitals in Sydney noted that, whilst variability in clinician diagnostic practices may reduce the sensitivity of pertussis coding, high specificity enables the codes to be useful for surveillance of infant pertussis trends.[19] In the National Clinical Coder Workforce Survey, most Australian coders (77%) nominated incomplete medical record content as the factor most likely to affect coding quality, followed by the principal diagnosis not being identified, complications/co-morbidities not being identified, illegible medical record entries and pressure to maintain coding throughput.[25] In Australia, 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) and for admissions with multiple diagnoses.[29]

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 rare diseases, such as tetanus and acute poliomyelitis, are likely to be due to coding errors. For some diseases, such as *Haemophilus influenzae* type b infection, both the previously used ICD-9-CM and current ICD-10-AM codes lack specificity. This is in contrast to the more stringent case definitions used for notification data. For example, Wood et al recently documented the poor specificity of hospitalisations coded as acute epiglottitis, with most cases on record review found not to be acute epiglottitis and, in the post-vaccination era, none of these admissions due to Hib disease.[30] Thus, care must be taken in ensuring the ICD codes accurately reflect diagnosis of the condition of interest. Generally, codes are most likely to be accurate when the disease has a clear definition with observable signs and symptoms, highly qualified physicians document information on the patient, experienced coders with full access to clinical information assign the codes and the codes are not new.[28]

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, thereby avoiding incomplete data for the latest available year. In recent years, less than 5% of deaths in a particular calendar year are registered in the subsequent year,[31] 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. Information on cause of death are reported routinely for each death on a Standard Medical Certificate of Cause of Death completed by a medical practitioner or coroner. The person completing the certificate must nominate the underlying (principal) cause of death and any associated conditions.[31] The accuracy of the ascertainment of the cause of death may clearly vary according to the experience of the practitioner, the complexity of the disease process and the circumstances of the death. The rate of hospital autopsy has been steadily declining (to approximately 12% in Australia in 2002/2003)[32] and inaccuracy in cause of death certification, compared to the gold standard of autopsy findings, is clearly documented,[33–36] with a recent meta-analysis estimating that around one third of deaths may be misclassified on death certificates.[37] In the case of pertussis and tetanus, studies have documented that deaths due to these diseases, that can be otherwise identified through disease surveillance systems and hospitalisation records, sometimes go unrecorded on death certificates.[38,39] In addition, the number of causes of death recorded by the ABS increased from 187 in 1907 to around 2,850 in 2000 as medical understanding increased.[31] Thus, despite comprehensive mapping algorithms, which attempt to take into account changing disease classification over time, caution is required in interpreting mortality trends.

In processing deaths registered from 1 January 1997, Australia adopted the use of the Automated Coding System (ACS) and introduced ICD-10 codes. As a result, there is now a break in the underlying causes of death series between 1996 and 1997. This is especially important where the death was recorded as hepatitis B. Prior to the use of ICD-10, acute, chronic and unspecified infections could not be differentiated. A large artefactual rise in deaths coded as due to pneumonia in 1997–1998 has also been ascribed to changes in coding practices during this period.[40]

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.[5,41] Another limitation of ACIR data is that records are only held for children up to seven years of age. Coverage is calculated only for children registered on Medicare; however, by the age of 12 months, it is estimated that over 99% of Australian children have been registered with Medicare.[16,41,42]

3. Vaccine preventable diseases

Diphtheria

Diphtheria is an acute toxin-mediated systemic disease caused by the bacterium *Corynebacterium diphtheriae*. Infection remains localised to the throat or skin but disease is mainly due to local inflammation and/or systemic toxaemia. Pharyngeal diphtheria presents with 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.[43,44] Non-toxigenic *C. diphtheriae* usually causes mild throat or skin infection, which is occasionally complicated by invasive disease including endocarditis or arthritis. *Corynebacterium ulcerans*, a bacterium found in cattle and more recently in cats, can also express diphtheria toxin and cause a zoonotic infection in humans that is similar to diphtheria.[45,46]

Case definitions

See Appendix 6 for pre-2004 definition

National definition from January 2004:[11]

Isolation of toxigenic *Corynebacterium diphtheriae* or toxigenic *C. ulcerans* (confirmed case)

OR

Isolation of *Corynebacterium diphtheriae* or *C. ulcerans* (toxin production unknown) and one of the following presentations as clinical evidence:

- pharyngitis and/or laryngitis (with or without membrane);or
- toxic (cardiac or neurological) symptoms (probable case);or
- clinical evidence as above and an epidemiological link to a confirmed case (probable case).

Hospitalisations

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

Deaths

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

Notifications, hospitalisations and deaths

There were no notifications of diphtheria during January 2003 to December 2005 and no deaths due to diphtheria in 2003–2004. For the three year period 2002/2003 to 2004/2005, there were 66 hospitalisations coded as diphtheria. Of these, most were cutaneous (A36.3; n=39), with the remainder coded as other (A36.8; n=16) or unspecified (A36.9; n=11) diphtheria. There were no hospitalisations coded as pharyngeal or laryngeal diphtheria. Of the hospitalisations, unspecified diphtheria was given as the principal diagnosis in three cases. Although numbers are low, the group with the most admissions coded as diphtheria were males aged 25–29 years (n=12).

Comment

Diphtheria has become rare in Australia. A cutaneous toxigenic case acquired in East Timor and notified in 2001 was the first case notified since 1993. Culture positive cutaneous and throat infections with non-toxigenic *C. diphtheriae* are endemic in the Northern Territory,[47] but these are not classified as diphtheria in the absence of relevant symptoms.

From 2004, all toxigenic isolates, including those from cutaneous cases, became notifiable. This report includes, for the first time, all ICD codes for diphtheria in hospitalisation data (previously restricted to codes for pharyngeal or nasopharyngeal diphtheria and laryngeal diphtheria in order to be consistent with notification data). In the absence of any notifications during the period 2003–2005, the 66 hospitalisations (three principal diagnosis) were presumably non-toxigenic or culture negative suspected diphtheria cases or coding errors.

Diphtheria is still a global problem with 21 countries in Asia, South America, Africa and Europe reporting 10 or more cases of diphtheria to the World Health Organization (WHO) in 2005,[48] with a total of 8,229 cases reported globally in 2005. In 2002, WHO estimated that there were 5,000 deaths due to diphtheria. The large outbreak of diphtheria in the Newly Independent States (NIS) of the former Soviet Union in the 1990s[49] has been gradually brought under control (from a peak of 50,425 cases in 1995 to 176 cases in the European Region in 2004). Four key strategies have been identified to maintain diphtheria control in the region: (1) ensuring high population immunity; (2) strengthened surveillance; (3) early diagnosis and high quality case management; and (4) rapid investigation and management of close contacts.[50] The NIS outbreak underscores the risk of diphtheria returning when high vaccination coverage in children (who are critical vectors of respiratory transmission) is not maintained.

In countries with high childhood vaccination coverage against diphtheria, such as Australia, the United Kingdom, Germany, the USA and Canada, cutaneous lesions are the most likely manifestation of *C. diphtheriae* infection. Cutaneous infection may be caused by local circulating non-toxigenic strains[51] (which can also cause invasive disease, including bacteraemia, endocarditis and arthritis, particularly in persons with risk factors such as homelessness, alcoholism, or diabetes)[52] or by imported toxigenic types due to overseas travel.[53,54] Cutaneous *C. diphtheriae* infection (due to toxigenic or non-toxigenic strains) may be difficult to diagnose due to a low index of suspicion, may cause chronic infection and may serve as a reservoir for ongoing transmission with greater efficiency than respiratory infection.[51,53] Unfortunately, the frequency of international travel now means that even in countries such as Australia, where diphtheria is rare, exposure to a toxigenic strain may occur, with potentially fatal consequences in unvaccinated individuals, or in those whose vaccine induced immunity has waned.[55,56] Waning immunity may be more of a problem in the small percentage of children of mothers born in countries overseas where cutaneous diphtheria is common, resulting in mothers having more circulating antibody, which, in turn, can reduce infant immune responses to vaccination through greater placental antibody transfer.[57] Australian serosurveillance data indicate that, whilst childhood protection is excellent (>99%), waning immunity in adults has resulted in a susceptible population.[58] Australians travelling to countries where diphtheria remains a problem should ensure that they are protected against diphtheria through booster immunisation as necessary.

Haemophilus influenzae type b (Hib) disease

Haemophilus influenzae is a Gram-negative bacterium which occurs in both encapsulated and unencapsulated forms. It is a commensal of the nasopharynx, especially in young children. Before Hib vaccines became available, one encapsulated serotype, type b (Hib), caused at least 95% of invasive disease due to *H. influenzae* in children.[59,60] 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.[59,60] Aboriginal and Torres Strait Islander children had a particularly elevated risk of Hib meningitis, with rates among the highest recorded anywhere in the world, but rarely developed epiglottitis.[61] Survivors of Hib meningitis commonly had neurological sequelae such as deafness and intellectual impairment.[59,60] Epiglottitis was the other major category of infection, most often occurring in children over the age of 18 months. Other manifestations of Hib disease include cellulitis, septic arthritis, pneumonia, pericarditis, osteomyelitis and septicaemia.

Case definitions

See Appendix 6 for pre-2004 definition

National definition from January 2004:[11]

a) Isolation of *Haemophilus influenzae* type b (Hib) from a normally sterile site where typing has been confirmed at an approved reference laboratory; *or*

b) Detection of Hib antigen in cerebrospinal fluid when other laboratory parameters are consistent with meningitis.

Hospitalisations and deaths

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

Secular trends

During the three years from January 2003 to December 2005, a total of 51 invasive Hib infections were notified. The average annual notification rate was 0.08 per 100,000 population (Table 3). A median of one case (range 0–5) was notified per month (Figure 1). There were 57 hospitalisations (average annual rate 0.10 per 100,000) recorded as *Haemophilus* meningitis, with a median of two cases (range 0–4) hospitalised per month.

Severe morbidity and mortality

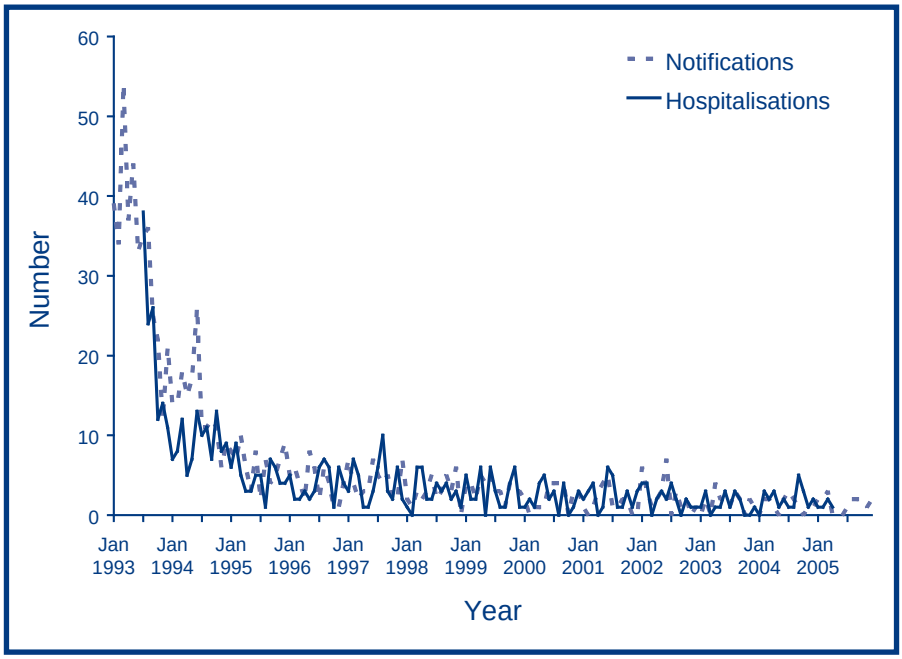
Overall ,within each age group, there were similar numbers of hospitalisations with *H. influenzae* meningitis and notifications of invasive Hib disease recorded (Table 3). Over the review period, a total of 780 hospital bed days (average 260 days per year) was recorded for patients with *Haemophilus* meningitis. The median length of stay for hospitalisations with a principal diagnosis of *Haemophilus* meningitis was 11 days.

In the two years 2003 to 2004, *H. influenzae* meningitis was certified as the underlying cause of death in one person (who was aged over 85 years) (Table 3). There were two deaths in notified cases reported to NNDSS between 2003 and 2005 (both in 2003). One case was in an infant and the other in a man aged 60–69 years.

Age and sex

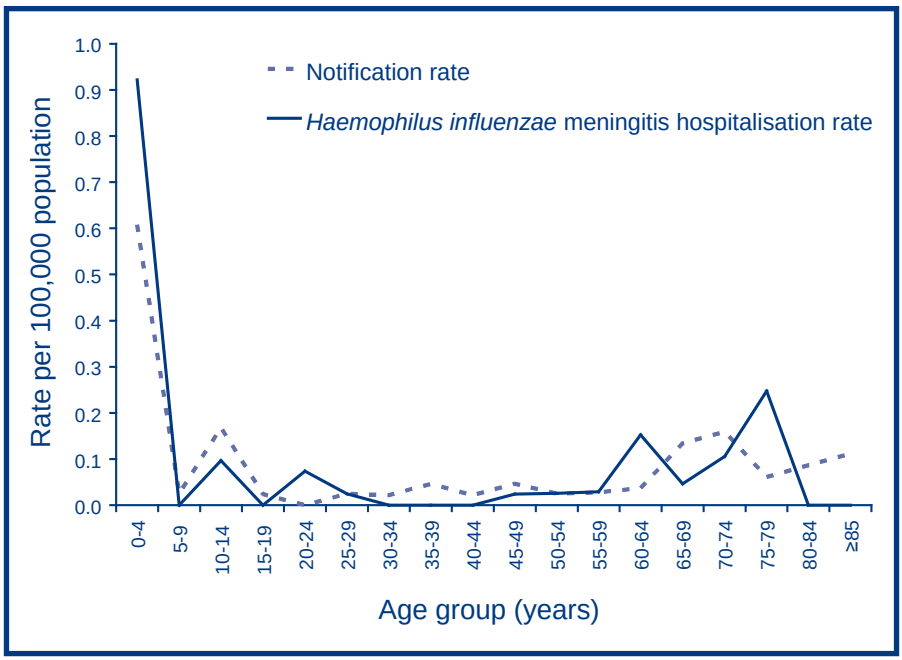
Hospitalisations for presumed Hib disease and notifications were higher in males than in females, with a male:female ratio of 1.21:1 and 1.33:1, respectively. In children aged 0–4 years, there were 23 *H. influenzae* meningitis cases. Overall, children aged 0–4 years accounted for 45% (23/51) of all notifications, 61% (35/57) of all presumed meningitis hospitalisations but no deaths (Table 3). The age-specific notification rate closely matched the age-specific *H. influenzae* meningitis hospitalisation rate (Figure 2).

Figure 1. *Haemophilus influenzae* type b notifications and *Haemophilus* meningitis hospitalisations for all ages, Australia, 1993 to 2005,* by month of diagnosis or admission



* Notifications where the month of diagnosis was between January 1993 and December 2005; hospitalisations where the month of admission was between 1 July 1993 and 30 June 2005.

Figure 2. *Haemophilus influenzae* type b notification and *Haemophilus* meningitis hospitalisation rates, Australia, 2003 to 2005,* by age at admission



* Notifications where the month of diagnosis was between January 2003 and December 2005; hospitalisations where the month of admission was between 1 July 2003 and 30 June 2005.

Table 3. *Haemophilus influenzae* type b (Hib) notifications, Hib meningitis hospitalisations and deaths, Australia, 2002 to 2005,* by age group

Age group (years)	Notifications 3 years (2003–2005)		Hospitalisations 3 years (July 2002–June 2005)				LOS† per admission (days)	Deaths 2 years (2003–2004)	
	n	Rate‡	n	n(§)	Rate‡	Rate‡(§)	Median(§)	n	Rate‡
0–4	23	0.61	35	26	0.92	0.68	10.0 (10.0)	0	0.00
5–14	8	0.10	4	4	0.05	0.05	8.5 (8.5)	0	0.00
15–24	1	0.01	3	1	0.04	0.01	n.p.	0	0.00
25–59	9	0.03	4	3	0.01	0.01	16.5 (19.0)	0	0.00
60+	10	0.09	11	9	0.11	0.09	15.0 (15.0)	1	0.01
All ages	51	0.08	57	43	0.10	0.07	11.0 (11.0)	1	0.003

* Notifications where the month of diagnosis was between January 2003 and December 2005; hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005; deaths where the death was recorded in 2003 or 2004.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

n.p. Not published due to small cell sizes.

Since 1993, all measures of invasive Hib disease in children aged 0–4 years, who had the highest disease incidence prior to the introduction of vaccination (and are the most highly immunised), have progressively fallen, though less steeply in recent years (Figure 3). In this age group, the average number of annual notified cases has decreased from approximately 27 in the late 1990s to approximately 10 between 2000 and 2002 to approximately 7 between 2003 and 2005. Six deaths were recorded in this age group in 1993 but none since 1999.

Vaccination status

Completion of the vaccination status field was expected for all notifications of Hib born after 31/12/1987 in NNDSS during 2003–2005. Overall, 94% of 31 cases had this field completed for this period. Of 20 cases classified as fully (n=10) or partially vaccinated (n=5), vaccination was validated from the Australian Childhood Immunisation Register or written records in 12/20 (60%). Among 14 cases 1–7 years of age notified between 2003 and 2005, nine (64%) were not vaccinated.

Geographical variation

As in previous years, there was little variation in notification and hospitalisation rates between the states and territories, except for the Northern Territory, where notification rates were substantially higher than other jurisdictions, but the absolute number of cases was small (Appendices 2 and 3).

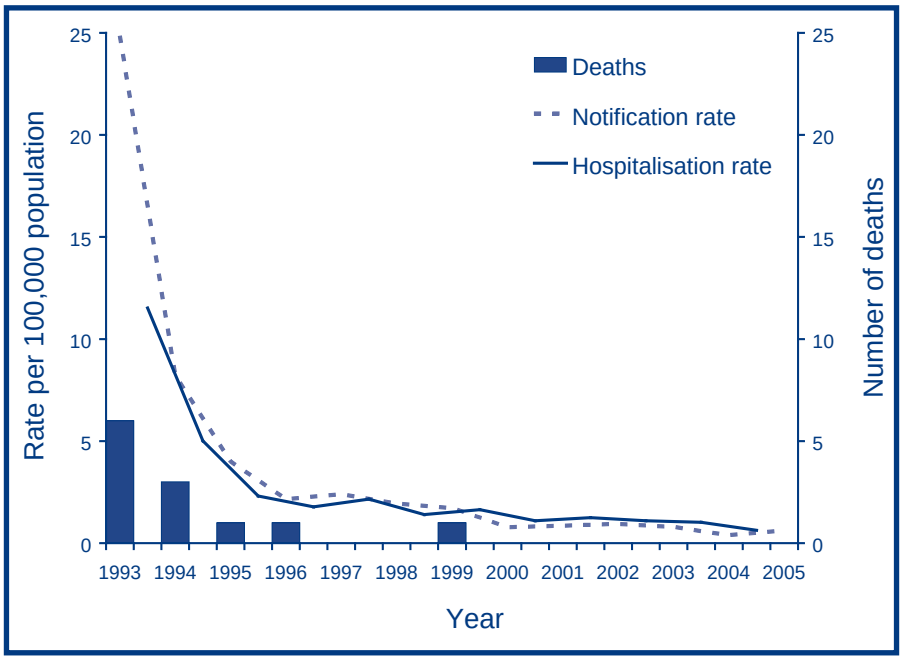
Comment

This report is the first in the series of NCIRS vaccine preventable diseases reports to exclude hospitalisations recorded as epiglottitis from presentation as a measure of Hib disease. This is because a review of hospitalisations coded as epiglottitis in Sydney from 1998 to 2000 showed none of these hospitalisations had Hib isolated from a sterile site, with one due to *Streptococcus pneumoniae* and a substantial proportion (32%) of incorrect coding.[30] Hospitalisation data now includes meningitis only, although as serotype-specific hospitalisation data are still not available, these cases, even when *Haemophilus* meningitis is the primary diagnosis, could be due to other serotypes.

In 2000, a new Hib immunisation schedule began in Australia with all children receiving PRP-OMP vaccine at 2 and 4 months of age, with a booster at 12 months of age. In this review period, vaccination status data are available for the first time and indicate that, consistent with the very high immunisation coverage reported for Hib vaccine, approaching 95%, the majority of confirmed Hib cases are occurring in unimmunised children. This is consistent with high vaccine effectiveness but also indicates that unimmunised children remain at risk of severe disease despite population herd immunity. Further, there is no evidence of any increase in Hib cases in older age groups, although the first cohort of children eligible to receive Hib vaccine are now approaching 20 years of age. Incidence of Hib may not decrease much

more than the very low incidence now reached, as it is consistent with the lowest disease rates reported internationally,[62,63] and in contrast to the increase in Hib cases seen in the United Kingdom where three doses of Hib vaccine were scheduled at 2, 3 and 4 months of age with no later booster.[64] The rarity of invasive Hib disease emphasises the importance of laboratory confirmation of all suspected cases, ideally by typing with polymerase chain reaction (PCR) in a reference laboratory.

Figure 3. *Haemophilus influenzae* type b notification and 'presumed invasive *Haemophilus influenzae* type b' hospitalisation rates and numbers of deaths* for children aged 0–4 years, Australia, 1993 to 2005†



* Hospitalisation and deaths include those for *Haemophilus* meningitis for the period up to 30 June 2005 (hospitalisations) and 31 December 2004 (deaths).

† Notifications where the month of diagnosis was between January 1993 and December 2005; hospitalisations where the month of separation was between 1 July 1993 and 30 June 2005; deaths where the death was recorded between January 1993 and December 2004.

Hepatitis A

Acute infection with the hepatitis A virus (HAV), a picornavirus, presents a clinical spectrum from malaise and diarrhoea to acute hepatitis with jaundice to fulminant liver failure. Onset of symptoms is usually abrupt with fever, anorexia, malaise, nausea and abdominal discomfort followed by jaundice and dark urine.[44] The single most important factor in determining the clinical presentation and outcome of HAV infection is age. Whilst only 10% to 50% of infections acquired before the age of 5 years are symptomatic, 70% to 95% of infected adults will show symptoms.[43]

Case definitions

See Appendix 6 for pre-2004 definition

National definition from January 2004:[11]

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

or

b) Detection of hepatitis A virus by nucleic acid testing;

or

c) Clinical hepatitis (jaundice and/or bilirubin in urine) without a non-infectious cause *and* an epidemiological link to a laboratory-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 1,075 hepatitis A notifications in the period January 2003 to December 2005 (average annual notification rate 1.8 per 100,000) (Table 4). A median of 27 cases (range 18–56) were notified per month. There were 755 hospitalisations (average annual hospitalisation rate 1.3 per 100,000) with a median of 20 admissions (range 10–36) per month.

Notification and hospitalisation rates continued to decline from 2003 to 2005 (Figure 4).These were the lowest levels recorded since national data have been collated on NNDSS from 1991 and hospitalisations from 1993, following large point-source and community epidemics in the 1990s.

Severe morbidity and mortality

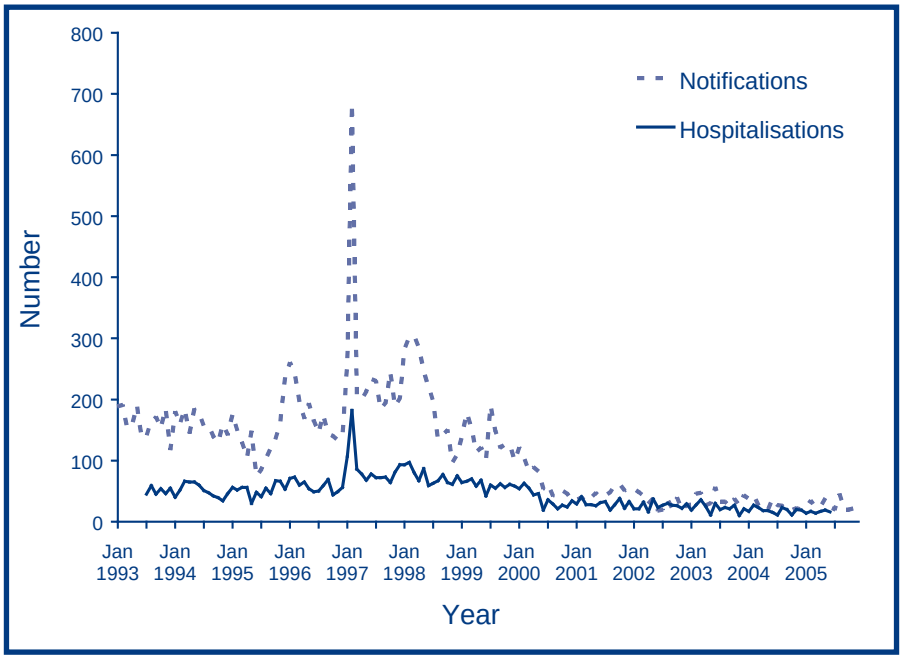
In the review period July 2002 to June 2005, there were 4,557 hospital bed days (average 1,519 per year) recorded for patients with an ICD-10-AM code for hepatitis A. Hepatitis A was the principal diagnosis in 49% of hospitalisations where hepatitis A was recorded (371 cases, average annual rate 0.6 per 100,000). This proportion was highest in those aged 0–4 years (88%), decreasing with increasing age to 24% in those aged 60 years and over. In this age group, more than 50% of hospitalisations had a principal diagnosis that was unlikely to be associated with hepatitis A infection, such as cancer, respiratory, ocular, or cardiac conditions or fractures. The median length of stay was longer for those aged 60 years and over than for younger age groups (Table 4). Hepatitis A with hepatic coma (ICD-10-AM B15.0) was recorded for six hospital admissions, one aged less than five years.

In 2003 to 2004, hepatitis A was recorded as the underlying cause of four deaths, all in people aged over 70 years. One of the cases notified to NNDSS between 2003 and 2005 was reported to have died from hepatitis A.

Age and sex distribution

The overall male to female ratio was 1.4:1 for notifications and 1.2:1 for hospitalisations. Three of four reported deaths were in females. The sex ratio was highest in those aged less than five years (notifications 2.5, hospitalisations 2.3) and lowest in those aged five to 24 years (1.2 and 0.9, respectively).

Figure 4. Hepatitis A notifications and hospitalisations, Australia, 1993 to 2005,* by month of diagnosis or admission



* Notifications where the month of diagnosis was between January 1993 and December 2005; hospitalisations where the month of admission was between 1 July 1993 and 30 June 2005

Table 4. Hepatitis A notifications, hospitalisations and deaths, Australia, 2002 to 2005,* by age group

Age group (years)	Notifications 3 years (2003–2005)		Hospitalisations 3 years (July 2002–June 2005)				LOS† per admission (days)	Deaths 2 years (2003–2004)	
	n	Rate‡	n	(§)	Rate‡	(§)	Median (§)	n	Rate‡
0–4	94	2.5	27	(24)	0.7	(0.6)	3.0 (3.5)	0	0
5–14	222	2.7	47	(43)	0.6	(0.5)	2.0 (2.0)	0	0
15–24	191	2.3	90	(60)	1.1	(0.7)	3.0 (3.0)	0	0
25–59	475	1.6	421	(203)	1.4	(0.7)	3.0 (3.0)	0	0
60+	93	0.9	170	(41)	1.7	(0.4)	6.0 (6.0)	4	0.06
All ages	1,075	1.8	755	(371)	1.3	(0.6)	3.0 (3.0)	4	0.01

* Notifications where the month of diagnosis was between January 2003 and December 2005; hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005; deaths where the death was recorded in 2003 or 2004.

† LOS = length of stay in hospital.

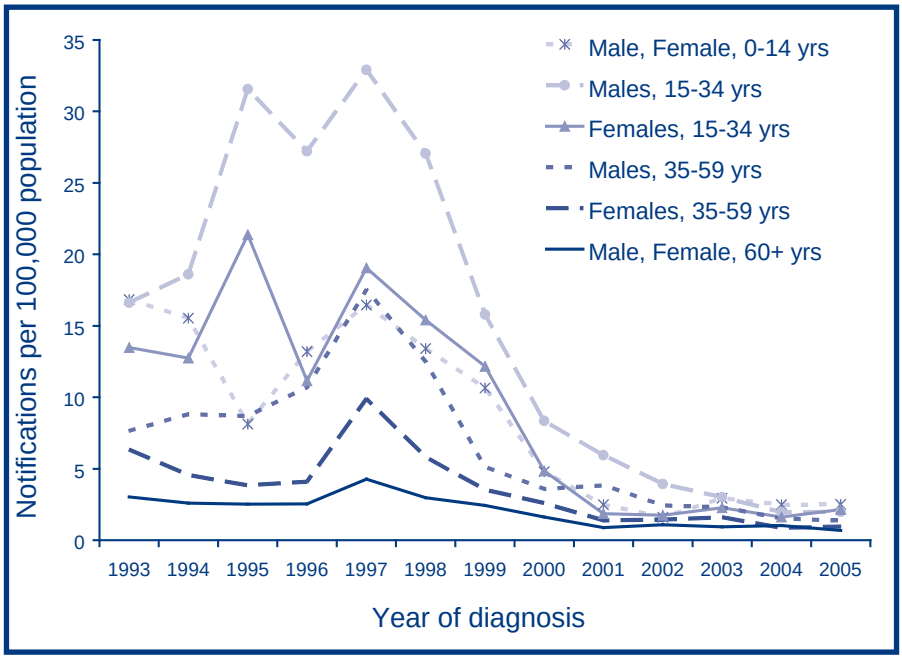
‡ Average annual age-specific rate per 100,000 population.

§ Principal diagnosis.

Notification and hospitalisation rates for all age and sex groups continued to fall compared with previous years (Figures 5 and 6). The previously high rates in males aged 15–34 years declined so that from 2003 to 2005 the highest notification rate occurred among males and females aged 0–14 years (average annual rate, 2.7 per 100,000), while the highest hospitalisation rates occurred in two age groups: males aged 34–59 years and males and females aged 60 years and over (average annual rates of 1.7 per 100,000).

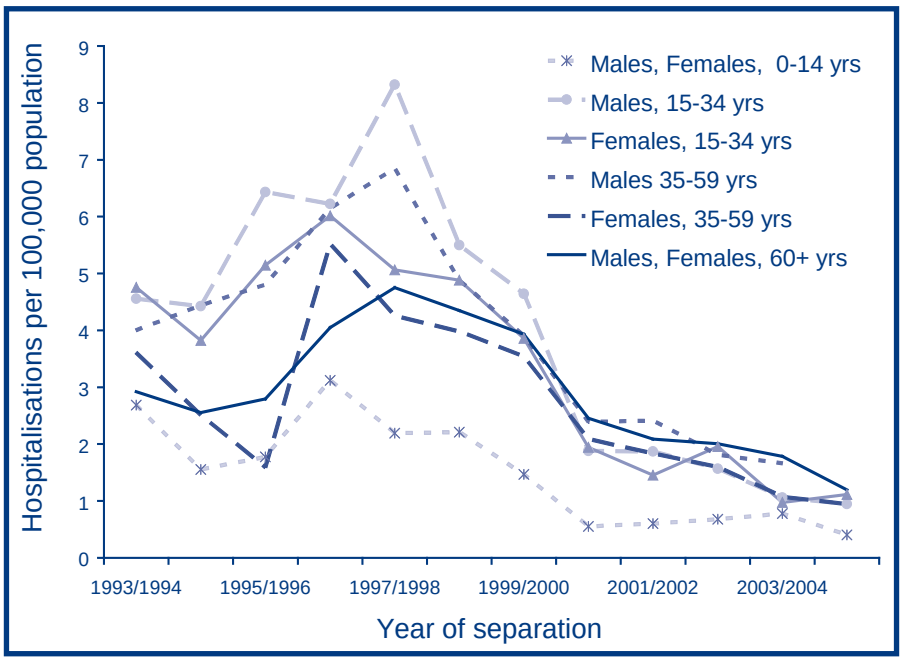
Notification rates tended to decrease with increasing age, while the reverse was true for hospitalisation rates. The 5–14 year age group had the highest notification rate and the lowest hospitalisation rate. Those aged 60 years and over had the lowest notification rate and the highest hospitalisation rate, and there were nearly twice as many reported hospitalisations with hepatitis A than there were notified cases in that age group.

Figure 5. Hepatitis A notification rates, Australia, 1993 to 2005,* by age group, sex and year of diagnosis



* Notifications where the month of diagnosis was between January 1993 and December 2005.

Figure 6. Hepatitis A hospitalisation rates, Australia, 1993 to 2005,* by age group, sex and year of separation



* Hospitalisations where the month of separation was between 1 July 1993 and 30 June 2005.

Geographical distribution

The highest rates were reported from the Northern Territory (average annual rates 20 per 100,000 for notifications and 8 per 100,000 for hospitalisations, Appendices 2 and 3). The jurisdiction with the next highest rates was Western Australia with 3.5 for notifications and 1.5 for hospitalisations, and there was comparatively little difference between the other jurisdictions, which ranged from 0.7 to 1.7 for notifications and 0.7 to 1.4 for hospitalisations (Appendices 2 and 3). There were no clear trends over time in any jurisdiction in the last three years of data.

Vaccination status

Vaccination status was reported for 244 (23%) of the 1,075 notifications on NNDSS from 2003 to 2005. Three of those were recorded as fully vaccinated, all adults, and there was no information on whether these were verified by written records or what the length of time was between vaccination and disease.

Comment

In Australia, as in other industrialised countries, hepatitis A occurs sporadically with periodic epidemic peaks related to point-source and community-wide outbreaks. The overall patterns are evident in hepatitis A notification and hospitalisation rates over the 13 years 1993–2005. Peaks in total hepatitis cases during the 1990s were due to a large outbreak associated with consumption of contaminated oysters in February 1997[65] and large community-wide epidemics mainly among men who have sex with men and injecting drug users.[66–69] The decline following these large outbreaks may have been due to a combination of a reduction in the number of people in high-risk groups who were susceptible to hepatitis A virus infection, and the promotion by local health authorities of vaccination and improved hygiene in target groups.[66] Notification and hospitalisation rates remained low during the last three years, with minor peaks associated with two outbreaks in Alice Springs – a food-borne outbreak in 2003 at an interstate gathering,[70] and a community outbreak in 2005 for which no source was identified.[71]

Infection in the elderly has been reported as much more likely to lead to hospitalisation with hepatitis A as the principal cause, to involve complications or to result in death, compared with that in younger adults.[72,73] However, the low proportion of hospitalisations in the elderly with hepatitis A as the principal diagnosis (24%) in these data suggests caution in interpreting hospitalisations with hepatitis A as a contributing cause in this age group. The majority of hospitalisations in this age group had principal causes unlikely to be related to hepatitis A, and, therefore, may reflect the high prevalence of co-morbidities in the elderly and incidental hepatitis A infection. A recent report from the USA of false positive IgM anti-hepatitis A virus tests in those without clinical hepatitis found this to be particularly common in the elderly.[74] The contrastingly high proportion with a principal diagnosis in young children (88%) emphasises that hepatitis A infection, although regarded as usually asymptomatic in young children, can require hospitalisation. The total number of hospitalisations recorded here was 70% of the total notifications for a similar period. A US study has estimated that 13% of cases resulted in hospitalisation,[72] suggesting substantial under-notification of hepatitis A in Australia over this period.

The most commonly reported risk exposures for notified hepatitis A cases in recent years have been travel to countries where hepatitis A is endemic, household contact with a case and attendance at child care centres.[12,13,75] Others at higher risk include sewage workers, men who have sex with men, injecting drug users and Indigenous Australians.[76]

The epidemiology of hepatitis A differs significantly for the Indigenous population, where it has been endemic. Among non-Indigenous Australians, as in other developed countries, adolescents and young adults have a lower seroprevalence than older adults.[68] In contrast, hospitalisation and notification rates are higher among Indigenous Australians, with rates in Indigenous children aged less than five years over twenty times as high as those of non-Indigenous children in the same age group.[77] The rate of hospitalisation is likely to be underestimated due to the known under-identification of Indigenous people in the hospitalisation data. This greater disease burden in Indigenous children has been particularly pronounced in more remote areas.[67,78,79] During 1999–2002, there were three deaths due to hepatitis A among children aged less than five years; all were Indigenous.[67,77]

Hepatitis A vaccines are effective in preventing disease in individuals[76] and in controlling outbreaks in some settings.[76,80] In Australia, vaccination is recommended for selected at-risk groups and occupations.[76] 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.[81] This was expanded in 2005 to include all Indigenous children aged 12 to 24 months in the Northern Territory, Queensland, South Australia and Western Australia.[82] In the United States, hepatitis A cases decreased substantially following the recommendation of vaccination of children in communities with high rates of disease in 1996, and for states and counties with high hepatitis A notification rates in 1999. In 2006, this was expanded to include all US infants, as part of a staged implementation of progressively expanded vaccination.[83] Continued monitoring should be a priority in Australia, both to assess the impact of these recent changes, and the need for any further expansion of vaccination.

Hepatitis B

The focus of this chapter is acute infection with hepatitis B virus (HBV), a hepadnavirus. It produces a range of conditions from subclinical infection to acute and, rarely, fulminant hepatitis. The majority of HBV infections are not clinically recognised, with less than 10% of children and 30%–50% of adults experiencing jaundice.[44,84] When illness occurs, it 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% of infants infected at birth, 20%–50% of children infected at 1–5 years of age, and about 1%–10% of persons infected as older children and adults.[44] Of people chronically infected with HBV, 15%–40% develop cirrhosis of the liver and/or hepatocellular carcinoma.[85,86]

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.[44] Major modes of transmission include sexual or close household contact with an infected person, perinatal transmission from mother to infant, injecting drug use and nosocomial exposure.[44] 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.[85,87–89]

Case definitions

See Appendix 6 for pre-2004 definition

National definition* for newly acquired hepatitis B from January 2004:[11]

a) Detection of hepatitis B surface antigen (HBsAg) in a patient shown to be negative within the last 24 months; *or*

b) Detection of HBsAg and IgM to hepatitis B core antigen, in the absence of prior evidence of hepatitis B virus infection; *or*

c) Detection of hepatitis B virus by nucleic acid testing, and IgM to hepatitis B core antigen, in the absence of prior evidence of hepatitis B virus infection.

* Queensland implemented a consistent but less comprehensive definition for laboratory notification in December 2005 for 'Hepatitis B (acute)': HBsAg positive AND Anti-HBc IgM positive. However the public heath protocol for notification in Queensland accepts cases meeting the broader national case definitions for notification.

Hospitalisations

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

As in the previous reports, hospitalisations were included only where the relevant ICD code was the principal diagnosis.

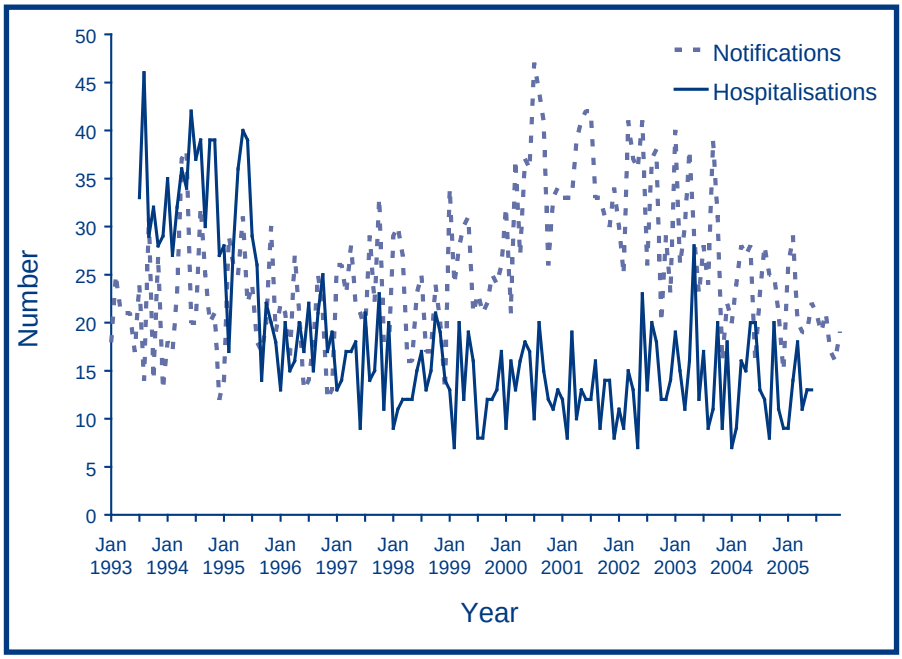
Deaths

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

Secular trends

In the three years from January 2003 to December 2005, there were 874 notifications (average annual rate 1.5 per 100,000) with a median of 23.5 notifications per month (range 15–40) (Figure 7, Table 5). The peak notification rate was in the 20–29 year age group (average annual rate 3.9 per 100,000). From 2002/2003 to 2004/2005, acute hepatitis B was the principal diagnosis in 35% of all hospitalisations with acute hepatitis B. There were 517 hospitalisations with a principal diagnosis of acute hepatitis B (average annual rate 0.9 per 100,000) with a median of 13 hospitalisations per month (range 7–28). Nearly all (97% (507/521)) of these hospitalisations were coded as 'acute hepatitis B without delta-agent and without hepatic coma' (ICD-10-AM B16.9). The national notification rate had an upward trend between 1997 and 2001, peaked in 2001 at 2.2 per 100,000 and is now in continuing decline from 1.7 per 100,000 in 2003, to 1.4 per 100,000 in 2004 and down to 1.2 per 100,000 in 2005 (Appendix 2). Hospitalisations have generally declined since 1993/1994 and, since 1999/2000, seem to have stabilised at a national hospitalisation rate of 0.8 per 100,000 (Appendix 3).

Figure 7. Acute hepatitis B notifications, and hospitalisations with a principal diagnosis of acute hepatitis B,* Australia, 1993 to 2005,† by month of diagnosis 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 diagnosis was between January 1993 and December 2005; hospitalisations where the month of admission was between 1 July 1993 and 30 June 2005. 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.

Table 5. Acute hepatitis B notifications, hospitalisations and deaths, Australia, 2002 to 2005,* by age group

Age group (years)	Notifications 3 years (2003–2005)		Hospitalisations† 3 years (July 2002–June 2005)		LOS‡ per admission (days)	Deaths 2 years (2003–2004)	
	n	Rate§	n	Rate§	Median	n	Rate§
0–4	6	0.2	0	0	0	0	0
5–14	16	0.2	12	0.2	1.5	0	0
15–24	229	2.8	98	1.2	3.0	0	0
25–59	586	2.0	360	1.2	3.0	8	0.04
60+	37	0.4	47	0.5	7.0	14	0.2
All ages	874	1.5	517	0.9	3.0	22	0.06

* Notifications where the month of diagnosis was between January 2003 and December 2005; hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005; deaths where the death was recorded in 2003 or 2004.

† 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.

Severe morbidity and mortality

For patients with a principal diagnosis of acute hepatitis B, 2,408 hospital bed days (914, 829 and 665 bed days in 2002/2003, 2003/2004 and 2004/2005, respectively) were recorded. The median length of stay was three days, with longer stays for adults aged 60 years and over (Table 5). There were 22 deaths from acute hepatitis B recorded in the two years 2003 to 2004, 17 in males and five in females. All of the deaths occurred in those aged 25 years and over, and nearly two thirds (14/22) were aged over 60 years, in whom

nine of the fourteen were males. In 2003, there were twice as many deaths (15/22) as in 2004 (7/22). There were four cases of hepatic coma recorded among hospitalisations with a principal diagnosis of acute hepatitis B, with none of these cases recorded as having delta co-infection (Table 6). There were three deaths reported to NNDSS in notified cases between 2003 and 2005.

Table 6. Hepatic coma* in hospitalised cases with principal diagnosis of acute hepatitis B

Age group (years)	Hepatic coma†	
	n	Hospitalisations (%)
0–4	0	0
5–14	0	0
15–24	0	0
25–59	2	0.6
60+	2	4.3
All ages	4	0.8

* Measured using National Hospital Morbidity data where the month of hospital separation was between 1 July 2002 and 30 June 2005.

† ICD-10-AM codes B16.0 and B16.2.

Age and sex distribution

Historically, notification rates have consistently been highest in young adults aged 15–19 years, 20–24 years and 25–29 years (Figure 8). Since 2001, notification rates in these three age groups have declined, particularly in 15–19 year olds, where for the first time in 2005 rates fell below 1 per 100,000 to 0.8 per 100,000. Rates have remained fairly stable in the other age groups from 1993 to 2005. As in previous years, there were more male than female notifications in almost all age groups in 2003, 2004 and 2005, with an overall male:female ratio of 1.7:1.

During the period 2002/2003 to 2004/2005, rates for hospitalisations with a principal diagnosis of acute hepatitis B were highest in adults aged 25–29 years (1.9 per 100,000) and 20–24 years (1.8 per 100,000) (Figure 9). Like notifications, and as in previous years, hospitalisations occurred predominantly in males with an overall male:female ratio of 1.8:1.

Geographical distribution

During the period 2003–2005, Victoria recorded the highest number of notifications (n=340; 39%), followed by New South Wales (n = 185; 21%). The Northern Territory had the highest average annual notification rate at 4.7 per 100,000. Victoria and Tasmania were next at 2.3 and 2.1 per 100,000, respectively, while rates were 1.8 per 100,000 or less in the other jurisdictions (Appendix 2). Rates in jurisdictions other than New South Wales, Queensland, South Australia, and Western Australia, declined between 2004 and 2005. (Appendix 2)

For the same period, Victoria also had the highest number of hospitalisations (n = 198; 198/517, 38%) followed by New South Wales (n = 148; 148/517, 29%).

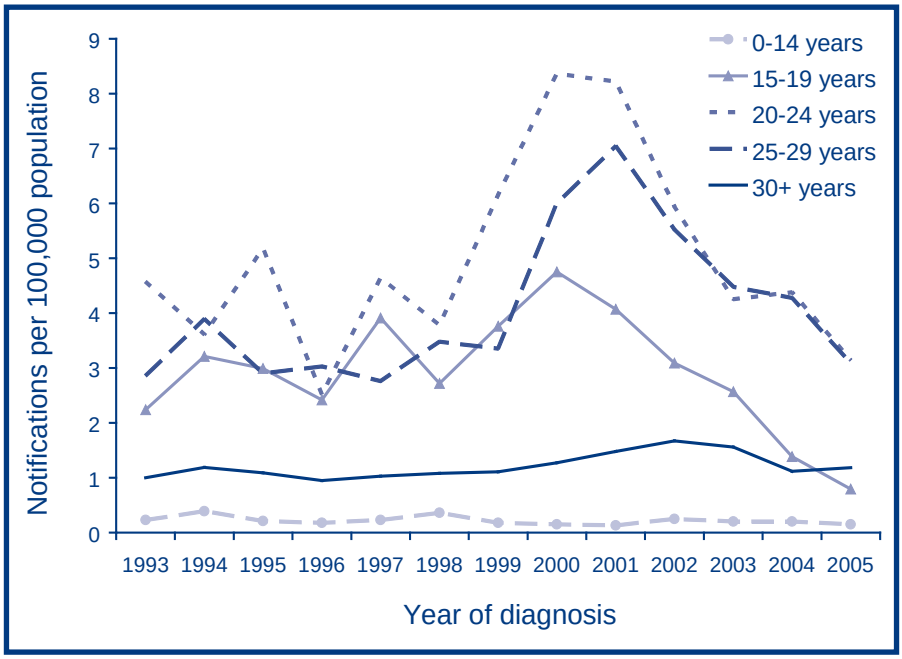
Vaccination status

There were six cases of acute hepatitis B in children aged 0–4 years notified between 2003 and 2005 and their vaccination status was recorded as "unknown" on NNDSS.

Comment

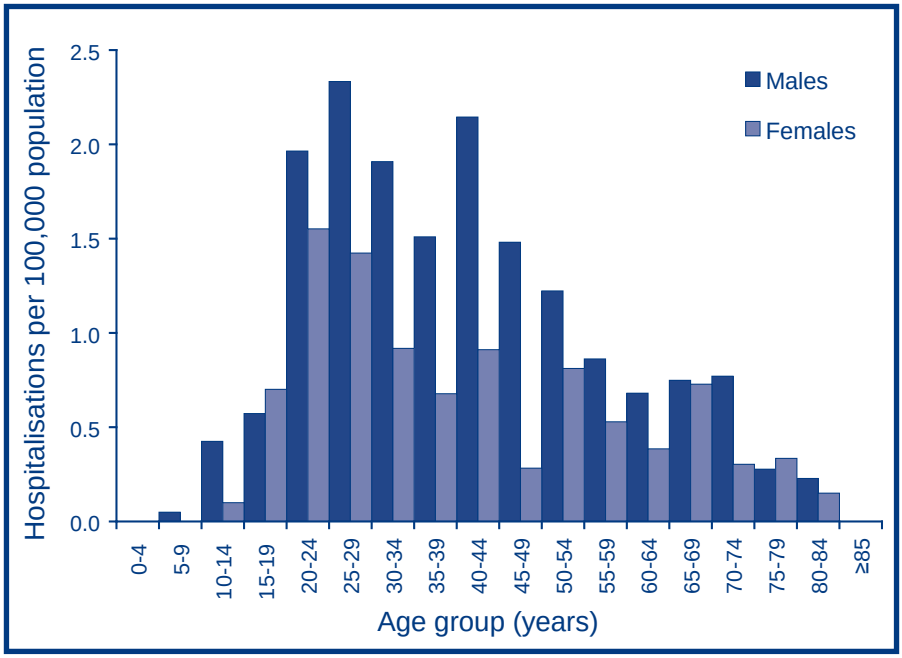
The national notification rate for newly acquired hepatitis B appears to have peaked in 2001 at 2.2 per 100,000, mirrored by corresponding peaks in incidence in the 15–24 year old age groups. The decrease in the national acute hepatitis B notification rate observed between 2003 and 2005 is largely confined to young adults aged 15–24 years, and, in particular, young adults aged 15–19 years. Notification rates in the latter group have fallen by 75%, from 3.1 per 100,000 in 2002 to 0.8 per 100,000 in 2005, while rates in 20–24 year olds have almost halved from 5.9 per 100,000 in 2002 to 3.1 per 100,000 in 2005. Rates

Figure 8. Acute hepatitis B notification rates, Australia, 1993 to 2005,* by age group



* Notifications where the month of diagnosis was between January 1993 and December 2005.

Figure 9. Acute hepatitis B hospitalisation rates, Australia, 2002/2003 to 2004/2005,* by age group and sex



* Hospitalisations where the principal diagnosis was acute hepatitis B and the month of separation was between 1 July 2002 and 30 June 2005.

of unspecified hepatitis B notifications[a] in 2000–2004 have also fallen by nearly 50% in the 15–19 year old age group.[12] This is important as this age group are not likely to meet NNDSS incident hepatitis B notification criteria (because development of a clinical illness consistent with acute viral hepatitis occurs

[a] Unspecified hepatitis B notifications – detection of hepatitis B surface antigen or hepatitis B virus by nucleic acid testing in cases who do not meet any of the criteria for a newly acquired case.

infrequently and they are unlikely to meet "absence of prior evidence of hepatitis B virus infection" criteria). The downward trend in notifications is not likely to be due to under-reporting of notifications as rates in other age groups, under 14 years and over 25 years, have remained unchanged. The change in national notification case definitions for newly acquired hepatitis B in January 2004 to include absence of prior evidence of hepatitis B infection may have resulted in reduced notifications since 2004; however, there was a trend to reduced notifications prior to this change. In addition, notification rates increased or remained the same from 2004 to 2005 in New South Wales, South Australia, Western Australia and Queensland, despite the change in notification definitions in 2004. One potential reason for the considerable reduction in notifications, particularly in 15–24 year olds, is the declining rates of intravenous drug use since 2000, consistent with trends in hepatitis C virus notifications.[90] The first Australian children received hepatitis B vaccines as infants in the late 1980s. In a national serosurvey in 2002, the prevalence of hepatitis B surface antibody detection amongst those aged 12–17 years was 45.5%, suggesting that catch-up programs have had some impact (Helen Quinn, NCIRS, personal communication).

In the Northern Territory, hepatitis B vaccine has been routinely given at birth to Aboriginal and Torres Strait Islander 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. Since 1997, most jurisdictions (New South Wales 2004, Northern Territory 1998 (catch-up program only), Tasmania 1998, Victoria 1998, South Australia 1999, Western Australia 2002 and Australian Capital Territory 1999) have also implemented hepatitis B catch-up immunisation school-based programs for adolescents in school years 6, 7 or 8 (aged 11–14 years).[91] The effect of this policy on the reported incidence of acute hepatitis B is not expected to become apparent until the first cohort of vaccinated infants, part of the universal program, reaches adolescence. 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.[88,92,93]

At both national and jurisdictional levels, notifications increased between 1993 and 2001 and since then have declined, while hospitalisations have remained steady since 1999. Overall, there were more hospitalisations than would be expected given the number of notifications and the epidemiology of the disease, as usually less than half of infections are clinically recognised. From 1999 to 2002, the notification rate was twice the hospitalisation rate. However, from 2003 to 2005, the ratio of notifications to hospitalisations fell to less than twofold higher.

The stabilisation in hospitalisations is likely to be a reflection of changes to coding practices, as well as misclassification of hospitalisations due to chronic infection as acute infection. In 1998/1999, ICD-10-AM, which can differentiate between acute and unspecified hepatitis B, replaced the four ICD-9-CM codes. 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 and, once established, have led to the stabilisation observed nationally since 1999/2000. 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 a sustained number of approximately 10 per year since 2001. Misclassification is likely to still be a problem, as only one of the 22 deaths recorded for 2003 and 2004 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. This is essentially unchanged from 2001 and 2002, when one out of 20 deaths had acute hepatitis B with hepatic coma. The age distribution of deaths (nearly two thirds over 60 years old) suggests that many of these cases may have been misclassified chronic HBV deaths.

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. Enhanced surveillance was instituted in Victoria in 2001, for six months, due to recognition that an outbreak of acute hepatitis B infections was occurring in injecting drug users.[94] This outbreak contributed to increased rates reported nationally in 2001[13] and has since ended.[94]

Only six cases of hepatitis B were notified to NNDSS for children born after 1 May 2000 (when universal infant hepatitis B vaccination commenced) and all had vaccination status recorded as "unknown". As this cohort (born after 1 May 2000) ages and increases in size, reporting of vaccination status will become increasingly important. Reporting of cases according to age and vaccination status will add evidence to measure the impact of vaccination. Data on the longevity of immunity following infant vaccination in

low endemic countries is sparse. After 2015, nearly all children will have received hepatitis B vaccines in infancy, and notification and hospitalisation rates in 15–19 year olds are likely to reflect the impact of the universal program, as well as the longevity of immunity.

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, as seen in Taiwan.[87,88,95] The recent reduction in hepatitis B notifications, particularly among the 15–24 year olds, may be due to declining intravenous drug use, as has also been seen in hepatitis C notifications. It is likely that the impact of both the targeted infant and catch-up adolescent vaccination programs will become more evident in the next 5–10 years.

Influenza

Influenza causes annual epidemics of respiratory disease often indistinguishable clinically from disease caused by other respiratory viruses. Symptoms include abrupt onset of fever, cough, malaise, myalgia, sore throat, and headache. 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 contributing to increased mortality, particularly among the elderly and those with high risk underlying conditions. In tropical climates, influenza infection may have two annual peaks, as illustrated in the Northern Territory.[96] Pandemics of influenza, occurring every 30 years or so, are caused by major antigenic shift. Antigenic drift, however, occurs more regularly, producing smaller epidemics.

Case definitions

Notifications

Laboratory-confirmed influenza is a nationally notifiable disease in all jurisdictions except South Australia. Although influenza is not a notifiable disease in South Australia, laboratory reports are collected and sent to NNDSS. Implementation of influenza notification occurred in all other jurisdictions, except Tasmania, during 2001.

Laboratory-confirmed infections are those in which influenza virus is isolated by cell culture, detected by nucleic acid amplification, or by influenza antigen testing, from appropriate respiratory tract specimens; a recent infection can also be demonstrated by serological methods.[11]

Hospitalisations

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, we did not make the distinction 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

In the period January 2003 to December 2005, there were 10,185 notifications, giving an average annual rate of 16.9 per 100,000. Notifications varied considerably by year, with a high of 4,569 in 2005 and a low of 2,133 in 2004. There was a clear seasonal distribution of notifications in all three years with peaks in August-September in 2003, September in 2004 and June-September in 2005.

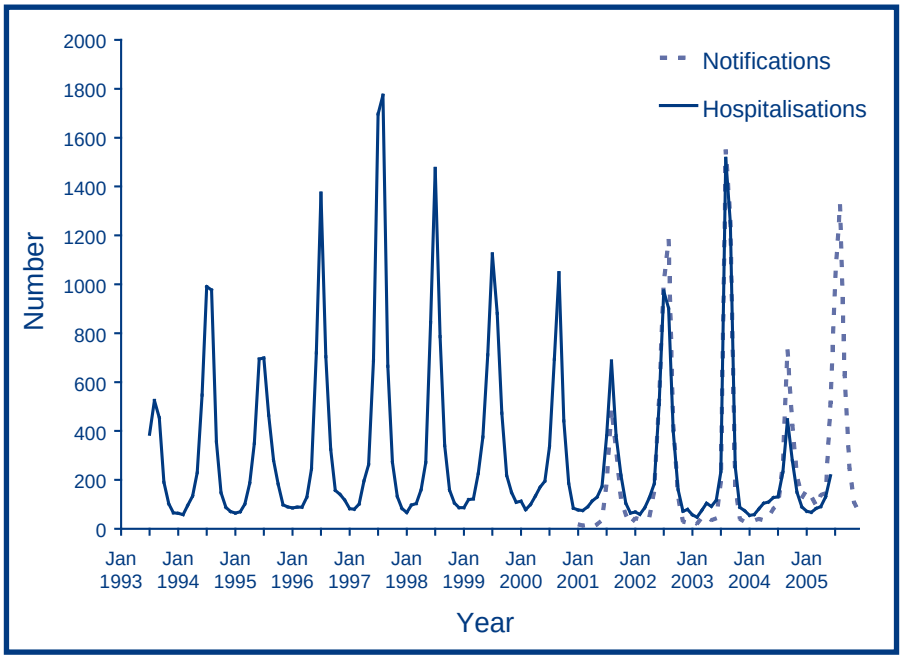
Between July 2002 and June 2005, there were 9,116 hospitalisations coded as influenza (an average annual rate of 15.3 per 100,000), with the greatest number of hospitalisations (3,956) recorded in 2003/04. There was a clear seasonal pattern, with dramatic increases over the winter months (Figure 10). The median number of admissions per month was 107 (range 47–1,515) with the highest number (973; 1,515 and 446) occurring in July 2002, August 2003 and September 2004, respectively.

Severe morbidity and mortality

A total of 56,955 hospital bed days with an ICD-10-AM code for influenza were recorded over the reporting period. The median length of stay was at least twice as long in the oldest age group (five days among people aged 60 years or over) than in the younger age groups (Table 7). Influenza was the principal diagnosis for 68% of the hospitalisations. Bed days peaked in 2003/2004 (n=24,156).

From 1 January 2003 to 31 December 2004, influenza was recorded on the death certificate as the underlying cause of death in 101 cases. Of these, 85 (84%) were aged 60 years or more, four (4%) were aged 25–59 years and five (5%) were aged 0–4 years. Deaths officially designated as due to influenza are known to be a gross underestimate of the true number of deaths attributable to influenza in the population.[97] Although there is no requirement for clinical information to be entered onto NNDSS for influenza cases, 13 deaths were recorded on NNDSS in influenza notifications 2003–2005; two deaths were recorded in children (age range for deaths in notified cases 1–89 years, median 69 years).

Figure 10. Influenza hospitalisations and notifications,* Australia, July 1993 to December 2005, by month of diagnosis or admission



* Notifications where the month of diagnosis was between January 2001 and December 2005; hospitalisations where the month of admission was between 1 July 1993 and 30 June 2005. Note that the Northern Territory, Queensland, Tasmania and Victoria did not notify influenza for the complete year in 2001.

Table 7. Influenza notifications, hospitalisations and deaths, Australia, 2002 to 2005,* by age group

Age group (years)	Notifications 3 years (2003–2005)		Hospitalisations 3 years (July 2002–June 2005)				LOS† per admission (days)	Deaths 2 years (2003–2004)	
	n	Rate‡	n	(§)	Rate‡	(§)	Median(§)	n	Rate‡
0–4	3,173	83.7	3,116	(2,458)	82.1	(64.7)	2.0 (2.0)	5	0.20
5–14	1,125	13.8	731	(544)	9.0	(6.7)	2.0 (2.0)	5	0.09
15–24	1,157	13.9	727	(488)	8.9	(6.0)	1.0 (1.0)	2	0.04
25–59	2,994	10.2	2,499	(1,542)	8.6	(5.3)	2.0 (2.0)	4	0.02
60+	1,724	16.4	2,043	(1,264)	19.9	(12.3)	5.0 (4.0)	85	1.23
All ages‖	10,185	16.9	9,116	(6,296)	15.3	(10.6)	2.0 (2.0)	101	0.25

* Notifications where the month of diagnosis was between January 2003 and December 2005; hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005; deaths where the date of death was recorded in 2003 or 2004.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

‖ Includes cases with unknown ages.

Age and sex distribution

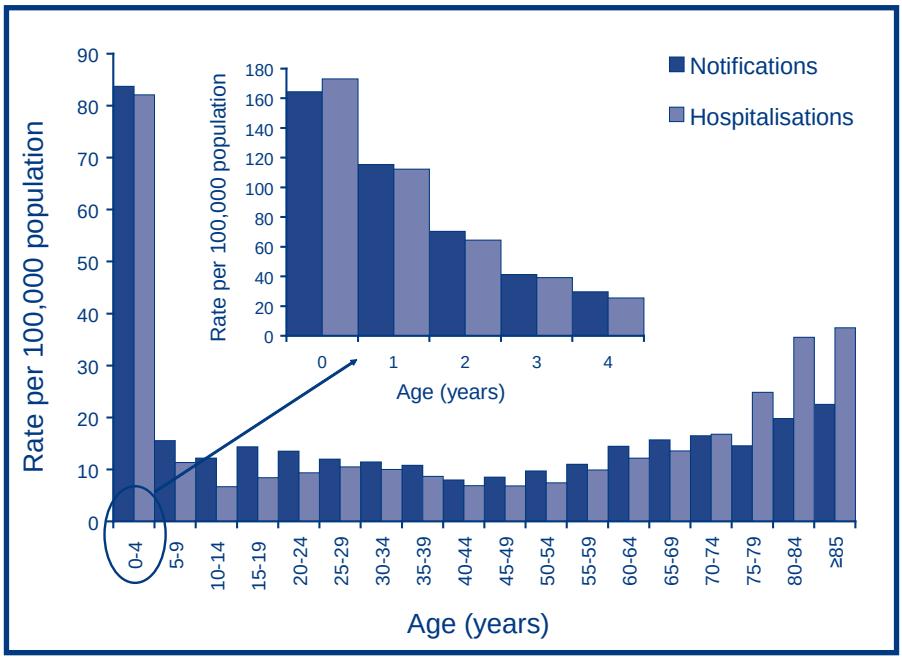
Influenza notification rates vary markedly by age, with peak rates in those under five years of age (Figure 11). In this age group, the highest rates of notifications are in those under one year of age and the rate declines progressively thereafter. The overall male to female ratio was 1.08:1.

Among the age groups specified in Table 7, hospitalisation rates were highest in children aged under five years (82.1 per 100,000). Although overall hospitalisation rates were lower than those in 0–4 year olds, among people aged 60 years and over, the rates increased with increasing age, ranging from 12.2 per 100,000 for those aged 60–64 years to 37.3 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 (annual average of 173 per 100,000 population aged less than one year, ranging from 97.2 per 100,000 in 2004/2005 to 261.8 per 100,000 in 2003/2004).

The overall male to female hospitalisation ratio was 0.92:1; however, this was not consistent across all age groups. In children under 10 years of age, male hospitalisations were more common (1.2:1).

Figure 11. Influenza notification rates 2003 to 2005 and hospitalisation rates 2002/2003 to 2004/2005, Australia,* by age group



* Hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005.

Geographical distribution

There was a wide variation by state or territory in the average crude hospitalisation rate recorded for the three year review period, ranging from 3.5 per 100,000 (n = 34) in the Australian Capital Territory to 30.0 per 100,000 in the Northern Territory (n = 179) (Appendix 3). Notification rates were similarly varied, with the highest rates reported in the Northern Territory and Queensland (41.8 and 27.5 per 100,000 population, respectively) and the lowest in Tasmania and the Australian Capital Territory (2.0 per 100,000 and 4.8 per 100,000, respectively) (Appendix 2).

In regard to annual hospitalisation rates, the distribution by state or territory was generally consistent with the national pattern, with the highest rates occurring in 2003/2004 in all states except Queensland, while the lowest rates occurred in 2004/2005 in all states except the Australian Capital Territory. Historically, the winter of 1997 remains the period between 1993/1994 and 2004/2005 during which most states and territories recorded the highest number of hospitalisations.

Influenza type and subtyping

Almost all of the influenza notifications reported to NNDSS (98%) recorded influenza type. Most of these were type A (n=8,284; 83%). A further 1,586 cases (16%) were recorded as type B and both A and B were documented in 91 notifications (1%). Subtype or strain information was recorded for 12.4% of notifications (n=1,267). Ninety-five per cent of subtyped influenza A was H3 and 5% was H1. Subtype information was only recorded on NNDSS from some notifications of influenza isolates received in Victoria, New South Wales, Queensland and the Northern Territory, with Queensland and Victoria reporting most of the notifications where subtyping was documented.

Comment

Both laboratory notifications and hospitalisations show a similar seasonal pattern. There is a strikingly higher notification and hospitalisation rate in children under one year or age, compared with even the extremely elderly. This is likely to reflect patterns of health care use, and, in particular, diagnostic testing for respiratory viruses, in this age group.

On the other hand, the role of influenza in exacerbating chronic cardiac and respiratory diseases in the elderly may not be adequately reflected in these surveillance data. Deaths and hospitalisations coded as influenza are widely acknowledged to underestimate deaths and hospitalisations due to influenza.[97–100] The proportion of deaths due to influenza occurring in people aged 60 years and over (84%) is lower than most other published studies.[101,102] This may be due to other contributing causes being preferentially coded, as deaths reported here are only those where influenza was recorded as the principal cause, or to lower rates of virological testing in the elderly age group in Australia in comparison to elsewhere.

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),[103] coronavirus[104] or picornavirus.[105,106] 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.

Both influenza A and B are well known to cause major epidemics of respiratory disease resulting in severe morbidity and mortality. In 2003, the predominant influenza isolate was influenza A (94%), with a majority of the A (99%) being H3 subtypes. Ninety-eight per cent of these were A/Fujian/411/2002 (H3N2) which had shown a significant antigenic drift. The 2003 Australian influenza vaccine contained A/Panama/2007/99, which induced two– to fourfold lower antibody response against the drifted strain.[107] An A/Fujian/411/2002 (H3N2)-like virus was incorporated in the Australian influenza vaccine for 2004.

In 2004, influenza A was again predominant (76.9%) but with increased influenza B activity compared to the previous season. Of the record low number of viable influenza isolates received (n=454), 98% of type A isolates were H3N2, which showed further antigenic drift, with one third of the isolates distinguishable from the A/Fujian/411/2002-like reference strain.[108]

In 2005, influenza A remained the predominant type notified (73%), with further increased influenza B activity. Antigenic analysis of 1,174 samples demonstrated that 689 (58.7%) were H3N2, 210 were A H1N1 (17%), and 275 (23.4%) were B viruses.[109]

Annual influenza vaccination is currently recommended as the primary method of influenza prevention in people aged 65 years and over, all Indigenous people aged 50 years and over, and all individuals aged 6 months and over with chronic medical conditions likely to be exacerbated by influenza or its complications, e.g. chronic pulmonary or cardiovascular disease.[76] Vaccination uptake in Australians aged 65 years and over was estimated at 76.9% and 79.1% for 2003[110] and 2004,[111] respectively. Extension of influenza vaccination to all adult Indigenous people should be considered as both influenza hospitalisation rates and mortality related to influenza and pneumonia are twice that documented in persons of non-Indigenous background across all age groups.[77] Influenza vaccination is also encouraged in health care workers and carers of people with high-risk conditions[76] as carer vaccination has been shown to reduce morbidity and mortality in those with high-risk conditions.[112]

As for the paediatric population, in 2003 the USA Advisory Committee on Immunization Practices (ACIP), recommended routine influenza vaccination[113,114] of healthy American children aged 6–23 months based on the high burden of illness.[99,115,116] More recently, in 2006, the ACIP extended its recommendation to include children up to the age of five years.[117] Whilst available Australian data suggest a similar significant burden of illness in young children,[118,119] examination of cost-effectiveness, efficacy and feasibility of universal immunisation of healthy children is required before implementing such a population level strategy.[120]

There is increasing concern regarding the likelihood of an influenza pandemic with the ongoing presence of H5N1 avian influenza in Asia and more recent spread to Europe and Africa.[121] Studies of H5N1 vaccines are beginning to show reassuring immunogenicity even in children and the elderly. Other control measures, such as enhanced infection control (handwashing, quarantine, cohorting), will be especially important early on should a pandemic arise as vaccine production will take some months. Furthermore, achieving high coverage now against seasonal influenza could usefully increase local production capacity of influenza vaccine through increased demand which could speed a switch of production to a pandemic vaccine.[122–125]

Measles

Measles is an acute and highly communicable disease caused by a morbillivirus. The clinical picture primarily includes prodromal fever and Koplik spots on the buccal mucosa, rash, and often conjunctivitis, coryza, and cough. Complications include otitis media, pneumonia and encephalitis. Subacute sclerosing panencephalitis (SSPE) occurs very rarely as a late sequel of wild infection but not vaccination.[44]

Case definitions

See Appendix 6 for pre-2004 definition

National definition from January 2004:[11]

a) Isolation of measles virus (confirmed case); *or*

b) Detection of measles virus by nucleic acid testing (confirmed case); *or*

c) Detection of measles virus antigen (confirmed case); *or*

d) Measles virus-specific IgG seroconversion or significant increase in IgG antibody level or a fourfold or greater rise in antibody titre to measles virus, with paired sera tested in parallel and in the absence of receipt of measles containing vaccine eight days to eight weeks prior to testing (confirmed case); *or*

e) Detection of measles virus-specific IgM antibody confirmed in an approved reference laboratory, in the absence of recent measles containing vaccination (confirmed case); *or*

f) A clinical illness characterised by a generalised maculopapular rash lasting at least three days, fever of at least 38°C at the time of rash onset and either cough, coryza, conjunctivitis or Koplik spots, together with an epidemiological link to a confirmed case (confirmed case); *or*

g) A clinical illness as in point (f) above, together with detection of measles-specific IgM antibody other than by an approved reference laboratory (in the absence of recent measles-containing vaccination) (probable 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

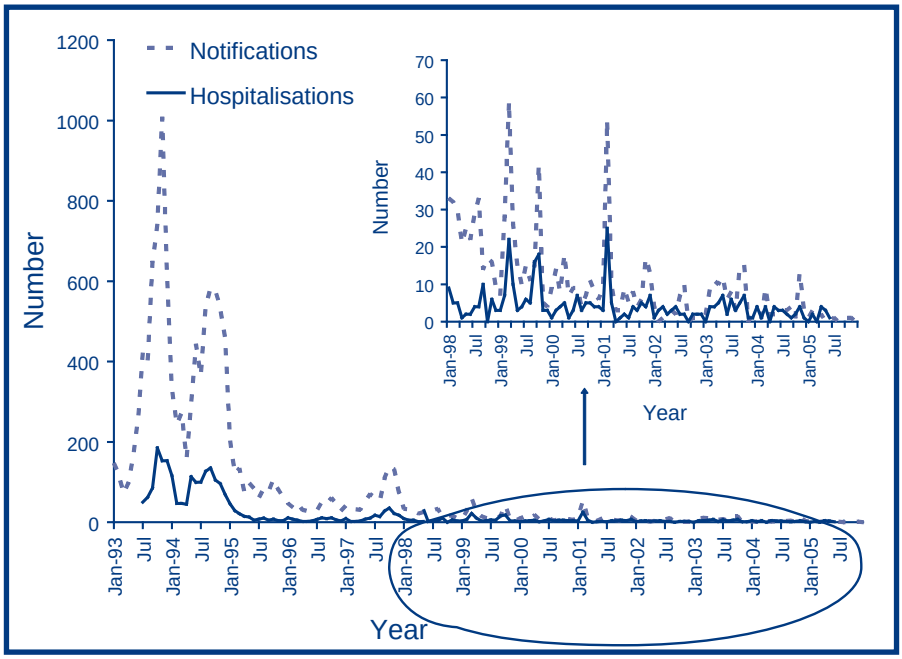
In the three year review period, there were 148 notified cases of measles, an average annual notification rate of 0.25 per 100,000 (Table 8). The number of measles notifications progressively decreased from 2003 (n=93) to 2004 (n=45), and in 2005 to the lowest on record (n=10). The median number of notifications per month was two (range 0–15).

In the period 2002/2003 to 2004/2005, there were 94 hospitalisations with the ICD-10-AM code B05 (measles). This equates to an average annual hospitalisation rate of 0.16 per 100,000. Since a decline in the mid-1990s, annual hospitalisation rates have been fluctuating at a low level, and in 2004/2005 were the lowest on record with only 23 separations, a rate of 0.11 per 100,000. The highest rates of hospitalisations were in the 0–4 and 20–35 year age groups. There were two peaks in the number of monthly hospitalisations during the review period, one in May 2003 (n=7), the other in October of 2003 (n=7) (Figure 12). The median number of hospitalisations per month was two (range 0–7).

Severe morbidity and mortality

In the three year review period, hospital separations for measles accounted for 375 hospital bed days. The median length of stay (LOS) was two days, with the highest median LOS (4.5) in the 60 years and over age group (Table 8). Of the 94 hospitalisations, 72 (77%) had measles recorded as the principal diagnosis. Complications arising from measles infection were recorded for 20 (21%) separations. There was one hospitalisation coded as having otitis media, one with neurological (encephalitis or meningitis) complications, six (6%) as having pneumonia, none with intestinal complications, and 12 (13%) as having other complications (Table 9). Adults aged 15 years and over accounted for nine of the 12 (75%) hospitalisations coded with other complications.

Figure 12. Measles notifications and hospitalisations, Australia, 1993 to 2005,* by month of diagnosis or admission



* Notifications where the month of diagnosis was between January 1993 and December 2005; hospitalisations where the month of admission was between 1 July 1993 and 30 June 2005.

In 2004, there was one death recorded in the National Mortality database where the underlying cause was measles; it was coded as measles complicated by encephalitis. However, further investigation demonstrated that the death was miscoded and was actually a result of subacute sclerosing panencephalitis rather than acute measles encephalitis (Peter Markey, Northern Territory Department of Health and Community Services, personal communication).

Table 8. Measles notifications, hospitalisations and deaths, Australia, 2002 to 2005,* by age group

Age group (years)	Notifications 3 years (2003–2005)		Hospitalisations 3 years (July 2002–June 2005)				LOS† per admission (days)	Deaths 2 years (2003–2004)	
	n	Rate‡	n	(§)	Rate‡	(§)	Median (§)	n	Rate‡
0–4	23	0.61	37	(29)	0.97	(0.76)	2.0 (1.0)	0	0.0
5–14	13	0.16	2	(2)	0.02	(0.02)	n.p.	0	0.0
15–24	42	0.51	19	(15)	0.23	(0.18)	2.0 (2.0)	0	0.0
25–59	70	0.24	32	(24)	0.11	(0.08)	3.0 (3.0)	0	0.0
60+	0	0.00	4	(2)	0.04	(0.02)	4.5 (2.0)	0	0.0
All ages	148	0.25	94	(72)	0.16	(0.12)	2.0 (2.0)	0	0.0

* Notifications where the month of diagnosis was between January 2003 and December 2005; hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005; deaths where the death was recorded in 2003 or 2004.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

n.p. Not published due to small cell size.

Table 9. Indicators of severe morbidity* for hospitalised cases of measles, Australia, 2002 to 2005,* by age group

Age group (years)	Measles encephalitis		Measles pneumonia	
	n	% total	n	% total
0–4	0	0.0	2	2.1
5–14	0	0.0	0	0.0
15–24	1	1.1	1	1.1
25–59	0	0.0	2	2.1
60+	0	0.0	1	1.1
All ages	1	1.1	6	6.4

* Measured using National Hospital Morbidity data where the month of hospital separation was between 1 July 2002 and 30 June 2005.

Age and sex distribution

By the end of the three year review period, notification rates for all age groups declined to their lowest recorded levels (Figure 13). Since 1999, there has been a decrease in measles notification rates in 0–12 month olds. Over the review period, notifications in this age group decreased from seven in 2003 to two in 2004 and zero in 2005. The notification rate for 20–34 year olds also decreased each year from 1.30 per 100,000 in 2003, to 0.70 per 100,000 in 2004 and was the lowest on record at 0.12 per 100,000 in 2005. For each year of the review period, the notification rate for the 20–34 year old age group was highest of all groups for the first time since the Measles Control Campaign (MCC) in 1998, followed by the 0–4 year age group which had the highest rates in previous reporting periods.

In 2005, hospitalisation rates were the lowest on record for the 0–4 (0.40 per 100,000), 5–9 (0.00 per 100,000) and 20–34 (0.21 per 100,000) year age groups. Hospitalisation rates for the 10–19 year and over 35 year age groups were higher at the end of the review period than at the beginning (Figure 14). Hospitalisation rates for the 0–4 year age group were consistently the highest of all age groups since reporting began. However, the rates of hospitalisation of 0–12 month olds decreased over the review period, from six in 2003 to zero in 2005 (data not shown).

Over the three year review period, there were more notifications for males than females (male:female ratio 1.5:1). There were also more hospitalisations of males than females (male:female ratio 1.4:1).

Geographical distribution

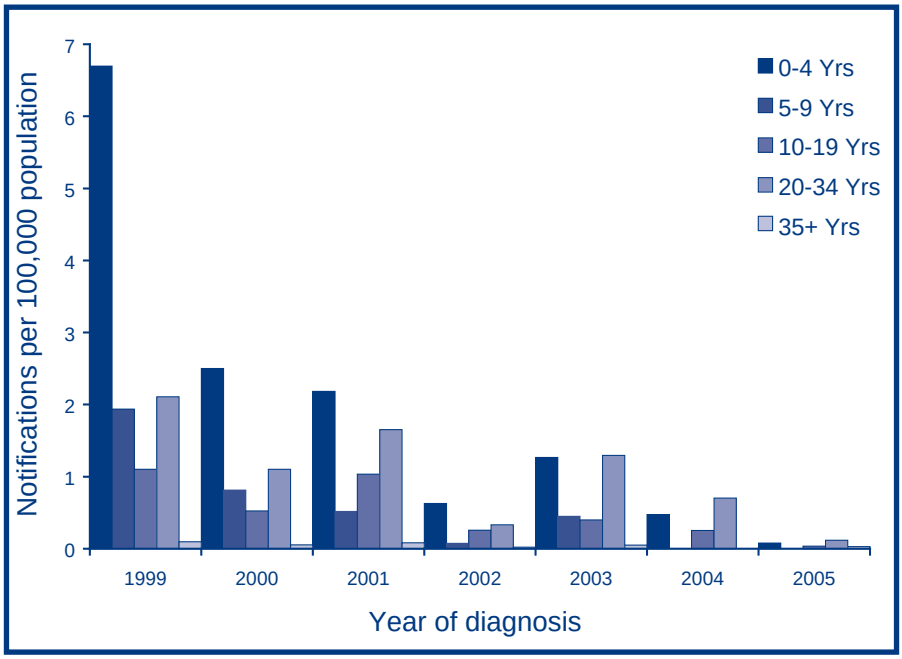
There were 93 measles notifications in 2003, of which 75 (81%) occurred in seven outbreaks.[13] Five of the seven outbreaks are known to have begun with an imported measles case. There were 45 cases of measles notified in 2004, of which 21 (47%) occurred in six outbreaks.[12] Four of the six outbreaks are known to have begun with imported measles cases. One of the 2004 outbreaks was an outbreak of measles in six Indigenous people in Western Australia, with no known link to a confirmed imported source case.[12]

The rate of notification over the reporting period was highest in the Northern Territory with 0.67 per 100,000 (n=4), followed by South Australia with 0.65 per 100,000 (n=30), more than twice the national rate (0.25 per 100,000). The increased notification rates in South Australia during the review period were largely due to one outbreak involving a returned traveller which began in August 2003 (n=21). The high notification rate in the Northern Territory was largely due to unrelated notifications in an overseas traveller and foreign tourists. All jurisdictions reported cases during the reporting period except the Australian Capital Territory (Appendix 2).

There was little variation between the rates of hospitalisation across the states and territories during the review period (0.1–0.2 per 100,000) (Appendix 3).

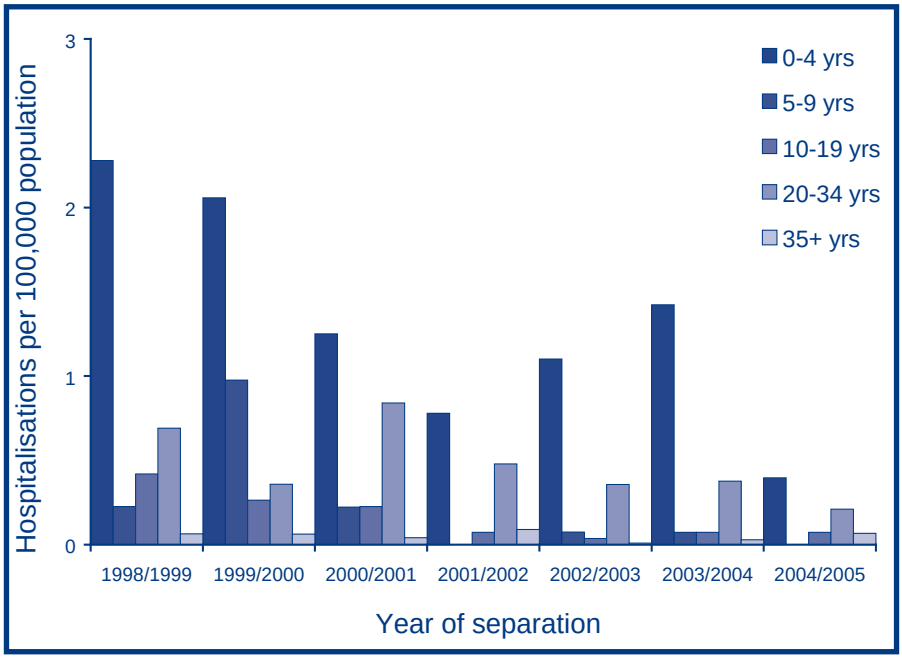
Victoria reported the highest percentage of notifications during the reporting period with 38% of cases (n=56). New South Wales reported the highest percentage of hospitalisations with 35% (n=33).

Figure 13. Measles notification rates, Australia, 1999 to 2005,* by age group and year of diagnosis



* Notifications where the date of diagnosis was between January 1999 and December 2005.

Figure 14. Measles hospitalisation rates, Australia, 1998 to 2005,* by age group and year of separation



* Hospitalisations where the date of separation was between 1 July 1998 and 30 June 2005.

Measles typing and vaccination status

In 2003, seven measles virus genotypes were identified by molecular analysis. The majority of these genotypes were associated with virus importations from countries in Southeast Asia and Japan (H1, D5, D8 and D9). Genotype D2 was also identified after importation from South Africa. The 2003 outbreak of measles in Central Northern Victoria was due to an imported measles strain, confirmed by the Victorian Infectious Diseases Reference Laboratory (VIDRL) as genotype H1. Molecular analysis enabled a second, overlapping outbreak in Melbourne, in which the primary case was a German traveller, to be distinguished from the rural outbreak by the identification of measles virus genotype D8 in clinical samples.[126] In 2004, genotyping identified importations of D4 and D5 (from Thailand), D9 (Malaysia and Indonesia), G2 (Singapore) and G3 (Indonesia). Genotypes D4, H1 and G3 were also detected but the index cases were not identified, thus no source country could be identified. In 2005, genotypes D4 (India) and D5 (Thailand) were detected, as were genotypes D8, D9, G3 and H1, although no source country was identified for these latter genotypes. In each year of the reporting period, molecular analysis of clinical specimens was able to confirm cases of vaccine associated illness (genotype A).

Completion of the vaccination status field was expected in NNDSS during 2003–2005 for all measles notifications born after 31/12/1969. Overall, 72% of cases born after 31/12/1969 (97/135) had this field completed for this period. None of the 97 cases reported receipt of two or more vaccine doses. Ten were reported as fully vaccinated for age (with half of these cases aged over four years and reporting only one dose), 18 as partially vaccinated for age, and 65 were not vaccinated (with the four remaining cases aged under one year and unvaccinated, with vaccination status recorded as "not applicable"). Vaccination status was validated by written records in 50% of the cases reported to be fully vaccinated, 28% of partially vaccinated cases and 8% of unvaccinated cases.

Comment

In the three year review period covered by this report, measles notifications and hospitalisations continued to decline to new record lows. Measles accounted for only 10 notifications in 2005 and 23 hospitalisations in 2004/2005. The downward trend is similar to that seen in other countries with high vaccination coverage, such as those of the Americas.[127] Caution is needed in interpreting hospitalisation data presented from this review period, as highlighted by the zero notifications with four hospitalisations in the 60 years and over age group.

Where endemic measles has been eliminated, enhanced surveillance, including a high level of confirmation, is required and recommended by the WHO.[128] By the end of this review period, a national case definition for measles requiring confirmation of all cases (either by laboratory tests or by linkage to a chain of transmission that includes a laboratory-confirmed case) had been adopted by all jurisdictions. The national case definition is more specific than previous jurisdictional case definitions, as notifications are no longer accepted based on clinical criteria alone, and the detection of measles virus-specific IgM antibody now requires confirmation in an approved reference laboratory. These changes are important for a country in the elimination phase and may have resulted in fewer notifications during the reporting period in states other than Victoria, where laboratory confirmation has been a requirement of notification since July 2001.

The current two-dose vaccination schedule began in 1998 following the mass vaccination of primary school aged children as part of the Measles Control Campaign (MCC).[129] Progress towards elimination of endemic measles in Australia has been underpinned by consistently high vaccination coverage with this schedule prior to school entry. As a result of these efforts, birth cohorts now aged 5–9 and 10–14 years have good coverage with two doses of MMR vaccine and notification rates in these age groups have reached record low levels; in the final year of the review period there was one notification and no hospitalisations from 5–14 year olds. Good vaccination coverage in these birth cohorts has also led to increased herd immunity and may explain why notification rates have remained low in children under 12 months of age (who are not targeted for vaccination).

In Australia, high vaccination coverage in children and reduced exposure to naturally circulating measles virus have left a residual cohort of susceptible young adults born in the 1970s and early 1980s, when measles vaccine was first introduced but coverage was low. Since the MCC, most outbreaks have involved a high proportion of young adults in this birth cohort. To improve immunity in this age group the young adult MMR vaccination campaign was conducted during 2001.[129] In 2002, a serosurvey, conducted by the NCIRS to evaluate this targeted program, indicated that the proportion immune in the young adult

birth cohort had not increased and that a relatively high proportion of targeted individuals remained susceptible.[130] The notification rate for 20–34 year olds in this review period was the highest of all age groups for the first time since the MCC in 1998.

The USA has recently declared their elimination of endemic measles.[131,132] The WHO Regional Committee for the Western Pacific determined 2012 as the target date for measles elimination in the Western Pacific region.[133] Modelling, using Australian serosurvey data, suggests elimination may already have been achieved[130,134] and, using estimates of vaccine uptake, the models predict endemic measles transmission will remain absent from Australia until at least 2012.[130] Continued monitoring is, however, still required as different geographic regions within Australia were projected to exceed the epidemic threshold at different times if vaccination coverage remained at current levels.[134]

Australia, and other countries of the region which have already eliminated indigenous measles transmission, remain at risk of measles importation from neighbouring countries where measles is still endemic. In Australia, the primary case of most recent measles outbreaks acquired their infection in another country and this is supported by molecular genotyping of clinical specimens or measles isolates from the resulting outbreaks.[126,135] However, during the review period, there have been several outbreaks for which the source case has not been identified, including a measles outbreak amongst Indigenous people in Western Australia in 2004 (n=6). In summary, the young adult MMR vaccination program does not appear to have increased the proportion immune to measles in this age group and young adults account for a higher proportion of notified cases than ever before. Therefore, to maintain elimination in the longer term, additional efforts may be needed to target this cohort. The other age group most at risk are children aged 0–4 years who continue to have the second highest notification and the highest hospitalisation rates of any age group. Therefore, high coverage with two doses, including better timeliness and completeness of childhood vaccinations among preschool children, should remain 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 as well as skin lesions. Clinical manifestations include meningitis, meningococcaemia without meningitis (which varies in presentation from fulminant to chronic), meningitis and meningococcaemia combined and septic arthritis. In culture-negative cases with a compatible clinical picture (such as fever, haemorrhagic rash and shock), a diagnosis of meningococcal disease can be supported by a range of other laboratory evidence. This includes the identification of Gram-negative intracellular diplococci or meningococcal antigen in blood or CSF, the identification of nucleic acid from *Neisseria meningitidis* in body fluids or demonstration of a serological response to *Neisseria meningitidis*.

Case definitions

See Appendix 6 for pre-2004 definition

National definition from January 2004:[11]

Confirmed cases require either laboratory definitive evidence or laboratory suggestive evidence and clinical evidence. Probable cases require specified clinical evidence only (as below) and are also notifiable.

a) Laboratory definitive evidence
   - Isolation of *Neisseria meningitidis* from a normally sterile site.

b) Laboratory suggestive evidence
   - Detection of meningococcus from a normally sterile site by nucleic acid testing; *or*
   - Detection of gram-negative diplococci in Gram stain of specimen from a normally sterile site or from a suspicious skin lesion; *or*
   - High titre IgM or significant rise in IgM or IgG titres to outer membrane protein antigens of *Neisseria meningitidis*; *or*
   - Positive polysaccharide antigen test in cerebrospinal fluid with other laboratory parameters consistent with meningitis.

c) Clinical evidence (for confirmed cases with laboratory suggestive evidence)
   - Disease which in the opinion of the treating clinician is compatible with invasive meningococcal disease.

d) Clinical evidence for notification of probable cases
   - The absence of evidence for other causes of clinical symptoms *and either*
   - Clinically compatible disease including haemorrhagic rash; *or*
   - Clinically compatible disease AND close contact with a confirmed case within the previous 60 days.

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 A39.0 (meningitis), then those without A39.0 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

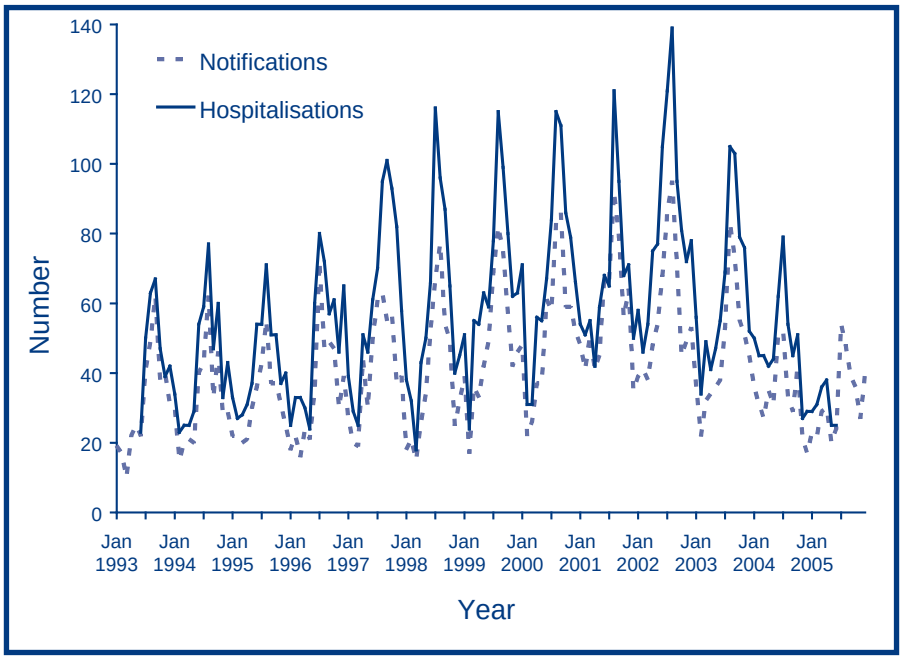
The ICD-10 code used to identify deaths was A39 (meningococcal infection).

Secular trends

There were 1,355 notifications of meningococcal disease in the three years January 2003 to December 2005, an average annual notification rate of 2.2 per 100,000 population (Table 10). A median of 35.5 cases was notified each month, with a range of 17 to 83 cases. Between July 2002 and June 2005, there were 2,135 hospital admissions recorded as ICD code A39 (average annual rate 3.6 per 100,000), and a median of 50.5 cases (range 25–139) per month. Coinciding with the introduction of the national meningococcal C immunisation program in January 2003,[136] both the notification and hospitalisation rates for meningococcal disease decreased each year, down from a peak in 2002 (Figure 15). The notification rate decreased by 39% from 2.8 cases per 100,000 in 2003 to 1.9 cases per 100,000 in 2005, while the hospitalisation rate decreased by 47% from 4.5 per 100,000 in 2002/2003 to 2.4 per 100,000 in 2004/2005.

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 2005,* by month of diagnosis or admission



* Notifications where the month of diagnosis was between January 1993 and December 2005; hospitalisations where the month of separation was between 1 July 1993 and 30 June 2005.

Severe morbidity and mortality

A total of 16,817 hospital bed days (average 5,606 days per year) were recorded for patients with an ICD-10-AM code of A39, of which 44% were coded as meningococcal meningitis (A39.0) and 46% were coded as septicaemia (A39.1–A39.4, not A39.0). The proportion where a meningococcal disease code was the principal diagnosis varied from 96% of cases among 0–14 year olds to 80% of cases for those aged 15–59 years and 67% for those aged 60 years and over. The average length of stay was six days and increased with age (Table 10).

From death certificate data, in 2003 and 2004 there were 46 deaths (0.12 per 100,000 population) with meningococcal disease recorded as the underlying cause of death (Table 10). Sixty-nine deaths were recorded for the 1,355 cases of meningococcal disease notified to NNDSS for the three years 2003–2005 (case fatality rate of 5%). Of the total 2,135 hospitalisations over three years (Table 10), 72 (3.4%) were recorded as dying before hospital discharge. The proportion of meningococcal infection hospitalisations that died before discharge increased steadily with age from approximately 2% for those aged 0–24 years to 5% for 25–59 year olds and 10% for those aged over 60 years.

Table 10. Meningococcal notifications, hospitalisations and deaths, Australia, 2002 to 2005,* by age group

Age group (years)	Notifications 3 years (2003–2005)		Hospitalisations 3 years (July 2002–June 2005)				LOS† per admission (days)	Deaths 2 years (2003–2004)	
	n	Rate‡	n	(§)	Rate‡	(§)	Median (§)	n	Rate‡
0–4	427	11.3	657	(631)	17.3	(16.6)	5.0 (5.0)	12	0.48
5–14	174	2.1	307	(296)	3.8	(3.6)	4.0 (4.0)	1	0.02
15–24	365	4.4	559	(509)	6.8	(6.2)	6.0 (6.0)	12	0.22
25–59	289	1.0	468	(376)	1.6	(1.3)	7.0 (7.0)	10	0.05
60+	100	0.9	144	(96)	1.4	(0.9)	10.0 (9.0)	11	0.16
All ages	1,355	2.2	2,135	(1,908)	3.6	(3.2)	6.0 (6.0)	46	0.12

* Notifications where the month of diagnosis was between January 2003 and December 2005; hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005; deaths where death was recorded in 2003 or 2004.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

Age and sex distribution

The highest meningococcal disease notification, hospitalisation and death rates occurred among children less than five years of age (Table 10), with the highest in this age group among those under one year of age (24.5 notifications, 35.2 hospitalisations and 2.0 deaths per 100,000 population). There was a second peak in notification (Figure 16), hospitalisation (Figure 17) and death rates among 15–19 year olds (5.3, 8.6 and 0.26 per 100,000, respectively), with rates in 20–24 year olds remaining elevated, declining to lower levels in those 25 years of age and over (Table 10, Figures 16 and 17).

Overall, there was a predominance of male cases (male:female ratio 1.2:1). This was consistent across all groups aged less than 30 years. Among cases 30 years and older, rates were higher in females in several age groups, particularly those aged 75 years and over (1:1.8)

Geographical distribution

The pattern of notification and hospitalisation rates for meningococcal disease varied across the country. The highest notification and hospitalisation rates were in the Northern Territory followed by Tasmania (Appendix 2 and Appendix 3). There was a decrease in notification rates between 2003 and 2005 in all states and territories. The largest decreases occurred in Tasmania (4.2 to 2.1 cases per 100,000 in 2003 and 2005, respectively) and Queensland (2.8 to 1.6 cases per 100,000 in 2003 and 2005, respectively).

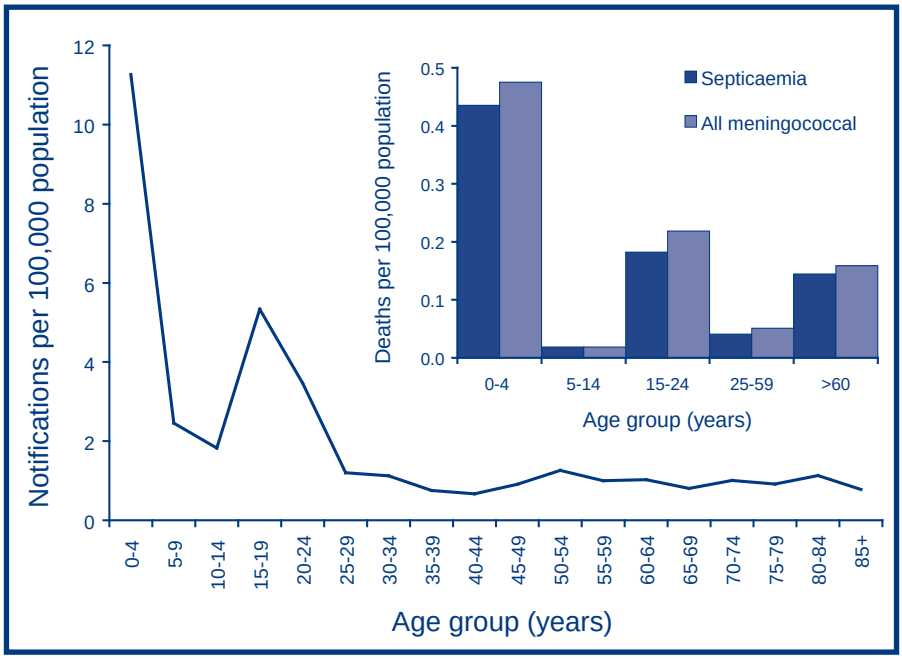
Hospitalisation rates also decreased between 2002/03 and 2004/05 for all jurisdictions except the Northern Territory and the Australian Capital Territory. This decease was greatest in Victoria (5.0 to 1.7 per 100,000 in 2002/03 and 2004/05, respectively) and Queensland (5.3 to 2.2 cases per 100,000 in 2002/03 and 2004/05, respectively) (Appendix 3).

In 2003–2005, among the jurisdictions that reported whether cases were part of an outbreak of meningococcal disease, there were 23 notified cases linked to 11 outbreaks. Outbreaks were recorded in South Australia, Victoria, Queensland and Western Australia.

Meningococcal serogrouping and vaccination

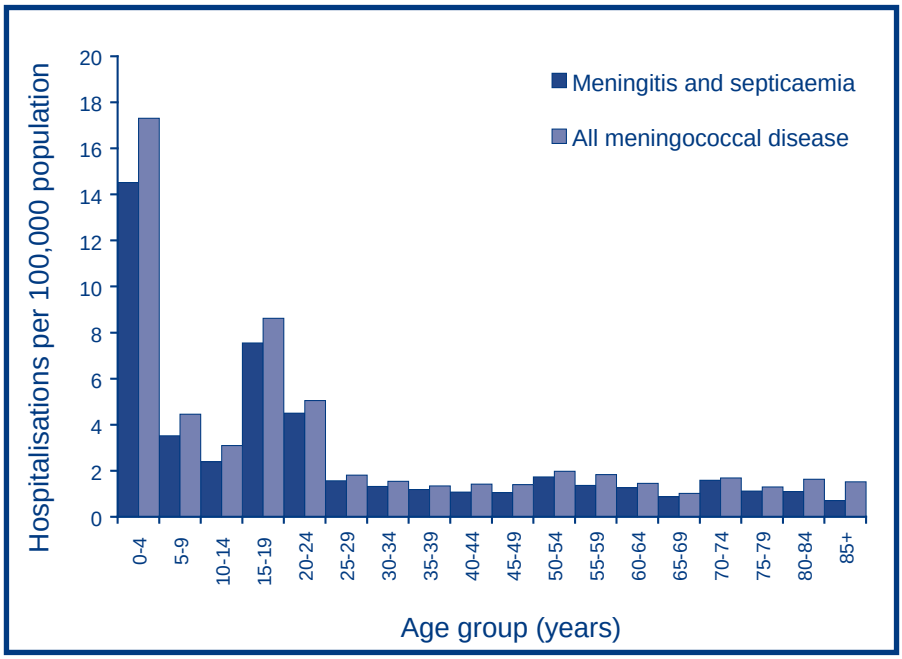
Meningococcal serogroup information was recorded for 84% of the 1,355 notified cases in 2003–2005, a higher proportion than in previous years (Figure 18). In 2003–2005, there were 795 notified cases (1.3 per 100,000 population) of serogroup B meningococcal disease and 280 cases (0.5 per 100,000 population) of serogroup C disease. Serogroup B disease notifications remained stable for all age groups while the number of notifications of serogroup C disease decreased substantially – by 80% from 225 in 2002 to 46 in 2005 (1.15 to 0.23 per 100,000 population) (Figure 18). As a proportion of total notifications for meningococcal disease, serogroup C decreased from 33% in 2002 to 12% in 2005.

Figure 16. Meningococcal disease notification and death rates, Australia, 2003 to 2005,* by age group



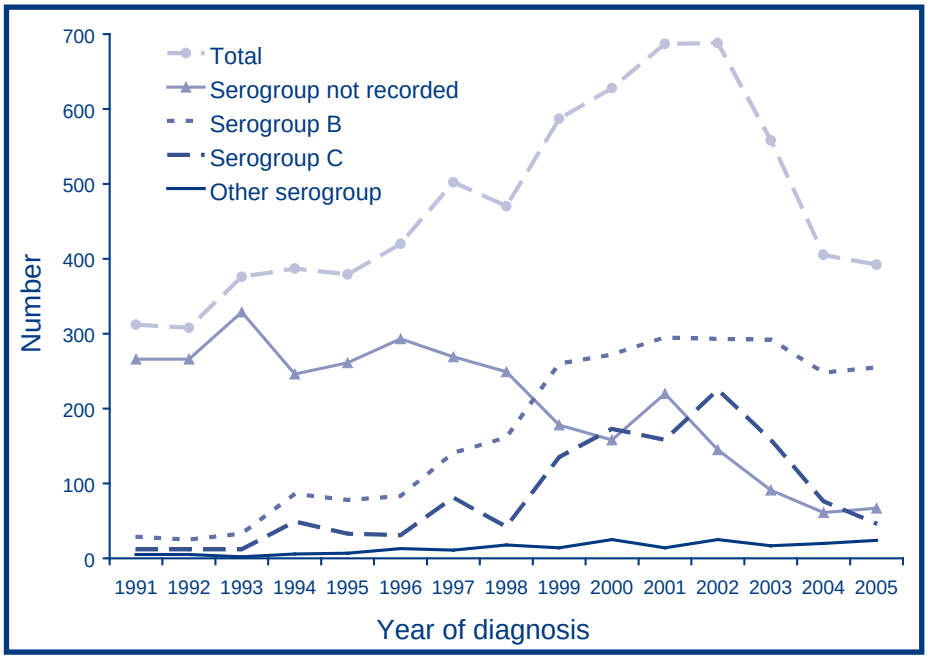
* Notifications where the month of diagnosis was between January 2003 and December 2005; deaths where the death was recorded in 2003 or 2004.

Figure 17. Meningococcal disease hospitalisation rates, Australia, July 2002 to June 2005,* by age group



* Hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005.

Figure 18. Meningococcal disease notifications, Australia, 1991 to 2005, by serogroup



Much of the decrease in serogroup C disease notifications occurred among the age groups targeted for the national meningococcal C vaccination program during 2003–2005 (Figure 19).[136] A decrease in serogroup C notifications was also evident among age groups not included in the vaccination program, including adults aged 25–39 years and those aged 65 years and over (Figure 20). Serogroup C notifications for infants aged less than one year remained low, with an average of four per year (1.7 cases per 100,000 population) compared with an average of 48 serogroup B notifications per year (19.1 cases per 100,000 population).

During 2003–2005, there was considerable heterogeneity between jurisdictions in the relative notification rates of serogroup B and serogroup C meningococcal disease (Figure 21). Following the introduction of the national meningococcal C vaccination program in January 2003, there was an impressive reduction in notification rates across most states and territories with the largest reductions in Tasmania and Victoria (Figure 21).

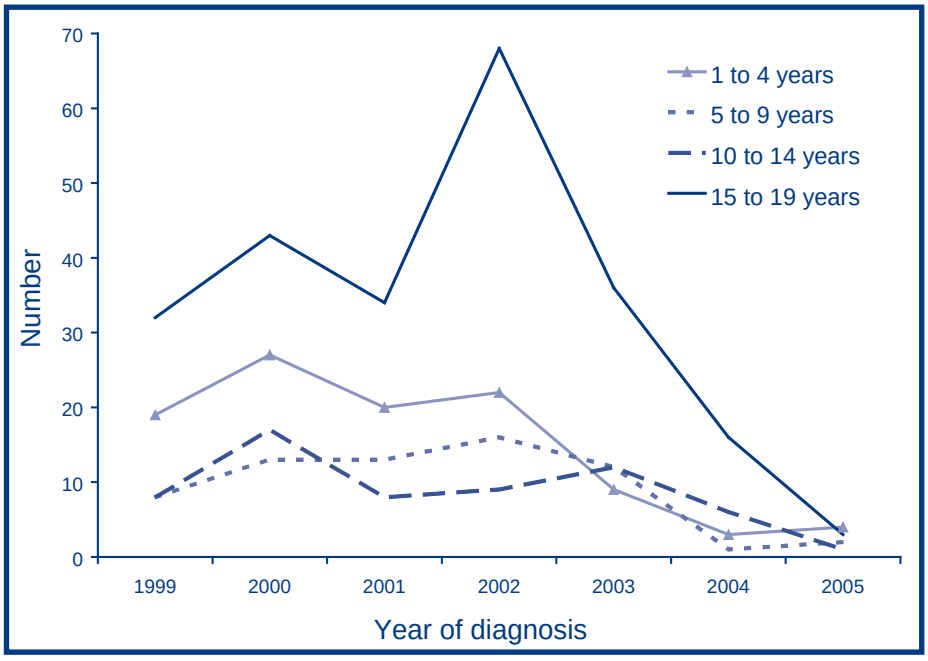
Meningococcal C vaccination status was recorded for 165 (59%) of the 280 notified cases of serogroup C disease and 84 (70%) of the 120 cases aged 19 years and under. There were two reports of vaccine failure in children aged 1–4 years; both occurred in 2003.

Comment

The incidence of meningococcal disease in Australia increased continuously between 1991 and 2002.[3] Following the introduction of the routine and catch-up meningococcal C vaccination programs in January 2003 for those born after 1983 (aged 19 years in 2003),[136] there has been a marked decrease in meningococcal disease notifications, hospitalisations and deaths. The reduction in notifications occurred for both serogroup C disease and those where serogroup information was not available, while notifications for serogroup B and other serogroups, mainly W135 and Y, remained relatively stable.

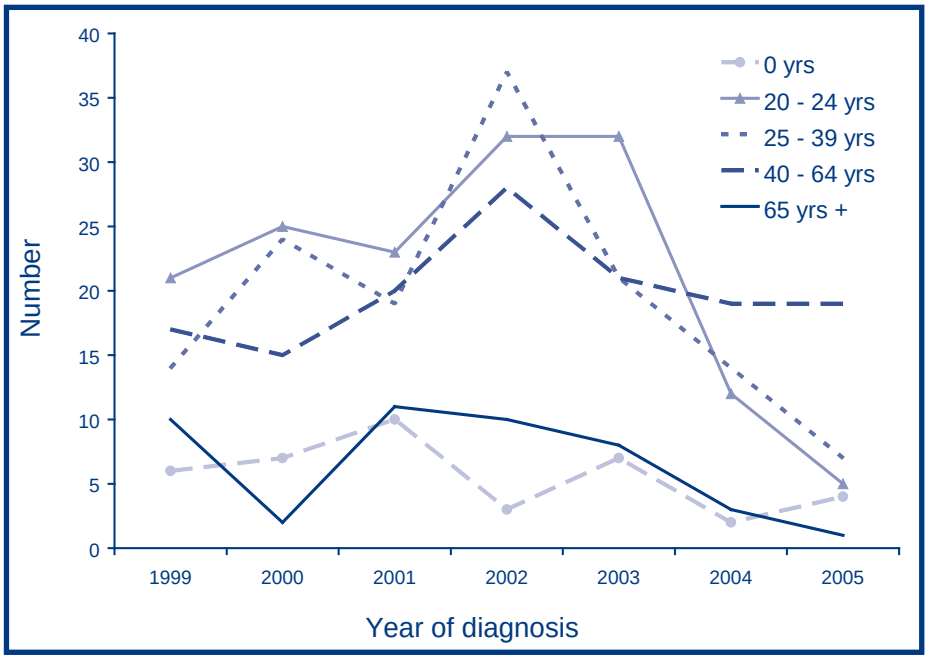
The decrease in serogroup C disease notifications occurred among all the age groups included in the mass vaccination program, particularly those aged 15–19 years at the time of diagnosis. There was also a smaller decrease in serogroup C notifications among two age groups not targeted by the national immunisation program, the 25–39 years and 65 years and over age groups. The 20–24 year age group (age at diagnosis) included some who would have received meningococcal C vaccine as part of the program, plus some who may have been protected by herd immunity, primarily as a consequence of the mass immunisation program and possibly to a lesser extent from natural exposure to pharyngeal infection/colonisation with serogroup C.[137] Federal funding for the serogroup C vaccination program has been extended until June 2007 to allow continued catch-up in 'hard to reach' groups such as those now aged in their early 20s.[138]

Figure 19. Meningococcal serogroup C disease notifications, Australia, 1999 to 2005, by age group eligible for the meningococcal C vaccination program*



* Shows the age groups eligible for the meningococcal C vaccination program from 1 January 2003 (those aged 1–19 years).

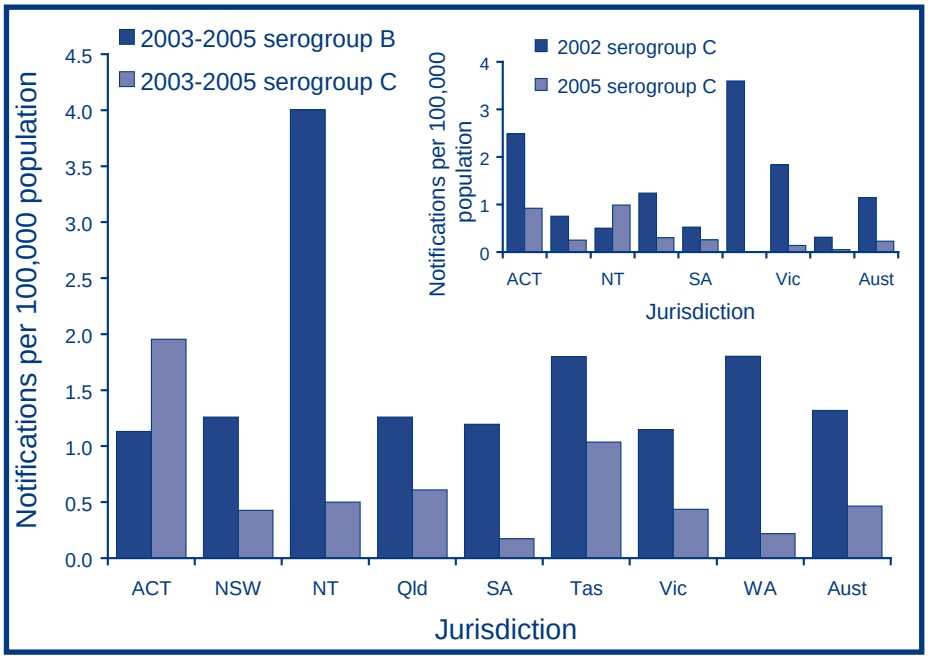
Figure 20. Meningococcal serogroup C disease notifications, Australia, 1999 to 2005, by age group*



* Shows the age groups not included in the meningococcal C vaccination program.

Note: different scales for Figures 19 and 20.

Figure 21. Meningococcal disease notification rates, Australia, by jurisdiction and serogroup



Data reported by the National Neisseria Network,[12] and related disease notification data reported elsewhere,[139] indicate that the majority (77% in 2005)[139] of meningococcal disease is due to serogroup B with the highest rate among children less than one year of age. Like other countries that have implemented national meningococcal C immunisation programs, there is no evidence to date of an increase in the incidence of serogroup B disease in Australia due to serogroup replacement in the vaccinated population.[12,140,141]

Meningococcal disease hospitalisations have been consistently higher than notifications since 1997 (Figure 15). A number of factors may contribute to this discrepancy including inter-hospital transfers (approximately 10% of separations for meningococcal disease), coding of some meningitis admissions as meningococcal disease and incomplete notification of hospitalised cases to public health authorities.

The high burden of meningococcal disease in infants, particularly non-vaccine preventable serogroup B disease, emphasises the importance of early recognition and appropriate clinical management of disease and the need for a vaccine to reduce the significant morbidity and mortality. Several candidate serogroup B vaccines are under investigation in international Phase II clinical trials.[142] However, availability of a universal serogroup B vaccine appropriate for use in Australia is still some way off. In New Zealand, a vaccine, based on a particular strain of meningococcus B that was responsible for a major upsurge in disease over an extended period, has proved successful. High rates of vaccine uptake were achieved (more than three million doses distributed) and the rate of disease has been substantially reduced in line with a vaccine effectiveness of about 80% against the epidemic strain.[143,144]

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. Symptomatic aseptic meningitis occurs in up to 10% of cases. Other potentially serious complications are less frequent and include pancreatitis, orchitis and encephalitis.[44]

Case definitions

See Appendix 6 for pre-2004 definition

National definition from January 2004:[11]

Confirmed cases require either laboratory definitive evidence, or laboratory suggestive evidence and clinical evidence, or clinical evidence and an epidemiological link to a laboratory-confirmed case.

a) Laboratory definitive evidence

- Isolation of mumps virus; *or*
- Detection of mumps virus by nucleic acid testing; *or*
- IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to mumps virus in the absence of recent vaccination.

b) Laboratory suggestive evidence

- Detection of mumps-specific IgM antibody in the absence of recent vaccination.

c) Clinical evidence

- A clinically compatible illness characterised by swelling of the parotid or salivary glands lasting two days or more without other apparent cause.

Hospitalisations and deaths

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

Secular trends

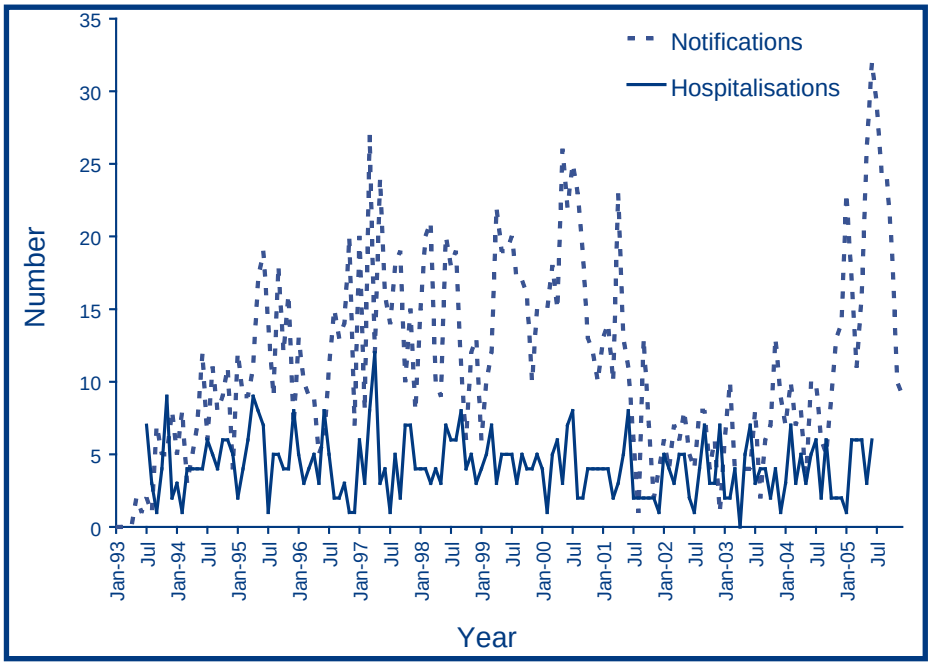
During the three years from January 2003 to December 2005, there were 419 notifications of mumps (an average annual notification rate of 0.69 per 100,000) (Table 11). Since 2002, when mumps notifications were the lowest on record, mumps cases have increased, and in 2005, reached the highest level since notification began in 1993, with a rate of 1.18 per 100,000 (Figure 22). Monthly numbers of notifications varied considerably, with a median of 9.5 (range 2–32) notifications per month. Notifications peaked in June 2005, with 32 notifications, and were above 10 per month between November 2004 and November 2005.

From July 2002 to June 2005, there were 138 hospitalisations coded as due to mumps (average annual rate of 0.23 per 100,000) (Table 11) with a median of 3.5 admissions per month (range 0–7). Hospitalisation rates were stable over the period, in contrast with the increase in notification rates (Figure 22). However, the hospitalisation rates for this review period were consistently above the record low levels seen in 2001/2002.

Severe morbidity and mortality

There were 549 hospital bed days (average 183 per year) recorded for patients with an ICD-10-AM code for mumps (Table 11). Of the 138 hospitalisations (average annual rate 0.23 per 100,000), 114 (83%) had mumps recorded as the principal diagnosis. Complications arising from mumps infection were recorded for 19 hospitalisations (14%). As in the past, the most commonly reported complication was orchitis. There were 10 (7%) hospitalised cases coded with orchitis; seven of these were between 15 and 59 years of age (Table 12). There were no hospitalisations coded as neurological (encephalitis or meningitis) or with multiple complications. The median length of stay (LOS) in hospital was two days, but adults aged 25 years and older had a longer median LOS compared with younger age groups (Table 11). The 15–24 year old age group had the highest hospitalisation rate and accounted for 20% of the hospitalisations. Adults over 15 years of age accounted for 75% of hospitalisations. Mumps was recorded as the underlying cause of death in one adult (aged 100 years) in 2004.

Figure 22. Mumps notifications and hospitalisations, Australia, 1993 to 2005,* by month of diagnosis or admission†



* Notifications where the month of diagnosis was between January 1993 and December 2005; hospitalisations where the month of admission was between 1 July 1993 and 30 June 2005.

† 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, 2002 to 2005,* by age group

Age group (years)	Notifications 3 years (2003–2005)		Hospitalisations 3 years (July 2002–June 2005)				LOS† per admission (days)	Deaths 2 years (2003–2004)	
	n	Rate‡	n	(§)	Rate‡	(§)	Median (§)	n	Rate‡
0–4	24	0.63	11	(9)	0.29	(0.24)	1.0 (1.0)	0	0.00
5–14	24	0.29	23	(20)	0.28	(0.25)	1.0 (1.0)	0	0.00
15–24	81	0.97	27	(24)	0.33	(0.29)	1.0 (1.0)	0	0.00
25–59	260	0.88	53	(50)	0.18	(0.17)	2.0 (2.0)	0	0.00
60+	30	0.28	24	(11)	0.23	(0.11)	6.0 (3.0)	1	0.01
All ages	419	0.69	138	(114)	0.23	(0.19)	2.0 (2.0)	1	0.003

* Notifications where the month of diagnosis was between January 2003 and December 2005; hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005; deaths where the death was recorded in 2003 or 2004.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

Age and sex distribution

Since 2002, low notification rates have continued in the 0–14 year age group but there has been a notable increase in older age groups (Figure 23). In particular, during 2005 there was a high rate of notifications in the 25–29 year age group (5.1 per 100,000) as well as elevated rates in the 20–24 and 30–34 year old age groups. An upward trend in hospitalisations is not apparent (Figure 24), with the highest rates of admissions in those aged 5–9 years (0.45 per 100,000) and 70–74 years (0.47 per 100,000) over the three year period between July 2002 and June 2005.

Table 12. Indicators of severe morbidity and mortality* for hospitalised cases of mumps, Australia, 2002 to 2005,* 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	0.00	0	0.00	0	0.00	1	9.09
5–14	0	0.00	0	0.00	1	4.35	1	4.35
15–24	0	0.00	3	11.1	0	0.00	1	3.70
25–59	0	0.00	4	7.55	1	1.89	1	1.89
60+	0	0.00	3	12.5	0	0.00	3	12.5
All ages	0	0.00	10	7.25	2	1.45	7	5.07

* Measured using National Hospital Morbidity data where the month of hospital separation was between 1 July 2002 and 30 June 2005.

The male:female ratio was 1.3:1 for notifications and 1.0:1 for hospitalisations over the three year review period. There was some variation by year in these ratios but the notifications and hospitalisations were broadly consistent with a similar level of mumps infection in males and females.

Geographical distribution

The rise in notifications across the three year period was primarily due to an increase in Queensland (0.26 per 100,000 in 2003 rising to 1.79 per 100,000 in 2005) and New South Wales (0.52 per 100,000 in 2003 rising to 1.62 per 100,000 in 2005), although there have been smaller increases in Victoria and Western Australia (Appendix 2).

No obvious trends were apparent in hospitalisations due to small numbers in each jurisdiction (Appendix 3).

Vaccination status

Vaccination status was recorded for all 112 notifications of individuals born after 31/12/1980 in the 2003–2005 period but with 35 of these recorded as "unknown". Of the 77 with vaccination status recorded, 24 were fully or partially vaccinated and 53 were unvaccinated. In the 7–15 year age range, 33% (6 of 18) of notifications were recorded as fully (5) or partially (1) vaccinated. Validation of vaccination status was recorded infrequently (8 of 77).

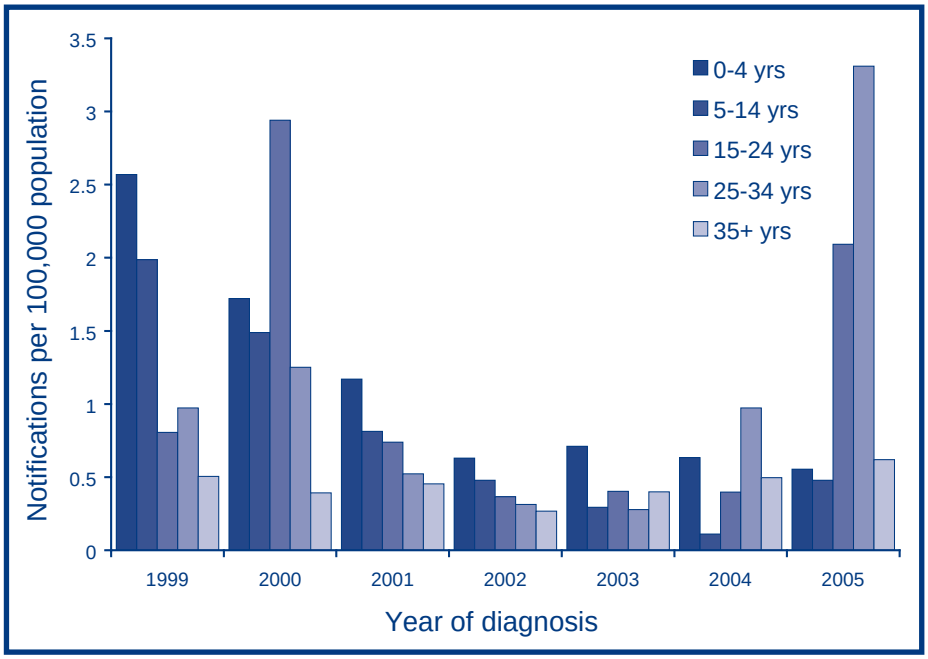
Comment

After declining to record lows in 2002, notifications of mumps increased considerably during the period 2003–2005. This rise was primarily due to increased notification rates in the 20–34 year age group, with rates remaining low in the 0–4 year age group. The highest notification rate was experienced in the 25–29 year age group during 2005, with 5.1 per 100,000. Individuals in this age group were not eligible for routine mumps vaccination as infants, although they were targeted as part of the young adult MMR vaccination campaign in 2001. However, results from the second national serosurvey conducted by NCIRS suggest that this campaign had little impact on population immunity for measles, a conclusion that is also expected to apply to mumps.[145]

The rise in notifications was not reflected in the hospitalisation rates over the review period. This difference might be explained by the increase in notifications being primarily in the 20–34 year age group, for whom mumps hospitalisation may be relatively uncommon. The hospitalisation rate in this age group increased from the record low of 2001/2002, and was similar to the rates experienced in the late 1990s. The male:female ratios for hospitalisations and notifications were similar but there was some geographic variation in notifications by state, with Queensland and New South Wales experiencing the largest numbers of cases. An increasing trend in hospitalisations was not seen in these states.

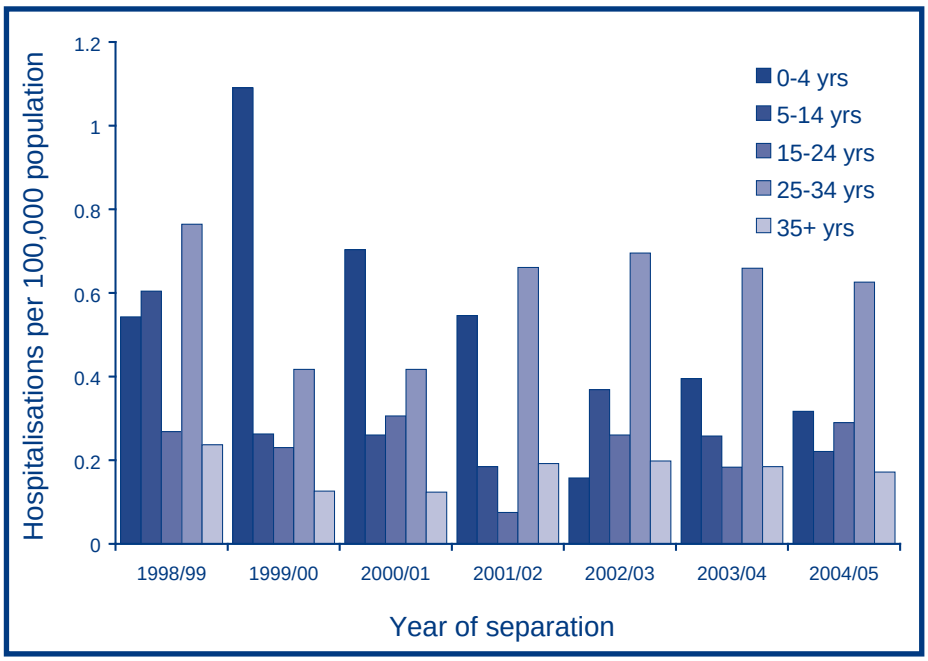
Despite the increase in mumps notifications, rates in Australia were low in comparison to the rates experienced in the United Kingdom during the epidemic of mumps in 2004/2005. During 2005, there were 56,390 preliminarily confirmed cases of mumps reported in the UK,[146] primarily affecting the 18–24 year age group in which the rate was above five per 1,000.[147] Geographic movements of college students and

Figure 23. Mumps notification rates, Australia, 1999 to 2005,* by age group and year of diagnosis



* Notifications where the month of diagnosis was between January 1999 and December 2005.

Figure 24. Mumps hospitalisation rates, Australia, 1999 to 2005,* by age group and year of separation



* Hospitalisations where the month of separation was between 1 July 1998 and 30 June 2005.

waning of vaccine-derived immunity presumably contributed to the scale of this outbreak. The cohort most affected, although included in the UK national campaign of 1994, received measles-rubella (MR) not measles-mumps-rubella (MMR) vaccine, would have been ineligible for routine infant MMR vaccination and had limited exposure to circulating mumps virus.

In the USA, there were also significant numbers of mumps cases reported in 11 states following an outbreak in Iowa, with 2,597 cases reported between January and May 2006.[148] Again, these cases were primarily in the 18–24 year age group. In Australia, notification rates have remained low. Nonetheless, in 2005, Australia exceeded the WHO incidence threshold for disease elimination of one case per 100,000. The higher notification rates in the 20–34 year age group suggest that some endemic transmission may be occurring in this age group. The rises in adult notifications of mumps over this reporting period and the recent experience in the USA and UK suggest that surveillance for mumps, with enhancement of immunisation efforts if necessary, is of continued importance in the Australian context.

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 of age, adolescents and adults often have fewer classical symptoms without paroxysms or whoop.[44]

Case definitions

See Appendix 6 for pre-2004 definition

National definition from January 2004:[11]

Confirmed cases are those with definitive laboratory evidence, laboratory suggestive evidence and clinical evidence, or clinical evidence and an established epidemiological link to a confirmed case. Probable cases require clinical evidence (as below) only and are also notified.

a) Laboratory definitive evidence

- Isolation of *B. pertussis* from a clinical specimen or detection of *B. pertussis* by nucleic acid testing

b) Laboratory suggestive evidence

- Seroconversion or significant increase in antibody levels or fourfold or greater rise in titre to *B. pertussis*, in absence of recent vaccination; *or*
- Single high IgA titre to whole cells; *or*
- Detection of *B. pertussis* antigen by immunofluorescence assay (IFA).

c) Clinical evidence

- A coughing illness lasting two or more weeks; *or*
- Paroxysms of coughing or inspiratory whoop or post-tussive vomiting.

Hospitalisations and deaths

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

Secular trends

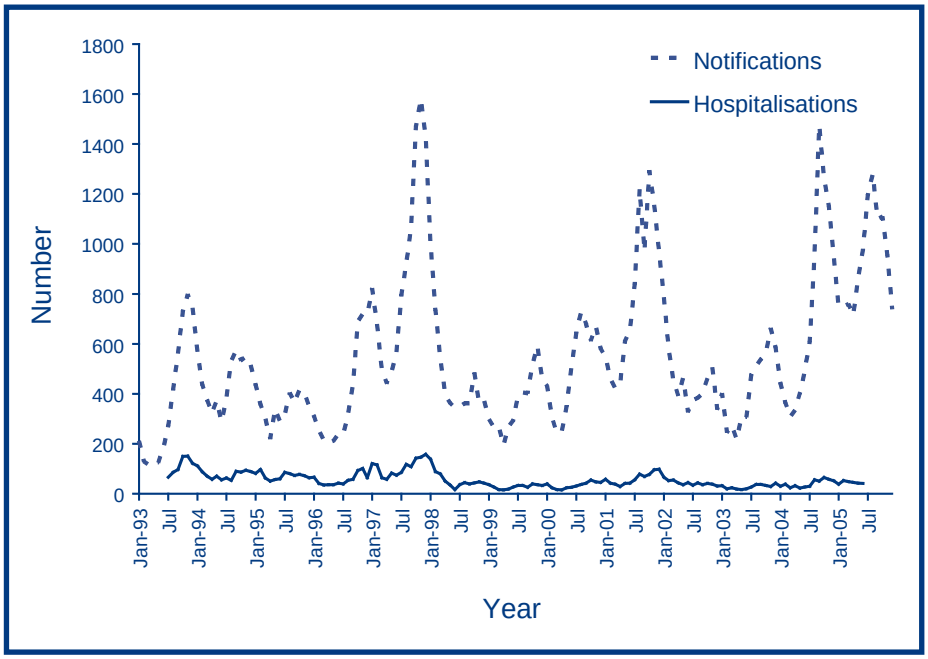
There were 25,035 notifications of pertussis received by the National Notifiable Diseases Surveillance System (NNDSS) with dates of onset in the three years January 2003 to December 2005 (average annual rate 41.5 per 100,000) (Table 13). A median of 635 cases was notified each month (range 217–1,474). Both 2004 and 2005 were epidemic years, with 8,750 and 11,191 notifications, respectively, compared with 5,094 notifications in 2003. Epidemic peaks have occurred every three to four years since national notifications became available in 1991. The national notification rate was 55.1 per 100,000 in 2005 and 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 2005 (Figure 25).

Hospitalisations followed a similar pattern to notifications. There were 1,319 hospital separations coded as pertussis during the review period, 359 in 2002/2003, 373 in 2003/2004 and 587 in 2004/2005 (Table 13 and Appendix 3). The median number of pertussis hospitalisations per month was 37 (range 17–65). The average annual national hospitalisation rate was 2.2 per 100,000 for this reporting period, compared with 3.3 per 100,000 for the previous two years 2000/2001 to 2001/2002.[3]

Severe morbidity and mortality

There were 8,038 hospital bed days recorded with an ICD-10-AM code for pertussis between July 2002 and June 2005 (2,500 for 2002/2003, 2,163 for 2003/2004 and 3,375 for 2004/2005). The median length of stay per admission was four days (Table 13). Of the 1,319 hospitalisations, 1,034 (78%) had a principal diagnosis of pertussis (average annual rate 1.7 per 100,000). The discharge diagnosis code A37.0 (*B. pertussis*) was recorded for 596 (45%) hospitalisations and was the principal diagnosis for 470 (79%)

Figure 25. Pertussis notifications and hospitalisations, Australia, 1993 to 2005,* by month of diagnosis or admission



* Notifications where the month of diagnosis was between January 1993 and December 2005; hospitalisations where the month of admission was between1 July 1993 and 30 June 2005.

of these. *Bordetella parapertussis* (A37.1) was recorded for 14 hospitalisations, and other *Bordetella* species (A37.8) for 22 hospitalisations. The remaining 687 (52%) hospitalisations were coded as whooping cough (organism unspecified – A37.9), and this was the principal diagnosis for 544 (79%) of these.

For the two years 2003 to 2004, two deaths were recorded where pertussis was the underlying cause (Table 13). Both occurred in 2004; one case was one month of age and the other was a 95 year old. Between 1993 and 2002, there were 16 deaths attributed to pertussis: all but one were younger than 12 months of age; six occurred in 1997.[1–3]

There were three deaths in notified cases reported to NNDSS between 2003 and 2005. Two of these are the cases identified in the 2004 death data and the other was a one month old case notified in 2005.

Table 13. Pertussis notifications, hospitalisations and deaths, Australia, 2002 to 2005,* by age group

Age group (years)	Notifications 3 years (2003–2005)		Hospitalisations 3 years (July 2002–June 2005)				LOS† per admission (days)	Deaths 2 years (2003–2004)	
	n	Rate‡	n	(§)	Rate‡	(§)	Median(§)	n	Rate‡
0–4	1,688	44.5	764	692	20.1	18.2	3.0 (3.0)	1	0.04
5–14	3,551	43.6	104	78	1.3	1.0	2.0 (2.0)	0	0.00
15–24	3,263	39.3	37	28	0.5	0.3	2.0 (2.0)	0	0.00
25–59	12,738	43.2	241	150	0.8	0.5	4.0 (3.0)	0	0.00
60+	3,789	36.0	173	86	1.7	0.8	7.0 (6.0)	1	0.01
All ages‖	25,035	41.5	1,319	1,034	2.2	1.7	4.0 (3.0)	2	0.01

* Notifications where the month of diagnosis was between January 2003 and December 2005; hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005; deaths where the death was recorded in 2003 or 2004.

† LOS = length of stay in hospital.

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

§ Principal diagnosis.

‖ Includes cases with unknown ages.

Age and sex distribution

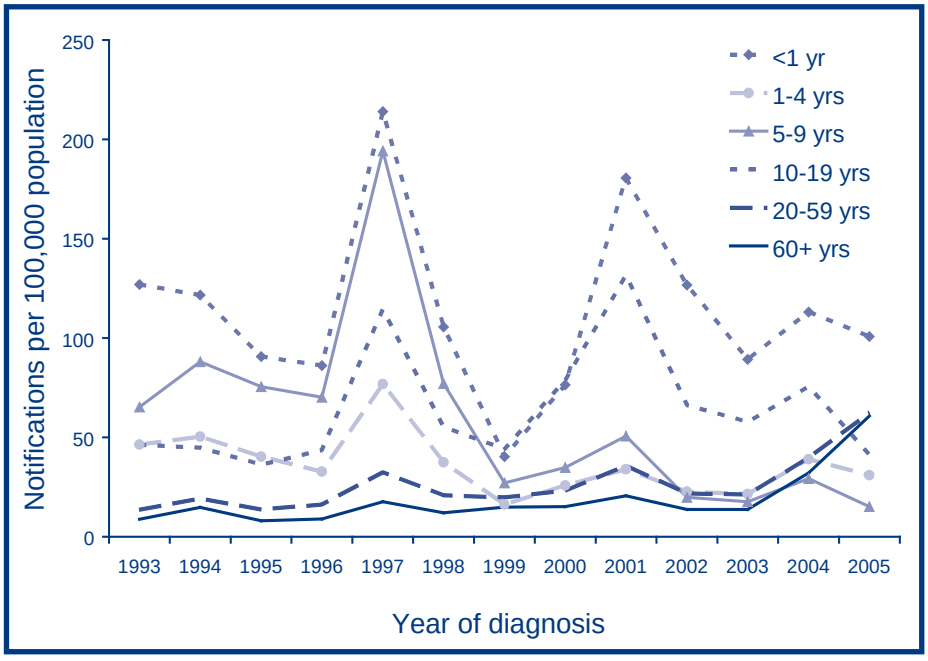
The highest notification rates were seen in infants aged less than one year (Figure 26), with annual average rates of 113.1 per 100,000 in 2004 and 100.8 per 100,000 in 2005. In the three year review period, infants aged less than one year accounted for 3% of all notifications (n=763) but 50% of hospitalisations (n=655). The average hospitalisation rate for infants was 88.1 per 100,000 in this reporting period compared with 154.1 per 100,000 for the previous two years, 2000/2001–2001/2002 (Figure 27 and Figure 28).[3]

The 10–19 year age group accounted for 19% of pertussis notifications in 2003–2005 (n=4,824) and 8% of all hospitalisations (n=100). The 10–19 year age group had very high notification rates in 2003 and 2004, similar to recent patterns of notifications in this age group[3] and second only to the notification rate in infants aged less than 1 year. In 2005, there was a decline in the notification rate for 10–19 year olds to 41.4 per 100,000, with the notification rate in this age group falling below that of people aged 20–59 years and 60 years and over.

People aged 20–59 years (adults) accounted for 56% of notifications (n =13,902) and 19% of hospitalisations (n =252, with an average annual hospitalisation rate of 0.8 per 100,000, the same as in the previous reporting period).[3] Elderly people aged 60 years and over accounted for 15% of notifications (n =3,789) and 13% of hospitalisations (n=173). The notification rates in those aged 20–59 years and 60 years and over have both recently risen to a record high of 62 notifications per 100,000 population, which is in contrast to the relatively steady annual rates previously seen in these age groups. The proportion of notifications in these groups has also been increasing, from 35%–45% between 1993 and 1998 to 83% in 2005.

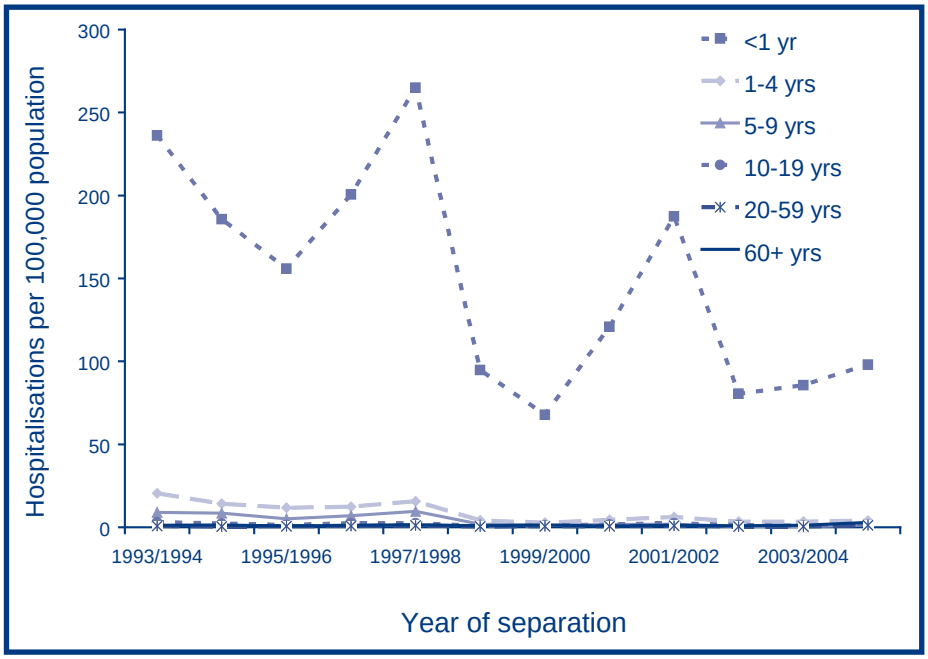
The overall male:female ratio was 1:1.3 for notifications and 1:1.1 for hospitalisations. Higher rates among females were apparent in most age groups for notifications and hospitalisations. The exception to this was for hospitalisations in people aged 70 years and over, where the male:female ratio was 1.6:1.

Figure 26. Pertussis notification rates, Australia, 1993 to 2005,* by age group



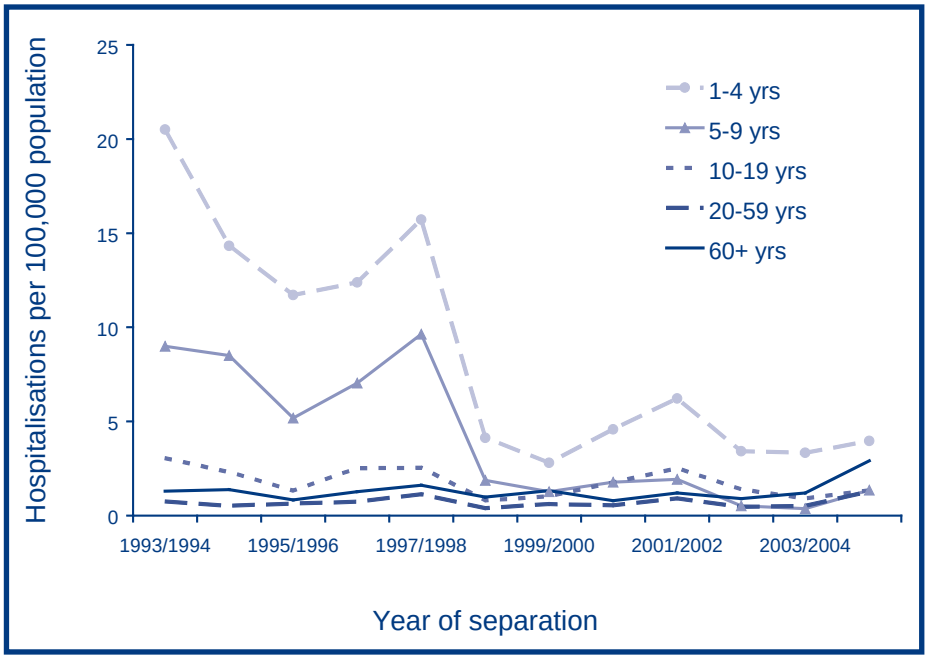
* Notifications where date of diagnosis was between 1 January 1993 and 31 December 2005.

Figure 27. Pertussis hospitalisation rates, Australia, 1993 to 2005,* by age group



* Hospitalisations where date of separation was between 1 July 1993 and 30 June 2005.

Figure 28. Pertussis hospitalisation rates, Australia, 1993 to 2005,* by age group (excluding <1 yrs)



* Hospitalisations where date of separation was between 1 July 1993 and 30 June 2005.

Geographical distribution

Periodic epidemics of pertussis occur in Australia at intervals of three to four years. However, the frequency and length of the epidemic cycles varies within states, particularly in the less populated and/or geographically isolated states and territories, such as Western Australia, the Northern Territory and Tasmania. During the study period, there was a large variation in notification (Appendix 2) and hospitalisation rates (Appendix 3) between regions and years. The Australian Capital Territory experienced a pertussis epidemic in 2003 and was the only state or territory to do so, with a notification rate of

110.4 per 100,000 population. In 2004, an elevated notification rate of 106.2 per 100,000 population signified a pertussis epidemic in Western Australia, seven years after the previous epidemic in this state. South Australia (97.7 per 100,000), the Australian Capital Territory (96.9 per 100,000), New South Wales (85.5 per 100,000), the Northern Territory (45.4 per 100,000) and Queensland (44.8 per 100,000) all experienced elevated notification rates in 2005.

Vaccination status

Completion of the vaccination status field was expected for all pertussis notifications aged under 15 years in NNDSS during 2003–2005. Overall, 72% of cases aged 0–14 years of age had this field completed for this period. Field completion rates varied by age group, ranging from 58% completion for cases aged 10–14 years, to 96% for those aged 6–11 months. The percentage of cases fully and partially vaccinated for age was calculated for people with a known vaccination status aged less than 9 years. The number of cases fully vaccinated for age rose from 37% in infants less than 6 months of age to 70% in those aged 1–4 years (Table 14).

Table 14. Vaccination status of notified pertussis cases aged six months to nine years, Australia, 2003 to 2005,* by age group

Age group	Vaccination status (2003–2005)			
	Fully vaccinated for age		Partially vaccinated for age	
	n	%†	n	%†
0–5 months	188	37	61	12
6–11 months	116	60	30	15
1–4 years	454	70	14	2
4–9 years	499	61	51	6

* Notifications where the month of diagnosis was between January 2003 and December 2005.

† Percentage of those with a known vaccination status.

Comment

Since 1993, pertussis has caused the greatest morbidity of any disease preventable by vaccines recommended for children on the National Immunisation Program (NIP) schedule. The highest numbers of pertussis notifications were seen in 2005, with many jurisdictions experiencing an epidemic in that year, followed by 1997 and 2001. Traditionally, hospitalisations in infants aged less than one year have exceeded notifications, indicating that notification rates tend to underestimate pertussis incidence.[3,149] However, in the 2003–2005 period, there were more notifications than hospitalisations in this age group, which may be a reflection of the increased use of PCR to diagnose pertussis in children. In children, hospitalisations coded as whooping cough have been shown to have a high correlation with clinical pertussis.[19] The high proportion (greater than 50%) of hospitalised cases aged less than one year is consistently observed each year and demonstrates the increased morbidity of pertussis in this age group.

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 10–19 years (Figure 26). 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.[150] The number of cases reported to be vaccinated for age may be an over-estimate, as the calculation did not include those with an "unknown" status (it is more likely that those recorded as unknown were not vaccinated). Studies show that a primary three-dose course of acellular pertussis vaccine provides 80%–85% protection.[76]

High incidence rates among 10–19 year olds continued to occur in 2003 and 2004. The susceptibility of this cohort is explained by a combination of low historical coverage (whole-cell vaccine safety concerns in the 1980s) and waning immunity (cohort not eligible for school entry booster dose).[150,151] There has been a downward trend in the notification rate for this age group since 2002 and a sharper decline in the rate since 2004. This is likely to reflect the impact of the fifth dose of pertussis vaccine reaching this older cohort and the impact of an adolescent booster vaccine (dTpa), introduced in November 2003. In response to the high incidence rate in adolescents, both New South Wales and Western Australia

conducted whole of high school dTpa vaccination programs in 2004. The combined incidence rate for 10–19 year olds in these states decreased from an average of 85.7 per 100,000 population for 1999–2003 to 37.2 per 100,000 population in 2005.[152] As Australian school-based dTpa programs mature and successive cohorts are vaccinated in future years, pertussis in adolescents should become well controlled, as occurred in 5–9 year olds following the introduction of the preschool booster.

In essence, pertussis is now a problem in two broad age groups: infants with the highest notification and hospitalisation rates, particularly those under 6 months of age who are too young to have received two or more doses of DTPa, and people aged 20 years and over, who account for 80% of pertussis notifications. The latter could be partly related to the increased use, especially in adults, of serology as a diagnostic tool (NNDSS data from 2000–2005 shows an increasing percentage of notifications diagnosed by serology (Quinn H et al, NCIRS, unpublished data)). In addition, recommendations for use of pertussis vaccine in adults make it increasingly likely that clinicians will consider pertussis as a potential cause of chronic cough in adults. Hospitalisations in adults are most likely to be related to complications, but could also be falsely inflated because of coding errors. Although severe morbidity and mortality are less likely in adults, increased circulation of pertussis can facilitate transmission to susceptible infants who are too young to be vaccinated.[153–155] The recent increase in the incidence and burden of pertussis notifications in persons aged 60 years and over warrants further investigation. As with parents, grandparents are an important source of pertussis transmission to infants.[153] It is also unclear whether the morbidity of pertussis in older people is more severe, or if complications are more likely to occur. With this in mind, it is interesting to note that two of eight pertussis deaths in the past five years have been recorded in people aged 60 years and over.[3] The current adult pertussis immunisation strategy in Australia is aimed at cocooning infants, by recommending immunisation in adults who are most likely to come into contact with them (such as family members, health care workers and child care workers). It is hoped that, in time, this may 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.[156]

Case definitions

Notifications

Invasive pneumococcal disease has been notifiable in the Northern Territory since 1995 and in Queensland 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.

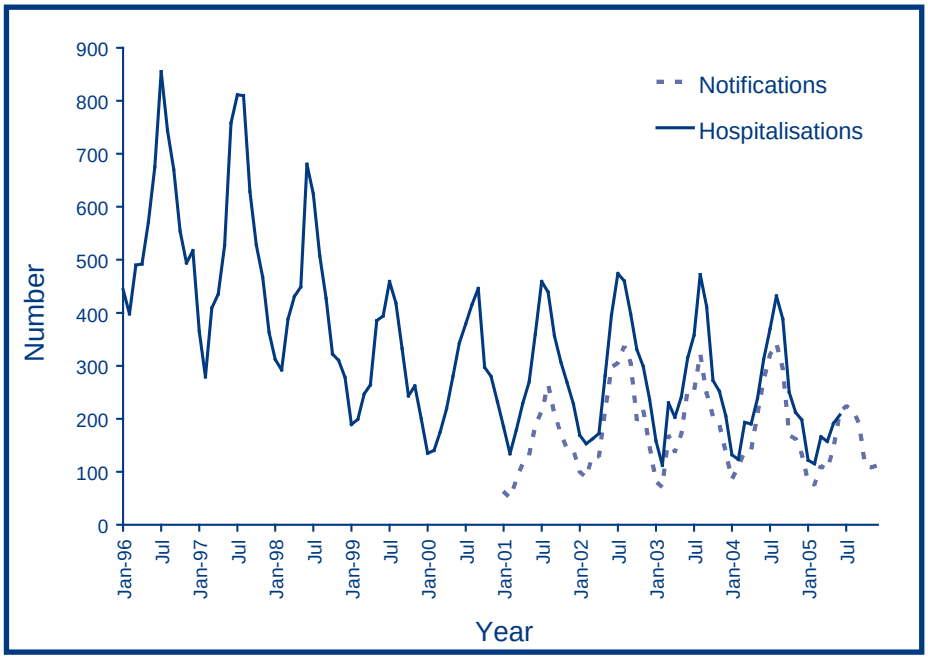
Secular trends

A total of 6,303 notifications of invasive pneumococcal disease were received for the three year period (2,237 in 2003, 2,377 in 2004 and 1,689 in 2005), an average annual notification rate of 10.5 per 100,000 (Table 15). In all years, there was a winter peak in pneumococcal notifications in August (Figure 29).

The total number of hospitalisations coded as pneumococcal meningitis, septicaemia or pneumonia between July 2002 and June 2005 was 9,543, an average annual rate of 16.0 per 100,000 (Table 15). In 69% of these, pneumococcal disease was recorded as the principal cause of the hospitalisation. Hospitalisations coded as meningitis or septicaemia accounted for 33% of total episodes giving a hospitalisation rate of 5.2 per 100,000. The median number of hospitalisations per month was 73 for meningitis or septicaemia (predominantly septicaemia) and ranged from 31 to 170. For pneumococcal pneumonia, the median number of hospitalisations per month was 161 (range 77–346). Hospitalisations showed a clear winter peak each year between July and September (Figure 29).

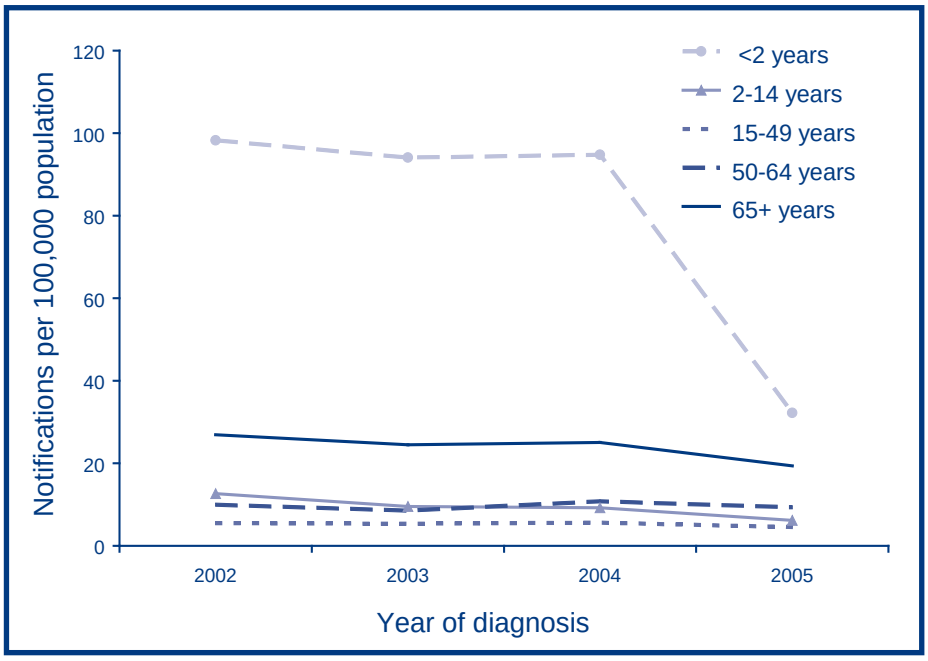
Notification rates and hospitalisation rates decreased in 2005 compared to previous years, coinciding with the commencement of universal funded vaccination for infants and the elderly. Notification rates in those aged less than two years were 66% lower in 2005 compared to the average for the previous three years (Figure 30), and 24% lower in those age 65 and over (Figure 31). There were also decreases in age groups not targeted for vaccination; 41% in those aged 2–14 years, 16% in those 15–49 years and 4% in those aged 50–64 years. Hospitalisation rates for the 2004/2005 financial year were also lower than the average of the previous five years, by 37% for those aged less than two years, 22% for those aged 65 and over, and 5% to 25% in other age groups.

Figure 29. Pneumococcal disease notifications and hospitalisations, Australia, January 1996 to December 2005,* by month of diagnosis or admission



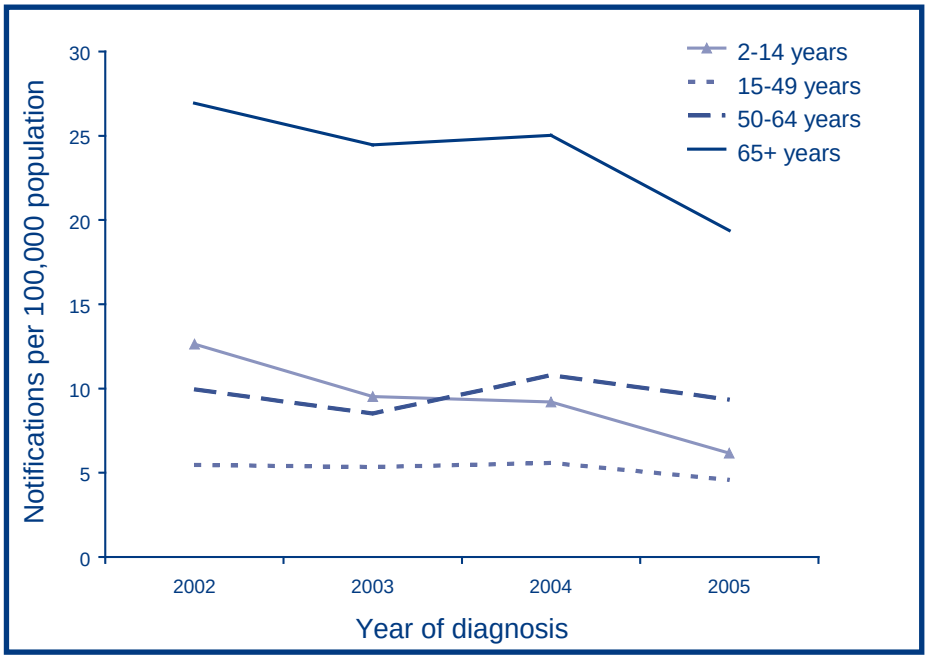
* Notifications where the month of diagnosis was between January 2001 and December 2005; hospitalisations where the month of admission was between 1 January 1996 and 30 June 2005. Hospitalisations include pneumonia, meningitis and septicaemia.

Figure 30. Pneumococcal disease notification rates, Australia, 2002 to 2005,* by age group



* Notifications where the date of diagnosis was between 1 January 2002 and 31 December 2005.

Figure 31. Pneumococcal disease notification rates, Australia, 2002 to 2005,* by age group (excluding <2 years)



* Notifications where the date of diagnosis was between 1 January 2002 and 31 December 2005.

Severe morbidity and mortality

A total of 93,691 hospital bed days (average 31,230 days per year) was recorded for hospital separations with an ICD-10-AM code corresponding to pneumococcal meningitis, septicaemia or pneumonia. The median length of stay increased with age in all categories of infection (Table 15). The average length of stay was greater than the median. For pneumococcal meningitis, the average was 13.5 days in all age groups, higher than that for septicaemia (10.4) or pneumonia (9.3).

Table 15. Pneumococcal disease notifications, hospitalisations and deaths, Australia, 2002 to 2005,* by age group

Age group (years)	Notifications 3 years (2003–2005)		Hospitalisations 3 years (July 2002–June 2005)				LOS† per admission (days)		Deaths 2 years (2003–2004)			
	n	Rate‡	n§	(M/S)‖	Rate‡,§	(M/S)‡,‖	Median§	(M/S)‖	n§	(M/S)‖	Rate‡,§	(M/S)‡,¶
0–4	1,712	45.2	1,396	(831)	36.8	(21.9)	3.0	(3.0)	13	(11)	0.5	(0.4)
5–14	258	3.2	302	(129)	3.7	(1.6)	3.0	(3.0)	0	(0)	0.0	(0.0)
15–24	247	3.0	357	(86)	4.4	(1.1)	4.0	(5.0)	2	(1)	0.04	(0.02)
25–59	1,935	6.6	3,255	(908)	11.1	(3.1)	5.0	(8.0)	20	(10)	0.1	(0.1)
60+	2,151	20.4	4,233	(1,161)	41.2	(11.3)	8.0	(9.0)	47	(16)	0.7	(0.2)
All ages	6,303	10.5	9,543	(3,115)	16.0	(5.2)	6.0	(7.0)	82	(38)	0.2	(0.1)

* Notifications where the month of diagnosis was between January 2003 and December 2005; hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005; deaths where the date of death was recorded between 2003 and 2004.

† LOS = length of stay in hospital.

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

§ All pneumococcal disease.

‖ (M/S) = meningitis and septicaemia.

Of the 82 registry-reported pneumococcal deaths in 2003 to 2004, 44 (54%) were from pneumonia, 20 (24%) from meningitis and 18 (22%) from septicaemia. Mortality rates from meningitis and septicaemia were highest in the very young and the elderly, while the pneumonia death rate was highest in the elderly (Table 15).

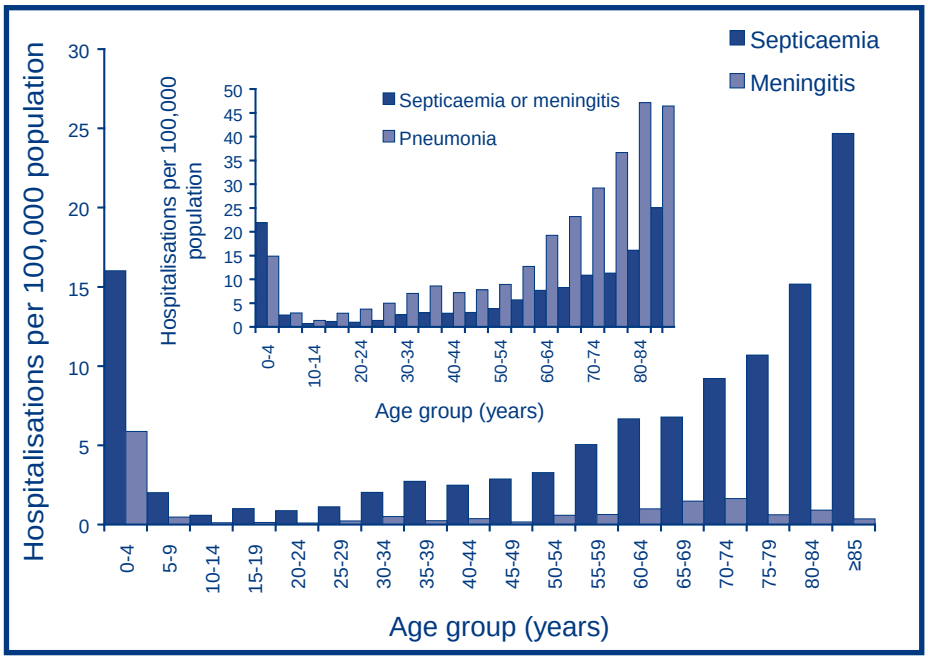
There were 361 deaths from pneumococcal disease in notified cases reported to NNDSS between 2003 and 2005, 6% of notified cases. The death rate increased with age, highest at 20% for those aged 85 years and over and 13% for those aged 65 years and over.

Age and sex distribution

Males had higher notification and hospitalisation rates compared to females in all age groups, and all ages combined (1.3 times and 1.4 times higher, respectively). Notification and hospitalisation rates were highest in the very young and the very old, although rates in infants decreased to a greater extent than other age groups in 2005 compared to previous years (Figure 30 and Figure 31). The hospitalisation rate for each age group varied with the focus of infection (Figure 32). For meningitis, rates were highest in those aged less than five years (6 per 100,000). By contrast, the incidence of hospitalisation for septicaemia without meningitis was also high in young children (16.0), but in adults increased with age to be highest in the elderly (24.7 in those 85 years and over).

When total hospitalisations (meningitis, septicaemia and pneumonia) were considered, adults aged 60 years and over had the highest total rate of hospitalisation (41.2 per 100,000, Table 15).

Figure 32. Pneumococcal meningitis, septicaemia and pneumonia hospitalisation rates,* Australia, 2002 to 2005, by age group



* Hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005.

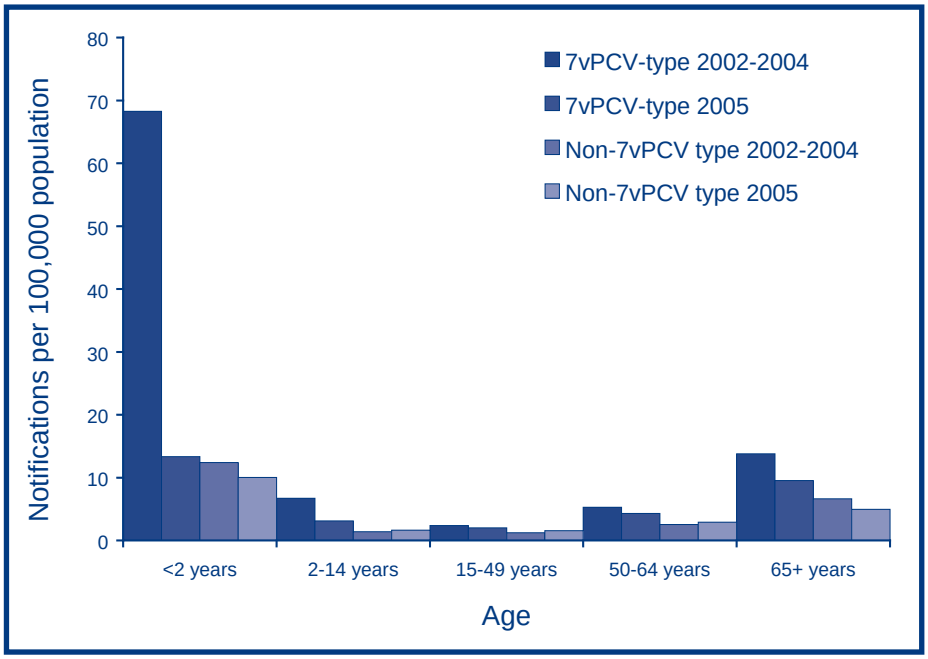
Geographical distribution

The average annual notification rate was 10.5 per 100,000 for Australia, highest in the Northern Territory (39.4, 3.8 times higher than the national rate) and ranged from 7.8 to 13.0 in other jurisdictions. A similar distribution was seen in the hospitalisation rate for pneumococcal meningitis or septicaemia (national rate 5.2), which was higher in the Northern Territory (22.1, 4.3 times higher than the national rate), with little difference between the other jurisdictions (4.2 to 5.8).

Pneumococcal typing

Data on the serotype of cases reported to NNDSS were available from 6,749 of 8,769 cases (77%) from 2002 to 2005, the period when surveillance has been carried out in all jurisdictions. In the pre-vaccination period of 2002 to 2004, 73% of serotyped cases were caused by serotypes contained in the 7-valent conjugate pneumococcal vaccine (7vCPV), used in infants, and 85% of serotyped cases in children aged less than two years. Cases caused by serotypes in the 23-valent pneumococcal polysaccharide vaccine (23vPPV), used in high-risk children and the elderly, were 92% of the total and 90% in those aged 65 years and over. In 2005, the notification rate in 7vPCV-type cases decreased by 80% in those aged less than two years, compared to the average of the previous three years (Figure 33), and by 43% in all ages combined (Table 16). Decreases were seen in all age groups, but they were less marked in younger adults. There was little change in all-age notification rates of cases with serotypes contained in the 23vPPV but not in the 7vPCV (23v-non-7v types), or in serotypes not in any vaccine, in 2005 compared to the previous three years. In those aged 65 years and over, there were decreases in all serotype groupings, i.e. 7vPCV types, 23v-non-7v types, and non-vaccine types. In age groups not included in universal funded vaccination programs (2–64 years), notification rates were higher in 2005 compared to 2002 to 2004 for serotypes not included in the 7vPCV, but total rates were lower, due to decreases in 7vPCV-type cases.

Figure 33. Notification rates of cases with serotypes contained in the 7-valent pneumococcal conjugate vaccine (7vPCV), versus rates for other serotypes, Australia, 2002 to 2005*



* Notifications where the date of diagnosis was between 1 January 2002 and 31 December 2005.

Vaccination status

Data on vaccination status were available for 89 (52%) of the 172 cases recorded on NNDSS in children eligible for a full course of the funded universal vaccination and catch-up programs (from 1 January 2005, children born from January 1 2003 and aged more than 6 months at disease onset). Of those, 22 (25%) were reported to be fully vaccinated, and this was validated by vaccination records for 18. Partially completed vaccination was reported by 26 (29%) and 41 (46%) were unvaccinated. In cases aged 65 years and over, vaccination status was reported for 936 of 1,782 (53%). Of those, 35% were reported as fully vaccinated, 11% partially vaccinated and 55% unvaccinated. Information on the source of vaccination status data was available in 709 (76%) of those records, and 94% were reported to be from written vaccination records.

Table 16. Invasive pneumococcal disease notification rates,* Australia, 2005 as a percentage of average annual rates from 2002 to 2004, by serotype grouping

Age group (years)	Serotype			Total‖
	7v†	23v-non-7v‡	Non-23v§	
<2	20	99	62	34
2–14	46	116	102	59
15–49	84	114	139	84
50–64	81	109	170	96
65+	69	85	62	76
All ages	57	103	98	70

* Notifications to the National Notifiable Diseases Surveillance System, per 100,000 population.

† Serotypes contained in the seven-valent pneumococcal conjugate vaccine (7vPCV); 4, 6B, 9V, 14, 18C, 19F, 23F.

‡ Serotypes contained in the 23-valent pneumococcal polysaccharide vaccine but not in the 7vPCV; 1, 2, 3, 5, 7F, 8, 9N, 10A, 11A, 12F, 15B, 17F, 19A, 20, 22F, 33F.

§ Serotypes not included in either vaccine.

‖ Includes untyped cases.

Comment

Invasive pneumococcal disease has historically been a disease predominantly affecting the very young, the very old and those with certain chronic diseases or other high-risk conditions. In particular, much higher rates of disease have been reported in Aboriginal and Torres Strait Islander young children and adults compared to non-Indigenous Australians.[157–159] Vaccination with the 23vPPV has been funded nationally since 1999 for Aboriginal and Torres Strait Islander adults aged 50 years and over, and those aged 15–49 years with high-risk conditions.[3] Nationally funded 7vPCV vaccination commenced in 2001 for all Aboriginal and Torres Strait Islander infants and other infants with high-risk conditions.[3] In 2005, the 7vPCV was funded for all Australian infants and the 23vPPV for all aged 65 years and over.

Reports on IPD national notification data have been published since 2001.[160–163] A decrease in rates in Indigenous children following the targeted infants vaccination program has been previously reported, with a threefold higher rate in Indigenous compared to non-Indigenous children under two years of age in 2001 reduced to parity in 2004.[163] Data in this report suggest a noticeable impact in the first year of universal infant vaccination, as the IPD notification rate declined by 66% in those aged less than two years in 2005 compared to the three previous years, and 80% in cases with serotypes contained in the vaccine. Smaller decreases occurred in all other age groups, but these should be interpreted with caution due to the smaller numbers of cases in these age groups. Hospitalisation rates were lower in 2004/2005 compared to previous years, although this included only the first six months of the funded vaccination program. There were decreases in all age groups, but the greatest decrease was seen in infants (37%). Similar impacts have been seen in the USA following the commencement of universal infant vaccination in 2001; an estimated 25,000 cases were prevented in 2003, and 49% of those were in non-immunised age groups, presumably due to reductions in nasopharyngeal carriage and, therefore, fewer transmissions to the unvaccinated.[164]

Hospitalisation rates for pneumococcal disease were approximately 30% higher than IPD notification rates. The hospitalisation codes selected were less specific for identifying invasive disease than the IPD case definition, as they include pneumococcal pneumonia without bacteraemia. In addition, hospitalisations coded as due to bacteraemia have been previously shown to have low sensitivity.[165] Hospitalisations for pneumococcal meningitis and septicaemia number approximately half those of IPD notifications. It is, therefore, likely that pneumococcal disease hospitalisations are under-estimated by these data.

In addition to preventing IPD, the 7vPCV has been shown to have some efficacy against the much more common manifestations of non-bacteraemic pneumococcal pneumonia and otitis media.[166,167] Therefore, decreases in IPD notification rates represent only a proportion of the total impact of this vaccine. Continued monitoring of hospitalisation rates for pneumococcal pneumonia will be valuable in monitoring some of this impact. The decrease in hospitalisation rates from 1996 to 1999 shown here is

likely to be related to coding practices rather than an actual decrease in incidence, as all-cause pneumonia hospitalisation rates did not change over time (data not shown). However, the more stable pattern from 1999 provides a useful baseline for monitoring the impact of vaccination in the future.

Concerns about replacement disease by serotypes not contained in the vaccine have been expressed.[167] In the USA, some replacement IPD appears to have occurred, but there is still a substantial overall decrease in IPD notifications.[164] The data presented here for Australia on the first year of the vaccination program suggest the possibility of some replacement disease in unvaccinated age groups, but a substantial overall decline in IPD rates. It will be important to monitor this closely in the future.

The 23vPPV has been shown to be effective against IPD in elderly populations without high rates of risk factors, but effectiveness against nasopharyngeal carriage and pneumococcal pneumonia have not been established.[43,168] Low vaccine effectiveness has been found in overseas studies in those with chronic disease and high-risk conditions, including indigenous populations.[169] Vaccination programs for Indigenous adults from the early 1990s in the Kimberley and mid-1990s in northern Queensland coincided with reductions in IPD incidence in Indigenous adults in those regions.[158,170] However, no decrease in incidence has been seen in Northern Territory Indigenous adults, despite vaccination programs targeting those aged 50 years and over since 1995 and all Indigenous adults since 2000.[171] Funded vaccination for all Victorians aged 65 years and over since 1998 has been effective in that state,[172] and a similar impact could be expected nationally. The data reported here include the period up to the end of the first year of nationally funded vaccination for those aged 65 years and over. Although decreases were seen in hospitalisation and notification rates in this age group in 2005 compared to previous years, the decreases in notifications occurred across a wide range of serotypes and may be attributable to adult vaccination, herd immunity from childhood vaccination, and/or other reasons. It will be necessary to examine this in later years when more data become available. Reported deaths were predominantly in adults, with the highest death rates in the oldest age groups. There were 2.9 times as many pneumococcal deaths reported through NNDSS compared to registry reports, even though registry deaths included the broader category of all pneumococcal pneumonia, suggesting under-reporting of this cause of death by this mechanism.

The Northern Territory has the highest disease burden and there is little difference between the other jurisdictions. Disparities between Indigenous and non-Indigenous children may re-emerge following universal vaccination, due to higher rates of non-vaccine type disease in Indigenous people.[163] New conjugate vaccines with greater serotype coverage may be necessary to address this and the persistently high rates in Northern Territory Indigenous adults.

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% of infections. More than 90% of infections are asymptomatic, with a minor illness characterised by fever, headache, malaise and nausea/vomiting occurring in about 10%. The maximum extent of paralysis is usually reached within three to four days of disease onset. Any paralysis still present after 60 days is likely to be permanent.[44]

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 Sabin live attenuated oral poliovirus vaccine (OPV)). A vaccine derived poliovirus (VDPV) is defined as having 1%–15% nucleic acid sequence variation from the prototype Sabin strain. The variation is due to long-term (more than one year) virus replication after administration of OPV. The virus replication may occur in an individual with an immunodeficiency (iVDPV) or through sustained person-to-person transmission in areas with low OPV coverage (circulating or cVDPV). VDPVs not clearly assigned to either of these categories are known as ambiguous VDPVs (aVDPV).[173]

Case definition

See Appendix 6 for pre-2004 definition

National definition from January 2004:[11]

Confirmed cases require laboratory definitive evidence and clinical evidence. Probable cases are also notifiable and require clinical evidence and that the case not be discarded as non-polio acute flaccid paralysis by the Polio Expert Committee.

a) Laboratory definitive evidence

- Isolation of wild poliovirus (or Sabin-like poliovirus for VAPP cases), confirmed in the WHO Western Pacific Regional Poliovirus Reference Laboratory; *or*
- Detection of wild poliovirus (or Sabin-like poliovirus for VAPP cases) by nucleic acid testing, confirmed in the WHO Western Pacific Regional Poliovirus Reference Laboratory.

b) Clinical evidence

- Acute flaccid paralysis (AFP): acute onset of progressive weakness and flaccidity of one or more limbs with decreased or absent tendon reflexes in the affected limbs or bulbar palsy without other apparent cause, and without sensory or cognitive loss.

Hospitalisations and deaths

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

Note: This code includes VAPP and specific codes for indigenous and imported wild-type poliovirus infection. Sequelae of poliomyelitis (ICD-10 code B91) were not included in these analyses.

Notifications, hospitalisations and deaths

No notifications or deaths were recorded for poliomyelitis in 2003, 2004 or 2005. From July 2002 to June 2005 there were 60 hospitalisations with a diagnosis of acute poliomyelitis. Of these, one was coded as VAPP, seven as acute non-paralytic poliomyelitis and 14 as other/unspecified acute paralytic poliomyelitis. The remaining 38 hospitalisations were coded as acute unspecified poliomyelitis. The majority of separations recorded as acute poliomyelitis were in older persons (92% aged 50 years and over). The number of hospitalisations reported as acute poliomyelitis decreased over the period with 39 cases in 2002/2003, 20 in 2003/2004 and only one case in 2004/2005. Only five hospitalisations were recorded as having a principal diagnosis of poliomyelitis (all from New South Wales in the period 2002/2003).

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.[174] Australia and the Western Pacific Region were declared polio-free in October 2000.[175] The most recent case of VAPP was reported in 1995.[176,177] 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, as indicated by the age distribution of the hospitalisations. The most recent hospitalisation data, with only one case coded as acute polio unspecified in an elderly male in 2004/2005, is encouraging in relation to possible improvements in coding practices. The hospitalisation case coded as VAPP in 2002/2003 would be worthwhile investigating, although it could be a coding error.

Although Australia has been declared polio free, achieving high quality AFP surveillance remains an important challenge. Such surveillance is required to detect any imported cases of wild-type polio infection, cases of VAPP, and outbreaks of circulating vaccine-derived polioviruses (cVDPV). In Australia, surveillance of AFP in children under 15 years of age is co-ordinated through the Victorian Infectious Diseases Reference Laboratory in collaboration with the Australian Paediatric Surveillance Unit (APSU). AFP cases are notified and stool specimens are referred to the Australian National Poliovirus Reference Laboratory for testing for polio and other enteroviruses. Cases are referred to the Polio Expert Committee for a determination as to the cause of the AFP. In 2004, Sabin-like poliovirus types 1 and 2 were isolated from a child presenting with AFP. However, the polioviruses were determined to be an incidental finding with the AFP determined as due to infant botulism.[178] In 2005, three Sabin-like polioviruses (two isolations of PV3 and one of PV2) were isolated from three patients with AFP, with all considered to be incidental isolations in patients recently vaccinated with OPV.[179] A recent review of all specimens referred for testing from AFP cases during 1996–2004 identified no cases as due to polio, with enterovirus 71 now the most common viral cause of AFP. Infant botulism is an important differential diagnosis.[180] Three cases of notified AFP were due to infant botulism in 2005.[181] AFP cases notified between 1995 and 1999 have also been reviewed, with Guillain-Barré syndrome and transverse myelitis identified as the most common diagnoses.[182] In 2005, these two diseases continued to be the primary diagnoses responsible for presentations with AFP.[181]

The WHO target for surveillance of AFP (one notified case of AFP per 100,000 children aged less than 15 years) has only been intermittently achieved in Australia (in 2000, 2001 and 2004). The WHO target of faecal sampling from 80% of AFP cases has never been achieved, with the 40% sampling rate achieved in 2004 the highest to date (previously 24–36% and falling to 19% in 2005).[178,179] Variability in the adequacy of AFP reporting amongst the populous states of Australia has been noted, perhaps relating to the degree of clinician awareness of AFP surveillance protocols locally, which in turn may be related to the differing structure of public health networks and laboratories in different states.[183] A capture-recapture study, comparing AFP surveillance reports with hospital records, has indicated that, in Victoria, failure to reach the WHO target rate of one per 100,000 is due to under-reporting rather than to local variation in AFP rates.[184]

The global aim to eradicate polio by 2000 has proven elusive. During the period 2002–2005, there was a resurgence of poliovirus 1 and importation into 21 previously polio free countries, due to postponement of eradication activities in Nigeria in 2003 and, to a lesser extent, importations from India.[185] In eight countries, wild poliovirus transmission was not sustained, whereas in the other 13 countries, sustained transmission occurred. Four countries (Indonesia, Somalia, Sudan, and Yemen) had outbreaks of over 100 polio cases. Countries where transmission was not sustained had a median three-dose vaccination coverage of 83%, compared with only 52% in those where transmission occurred, underscoring the ongoing importance of high polio vaccine coverage even in countries where polio has been locally eradicated.[185] Control strategies for affected countries have involved large scale supplementary immunisation activities and underscored the critical role of sensitive AFP surveillance.

In 2006, endemic transmission of wild-type poliovirus is now constrained to four countries (an all-time low): Afghanistan, India, Nigeria and Pakistan.[186] However, 10 countries have reported polio cases due to importations in 2006.[187] The Global Polio Eradication Initiative Strategic Plan 2004–2008[188] has four main objectives. The most urgent is the rapid interruption of polio transmission in the remaining endemic countries. This requires high level political and resource commitments and, since 2005, the use of monovalent oral poliovirus vaccine (OPV) in order to provide sufficiently high levels of immunity.[186] The other three objectives underscore the need to ensure adequate surveillance (Objective 2: Achieve certification of global polio eradication); to move away from OPV to inactivated poliovirus vaccine (IPV) once the risk of VAPP exceeds that from wild poliovirus (Objective 3: Develop products for the global OPV cessation phase); and to integrate poliovirus control and maintenance strategies into routine disease control structures (Objective 4: Mainstream the Global Polio Eradication Initiative).

In 2003, the Australian Government recommended, but did not fund, the use of IPV in place of OPV.[76] From November 2005, IPV became a funded part of the routine childhood immunisation schedule in Australia with doses given at 2, 4 and 6 months and 4 years of age. This policy was facilitated by the availability of combination IPV vaccines and removes the risk of VAPP while providing protection against polio importations.[189] With the replacement of OPV with IPV in Australia, incidental detection of polioviruses in faecal specimens should no longer occur. Future poliovirus isolations will, therefore, require full investigation.[179] An important goal in the diagnosis of all AFP cases is the exclusion of an imported wild or vaccine-associated poliovirus as the cause. The high frequency of arrivals from countries where poliovirus remains endemic and where cVDPV cases have been recently documented (such as China),[190,191] by Australian travellers, visitors and refugees, make such importations a distinct possibility. The likelihood of local transmission following importation will be dependent upon the vaccination coverage locally and living conditions primarily relating to the likelihood of faecal contamination of the water supply. Such contamination remains a possibility in rural and remote areas of Australia.[192] Travellers should be reminded to ensure that they are vaccinated against polio.[193]

Q fever

Q fever is a zoonotic disease caused by *Coxiella burnetii*. It infects both wild and domestic animals and their ticks, with cattle, sheep and goats the main source of human infection. Infected animals shed *C. burnetii* into the environment through their products of conception (in especially high numbers) but also in their urine, milk and faeces. Transmission to humans is via inhalation of infected droplets or dust. Q fever can be acute or chronic,[194] and there is increasing acceptance of an association with long-term sequelae including post Q fever fatigue syndrome.[195–197] However, in many instances infection is asymptomatic. Symptoms of the acute illness resemble those of influenza but can be complicated by encephalitis, pneumonitis, hepatitis, myocarditis and pericarditis. Chronic Q fever, which may occur years after infection, is most often characterised by endocarditis.[198]

A vaccine against Q fever, Q-VAX® (CSL Limited) was licensed in Australia in 1989 and, following successful Australian trials,[199–201] vaccination programs have been operating in most large abattoirs since the mid 1990s.[202] To improve vaccination coverage in the wider at-risk population, the Commonwealth Government funded the National Q fever Management Program (NQFMP).[203] The program was funded and delivered in two phases. Phase one targeted the meat and livestock industry, promoting screening and vaccination services to abattoir workers, workers contracted to abattoirs and sheep shearers ($10.6 million funding over three years). Phase two targeted sheep, dairy and beef cattle farmers, their employees and unpaid family members working on farms ($8 million funding over three years). The program was implemented in all states and the Australian Capital Territory during 2001–2002, with vaccination activity peaking in 2002.

Case definitions

See Appendix 6 for pre-2004 definition

National definition from January 2004:[11]

Confirmed cases require either laboratory definitive evidence or laboratory suggestive evidence and clinical evidence.

a) Laboratory definitive evidence
- Detection of *Coxiella burnetii* by nucleic acid testing; *or*
- Seroconversion or significant increase in antibody level to Phase II antigens in paired sera tested in parallel in absence of recent Q fever vaccination; *or*
- Detection of *C. burnetii* by culture.

b) Laboratory suggestive evidence
- Detection of specific IgM in the absence of recent Q fever vaccination.

c) Clinical evidence
- A clinically compatible disease.

Hospitalisations and deaths

The ICD-9 code 083.0 (for historical data) and ICD-10 code A78 were used to identify hospitalisations and deaths.

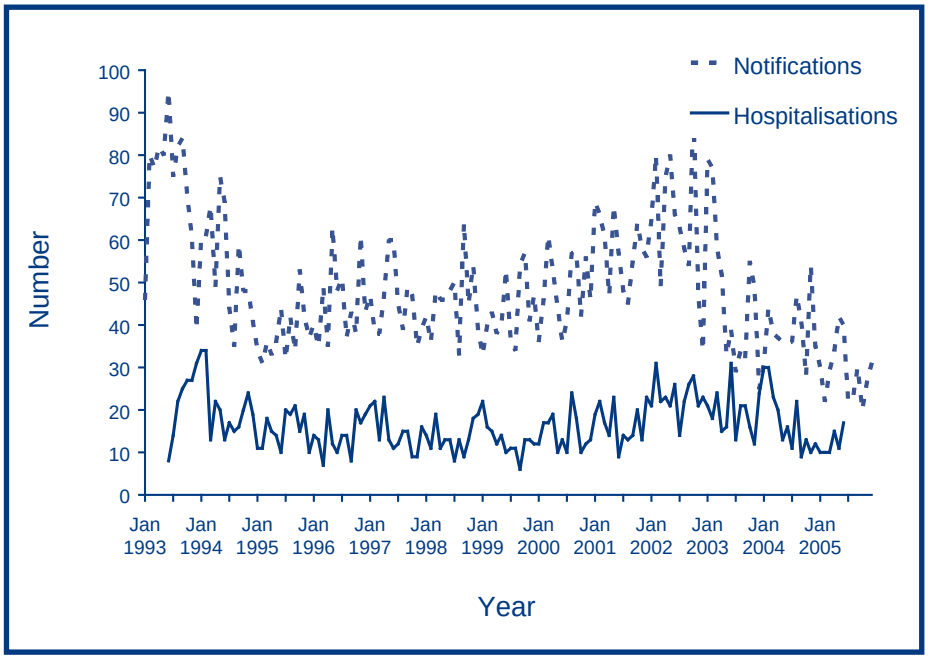
Secular trends

In the three year review period there were 1,375 notified cases of Q fever (average annual rate of 2.3 per 100,000) and 652 hospitalisations with the ICD-10 code A78 for Q fever (average annual rate 1.1 per 100,000). Both notification and hospitalisation rates declined over the review period, following a peak in 2002, to the lowest levels on record in the most recent year reviewed (1.7 and 0.8 per 100,000, respectively). There was a median of 36 (range 20–79) notifications and 17 hospitalisations (range 9–31) per month. The considerable monthly variation did not appear to be seasonal.

Severe morbidity and mortality

In the three year review period, hospital separations for Q fever accounted for 3,807 hospital bed days. The median length of stay (LOS) overall was 4 days but, in adults, the LOS increased with age (Table 17).

Figure 34. Q fever notifications and hospitalisations, Australia, 1993 to 2005,* by month of diagnosis or admission



* Notifications where the month of diagnosis was between January 1993 and December 2005; hospitalisations where the month of admission was between 1 July 1993 and 30 June 2005.

Of the 652 hospitalisations, 502 (77%) had Q fever recorded as the principal diagnosis. However, only 62 (54%) of the 115 hospitalisations in the 60 years and over age group were recorded as the principal diagnosis.

In the National Mortality database, four deaths were recorded with an underlying cause of death coded as Q fever in 2003–2004. All were in adults aged at least 25 years. Three were female and one was male. There were two deaths in notified cases reported to NNDSS between 2003 and 2005.

Table 17. Q fever notifications, hospitalisations and deaths, Australia, 2002 to 2005,* by age group

Age group (years)	Notifications 3 years (2003–2005)		Hospitalisations 3 years (July 2002–June 2005)				LOS† per admission (days)	Deaths 2 years (2003–2004)	
	n	Rate‡	n	(§)	Rate‡	(§)	Median (§)	n	Rate‡
0–4	5	0.1	3	(2)	0.1	(0.1)	5.0 (3.0)	0	–
5–14	39	0.5	14	(13)	0.2	(0.2)	3.0 (3.0)	0	–
15–24	173	2.1	88	(78)	1.1	(1.0)	2.0 (3.0)	0	–
25–59	953	3.2	432	(347)	1.5	(1.2)	4.0 (4.0)	3	0.02
60+	205	2.0	115	(62)	1.1	(0.6)	7.0 (7.0)	1	0.01
All ages	1,375	2.3	652	(502)	1.1	(0.8)	4.0 (4.0)	4	0.01

* Notifications where the month of diagnosis was between January 2003 and December 2005; hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005; deaths where the death was recorded in 2003 or 2004.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

Age and sex distribution

The male to female ratio for both notifications and hospitalisations has generally been decreasing over time since the early 1990s, but remained relatively constant over the three year review period at 3.3:1 and 3.8:1, respectively.

As in previous years, adult males aged 15–39 and 40–64 years had the highest notification and hospitalisation rates (Figure 35). In the 1990s males aged 15–39 years had the highest rates, but, since then, rates have been highest in males aged 40–64 years.

All age/sex groups showed a decline in notification and hospitalisation rates over the three year review period except the 65 years and over age group which had a peak in hospitalisations in 2003/2004. This oldest age group also contributed proportionally more hospitalisations (13%) than notifications (8%) during the review period.

Geographical distribution

As in previous years, notification and hospitalisation rates were highest in Queensland followed by New South Wales (Appendix 2 and Appendix 3). Together, these two states contributed 86% of the notifications and 83% of the hospitalisations for Q fever over the review period.

Consistent with national trends, notification rates in both of these states declined to record low levels over the three years reviewed with only a few outbreaks reported. There were six small clusters of Q fever cases reported nationally in 2003 with five of these in Queensland.[13] In 2004, a cluster of ten cases associated with a shearing team was reported in New South Wales.[204] Rates in other jurisdictions remained low over the review period. The exceptions were: South Australia, which experienced an outbreak associated with a sheep saleyard in late 2004;[12] and the Northern Territory which reported three cases in each of 2004 and 2005 when prior to 2004 there had only been two cases in total reported since 1991. The three cases in 2004 were unrelated. One was a foreign national working on a cattle boat[205] and two cases were from Central Australia, one of whom died.[206]

Hospitalisation rates tended to follow the same pattern as notifications, except that there were few hospitalisations reported from South Australia during the 2004 outbreak.

Vaccination status

Vaccination status is requested on all Q fever notifications but was recorded only for 903 (66%) of the 1,375 notified cases in 2003–2005. Fourteen cases were recorded as being fully vaccinated, six of whom had their vaccination status validated by written records.

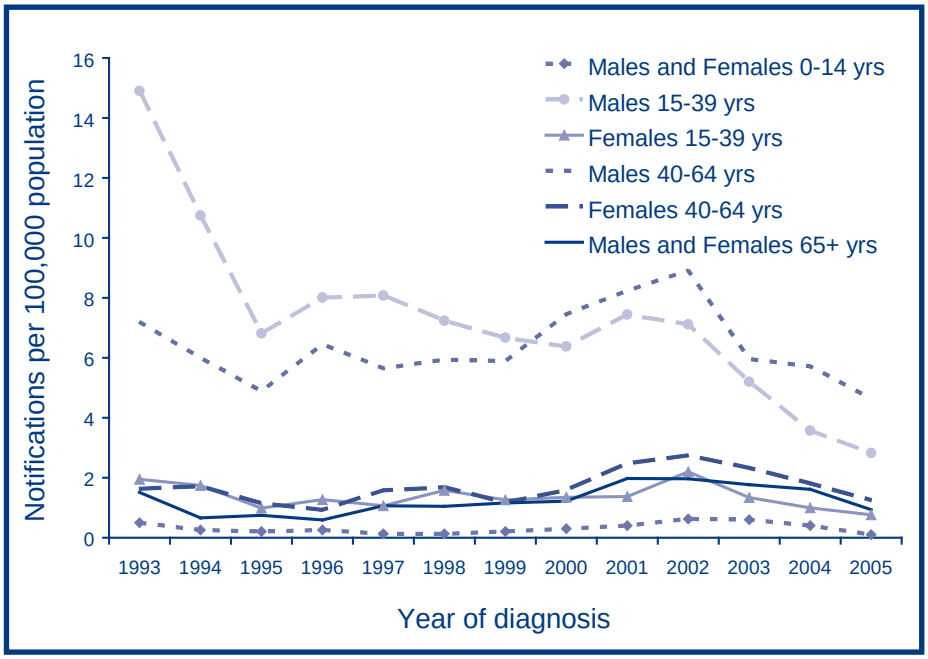
Comment

Q fever notification and hospitalisation rates declined to record low levels during the review period. The decline follows an increase in screening and vaccination activity associated with the NQFMP; phase one was fully implemented by the end of 2002 while phase two activity was predominantly in 2003. These data are supported by a decline in claims for workers' compensation for Q fever over this period.[207] It appears, therefore, that the NQFMP has contributed to a reduction in the burden of Q fever in Australia, although it is difficult to estimate the contribution of non-program factors such as an improvement in drought conditions (droughts promote airborne dissemination) and variation in livestock slaughtering. However, the reductions are most noticeable in young adult males who were the main target in phase one of the program.

Despite these promising findings, longer term and enhanced surveillance is required to fully evaluate the impact of the NQFMP. Most jurisdictions completed the program by 2005, but Victoria and South Australia continued until 30 June 2006, and Queensland will continue until 30 June 2007.[12] Ongoing surveillance is, therefore, required to detect variations between states and following completion of the program. Although there have been recent publications using enhanced state notification data to document trends in Q fever notifications by occupation,[208–211] there is a need for enhanced data at a national level, including the occupation of cases and complete information about their vaccination status. Such data will help to better determine the targeted impact of the NQFMP, further understand the epidemiology of Q fever, and identify the most appropriate vaccination strategies for the future.

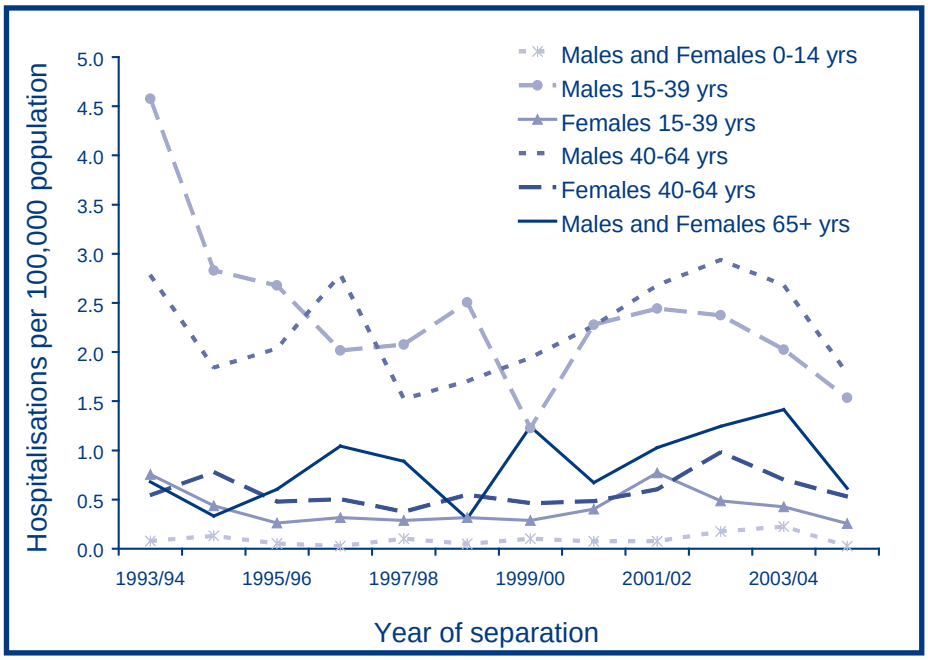
Australia is the only country to have implemented a vaccination program of any kind, even though Q fever has been found world-wide (except in New Zealand).[195,212,213] Large outbreaks continue to be reported from many countries.[214–216] However, annual rates in North America, the United Kingdom and most of Europe

Figure 35. Q fever notification rates, Australia, 1993 to 2005,* by age group and sex



* Notifications where the date of diagnosis was between 1 January 1993 and 31 December 2005.

Figure 36. Q fever hospitalisation rates, Australia, 1993 to 2005,* by age group and sex



* Hospitalisations where date of separation was between 1 July 1993 and 30 June 2005.

are low compared with Australia. In 2000–2004, an average of only 51 cases was reported each year in the USA[217] and, since 2000, the number of reported cases in the UK has declined annually to less than 50 sporadic cases each year.[218] The variation between countries may be the result of true epidemiological differences, but could also be due to differences in awareness, surveillance, or diagnostic methods.

In summary, there appears to be an impact of the NQFMP on the burden of Q fever in Australia, but confounding factors are difficult to exclude. Q fever surveillance data must, therefore, be monitored over a longer period of time to fully assess the impact of the program. In addition, more complete national surveillance data on the occupation and vaccination status of cases is recommended.

Rotavirus

Rotavirus is a non-enveloped virus that is the major cause of acute gastroenteritis in young children and infants. Virtually all children worldwide are infected with rotavirus by 3–5 years of age. However, rotavirus occurs in all age groups.[219] Rotaviruses are primarily spread by faecal-oral transmission. Infection can be asymptomatic, cause mild to moderate gastroenteritis, or severe gastroenteritis with dehydration requiring hospitalisation.[219] Infection with rotavirus often confers some protection against subsequent disease.[220] Severe disease occurs most commonly in those aged 6 months to 2 years.[221,222] Rotaviruses are typed based on two surface proteins, VP7 (G protein) and VP4 (P protein). Viruses that contain either G1, 2, 3, 4 or 9 (and either P1a or P1b) are the five most common virus types currently circulating in Australia.[223]

Two vaccines became available in Australia in 2006 for the prevention of rotavirus gastroenteritis. They are to be funded under the National Immunisation Program (NIP) for all infants born from 1 May 2007. The Northern Territory has made one of the rotavirus vaccines available free of charge since 1 October 2006 for infants born after 1 August 2006. Both products are oral live attenuated vaccines for use in infants in either a two-dose course at 2 and 4 months of age (Rotarix®, GlaxoSmithKline), or a three-dose course at 2, 4, and 6 months of age (RotaTeq®, CSL Limited/Merck and Co Inc). Overall, vaccination is likely to prevent around 70% of rotavirus gastroenteritis of any severity and between 85%–100% of cases of severe gastroenteritis in immunised infants/children.[224,225]

Case definitions

Notifications

Rotavirus is not a nationally notifiable disease. Rotavirus became notifiable in the Northern Territory in 1994. Rotavirus became laboratory notifiable in Queensland in December 2005. Other states and territories are currently adopting passive surveillance for rotavirus infections.

Hospitalisations and deaths

The ICD-9 code 008.61(for historical data) and ICD-10 code A08.0 (rotaviral enteritis) were used to identify hospitalisations and deaths. Historical death data were reviewed from 1990–2005.

Northern Territory notification data

Northern Territory notification data for the period January 2003 to December 2005 were included in this report. Rotavirus has been notifiable in the Northern Territory since 1994. Historical notification data is published elsewhere.[226] Cases notified in the Northern Territory meet the following case definition: 'Detection of human rotavirus in stool, unless typing reveals it is rotavirus from a vaccine'.

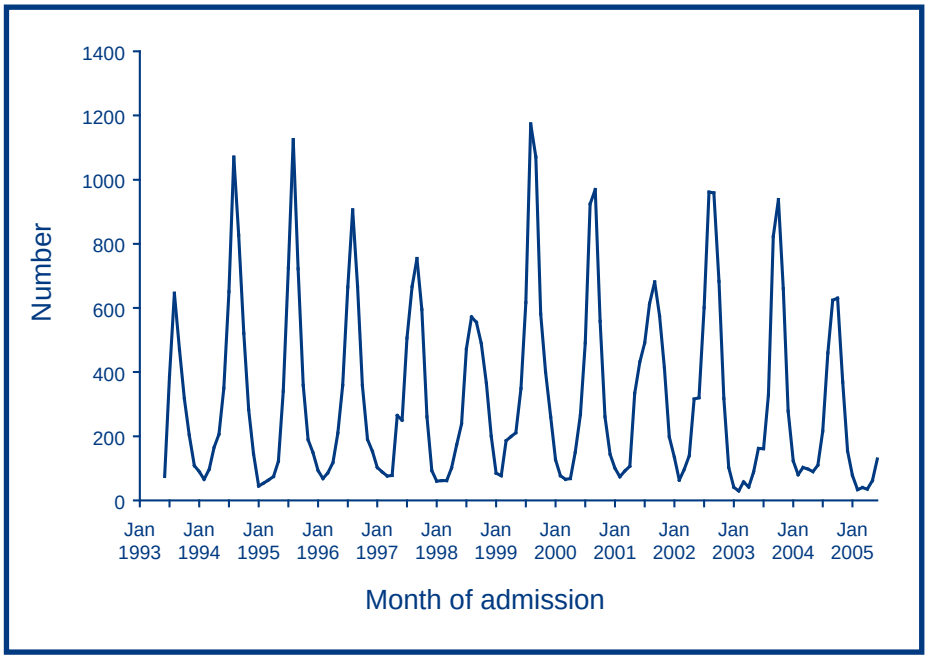
Secular trends

In the three year review period (2002/2003 to 2004/2005), there were 10,733 hospitalisations for rotavirus (average annual rate of 18.0 per 100,000 total population). Rotavirus was recorded as the principal diagnosis in 89% of these hospitalisations (Table 18). There were more hospitalisations recorded in the first year of the review period than in the subsequent two years (2002/2003 n=4,071, 2003/2004 n=3,803, 2004/2005 n=2,859). In the previous three year period (1999/2000–2001/2002), the number of hospitalisations for rotavirus gastroenteritis was greatest in 1999/2000 before declining (Figure 39), consistent with known fluctuations in rotavirus disease activity between years.[227] Rotavirus hospitalisations in temperate regions in Australia have a consistent seasonal pattern, with higher rates in the cooler months of the year from June to November. Data in the period reviewed here was consistent with this observation, with a low of 30 hospitalisations in February 2003 and a peak of 961 hospitalisations in August 2002 (Figure 37).

Severe morbidity and mortality

The number of bed days and median length of stay (LOS) were calculated only for those hospitalisations with a principal diagnosis of rotavirus. There were a total of 23,454 bed days recorded for rotavirus (average 7,818 per year). The median LOS was two days.

Figure 37. Rotavirus hospitalisations, Australia, 1993 to 2005,* by month of admission



* Hospitalisations where the month of admission was between 1 July 1993 and 30 June 2005.

The National Mortality Database 'cause of death' mortality records indicate a total of 14 deaths due to rotavirus over the last 15 years (1990–2004). Seven of the deaths occurred in children under five years of age, three in those aged 5–69 years, and four in adults aged over 70 years. Since 2001, only one death has been recorded.

Table 18. Rotavirus hospitalisations and deaths, Australia, 2002 to 2005,* by age group

Age group (years)	Hospitalisations 3 years (July 2002–June 2005)				LOS† per admission‡ (days)	Deaths 2 years (Jan 2003–Dec 2004)	
	n	(‡)	Rate§	(‡)	Median	n	Rate§
0–4	9,662	(8,601)	254.5	(226.5)	2.0	0	0
5–14	841	(779)	10.3	(9.6)	2.0	0	0
15–24	26	(18)	0.3	(0.2)	2.0	0	0
25–59	64	(50)	0.2	(0.2)	3.0	0	0
60+	140	(86)	1.4	(0.8)	3.0	1	0.01
All ages	10,733	(9,534)	18.0	(16.0)	2.0	1	0.003

* Hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005; deaths where the death was recorded in 2003 or 2004.

† LOS = length of stay in hospital.

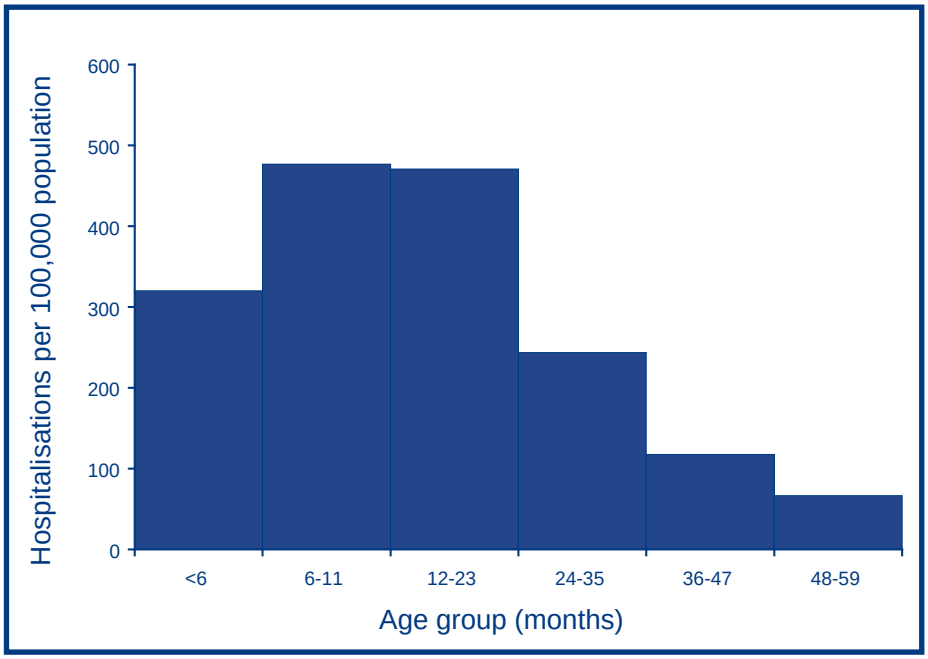
‡ Principal diagnosis (hospitalisations).

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

Age and sex distribution

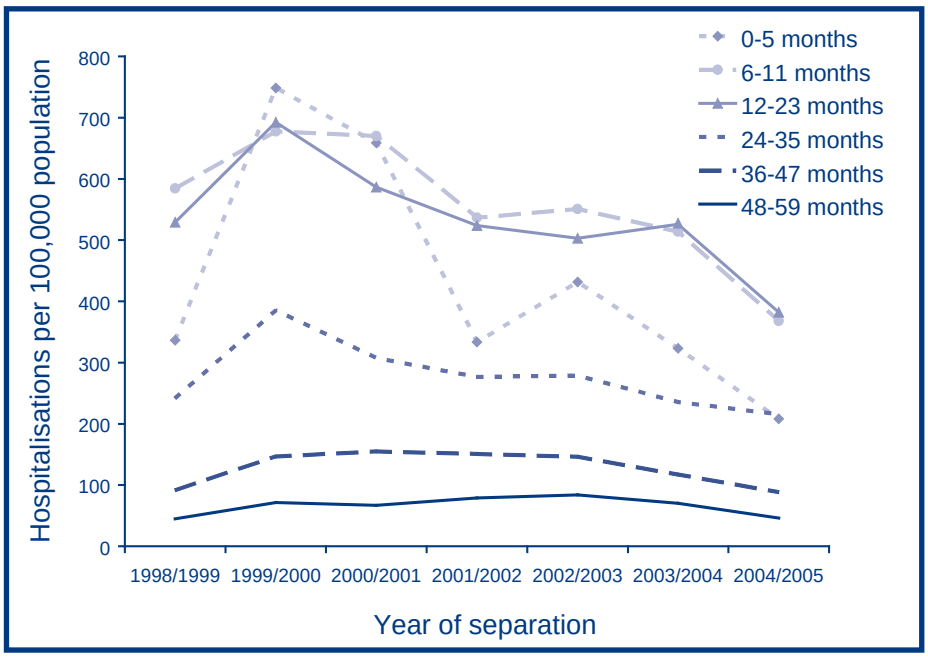
Across all age groups slightly more males were hospitalised with rotavirus (male:female ratio 1.18:1). The vast majority of rotavirus hospitalisations occur in those under five years of age (n=9,662, Table 18). The age distribution of hospitalisations in children under 5 years of age can be seen in Figure 38. The highest rates of admission occurred in those aged 6–23 months (Table 19).

Figure 38. Rotavirus hospitalisation rates, Australia, 2002 to 2005,* by age group (under 5 years)



* Hospitalisations where rotavirus was recorded as either a principal or any other diagnosis and the month of separation was between 1 July 2002 and 30 June 2005.

Figure 39. Rotavirus hospitalisation rates, Australia, 1998 to 2005,* by age group (under 5 years) and year of separation



* Hospitalisations where rotavirus was recorded as either a principal or any other diagnosis and the month of separation was between 1 July 1998 and 30 June 2005.

Table 19. Rotavirus hospitalisations and deaths, Australia, 2002 to 2005,* by age group (under 5 years)

Age group (months)	Hospitalisations 3 years (July 2002–June 2005)				LOS† per admission‡ (days)	Deaths 2 years (Jan 2003–Dec 2004)	
	n	(‡)	Rate§	(‡)	Median	n	Rate§
0–5	1,090	(768)	320.1	(206.6)	2.0	0	0
7–11	1,773	(1,526)	476.9	(410.5)	2.0	0	0
12–23	3,519	(2,951)	470.7	(394.7)	2.0	0	0
24–35	1,849	(1,609)	243.6	(212.0)	2.0	0	0
36–47	906	(801)	117.5	(103.9)	2.0	0	0
48–59	517	(452)	66.7	(58.3)	2.0	0	0
All ages	9,654	(8,107)	256.9	(213.9)	2.0	0	0

* Hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005; deaths where the death was recorded in 2003 or 2004.

† LOS = length of stay in hospital.

‡ Principal diagnosis (hospitalisations).

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

Geographical distribution

Over the period 2002/2003 to 2004/2005, the Northern Territory recorded over five times the Australian average rate of hospitalisation for rotavirus gastroenteritis (Northern Territory rate: 99.3 per 100,000 population) (Appendix 3). Victoria had the lowest rate of hospitalisation (8.5 per 100,000 population), less than half the Australian average. However, comparison of hospitalisation rates between jurisdictions is complicated by likely differences in testing practices for rotavirus, which may, in part, explain the differences in observed rates of hospitalisation.[221,228]

Northern Territory notification data, rotavirus

A total of 901 cases were notified from January 2003 to December 2005, an average annual rate of 149.7 per 100,000 total population. In 2004, there were almost twice as many notifications (n=407) as in 2002 (n=238) or 2005 (n=256). The male to female ratio amongst notified cases was 1.26:1.

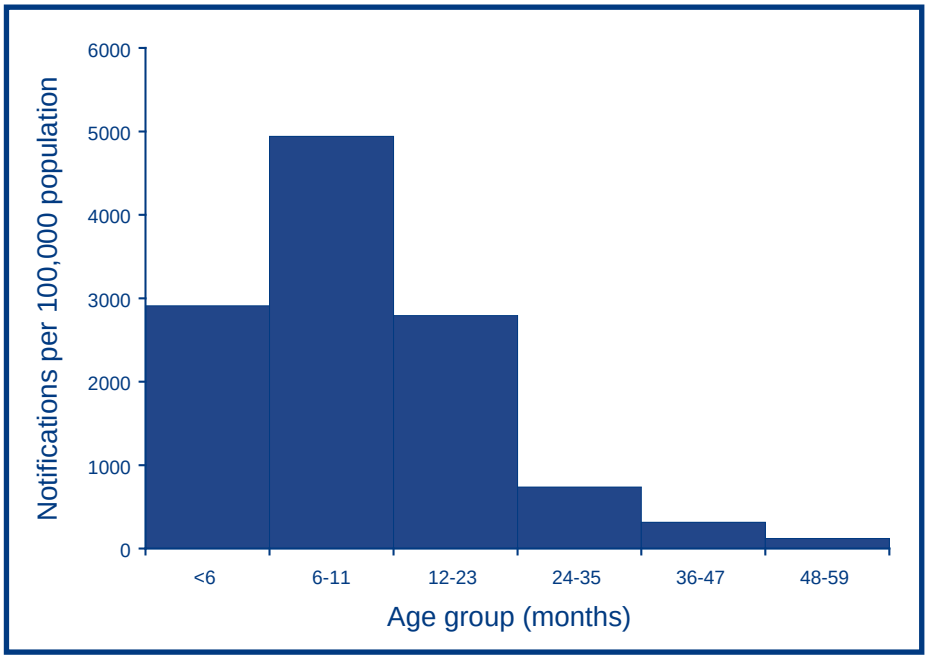
The rate of notification for rotavirus was significantly higher than the hospitalisation rate in the Northern Territory. The notification data from the Northern Territory generally has a younger age structure than hospitalisation data from all of Australia (Figure 40).

Comment

Rotavirus is responsible for a significant number of hospitalisations in Australia each year (around 3,500 hospitalisations were recorded annually with either a principal or non-principal diagnosis of rotavirus). The primary disease burden is in those under five years of age, with hospitalisations most common in the first two years of life. Universal immunisation of infants in the first six months of life under the NIP should prevent the majority of severe cases of rotavirus and it is anticipated that hospitalisation rates will be substantially reduced. However, it will be important to encourage timeliness of vaccination in order to ensure a maximal impact upon disease. Ongoing analysis of national hospitalisation data will provide valuable data to help assess the impact of a national vaccination program.

In addition to the caveats required in the interpretation of hospitalisation data, the use of the specific rotavirus code (A08.0) for quantifying rotavirus hospitalisations has substantial limitations. Although hospitalised patients with laboratory-confirmed rotavirus infection are likely coded as rotavirus gastroenteritis, laboratory testing for rotavirus antigen in stool specimens of children hospitalised with acute gastroenteritis is not often conducted. Clinical guidelines for the management of acute uncomplicated gastroenteritis do not recommend routine stool testing for confirmation of the aetiological agent.[229] Several international studies have shown that measurement of rotavirus hospitalisation rates utilising the specific rotavirus code underestimated the true number of rotavirus-associated hospitalisations.[230,231] Two Australian studies have estimated the burden of rotavirus hospitalisations, as a percentage of the

Figure 40. Rotavirus notifications, the Northern Territory, 2003 to 2005,* by age group (under 5 years)



* Notifications that were recorded between 1 January 2003 and 31 December 2005.

number of acute gastroenteritis (AGE) hospitalisations using distinct methodologies. Both studies, conducted a decade apart, estimated that rotavirus is responsible for approximately 10,000 hospitalisations annually.[228,232] In addition, emergency department visits for rotavirus were estimated as a percentage of AGE emergency department visits at approximately 21,500 annually.[232] These estimations may be a more accurate picture of the true hospitalisation disease burden.

The Australian Rotavirus Surveillance Program was initiated in June 1999 to monitor changes in the distribution of rotavirus serotypes over time. Historically, the dominant serotype in Australia and worldwide has been G1.[233] Recent surveillance reports noted a dramatic increase in the prominence of the G9 serotype in the years 2002–2003,[234,235] followed by a decline in G9 prevalence in subsequent years.[223,236] It appears likely that both rotavirus vaccines are effective against the G9 serotype, as well as the majority of other serotypes detected in Australia.[224,225]

Data from the Northern Territory hospitalisations and notifications emphasises the higher burden of disease in the region, particularly in Indigenous infants and children. Historically, the Northern Territory has experienced epidemics of rotavirus on the background of endemic disease.[226] These epidemics are thought to result from the relative isolation of remote communities with a lack of circulating strains, which then see a rapid spread of infection upon reintroduction of the virus.[226,237,238]

Compared with other developed countries, Australia has a relatively high rate of hospitalisation, with an estimated one in 26 children hospitalised by five years of age.[232] In Europe it is estimated that one in 50 children are hospitalised by the age of five years, although estimates vary by country and study methods.[239] Although there are limited data on nosocomial rotavirus infections in Australia, it is recognised as a significant problem in paediatric wards and hospitals, with at least 14%–19% of all rotavirus infections being hospital acquired.[240,241] Timely vaccination should control this.

Acknowledgements: Rosalie Schultz, Peter Markey and Christine Selvey, Centre for Disease Control, Northern Territory.

Rubella

Rubella is caused by the rubella virus (family togaviridae). It is usually a mild, febrile, viral disease characterised by a rash, conjunctivitis, coryza, postauricular and suboccipital lymphadenopathy, and nausea, but may be subclinical in up to 50% of cases. Arthralgia and arthritis may also occur, particularly in women. More severe disease manifestations, such as encephalitis, haemorrhage and Guillain-Barré syndrome, may also rarely occur. Rubella is important because of its ability to produce death or abnormalities in the developing fetus (congenital rubella syndrome (CRS)) when acquired in early pregnancy.[242]

Case definition

See Appendix 6 for pre-2004 definition

National definition from January 2004:[11]

A confirmed case requires laboratory definitive evidence. A probable case requires clinical evidence and either laboratory suggestive evidence or an epidemiological link to a laboratory-confirmed case.

a) Laboratory definitive evidence

- Isolation of rubella virus; *or*
- Detection of rubella virus by nucleic acid testing; *or*
- IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to rubella virus in the absence of recent rubella vaccination in paired sera tested in parallel; *or*
- Detection of rubella-specific IgM antibody in the absence of recent rubella vaccination (must be confirmed in a reference laboratory in pregnant women).

b) Laboratory suggestive evidence

- In a pregnant patient, the detection of rubella-specific IgM antibody that has not been confirmed in a reference laboratory, in the absence of recent rubella vaccination.

c) Clinical evidence

- A generalised maculopapular rash and fever, and one or more of: arthralgia/arthritis or lymphadenopathy or conjunctivitis.

Hospitalisations and deaths

The ICD-10-AM/ICD-10 code B06 (rubella [German measles]) was used to identify hospitalisation 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 2004 are available elsewhere.[243,244]

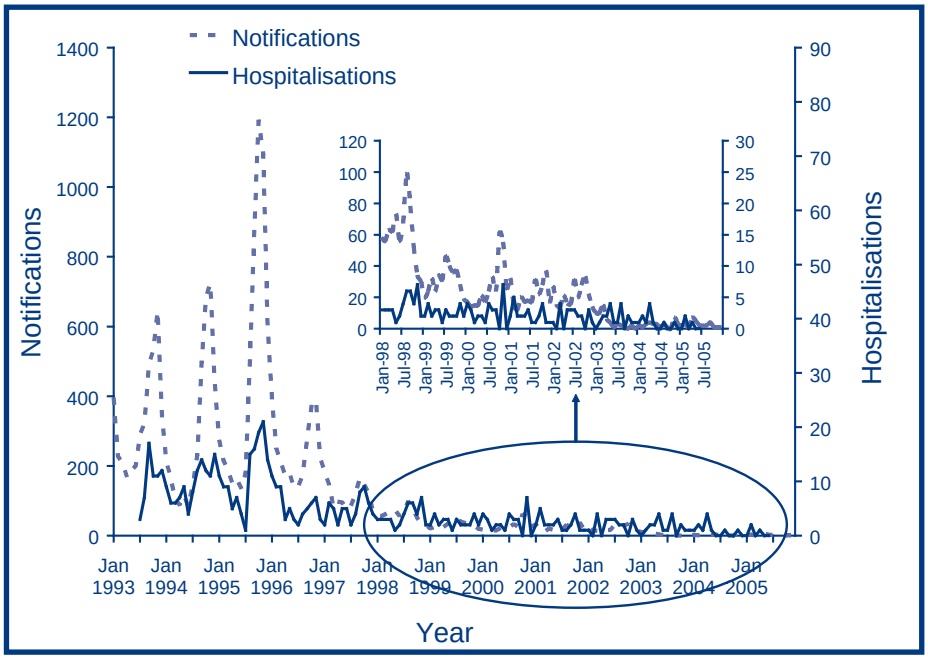
Secular trends

Between January 2003 and December 2005, there were 116 notified cases of rubella, an average annual notification rate of 0.2 per 100,000 (Table 20). There was a continuing and marked decline in the number of notifications with a more than sevenfold decrease between the current and previous three year period (2000–2002) when there were 844 cases notified. There were 54 notifications in 2003 (0.27 per 100,000) and 31 notifications (0.15 per 100,000) in 2004 and again in 2005. Between July 2002 and June 2005, 44 hospitalisations were coded as being due to rubella (an average annual rate of 0.07 per 100,000). Hospitalisations coded as rubella fell to historical lows, with 21 separations in 2002/2003, 18 in 2003/2004 and only five in 2004/2005. The current low incidence of rubella now means that the previously discernible spring peaks[3] in rubella activity are no longer observed (Figure 41).

Severe morbidity and mortality

In the period 2002/2003–2004/2005, 131 hospital bed days (average 44 per year) were recorded for patients with an ICD-10-AM code for rubella. In 2004/2005, there were only 10 hospital bed days coded with rubella. Of the hospital separations, 75% had a principal diagnosis of rubella (average annual rate 0.06 per 100,000). The median length of stay in hospital was two days, but increased with age (Table 20). In 2003 to 2004, there were no deaths with rubella recorded as the underlying cause.

Figure 41. Rubella notifications and hospitalisations, Australia, 1993 to 2005,* by month of diagnosis or admission



Note: varying scales between notifications and hospitalisations.

* Notifications where the month of diagnosis was between January 1993 and December 2005; hospitalisations where the month of admission was between 1 July 1993 and 30 June 2005.

Complications arising from rubella infection were recorded for five (11%) hospitalisations (Table 21). Only one case with complications was recorded in a child. Neither of the two cases with neurological complications was also coded as having other complications of rubella.

Table 20. Rubella notifications, hospitalisations and deaths, Australia, 2002 to 2005,* by age group

Age group (years)	Notifications 3 years (2003–2005)		Hospitalisations 3 years (July 2002–June 2005)				LOS† per admission (days)	Deaths 2 years (2003–2004)	
	n	Rate‡	n	(§)	Rate‡	(§)	Median (§)	n	Rate‡
0–4	12	0.32	21	(17)	0.55	(0.45)	1.0 (1.0)	0	0
5–14	2	0.02	3	(2)	0.04	(0.02)	n.p.	0	0
15–24	43	0.52	7	(6)	0.09	(0.07)	2.0 (2.0)	0	0
25–59	56	0.19	10	(6)	0.03	(0.02)	3.0 (1.0)	0	0
60+	3	0.03	3	(2)	0.03	(0.02)	n.p.	0	0
All ages	116	0.19	44	(33)	0.07	(0.06)	2.0 (1.0)	0	0

* Notifications where the month of diagnosis was between January 2003 and December 2005; hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005; deaths where the death was recorded in 2003 or 2004.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

n.p. Not published due to small cell sizes.

Table 21. Indicators of severe morbidity* for hospitalised cases of rubella

Age group (years)	Complication neurological		Complication other	
	n	% total	n	% total
0–4	0	0.0	1	4.8
5–14	0	0.0	0	0.0
15–24	0	0.0	0	0.0
25–59	1	10.0	0	0.0
60+	1	33.3	2	66.7
All ages	2	4.5	3	6.8

* Measured using National Hospital Morbidity data where the month of hospital separation was between 1 July 2002 and 30 June 2005.

Age and sex distribution

For the three year review period, notification rates were highest in the 20–24 year old age group, who accounted for nearly a quarter (24%) of cases (average annual rate 0.67 per 100,000). Within this age group, males predominated (male:female ration 3.5:1) and it was the only group where the notification rate was over one per 100,000. Together, the 25–29 year and 30–34 year age groups accounted for a further 31% of rubella notifications, with 18 notifications in each of these groups over the three year period (data not shown). The progressive increase seen in the median age of notified cases since the Measles Control Campaign (MCC) in 1998 has plateaued in the last three years, with the median age for notified cases of 25 years in each year 2003–2005 (up from 22 years in 2002.)

In 2003–2005, the male to female ratio for notifications was 1.3:1. This ratio disguises some variation by age group, with males predominating both in infants 0–4 years (10/12 cases) and in those aged over 20 years, but more female than male cases in the 15–19 year age group (11/15 cases). However, due to the low number of cases, these differences should be interpreted with caution.

For the three years combined, 2002/2003–2004/2005, children aged 0–4 years continued to have the highest hospitalisation rates (average annual rate 0.6 per 100,000). Whilst two thirds of hospitalisations in this group were males, the overall male to female ratio for hospitalisations was identical to the ratio for notifications (1.3:1). Whilst just over half of the hospitalisations were in infants too young to be vaccinated (11/21 aged under one year), no notifications were received for infants under one year of age in 2003–2005.

There were 40 notified cases of rubella in women of child bearing age (15–44 years) in 2003–2005, an average annual rate of 0.3 per 100,000. This rate has been declining each year since the outbreak in 1995 (rate 16.0 per 100,000) and is a notable decline from the previous reporting period 2001–2002 when the rate was 1.1 per 100,000. Within this age range, and indeed for any female age group, highest rates were reported in those aged 15–19 years (0.6 per 100,000).

Geographical distribution

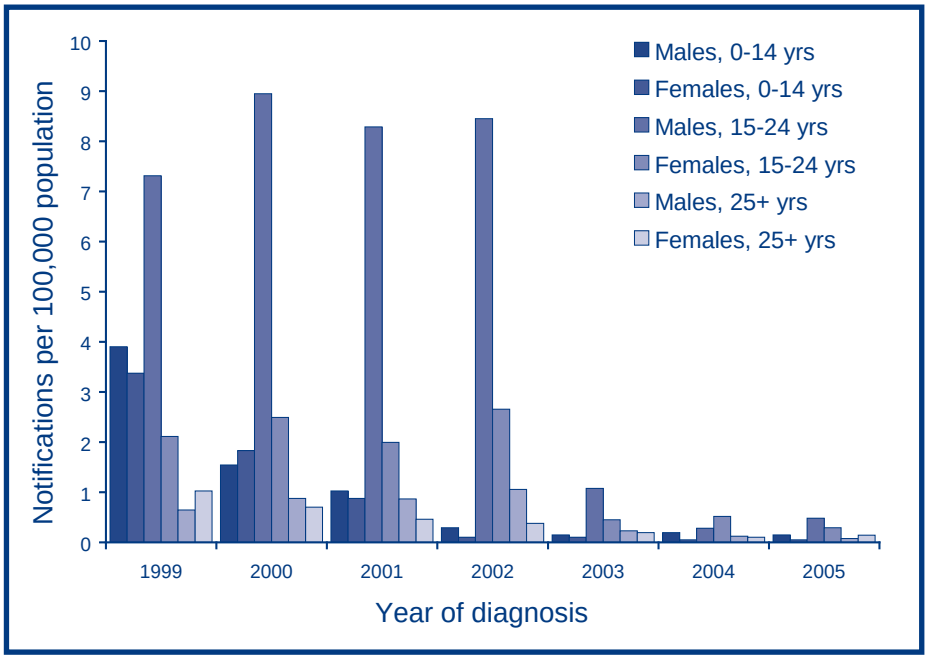
Most of the notifications received in 2003–2005 were from New South Wales (n=50; rate 0.25 per 100,000) or Queensland (n=43; rate 0.37 per 100,000). In both states, notifications were highest in 2003, following a peak in rubella activity in Queensland in 2002 which was associated with two cases of locally acquired congenital rubella syndrome, and then declined.

No notifications of rubella were received in the Northern Territory or the Australian Capital Territory during 2003–2005. Very low numbers of cases were reported sporadically in Victoria, South Australia and Tasmania (rates of 0.07 per 100,000 or less.) In Western Australia, 12 cases were reported through the period (0.2 per 100,000) (Appendix 2).

Four notifications, all in persons aged 18–36 years, were recorded on NNDSS as imported cases (i.e. acquired whilst overseas).

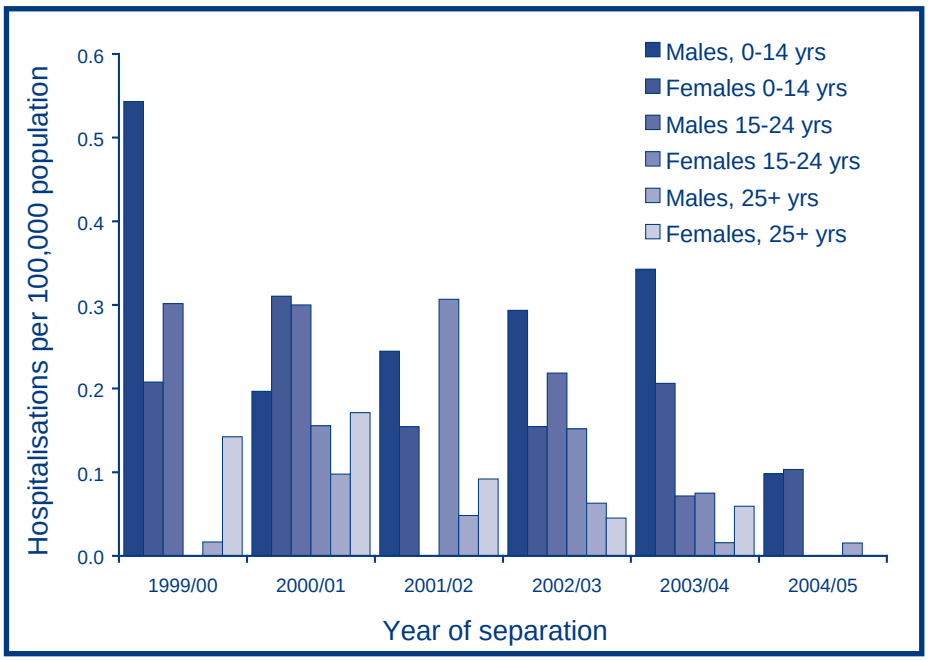
Hospitalisation rates varied over time and between states and territories (Appendix 3). However, there were too few cases in each jurisdiction to identify any trends.

Figure 42. Rubella notification rates, Australia, 1999 to 2005,* by age group, sex and year of diagnosis



* Notifications where date of diagnosis was between 1 January 1999 and 31 December 2005.

Figure 43. Rubella hospitalisation rates, Australia, 1999/2000 to 2004/2005,* by age group, sex and year of separation



* Hospitalisations where date of separation was between 1 July 1999 and 30 June 2005.

Vaccination status

Vaccination status for rubella cases is to be recorded on NNDSS for women of child bearing age (15–45 years). Whilst the vaccination status field was completed for all 40 cases, it was categorised as "unknown" in about one third of these (35%). Most women with known vaccination status were

unvaccinated (16/26; 62%). Whilst five women reported being fully vaccinated against rubella (i.e. two doses), this was not validated by written records. Vaccination status was confirmed in three of five women who reported partial vaccination (i.e. one dose).

Comment

Rubella notification and hospitalisation rates continue to decline and, in the most recent year reviewed, were the lowest on record. The adoption of a more specific national case definition in 2004 may have in part contributed to the ongoing decline in notifications. Hospitalisation data reported here suggest that either infants too young to be immunised are still at some (albeit small) risk of rubella in Australia or that coding errors or misdiagnosis are more likely to occur in paediatric age groups. There is no doubt that rubella control is a great immunisation success story in Australia but what challenges remain?

Notification data suggest that there remain vulnerable groups within the Australian population, particularly young males who missed out on both school based immunisation programs and natural infection.[245] The Queensland rubella 'outbreak' of 2001/2002 clearly demonstrated that there are sufficient pockets of susceptibility to rubella within the Australian population to maintain rubella circulation amongst vulnerable individuals for some time and, unfortunately in this context, the risk of locally acquired CRS remains.[246,247] Young women who have migrated to Australia may also be vulnerable due to lack of rubella vaccination programs in their countries of origin; in the last two years, six imported CRS cases were notified to the APSU.[243,244] Two recent studies of rubella serology records from women's hospitals in Melbourne and Sydney confirm that overseas born women, particularly from Asia but also from South America and sub-Saharan Africa, are more likely to be non-immune to rubella than other Australian women.[248,249] Also of concern, a study of antenatal screening records identified that rural and remote Indigenous women in the Northern Territory have low rates of rubella immunity and, thus, remain susceptible to rubella.[250] Antenatal rubella screening and post-natal vaccination, and rubella programs targeting women in these risk groups, will be important components of ongoing rubella control.

Those travelling within the Western Pacific region (and internationally) are at risk of rubella exposure. In 2005, 117 countries, covering 26% of the international birth cohort, were using rubella vaccines in their national immunisation programs.[251] Although very few cases on NNDSS were identified as imported cases (n=4), importation status was poorly completed on NNDSS (66% of records had this field missing). WHO received 28,659 notifications of rubella from the Western Pacific Region in 2005. This is a gross underestimate of the disease burden given that many cases are asymptomatic and many countries within the region do not have rubella surveillance or rubella vaccination programs. Rubella outbreaks have been documented in recent years in Tonga and Samoa, and these outbreaks were associated with high rates of rubella encephalitis.[252,253] The major regional focus of the Expanded Program on Immunization has been measles control and hepatitis B immunisation. With the increasingly successful achievements of the former target comes the opportunity for rubella programs, which require either targeting of women of child bearing age or sustained high coverage rates of the population. Guidelines to assist countries in assessing the appropriateness of adding rubella control strategies have recently been prepared by the WHO Western Pacific Regional Office. An assessment of local epidemiology, proper surveillance and coverage levels and an ability to meet and sustain the cost of MR vaccine over measles vaccine are critical.[252,253]

The data reviewed here suggest that Australia may be close to elimination of locally acquired rubella. In this respect, the recent elimination of rubella in the USA[254] and the European plan to eliminate rubella by 2010[255] will serve as good examples of possible ways forward. The United States declaration of rubella elimination could only occur in the context of a Pan American regional approach, in which successful mass immunisation campaigns, using the 'catch-up, keep-up and follow-up' strategies, achieved high population immunity and low disease incidence in neighbouring countries such as Mexico.[256] Similar to US requirements, Australia might consider targeted immunisation campaigns for immigrants from low prevalence countries. Whilst Australia already conducts measles genotyping, which provides molecular evidence that there is no indigenous strain of measles circulating, rubella genotyping is not performed. Internationally, the standardisation of nomenclature and genotyping methodology for rubella are continuing to be developed and, at this time, genotyping of rubella has not been performed in Australia.[257–259] Despite its continuing development, rubella genotyping has proven a critical tool in declaring the absence of local rubella circulation in the US.[260] In Australia, it is increasingly important that suspected rubella cases have specimens collected for PCR which, if positive, can be referred for genotyping.

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% to 90%, with the highest rates in infants and the elderly.[44]

Case definition

See Appendix 6 for pre-2004 definition

National definition from January 2004:[11]

Confirmed cases require either laboratory definitive evidence or clinical evidence.

a) Laboratory definitive evidence

- Isolation of *Clostridium tetani* from a wound in a compatible clinical setting and prevention of positive tetanospasm in mouse test from such an isolate using specific tetanus antitoxin.

b) Clinical evidence

- A clinically compatible illness without apparent cause.

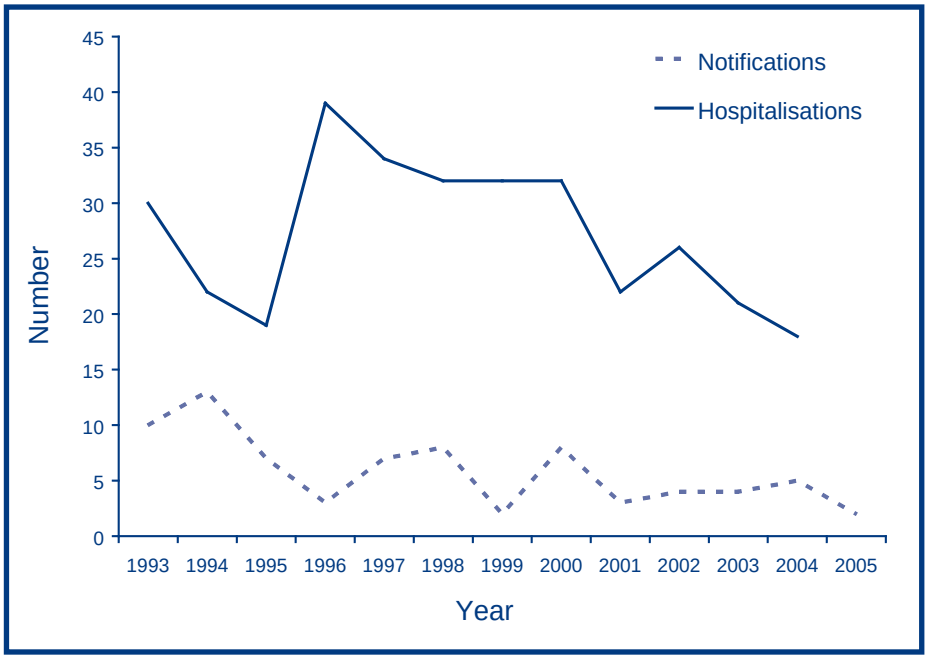
Hospitalisations and deaths

The ICD-10-AM/ICD-10 codes A34 (obstetrical tetanus) and A35 (other tetanus) were used to identify hospitalisations and deaths.

Secular trends

There were 11 notifications of tetanus in the January 2003 to December 2005 review period (an average annual notification rate of 0.02 per 100,000). However, in the period July 2002 to June 2005, there were 66 hospitalisations coded as tetanus (an average annual rate of 0.11 per 100,000). Notifications for tetanus remained relatively stable between 2002 and 2005, ranging from two to five notifications annually. Hospitalisations for tetanus declined between 2002/2003 and 2004/2005 (Figure 44).

Figure 44. Tetanus notifications and hospitalisations, Australia, 1993 to 2005,* by year of diagnosis or admission



* Notifications where the year of diagnosis was between January 1993 and December 2005; hospitalisations where the month of admission was between 1 July 1993 and 30 June 2005.

Severe morbidity and mortality

A total of 1,061 hospital bed days (average 354 per year) were recorded for patients with an ICD-10-AM code for tetanus. Of the 66 separations, 46 (70%) had tetanus recorded as the principal diagnosis. The median length of stay in hospital was 4.5 days and varied depending on age. Adults aged 60 years and over had longer median lengths of stay (Table 22).

Table 22. Tetanus notifications, hospitalisations and deaths, Australia, 2002 to 2005,* by age group

Age group (years)	Notifications 3 years (2003–2005)		Hospitalisations 3 years (July 2002–June 2005)				LOS† per admission (days)	Deaths 2 years (2003–2004)	
	n	Rate‡	n	(§)	Rate‡	(§)	Median (§)	n	Rate‡
0–4	0	0.00	0	(0)	0.00	(0.00)	0.0 (0.0)	0	0.00
5–14	0	0.00	1	(1)	0.01	(0.01)	n.p.	0	0.00
15–24	0	0.00	6	(4)	0.07	(0.05)	1.0 (1.0)	0	0.00
25–59	0	0.00	15	(9)	0.05	(0.03)	2.0 (2.0)	0	0.00
60+	11	0.10	44	(32)	0.43	(0.31)	12.0 (10.0)	0	0.00
All ages	11	0.02	66	(46)	0.11	(0.08)	4.5 (3.5)	0	0.00

* Notifications where the month of diagnosis was between January 2003 and December 2005; hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005; deaths where the death was recorded in 2003 or 2004.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

n.p. Not published due to small cell size.

In the review period (2003–2004), there were no deaths in the AIHW mortality database with tetanus recorded as the underlying cause. However, there was one death (in 2004) amongst notified cases reported to NNDSS for the period 2003–2005.

Age and sex distribution

All of the notified case patients and the majority of the hospitalised patients (44/66, 67%) were aged 60 years and over. The youngest hospitalised case was a child aged 5–14 years. The male:female ratio of notified cases was 0.84:1, but there were fewer hospitalised female patients, with a male:female ratio of 1.2:1. In the age group 70 years and over, 66% of the hospitalised cases (23/35) were males.

For both notifications and hospitalisations, rates increased with increasing age (Figure 45). Males aged 70 years and over had the highest average annual hospitalisation rate (0.98 per 100,000).

Geographical distribution

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

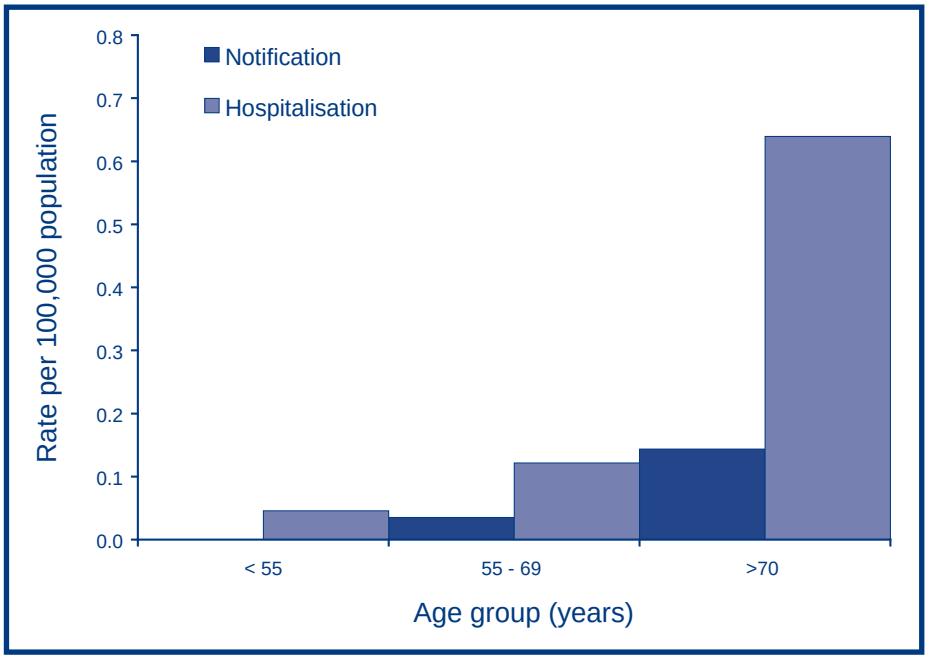
Vaccination status

The vaccination status of 73% (8/11) of notified cases in NNDSS was "unknown" in 2003–2005. No case was reported as fully vaccinated.

Comment

There has been a downward trend in tetanus hospitalisation rates. Hospitalisation rates remain higher for tetanus than notification rates. It is likely that this discrepancy is principally due to under-reporting, together with multiple hospital admissions for the same case and coding errors.[261] Coding errors may have resulted from misclassification of other conditions as tetanus, especially where tetanus was not the

Figure 45. Tetanus notification and hospitalisation rates, Australia, 2003 to 2005,* by age group



* Notifications where the month of diagnosis was between January 2003 and December 2005; hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005.

principal diagnosis. Notifications of tetanus rely heavily on clinicians rather than laboratories, as laboratory confirmation of the diagnosis is rarely possible. Clinicians are known to under-notify hospitalised cases of disease;[19] thus, under-notification for tetanus is likely.

Tetanus is a disease of older adults. In Australia, booster doses of tetanus vaccine are thought to be poorly utilised, as noted in Canada and Switzerland.[262,263] The major impetus for tetanus immunisation in adults is injury,[262] but tetanus occurs in cases with trivial or no known injury and the definition of a 'tetanus prone' wound is unclear.[264,265] International serosurveys and the Australian National Serosurvey have shown progressively lower prevalence of levels of tetanus antibody in older age groups, particularly in women.[58,266–269] Although the tetanus organism is ubiquitous in the environment, and the vaccine only provides individual level protection against the toxin, tetanus vaccination programs have had a significant impact upon the disease burden in Australia. The current tetanus notification rate in Australia is similar to that in other developed countries.[264,270–272] A tetanus booster is recommended at the age of 50 unless a booster has been documented within 10 years.[76] Whilst the data presented in this report suggest that this is an appropriate recommendation, strategies to improve vaccine uptake at this age need to be investigated. As both notifications and hospitalisations predominate in those aged 65 years and over, review of tetanus immunisation status at the time of annual influenza vaccination is clearly appropriate. 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.[271,273] 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% of infections are subclinical. Acute varicella may be complicated by cerebellitis, aseptic meningitis, transverse myelitis, thrombocytopenia and pneumonia.[44]

In unvaccinated populations, varicella is primarily a childhood illness with more than 90% of the population in temperate countries developing clinical or serological infection by adolescence.[274] In Australia, however, seropositivity was 83% by age 10–14 years.[275] Varicella is generally a benign, self-limiting illness in children, but morbidity and mortality rates are higher in adults,[276] at the extremes of ages, and in the immunocompromised.[277] A universal infant varicella vaccination program was introduced in Australia in 2005.

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.[278] Although herpes zoster can occur at any age, most cases occur after the age of 50 and incidence increases with age.[279] 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.[280–282] A new herpes zoster vaccine which is over 60% effective in reducing the burden of herpes zoster and post-herpetic neuralgia[283] is likely to be available in Australia in 2007.

Case definitions

Notifications

Varicella is not a nationally notifiable disease. Varicella and herpes zoster became notifiable in South Australia in 2002. Varicella zoster virus infection became laboratory notifiable in Queensland in December 2005. Other jurisdictions are considering varicella zoster surveillance.

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.

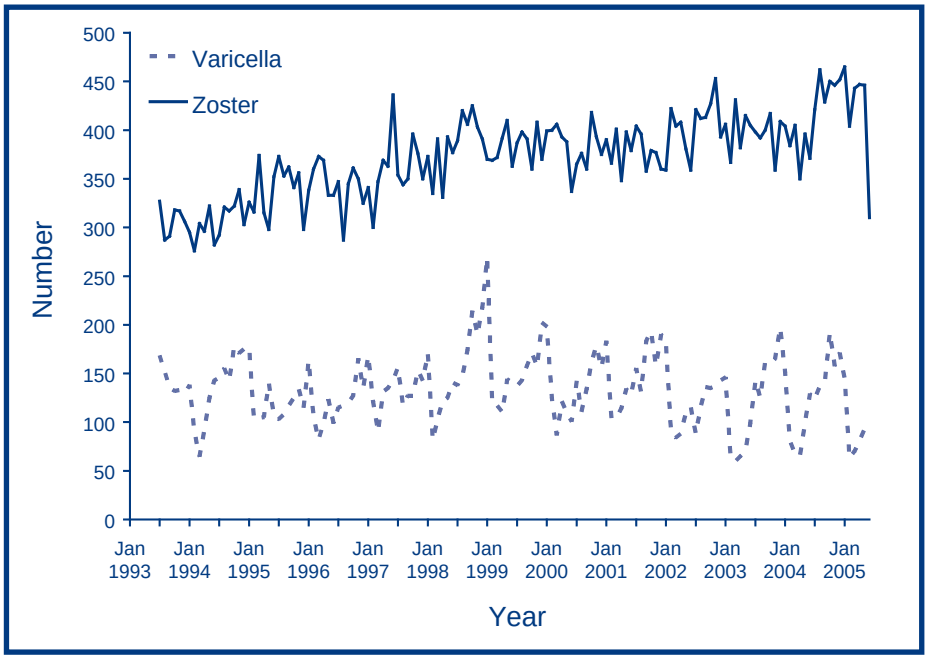
Secular trends, varicella and herpes zoster

There were 4,281 hospitalisations (average annual hospitalisation rate 7.2 per 100,000) for varicella between 1 July 2002 and 30 June 2005 (Table 23). A median of 124 cases of varicella (range 60–197) was hospitalised per month (Figure 46). The rate and median monthly admissions are slightly lower than the period 2000–2002, but longer term surveillance is required to determine if this is random fluctuation or a meaningful decrease.

Figure 46 shows that there are significantly more hospitalisations for herpes zoster than varicella. There were 14,926 hospitalisations (average annual hospitalisation rate 25 per 100,000 for all herpes zoster and 10.7 per 100,000 for herpes zoster as a principal diagnosis), between 1 July 2002 and 30 June 2005 (Table 25). A median of 410 cases of herpes zoster (range 310–465) were hospitalised per month (Figure 46). The rate and median monthly admissions are very slightly higher than the period 2000 to 2002, but longer term surveillance is required to determine if this is random fluctuation or a meaningful increase.

There was demonstrable seasonality, with hospitalisations for varicella peaking in late spring/early summer and dropping in late summer/early autumn.

Figure 46. Varicella and herpes zoster hospitalisations, Australia, July 1993 to June 2005,* by month of admission



* Hospitalisations where the month of admission was between 1 July 1993 and 30 June 2005.

Severe morbidity and mortality, varicella

For patients with an ICD-10-AM code for chickenpox, 21,872 hospital bed days (average 7,290 per year) were recorded from 2002–2005. Of the 4,281 varicella hospitalisations, 2,816 (66%) had a principal diagnosis of varicella (average annual rate 4.7 per 100,000) (Table 23). Complications arising from varicella infection were recorded for 1,374 hospitalisations (32%). Of all varicella hospitalisations, 115 (2.7%) were coded as having encephalitis and 433 (10.1%) were coded as having pneumonitis (Table 24). Although most hospitalisations were in the youngest age group, people aged 60 years and over had the longest median length of stay. There were 11 deaths recorded with varicella as the underlying cause in the calendar years 2003–2004, six (54%) of them for people aged 60 years and over. The highest death rate was in children aged 0–4 years.

Table 23. Varicella hospitalisations and deaths, Australia, 2002 to 2005,* by age group

Age group (years)	Hospitalisations 3 years (July 2002–June 2005)				LOS† per separation (days)	Deaths 2 years (2003–2004)	
	n	(§)	Rate‡	(§)	Median (§)	n	Rate‡
0–4	1,597	(1,076)	42.1	(28.4)	2.0 (2.0)	3	0.12
5–14	668	(421)	8.2	(5.2)	2.0 (2.0)	0	0.00
15–24	403	(274)	4.9	(3.4)	2.0 (2.0)	0	0.00
25–59	1,258	(885)	4.3	(3.0)	3.0 (3.0)	2	0.01
60+	355	(160)	3.5	(1.6)	9.0 (7.0)	6	0.09
All ages	4,281	(2,816)	7.2	(4.7)	2.0 (2.0)	11	0.03

* Hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005; deaths where the death was recorded in 2003 or 2004.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

Table 24. Indicators of severe morbidity* for hospitalised cases of varicella, Australia, 2002 to 2005,* by age group

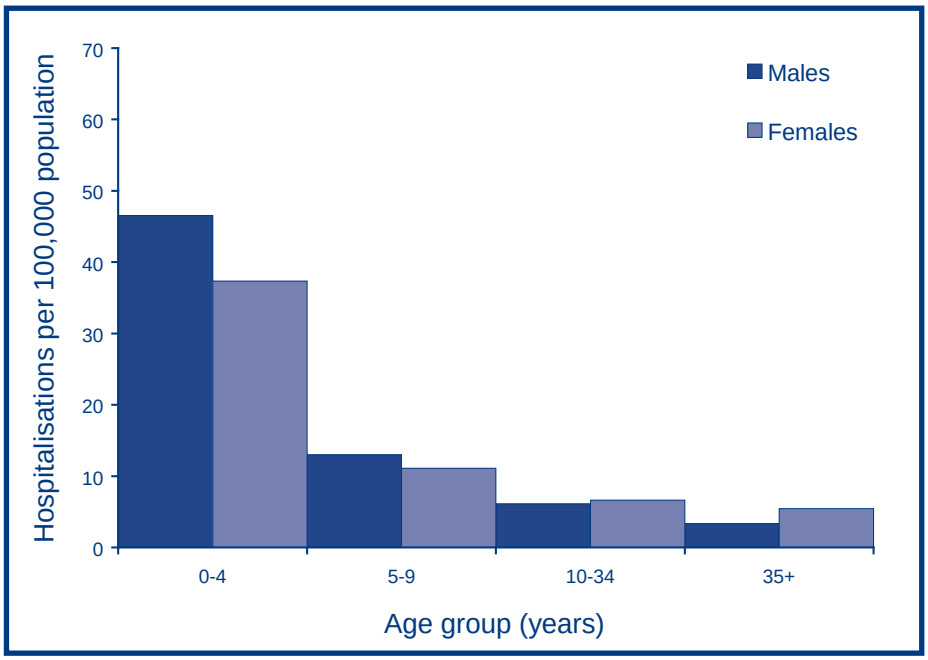
Age group (years)	Varicella encephalitis		Varicella pneumonitis	
	n	% of cases	n	% of cases
0–4	22	1.4	51	3.2
5–14	33	4.9	15	2.2
15–24	10	2.5	41	10.2
25–59	24	1.9	295	23.4
60+	26	7.3	31	8.7
All ages	115	2.7	433	10.1

* Measured using National Hospital Morbidity data where the month of separation was between 1 July 2002 and 30 June 2005.

Age and sex distribution, varicella

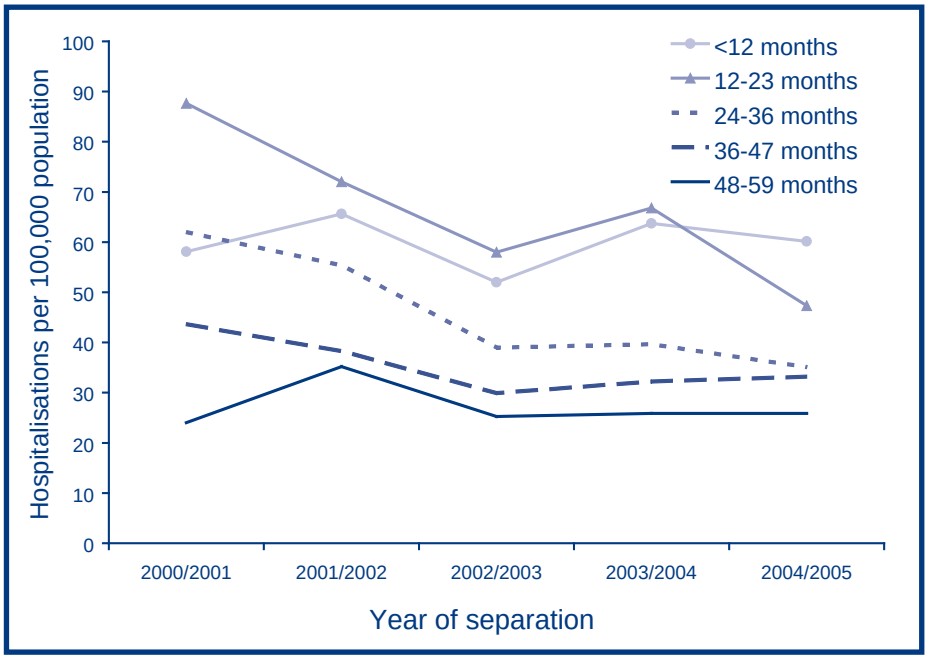
The highest number and rate of varicella hospitalisations occurred in the youngest age groups, especially the 0–4 years age group (Table 23, Figure 47). Within the age group 0–4 years (Figure 48), a decrease in the 12–23 months age group was observed in 2004/2005, a period which includes the introduction of universal varicella immunisation at 12 months of age, compared to the previous years. For the first time, hospitalisation rates were higher in infants aged less than 12 months than in those aged 12–23 months. The overall male:female ratio of hospitalisations was 1.2:1. Males predominated in all age groups except in young adults aged 15–29 years where there was a slight female predominance (data not shown). Of the 11 varicella deaths, five were males.

Figure 47. Varicella hospitalisation rates, Australia, 2002 to 2005,* by age group and sex



* Hospitalisations where the date of separation was between 1 July 2002 and 30 June 2005.

Figure 48. Varicella hospitalisation rates, Australia, 2000 to 2005,* by age group (0–4 years) and year of separation



* Hospitalisations where the date of separation was between 1 July 2000 and 30 June 2005.

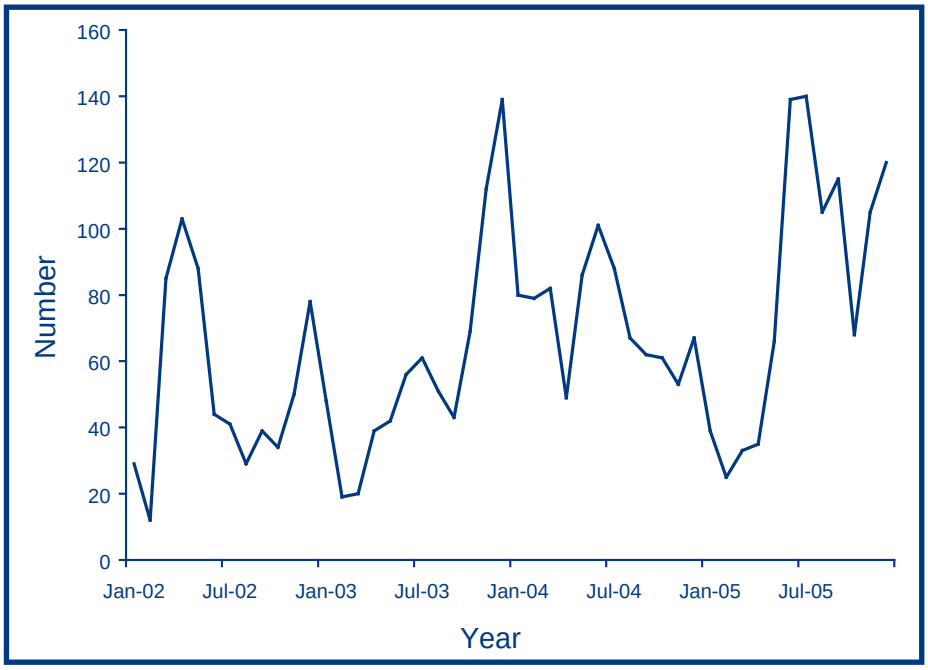
Geographical distribution, varicella

For the years 2002/2003–2004/2005, the Northern Territory had the highest average annual hospitalisation rate (10.6 per 100,000) with all other states recording average annual rates between 6 and 8 per 100,000. Hospitalisation rates in the Australian Capital Territory were lower (average annual rate 3.2 per 100,000) (Appendix 3).

South Australian surveillance data, varicella

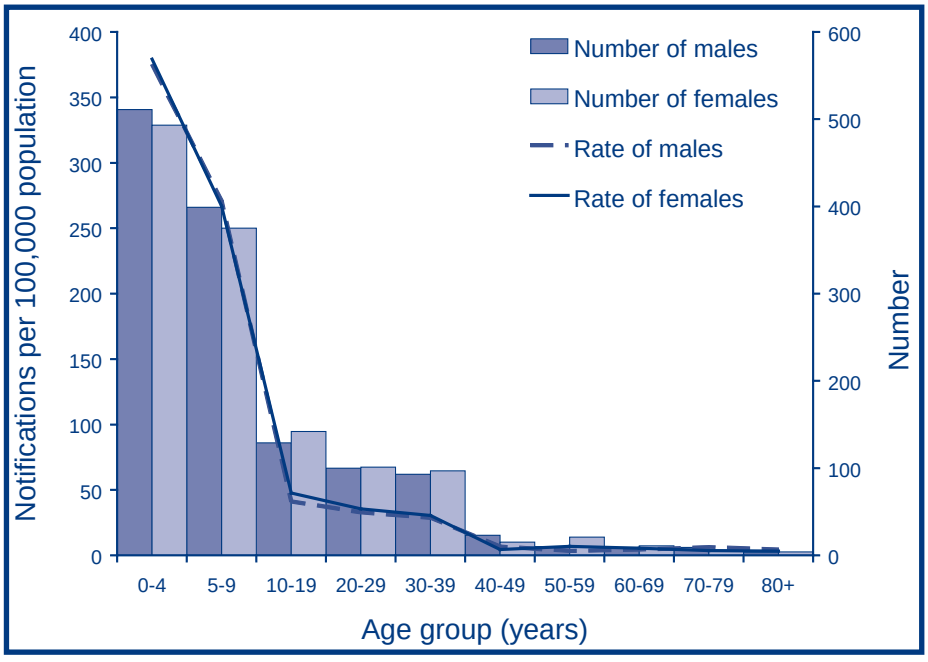
Figure 49 shows the notifications of varicella by month of notification from January 2002 to December 2005. There is a clear seasonality in the reported incidence of varicella. A total of 2,564 cases of chickenpox were notified in the current review period (2003–2005), an average annual rate of 56 per 100,000. Figure 50 shows the notifications by gender and age for the current review period. In addition to clinical diagnoses of chickenpox and zoster (reported below), 242 laboratory diagnoses of varicella-zoster were received.

Figure 49. Varicella notifications, South Australia, 2002 to 2005, by month of notification



* Notifications where the date of notification was between 1 January 2002 and 31 December 2005.

Figure 50. Varicella notifications, South Australia, 2003 to 2005,* by age group and sex



* Notifications where the date of notification was between 1 January 2003 and 31 December 2005.

Severe morbidity and mortality, herpes zoster

For patients with an ICD-10-AM code for herpes zoster, 175,164 hospital bed days (average 58,388 per year) were recorded. Of the 14,926 herpes zoster hospitalisations, 6,387 (43%) had a principal diagnosis of HZ (average annual rate 10.7 per 100,000) (Table 25). Complications arising from HZ infection were recorded for 7,576 hospitalisations (50.7%). Of all HZ hospitalisations, 139 (0.93%) were coded as having disseminated HZ and 1,817 (12.2%) were coded as having ocular complications (Table 26). By far the greatest number of hospitalisations were in the oldest age group, who also had the longest median length

of stay. There were 38 deaths recorded with herpes zoster as the underlying cause in the calendar years 2003–2004, 36 of them for people 60 years and over. The highest death rate was also recorded in people 60 years and over.

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, where the rate was over 112 per 100,000 (Table 25). Across all ages, the male:female rate ratio of hospitalisations was 0.71:1. The male:female rate ratio for deaths due to herpes zoster was 0.53:1.

Geographical distribution, herpes zoster

For the years 2002/2003–2004/2005, South Australia had the highest crude average annual hospitalisation rate for herpes zoster (30.6 per 100,000), followed by Tasmania (29.0 per 100,000). The Northern Territory and the Australian Capital Territory had the lowest rates at around 15 per 100,000, with the other states having rates close to 25 per 100,000 (Appendix 3). That the Northern Territory has the lowest hospitalisation rates for herpes zoster and the highest for varicella presumably reflects the younger age structure of the population of the Northern Territory.

Table 25. Herpes zoster hospitalisations and deaths, Australia, 2002 to 2005,* by age group

Age group (years)	Hospitalisations 3 years (July 2002–June 2005)				LOS† per separation (days)	Deaths 2 years (2003–2004)	
	n	(§)	Rate‡	(§)	Median (§)	n	Rate‡
0–4	108	(81)	2.8	(2.1)	3.0 (3.0)	0	0.00
5–14	269	(183)	3.3	(2.3)	3.0 (3.0)	0	0.00
15–24	224	(120)	2.7	(1.5)	3.0 (3.0)	1	0.02
25–59	2,762	(1,240)	9.5	(4.2)	4.0 (3.0)	1	0.01
60+	11,562	(4,763)	112.6	(46.4)	7.0 (5.0)	36	0.52
All ages‖	14,926	(6,387)	25	(10.7)	6.0 (4.0)	38	0.10

* Hospitalisations where the month of separation was between 1 July 2002 and 30 June 2005; deaths where the death was recorded in 2003 or 2004.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

‖ Includes cases with unknown ages.

Table 26. Indicators of severe morbidity* for hospitalised cases of herpes zoster, Australia, 2002 to 2005,* by age group

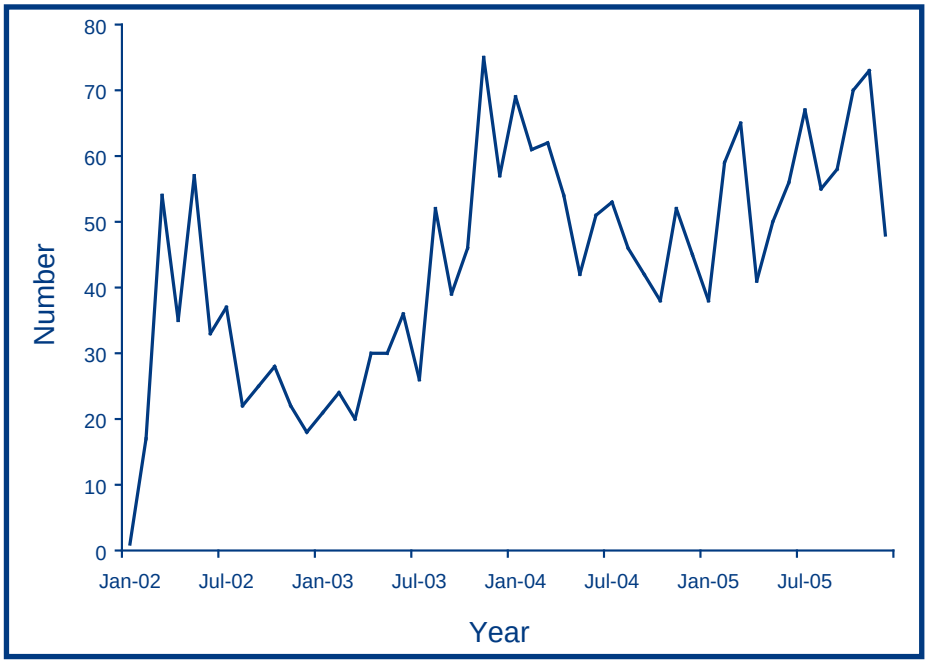
Age group (years)	Zoster encephalitis or meningitis		Zoster with other nervous system involvement		Disseminated zoster		Ocular complications of herpes zoster	
	n	% of cases	n	% of cases	n	% of cases	n	% of cases
0–4	4	3.7	5	4.6	1	0.9	27	25.0
5–14	9	3.3	20	7.4	1	0.4	42	15.6
15–24	13	5.8	20	8.9	4	1.8	29	12.9
25–59	125	4.5	507	18.4	44	1.6	346	12.5
60+	145	1.3	3,744	32.4	89	0.8	1,373	11.9
All ages	296	2.0	4,296	28.8	139	0.9	1,817	12.2

* Measured using National Hospital Morbidity data where the month of separation was between 1 July 2002 and 30 June 2005.

South Australian surveillance data, herpes zoster

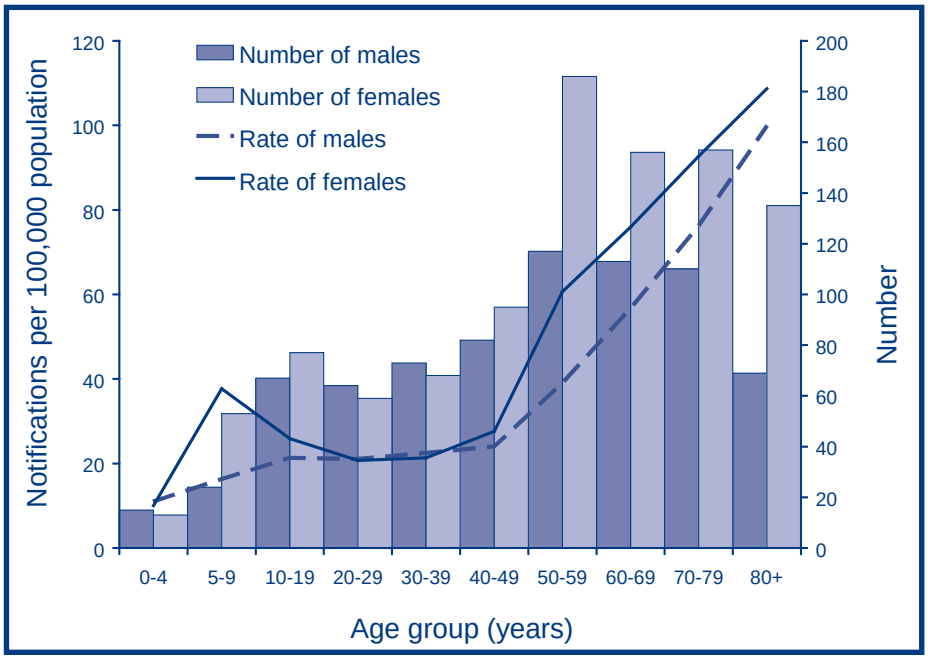
Figure 51 shows the notifications of herpes zoster by month from January 2002 to December 2005. A total of 1,751 cases were notified in the current review period (2003–2005), an average annual rate of 38.4 per 100,000. Figure 52 shows the notifications by gender and age for the current review period.

Figure 51. Herpes zoster notifications, South Australia, 2002 to 2005, by month of notification



* Notifications where the date of notification was between 1 January 2002 and 31 December 2005.

Figure 52. Herpes zoster notifications, South Australia, 2003 to 2005, by age group and sex



* Notifications where the date of notification was between 1 January 2003 and 31 December 2005.

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 four days longer than for varicella, so that the burden of disease caused by severe herpes zoster, as indicated in these hospitalisation data, 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, 32% of hospitalised cases had a recorded complication. For herpes zoster, just over half of the cases had a complication. This routine data is in agreement with a more detailed study that found that over 50% of herpes zoster hospital episodes had a documented complication, the majority of which were neurological.[284] In that study, 16% had ophthalmic zoster, which is a serious complication because it threatens vision.[284]

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 declined dramatically. This is evident in all age groups, and is most marked among those aged 1–4 years.[285] Universal VZV vaccination was recommended at 18 months of age in Australia in September 2003, and implemented as a national program in 2005, making it important to have a good understanding of the local epidemiology of disease at baseline. Our early data suggests an impact of the program in 2005 in the age group 12–24 months, with hospitalisations in this group falling below those in the under one year age group for the first time, but longer term trends will need to be observed to confirm this.

In 1952, Hope-Simpson proposed the hypothesis that exposure to varicella may boost immunity against HZ.[286] 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.[287] 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.[288,289] Mathematical modelling suggests that widespread infant VZV vaccination might result in a significant increase in the incidence of HZ, affecting more than 50% 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.[287] These predictions might not be correct, particularly if vaccine efficacy is less than that suggested by clinical trial data. Currently surveillance data from the USA, where varicella immunisation has been recommended for over a decade, indicates a large reduction in varicella morbidity with no increase in zoster disease yet demonstrated.[290,291]

The South Australian notification data show no apparent impact on the transmission of varicella by the availability of VZV vaccine on the private market with a slight increase in reported cases noted per year, although this may well be due to increasing awareness amongst clinicians of the need to notify cases rather than any real change in disease prevalence. Interestingly, the South Australian surveillance system, in contrast to hospitalisations, detects varicella more frequently than herpes zoster. The gender-specific and age-specific data from South Australia show a similar epidemiology to the hospitalised cases, suggesting that hospitalisation data provides a useful measure of trends in varicella and herpes zoster.

Varicella-zoster surveillance has been funded as part of the national varicella immunisation program[292] and will include notification to the NNDSS (in five jurisdictions surveillance will comprise passive notification through GPs and labs; in two it will include passive notification as well as syndromic surveillance, with specimen collection through sentinel GPs/emergency departments; and in one state varicella will not be notifiable, with emergency department syndromic surveillance only).[293] Additionally trends in disease burden will continue to be reviewed through hospitalisation data and Australian Paediatric Surveillance Unit data on neonatal and congenital varicella infections.

Acknowledgement: Dr Rod Givney, Communicable Diseases Control Branch, Department of Human Services, South Australia.

4. Vaccination coverage

Childhood Vaccination Schedule 1998 to 2005

There have been a series of changes to recommended vaccines for children 0–6 years of age between 1998 and 2005.

1998–2000

The first change was in the second half of 1998, when the second dose of measles-mumps-rubella (MMR) vaccine (previously recommended at 10–16 years of age) was moved to 4 years of age at the time of the national Measles Control Campaign. The next change occurred in May 2000, when two distinct paths were introduced for children born on or after 1 May 2000,[76] using different combination vaccines, outlined in Table 27. Pathway 1, followed by New South Wales, Queensland, South Australia, the Australian Capital Territory and the Northern Territory, used hepatitis B (Hep B) vaccine in a combination with diphtheria-tetanus-acellular pertussis (DTPa) vaccine, while Pathway 2, followed by Tasmania, Victoria and Western Australia, used Hep B in combination with *Haemophilus influenzae* type b (Hib) vaccine. This meant that from May 2000, full vaccination at 12 months of age (first milestone) required three doses of DTPa and oral poliomyelitis (OPV) vaccines, two doses of Hib and either two or three doses of Hep B vaccine, depending on the pathway adopted. Full Hib immunisation at 12 months of age required two doses of PRP-OMP. Full Hep B immunisation at 12 months required either three doses of combined DTPa-Hep B (Pathway 1) or two doses of combined Hib-Hep B vaccine (Pathway 2). The neonatal dose of HepB vaccine (scheduled for all newborns since May 2000) was not included in ACIR coverage estimates. In the second year of life, a dose of MMR vaccine remained at 12 months of age as well as booster doses of Hib vaccine (at 12 months) and DTPa (at 18 months) and under Pathway 2 this Hib vaccine was given with Hep B vaccine. In the fifth year of life, a second dose of MMR vaccine was scheduled as well as booster doses of DTPa and OPV.

2001–2005

Meningococcal C conjugate vaccine was recommended and funded at 12 months of age from January 2003. In September 2003, the DTPa booster dose at 18 months of age was no longer recommended. Consequently all vaccines in the second year of life for non-Indigenous children were given at 12 months of age. Also in September 2003, the recommended 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 of age, and inactivated poliomyelitis vaccine in place of oral vaccine, although these recommendations were not funded. In January 2005, universal 7-valent conjugate pneumococcal vaccine was funded and, in May 2005, the National Immunisation Program (NIP) schedule replaced the Australian Standard Vaccination Schedule (ASVS) for children aged 0–6 years, with all recommended vaccines to be funded, including varicella-zoster vaccine and inactivated poliomyelitis vaccine from November 2005.

Table 27. Australian Standard Vaccination Schedule 2005 for children

Age	Vaccine							
Birth	Hep B							
2 months	Hep B*,†	DTPa	Hib†,‡	IPV			7vPCV	
4 months	Hep B*,†	DTPa	Hib†,‡	IPV			7vPCV	
6 months	Hep B*	DTPa	Hib‡	IPV			7vPCV	
12 months	Hep B†		Hib†		MMR			MenCCV
18 months						VZV	23vPPV§	
4 years		DTPa		IPV	MMR			

* Diphtheria-tetanus-acellular pertussis/Hep B vaccine from May 2000 (Pathway 1) (three doses, at 2, 4 and 6 months of age).

† Hib PRP-OMP/hep B from May 2000 (Pathway 2) (three doses, at 2, 4 and 12 months of age).

‡ Hib PRP-OMP (Pathway 1) from May 2000 (three doses, at 2, 4 and 6 months of age).

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

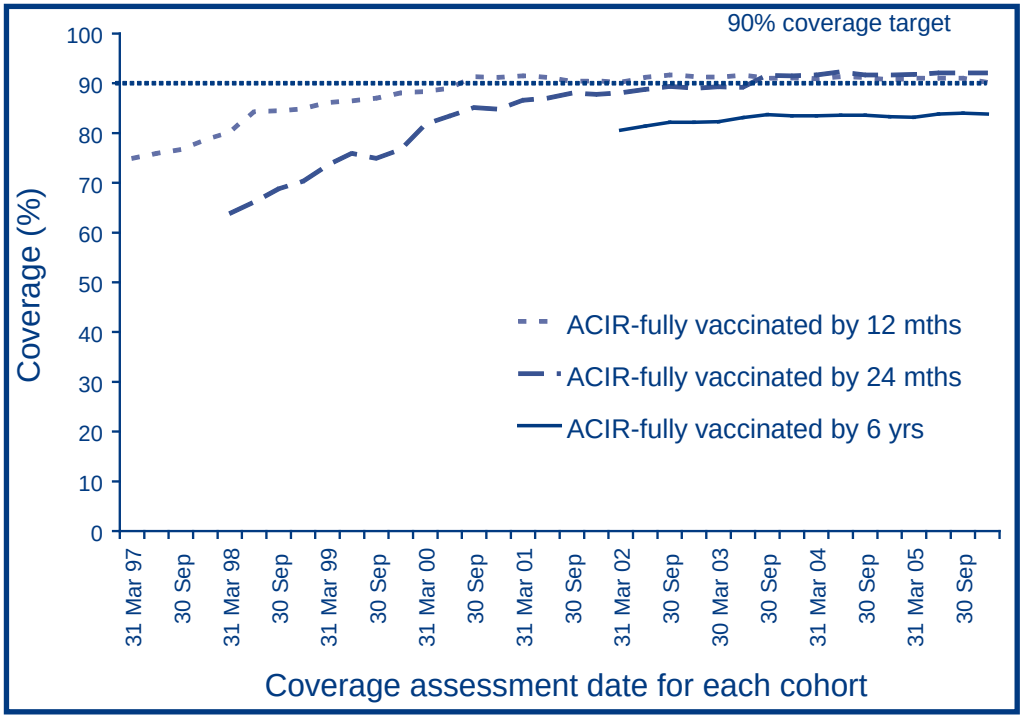
Vaccination coverage estimates from the ACIR 1996 to 2005

The methodology for calculating cohort-based vaccination coverage from the ACIR was published with the first coverage estimates in 1998.[5] 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.[16] The vaccination status of each cohort is assessed at the three key milestones of 12 months, 24 months, and 72 months 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 (approximately 99% of children).[5] 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').[16] 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 a third dose of Hep B vaccine, depending on the pathway taken on the schedule. ACIR coverage estimates (using the 'assumption') for the first vaccination milestone (the first three scheduled doses of DTPa, OPV and, recently, two or three doses of Hep B and two or three doses of Hib) have been reported in *Communicable Diseases Intelligence* since 1998.[294] The coverage for MMR has been reported in *Communicable Diseases Intelligence* since 1998.[294] Coverage for the third vaccination milestone at 72 months of age has been reported in *Communicable Diseases Intelligence* since 2002.[295]

Trends in vaccination coverage estimates from the ACIR

The trends in childhood vaccination coverage in Australia for three doses of DTPa, OPV and two or three doses of Hib and Hep B assessed at 12 months of age, and for three or four doses of DTPa (according to the vaccine schedule in use), two or three doses of Hib and Hep B, three doses of OPV, and one dose of MMR assessed at 24 months, and for two doses of MMR, four or five doses of DTPa, and four doses of OPV assessed at 72 months of age are shown in Figure 53. Coverage was calculated for 36 consecutive three-month cohorts born from 1 January 1996 to 31 December 2004. For all vaccines due by one year of age, coverage estimates increased steadily from 75% for the first cohort, to 90% by the thirty-sixth cohort, assessed on 31 December 2005. For all vaccines due by 24 months of age, coverage estimates also

Figure 53. Trends in vaccination coverage estimates from the Australian Childhood Immunisation Register for 12, 24 and 72 month olds*



Source: Australian Childhood Immunisation Register.

* By three-month birth cohorts born between 1 January 1996 and 31 December 2004. Coverage assessment date was 12 months, 24 months or 72 months after the last birth date of each cohort.

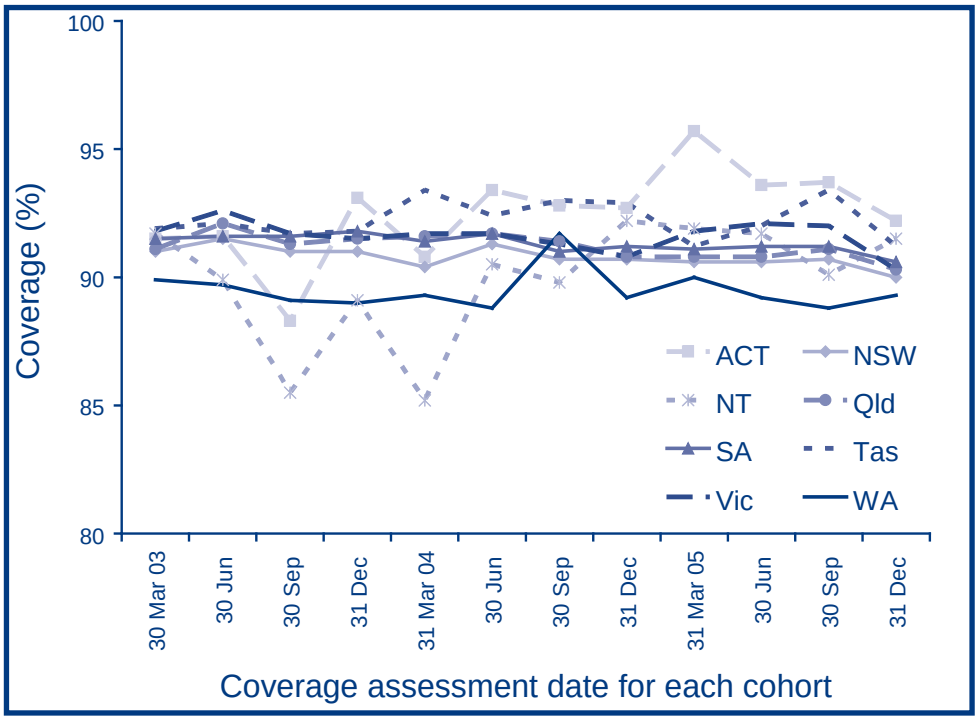
increased steadily from 64% for the first cohort to 92.1% by December 2005. Coverage estimates for all vaccines due by 72 months of age were first reported in *Communicable Diseases Intelligence* in 2002, and have also increased steadily from 80.6% in early 2002 to 83.8% in late 2005.

Coverage estimates for the 24-month age group had a noticeable increase in September 2003, largely as a result of a more liberal definition of completeness following the removal of the fourth dose of DTPa (due at 18 months) from the immunisation schedule from this quarter onwards. Coverage estimates for the 12-month age group have, however, remained steady over the past five years fluctuating around the 91% level. With up to 3% of Australian parents not immunising their children because they object to, disagree with, or are concerned about immunisation,[296] it will be difficult for 'fully immunised' coverage estimates to exceed 95%, especially as the reporting of immunisation encounters is still not totally complete due to poor reporting by some providers and late immunisation by some parents.

Vaccines scheduled in the first year of life

Differences between states and territories in estimates of the proportion of children classified as 'fully immunised' are shown in Figure 54. 'Fully immunised' 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% 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, who have relatively small populations, are likely to be the result of small numbers of unimmunised children having large impacts on the coverage percentages.

Figure 54. Trends in vaccination coverage estimates, by jurisdiction: children 'fully immunised' at the age of 12 months*

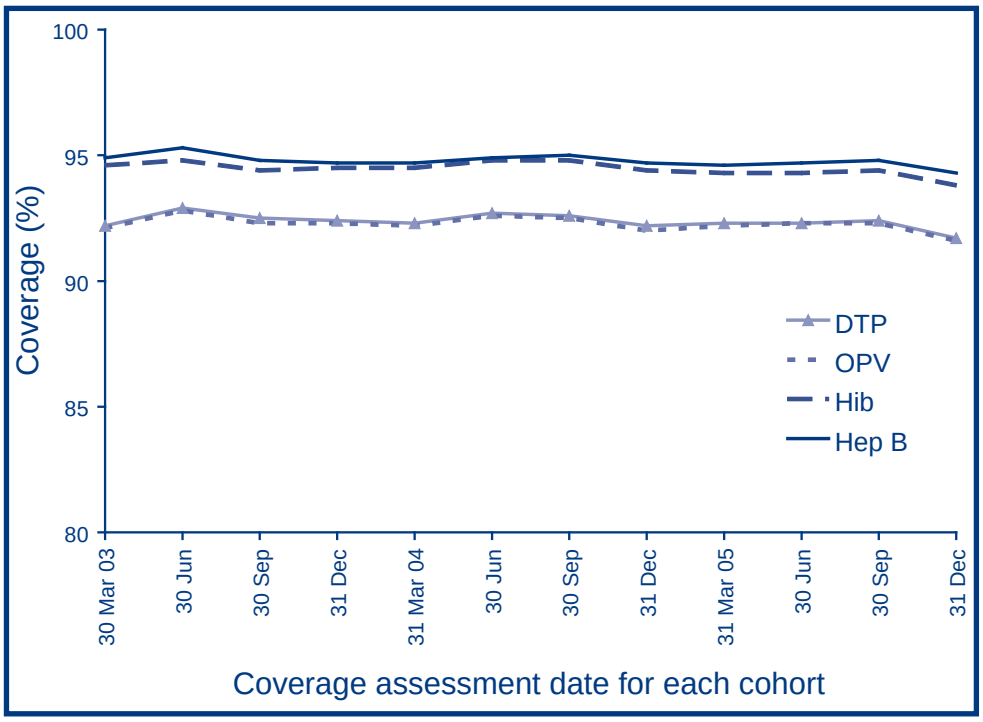


Source: Australian Childhood Immunisation Register

* By three-month birth cohorts born between 1 January 2002 and 31 December 2004. Coverage assessment date was 12 months after the last birth date of each cohort.

The trends in childhood vaccination coverage in Australia for individual vaccines (DTPa, OPV, Hib and Hep B assessed at 12 months) are shown in Figure 55, calculated for 12 consecutive three-month cohorts born from 1 January 2002 to 31 December 2004. Coverage estimates for all vaccines remained stable throughout the 2001 to 2003 period, hovering around the 92% to 95% mark. Coverage for the Hib and Hep B vaccines is greater than DTPa and OPV coverage largely due to the spurious effect of the change in the immunisation schedule in mid 2000, altering the algorithm used to calculate coverage at 12 months of age where a record of two doses of Hib and Hep B on the ACIR is enough for a child to be considered 'fully immunised'.

Figure 55. Trends in vaccination coverage estimates for individual vaccines: children vaccinated with DTPa, OPV, HepB and Hib at the age of 12 months*



Source: Australian Childhood Immunisation Register.

* By three-month birth cohorts born between 1 January 2002 and 31 December 2004. Coverage assessment date was 12 months after the last birth date of each cohort.

Figure 56 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% in almost all jurisdictions, there exist a significant number of areas that have low levels of coverage, below 90%, and even below 85% in a few areas such as Richmond-Tweed and Lismore (NSW).

The timeliness of the third dose of DTPa, due at six months of age, by jurisdiction, for the cohort born 1 January 2004 to 31 March 2004 is shown in Figure 57. Timeliness varied by jurisdiction, with children in Western Australia and the Northern Territory experiencing greater vaccination delay, and children in the Australian Capital Territory being more on time for their third dose of DTPa. However, whilst timeliness varies between jurisdictions, eventually all jurisdictions catch up by 12 months of age (Figure 57).[297]

Figure 56. Immunisation coverage for 'fully immunised' at 12 months of age, Australia, December 2005

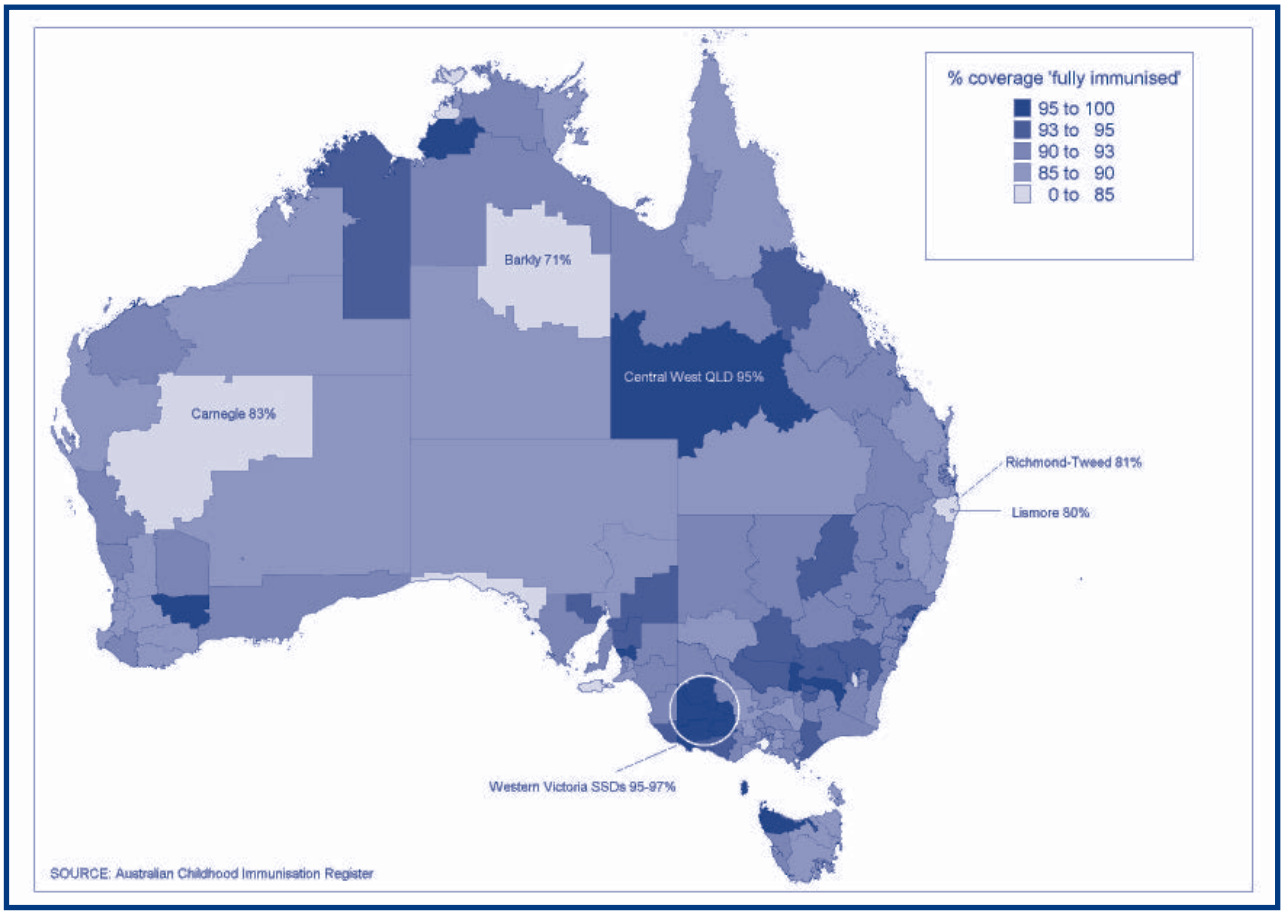
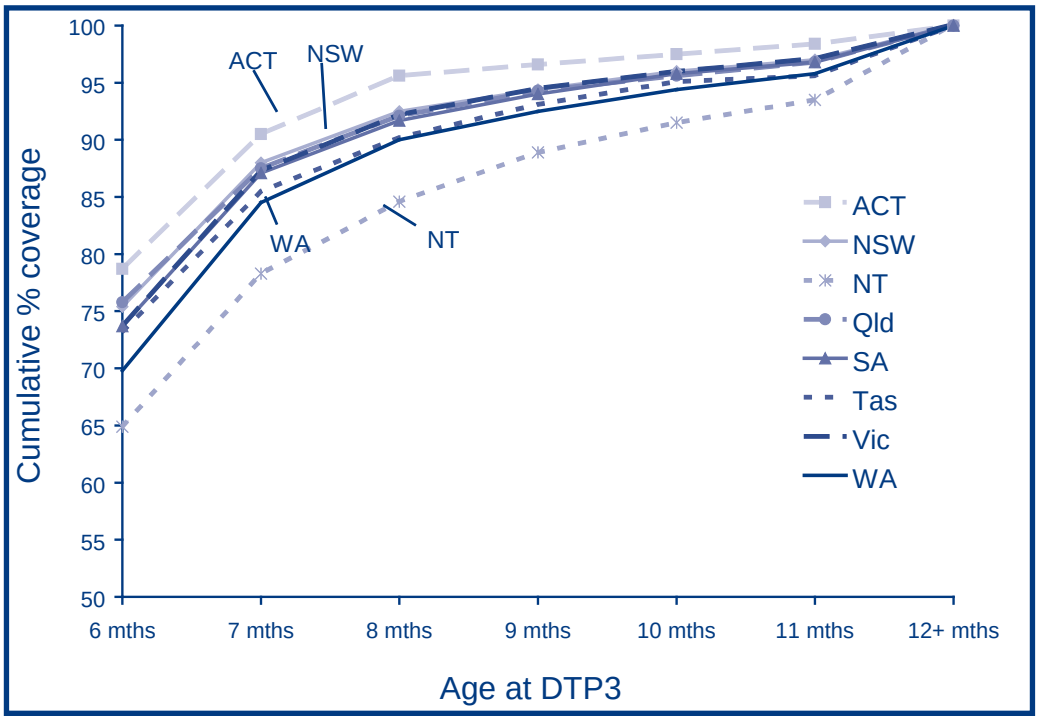


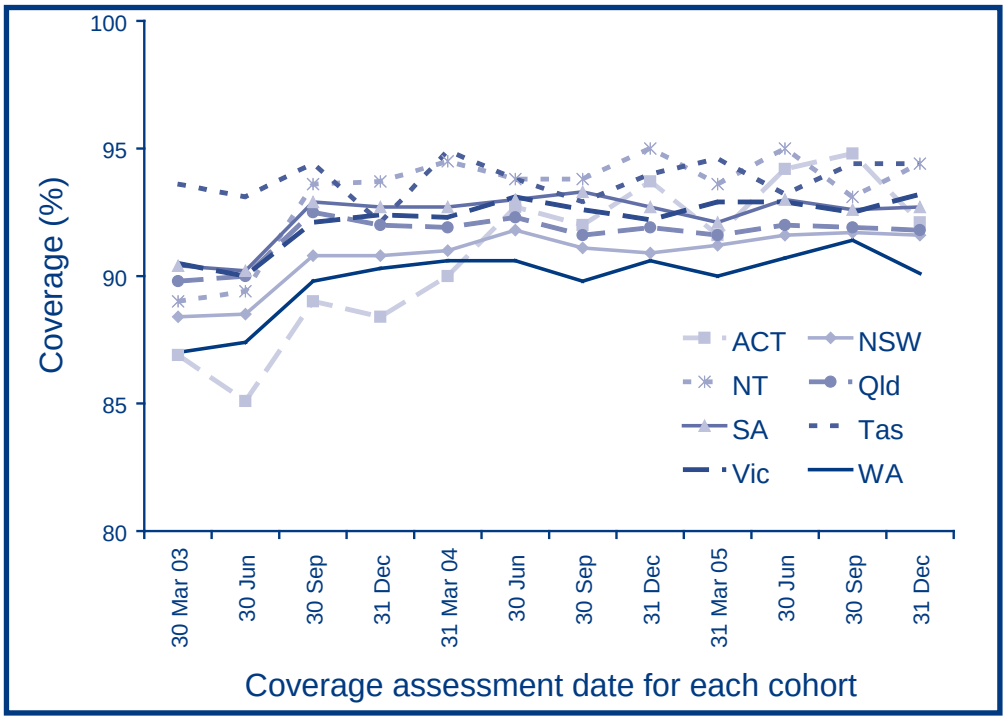
Figure 57. Timeliness of the third dose of DTPa (due at 6 months of age), by jurisdiction – cohort born 1 January 2004–31 March 2004



Vaccines scheduled in the second year of life

Differences between states and territories in estimates of the proportion of children classified as 'fully immunised' at 24 months of age are shown in Figure 58. 'Fully immunised' coverage at 24 months of age for consecutive cohorts increased markedly for all jurisdictions between the June and September quarters in 2003, mainly due to the removal of the fourth dose of DTPa vaccine at 18 months from the immunisation schedule in September 2003. The coverage assessment for the 24-month cohort now excludes the requirement for the 18-month dose of DTPa. There was significant variation between the jurisdictions across the three-year period with Tasmania, the Northern Territory, South Australia and Victoria consistently having higher coverage levels than the other four jurisdictions. However, by the end of 2005, all jurisdictions had reached the 90% coverage target.

Figure 58. Trends in vaccination coverage estimates, by jurisdiction: children 'fully immunised' with DTPa, OPV, HepB, Hib and MMR at the age of 24 months*



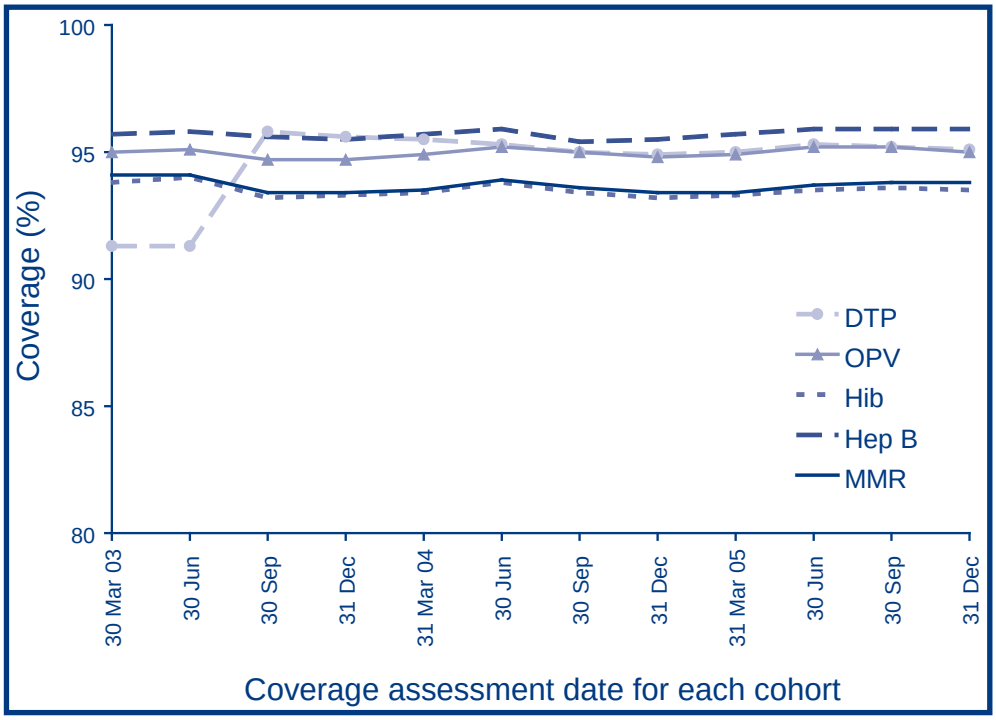
Source: Australian Childhood Immunisation Register.

* By three-month birth cohorts born between 1 January 2001 and 31 December 2003. 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, Hib, Hep B and MMR assessed at 24 months) are shown in Figure 59, calculated for 12 consecutive three-month cohorts born from 1 January 2001 to 31 December 2003. As discussed previously, there was a significant increase in coverage for DTPa at 24 months of age during 2003 due to the removal of the fourth dose of DTPa from the schedule. For most of the three-year period, Hep B coverage was higher than for all other vaccines (most likely due to the coverage algorithm accepting two or three doses of Hep B as an indication of fully immunised status), hovering just below 96%, whilst coverage at 24 months of age was lowest for the MMR and Hib vaccines. However, there has been a real lack of movement in coverage for any of the vaccines assessed at this age, as evidenced by the very flat curves for all vaccines.

Figure 60 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 with Figure 56, that whilst coverage is over 90% in all jurisdictions, there exist many small areas within jurisdictions that have levels of coverage below 90%, such as regional areas in Western Australia and New South Wales.

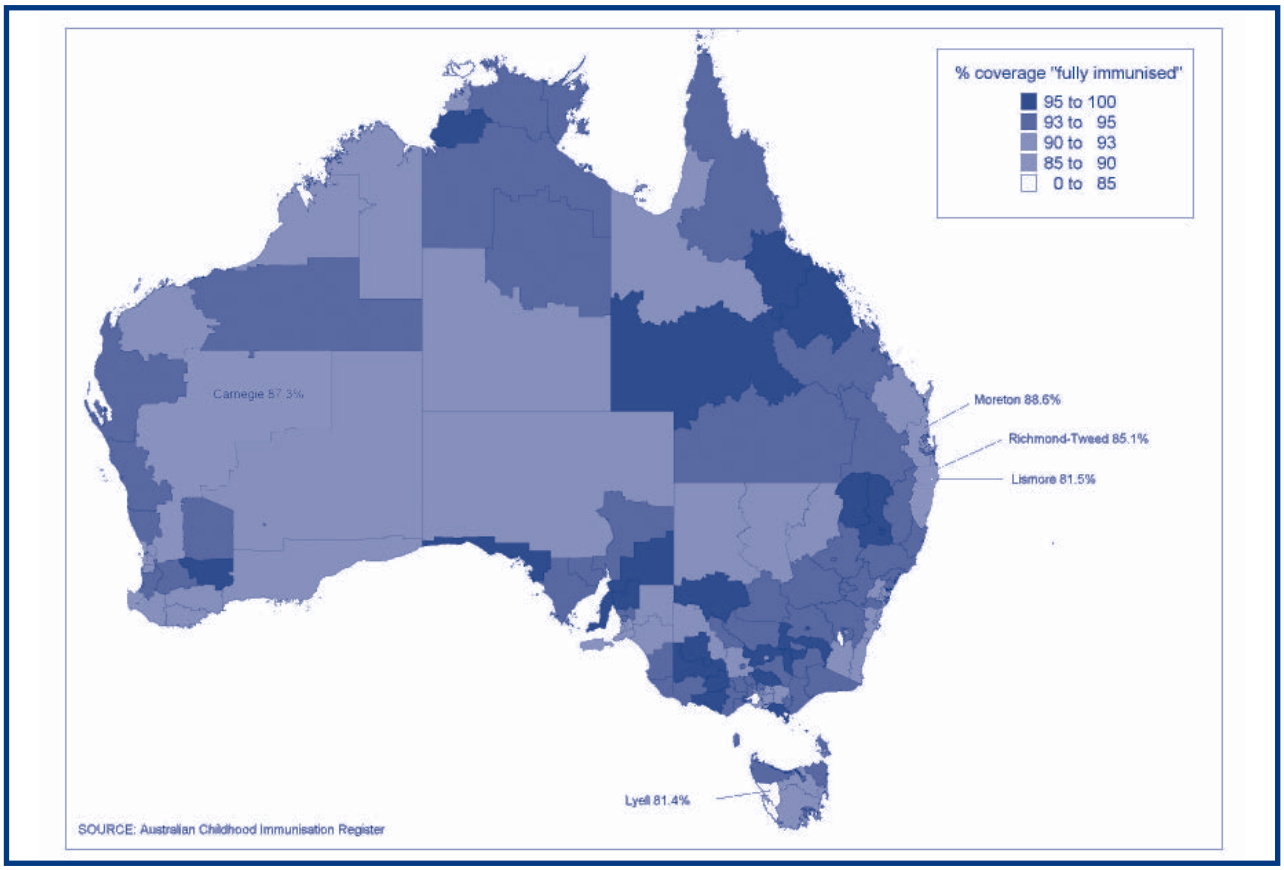
Figure 59. Trends in vaccination coverage estimates for individual vaccines: children vaccinated with DTPa, OPV, HepB, Hib and MMR at the age of 24 months*



Source: Australian Childhood Immunisation Register.

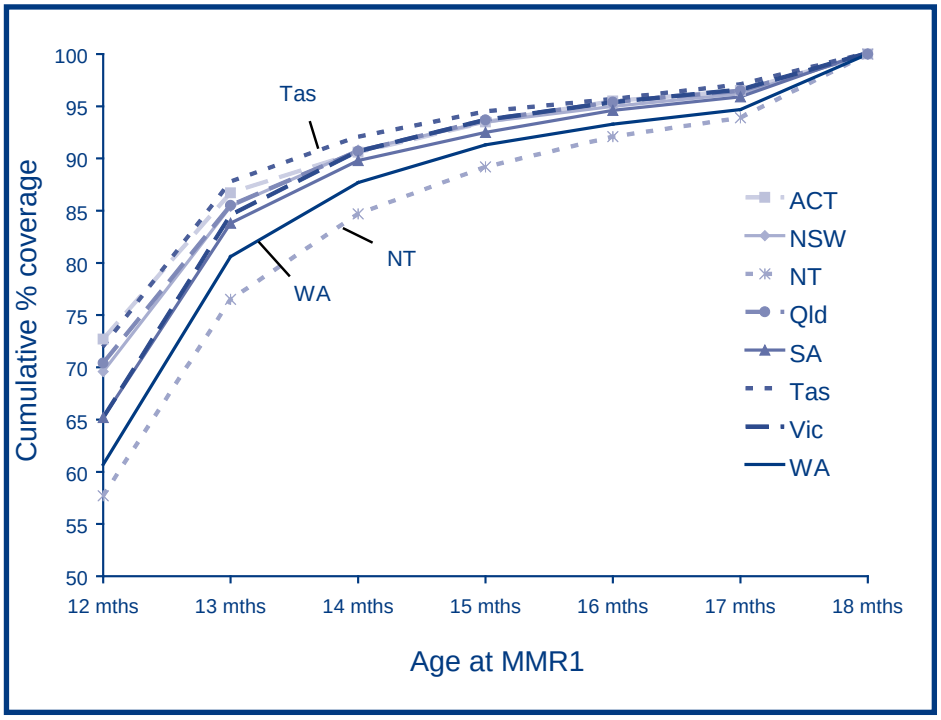
* By three-month birth cohorts born between 1 January 2001 and 31 December 2003. Coverage assessment date was 24 months after the last birth date of each cohort.

Figure 60. Immunisation coverage for 'fully immunised' at 24 months of age, Australia, December 2005



The timeliness of the first dose of MMR by jurisdiction for the cohort born 1 January 2003 to 31 March 2003 is shown in Figure 61. As with the third dose of DTPa, the timeliness of the first dose of MMR varied by jurisdiction, with children in Western Australia and the Northern Territory again experiencing greater vaccination delay, and children in Tasmania being more on time for this vaccine dose. Again, whilst timeliness of MMR vaccination varies between jurisdictions, eventually all jurisdictions catch up by 18 months of age (Figure 61).[297]

Figure 61. Timeliness of the first dose of MMR (due at 12 months of age), by jurisdiction – cohort born 1 January 2003–31 March 2003



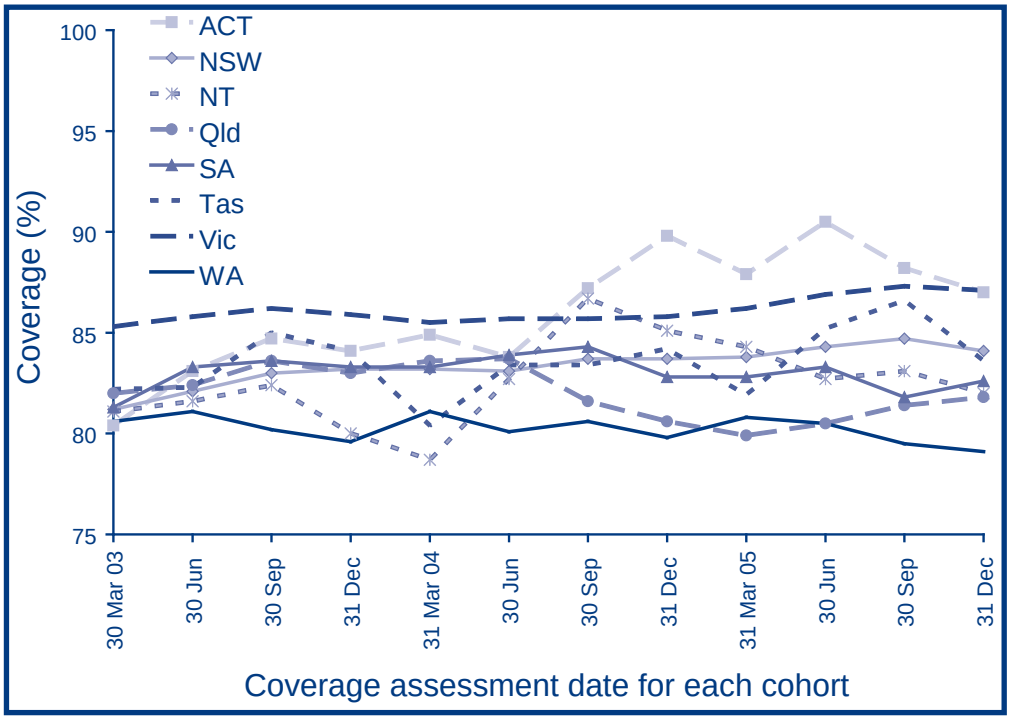
Vaccines given at 4–5 years of age

Differences between states and territories in estimates of the proportion of children classified as 'fully immunised' by 72 months of age are shown in Figure 62. 'Fully immunised' coverage increased only slightly over the three year assessment period for most jurisdictions, with some jurisdictions experiencing a greater increase than others, and a couple (Queensland and Western Australia) experiencing an overall slight decrease. Coverage was highest in Victoria and the Australian Capital Territory.

The trends in childhood vaccination coverage in Australia for individual vaccines (DTPa, OPV and MMR assessed at 72 months) are shown in Figure 63, calculated for 12 consecutive three-month cohorts born from 1 January 1997 to 31 December 1999. Coverage for all three vaccines was almost identical and remained steady across the whole period hovering around 85% with very little change.

Figure 64 presents a map of immunisation coverage at 72 months of age in Australia by Australian Bureau of Statistics (ABS) Statistical Subdivision. The map demonstrates that coverage for this age group, compared with other milestones, is below target levels in many areas of Australia, and is less than 85% in a majority of areas. However, there are a few areas in regional Victorian and Western Australia where coverage has reached levels achieved at 12 and 24 months of age.

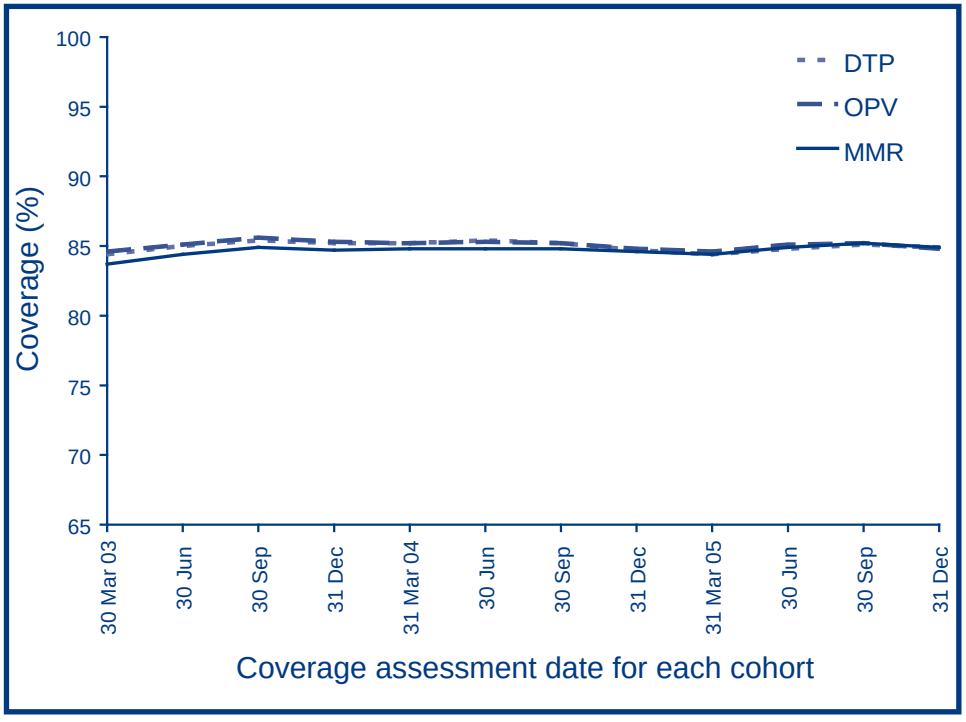
Figure 62. Trends in vaccination coverage estimates, by jurisdiction: children 'fully immunised' with DTPa, OPV and MMR at the age of 72 months*



Source: Australian Childhood Immunisation Register.

* By three-month birth cohorts born between 1 January 1997 and 31 December 1999. Coverage assessment date was 72 months after the last birth date of each cohort.

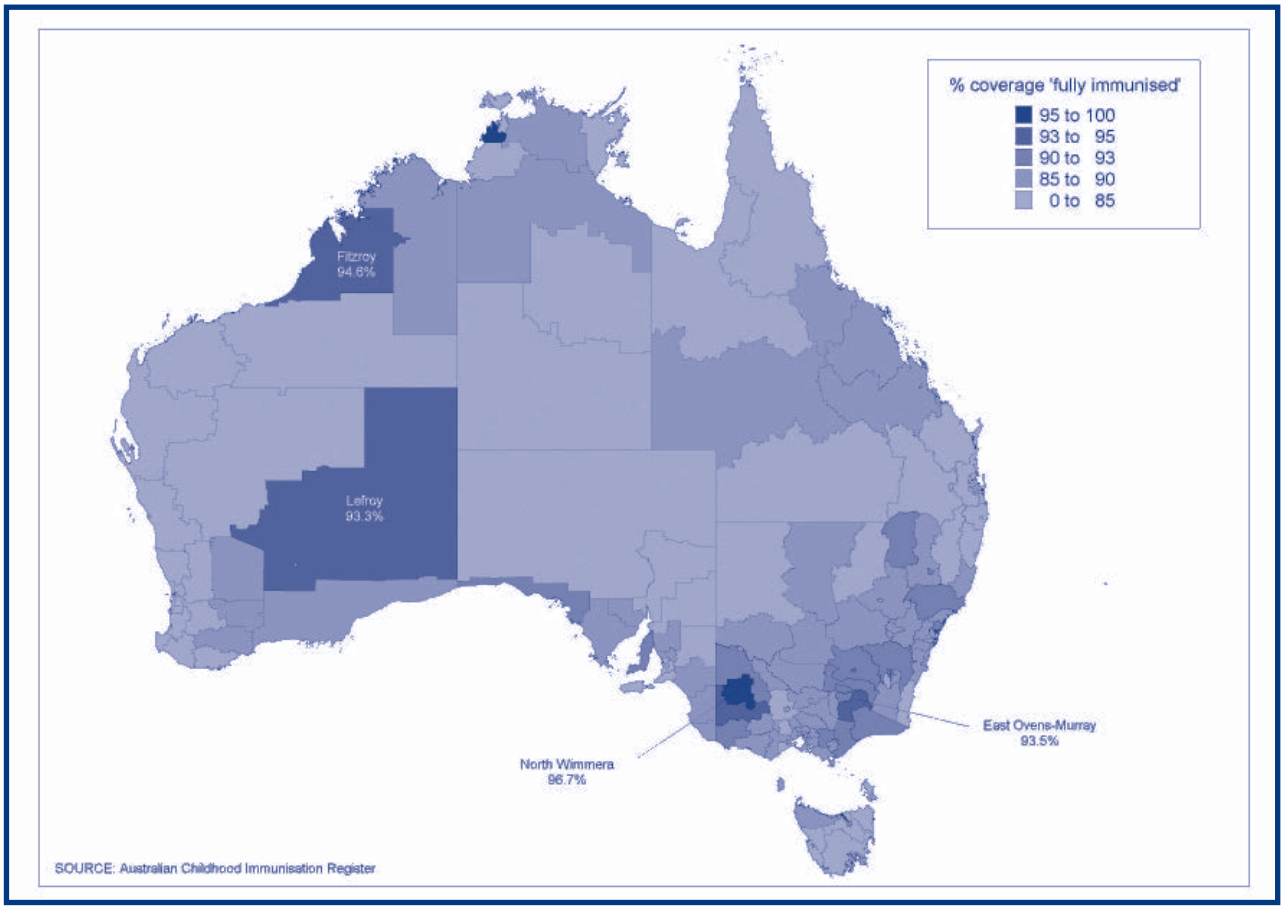
Figure 63. Trends in vaccination coverage estimates for individual vaccines: children vaccinated with DTPa, OPV and MMR at the age of 72 months*



Source: Australian Childhood Immunisation Register.

* By three-month birth cohorts born between 1 January 1997 and 31 December 1999. Coverage assessment date was 72 months after the last birth date of each cohort.

Figure 64. Immunisation coverage for 'fully immunised' at 72 months of age, Australia, December 2005



Comment

Australia is uniquely well placed to monitor immunisation coverage and timeliness of immunisation routinely and regularly because of its universal national childhood immunisation register, the ACIR, the only national register of its kind in the world. The ACIR records immunisation data on all children under the age of 7 years enrolled in the Australian universal health insurance scheme, Medicare, and constitutes a nearly complete population register, as approximately 99% of the 250,000 children born each year are registered with Medicare by 12 months of age.

Estimates of vaccination coverage in Australia for all jurisdictions have increased steadily since the ACIR commenced in 1996 but less so in recent years. There have been increases in coverage for 12 month olds, 24 month olds and 72 month olds, with 'fully immunised' coverage for 12 and 24 month olds reaching the *Immunise Australia Program* target of 90% coverage for the first and second milestone vaccines.

The ACIR is now likely to be performing at close to its maximum achievable capacity in terms of data administration, following the impact of the General Practitioner Immunisation Incentive Scheme, parental incentives, the recording of overseas-given immunisations on the ACIR and data cleaning initiatives such as the ACIR Field Officers. Limitations of the ACIR, 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, especially at the 12 and 24 month old milestones, 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%–3% of parents who are officially opposed to immunisation and a further slightly larger percentage that are opposed to immunisation but who do not object through official channels.

To maintain the current high levels and to achieve further increases in coverage, especially in the 72 month old group, 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 and specific educational initiatives for those parents and providers concerned about contraindications to immunisation.

Analysis of age appropriateness (timeliness) of childhood immunisation in Australia is not routinely undertaken using ACIR data, with regular reports limited to immunisation coverage for scheduled vaccines at the age milestones of 12, 24 and 72 months for each three-month birth cohort.[295] With coverage estimates for most vaccines reaching levels required for herd immunity, the next benchmark for program success is timeliness, which can be regularly reported with an immunisation register, in contrast to the intermittent reports possible from national surveys. In addition, failure to commence the immunisation schedule on time has been shown to be a powerful predictor of failure to complete it.[298,299] Measurement of timeliness should be incorporated into routine monitoring of the success of immunisation program delivery in countries with high levels of immunisation coverage.

It should be noted that there are several national, publicly funded, targeted immunisation programs for which systematically collected data on vaccine coverage are not currently available or published. These include meningococcal, pneumococcal and varicella vaccines for infants, Hep 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[91] or national special purpose surveys and studies[110,111,300,301] for three of these programs, lack of widely applicable data inhibits planning and evaluation at the regional and national level. 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 January 2003 to December 2005 were marked in the main by continuing gains in the control of vaccine preventable diseases and further expansion in routine vaccination programs to cover meningococcal C disease, chickenpox and pneumococcal disease. A particular challenge of the period was that, for the first time, recommendations for the routine use of some new childhood vaccines preceded their provision free of charge to parents (varicella-zoster, inactivated poliomyelitis and 7-valent conjugate pneumococcal vaccine).[302] In December 2000, the Australian Childhood Immunisation Register (ACIR) documented that the 90% coverage target for immunisation of 12 month olds had been achieved for the first time. This has been maintained and exceeded through to the end of 2005. This target has now also been met for immunisation of two year olds. Such coverage is likely to have been facilitated by the now routine use of combination vaccines.

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 (e.g. against rotavirus, human papillomavirus and shingles) and the incremental 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, mortality and vaccine coverage) 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 three years prior to the current report (2000 to 2002) is shown in Table 28 and for the three years 2003 to 2005 in Table 29. While the limitations of the data sources for notification, hospitalisation and death 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 continued in the period 2003 to 2005. Among diseases currently targeted by immunisation, hospitalisations due to measles, rubella, mumps, and presumed Hib disease have all continued to decrease. Although vaccination programs are relatively recent for these diseases, decreases in hospitalisations for meningococcal disease, pneumococcal disease, and varicella disease have also occurred. Hospitalisations due to pertussis continue to be a significant burden in young infants. Influenza, varicella, pneumococcal disease, pertussis 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 commonly coded vaccine preventable diseases in hospitalised patients also included influenza and varicella, but zoster was the most common recorded cause. The vaccine preventable diseases most frequently recorded as the underlying cause of death were influenza in adults and meningococcal disease in infants. 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 2005.

Diseases on the Australian Standard Vaccination Schedule in 2003–2005

Measles and rubella

During 2003–2005, measles and rubella notifications and hospitalisations continued their decline, reaching record lows, with about 90% and 80% reductions, respectively, compared with earlier in the decade. These achievements reflect the success of the Measles Control Campaign as well as ongoing high childhood vaccination coverage with measles-mumps-rubella (MMR) vaccine. Australia's remaining susceptible cohort of young adults born in the 1970s and early 1980s were targeted by a young adult MMR vaccination campaign in 2001. Both a serological evaluation[130] and data presented in this report suggest that the campaign failed to significantly improve immunity in this age group. Improving immunity in this cohort and communicating the potentially serious nature of infection with measles and rubella in

Table 28. Average annual morbidity and mortality from vaccine preventable diseases in Australia for 3 years 1999/2000–2001/2002*

Disease†	Notifications 2000–2002 (average no.)		Notification rate/100,000 (average rate)		Hospitalisations 1999/00–2001/02 (average no.)		Hospitalisation rate/100,000 (average rate)		Deaths‡ 2000–2002 (average no.)		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	0	0§	0§	0	0	0	0	0	0
Hib‖	11	15	0.9	0.4	17	18	1.3	0.4	0	0	0	0
Hepatitis A	38	587	3	3	13	442	1.0	2.3	0.3	1.3	0.03	0.01
Hepatitis B‡,¶	2	407	0.13	2.1	0.3	154	0.0	0.81	0	9	0	0.05
Influenza**	1,510	3,671	119	19	696	3,425	54	18	2	51	0.16	0.26
Measles	22	94	1.7	0.5	17	58	1.4	0.3	0	0	0	0
Meningococcal disease	199	667	16	3	269	854	21.0	4.5	11.3	39	0.89	0.20
Mumps††	15	132	1.2	0.7	10	46	0.8	0.2	0	1	0	0.005
Pertussis	602	7,013	47	36	363	542	28.3	2.8	2	2.3	0.16	0.012
Pneumococcal (invasive)‡‡,**	802	2,466	63	13	341	1,021	26.6	5.3	2.3	14.3	0.18	0.07
Poliomyelitis‡	0	0	0	0	0	1.3	0	0.007	0	0	0	0
Rubella	26	281	2.1	1.4	9	28	0.7	0.14	0	0	0	0
Tetanus	0.3	5	0.03	0.03	0.7	29	0.05	0.15	0	1	0	0.005
Varicella	NN	NN	NN	NN	774	1,686	60	9	0.7	8	0.05	0.04
Zoster	NN	NN	NN	NN	45	4,790	3.5	25	0	15.3	0	0.08

* Notification data, National Notifiable Diseases Surveillance System, January 2000–December 2002; hospitalisation data, Australian Institute of Health and Welfare (AIHW) National Morbidity database, July 1999–June 2002; death data, AIHW National Mortality database, January 2000–December 2002.

† See Chapter 3 for case definitions.

‡ Principal diagnosis only for hospitalisations (hepatitis B, poliomyelitis, diphtheria), and for deaths, only cases with disease classified as underlying cause of death.

§ Only includes hospitalisations coded as pharyngeal, nasopharyngeal or laryngeal diphtheria.

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

¶ Includes only acute hepatitis B notifications, hospitalisations and deaths.

** Notifications only complete for 2002 (influenza and pneumococcal disease)—notification rate for 2002 only.

†† In Queensland, mumps not notifiable in 2000.

‡‡ Includes pneumococcal meningitis and septicaemia only.

NN Not Notifiable.

Table 29. Average annual morbidity and mortality from vaccine preventable diseases in Australia for 3 years 2002/2003–2004/2005*

Disease†	Notifications 2003–2005 (average no.)		Notification rate/100,000 (average rate)		Hospitalisations (average no.)		Hospitalisation rate/100,000 (average rate)		Deaths‡ 2003–2004 (average no.)		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	0	0§	0§	0	0	0	0	0	0
Hib‖	8	10	0.6	0.3	12	13	0.9	0.3	0	0	0	0
Hepatitis A	31	358	2.5	1.8	9	252	0.7	1.3	0	0	0	0
Hepatitis B‡,¶	2	291	0.16	1.5	0	172	0	0.87	0	11	0	0.06
Influenza	1,058	3,395	84	17	1,039	3,039	82	15	2.5	51	0.20	0.25
Measles	8	49	0.6	0.3	12	31	1.0	0.2	0	0	0	0
Meningococcal disease	142	452	11	2.3	219	712	17	3.6	6	23	0.5	0.1
Mumps	8	140	0.6	0.7	4	46	0.3	0.2	0	0.5	0	0.003
Pertussis	563	8,345	45	42	255	440	20	2.2	0.5	1	0.04	0.005
Pneumococcal (invasive)**	571	2,101	45	10	277	1,038	22	5.2	5.5	19	0.4	0.095
Poliomyelitis‡	0	0	0	0	0.3	2	0.03	0.01	0	0	0	0
Rubella	4	39	0.3	0.2	7	15	0.6	0.07	0	0	0	0
Tetanus	0	4	0	0.02	0	22	0	0.11	0	0	0	0
Varicella	NN	NN	NN	NN	532	1,427	42	7.2	1.5	5.5	0.12	0.03
Zoster	NN	NN	NN	NN	36	4,975	2.8	25	0	19	0	0.095

* Notification data, National Notifiable Diseases Surveillance System, January 2003–December 2005; hospitalisation data, Australian Institute of Health and Welfare (AIHW) National Morbidity database, July 2002–June 2005; death data, AIHW National Mortality database, January 2003–December 2004.

† See Chapter 3 for case definitions.

‡ Principal diagnosis only for hospitalisations (hepatitis B, poliomyelitis, diphtheria) and for deaths only cases with disease classified as underlying cause of death.

§ Only includes hospitalisations coded as pharyngeal, nasopharyngeal or laryngeal diphtheria as per included codes for previous review period.

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

¶ Includes only acute hepatitis B notifications, hospitalisations and deaths.

** Includes pneumococcal meningitis and septicaemia only.

NN Not Notifiable

the face of near absent disease remain important and far from hypothetical challenges. Whilst regional measles elimination would appear to be in reach, regionally rubella control remains at an early stage. The laboratory identification and typing of isolates from measles and rubella cases is now vital in order to monitor the origin and any transmission of these diseases in Australia.

Mumps

Since 2002, when mumps notifications were the lowest on record, mumps notifications have increased, and in 2005 reached the highest level since notification began in 1993. This rise was primarily due to increased notification rates in the 20–34 year age group, especially in Queensland and New South Wales, with the highest incidence occurring during 2005 in the 25–29 year old age group (5.1 per 100,000). Notification rates in children remained low. Despite the increase in mumps notifications, rates in Australia were low in comparison to the rates experienced in the United Kingdom during the epidemic of mumps in 2004/2005.[146] The peak age group affected in the UK, and also in recent outbreaks in the USA,[148] were 18–24 year olds. Thus, Australia faces similar issues to other developed countries that are also experiencing mumps resurgence in older susceptible cohorts. The high notification rates in the 20–34 year age group suggest that some endemic transmission may be occurring in this age group. Surveillance for mumps and further consideration of initiatives to effectively target young adults for immunisation are of continued importance in the Australian context.

Hib disease

The virtual disappearance of invasive Hib disease among children less than five years old has been an ongoing success story for vaccination with continued year on year falls in notifications of invasive Hib disease during 2003–2005. Laboratory confirmation with definitive typing remains very important, now that Hib disease is even rarer and as the relative incidence of non-type b invasive *Haemophilus influenzae* increases. Surveillance of Hib disease, through hospitalisation data, would greatly benefit from a specific ICD code for type b disease, which would provide an additional source of information independent from notifications.

Pertussis

Trends in pertussis are difficult to interpret as the disease is by nature variable with peaks every 3–4 years. It stands out as an ongoing challenge, with an epidemic year experienced in 2005 greater in size than the previous peak (>11,000 notifications compared with about 9,600 in 2001). However, it is encouraging that pertussis notifications and hospitalisations have fallen in the most immunised age group (1–10 years), with most notifications occurring either in those too young to be immunised or in adolescents and adults whose vaccine-derived or infection-induced immunity has waned over time. The review period saw the incorporation of a booster dose of pertussis vaccine in adolescence and, although it is still early, there are data to support the success of this strategy already, driven perhaps by the active catch-up adolescent programs implemented in two states. Current recommendations also encourage dTpa vaccination for prospective and recent parents and for adults working with young children, but uptake is uncertain and unlikely to be high.[76]

Influenza

The data presented in this report, although minimal estimates of influenza cases, indicate that the disease burden from influenza is large, with the highest number of hospitalisations and bed days occurring at the extremes of age, in children under five years of age and in the elderly. There are intermittent surges in influenza every few years but data from Australia and the UK are consistent with a gradual decline in such peaks; these are largely due to influenza A H3N2 that started as a pandemic strain in 1968 and to which (and its many drift variants) substantial herd immunity has developed. Influenza notifications commenced nationally in 2001 for laboratory-confirmed cases and, although a gross underestimate of disease burden, these provide useful information about the 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. Annual influenza vaccination is currently recommended as the primary method of influenza prevention in people aged 65 years and over, all Indigenous people aged 50 years and over, and all individuals aged 6 months and over with chronic medical conditions likely to be exacerbated by influenza or its complications, e.g. chronic pulmonary or cardiovascular disease.[76] Vaccination uptake in Australians aged 65 years and over was estimated at 76.9% and 79.1% for 2003[110] and 2004,[111] respectively. Extension of influenza vaccination to all adult Indigenous people should be considered as both influenza hospitalisation rates and mortality related to influenza and pneumonia are twice that documented in persons

of non-Indigenous background across all age groups.[77] As for the paediatric population, in 2003, the USA Advisory Committee on Immunization Practices (ACIP) recommended routine influenza vaccination[113,114] of healthy American children aged 6–23 months based on the high burden of illness.[99,115,116] More recently, in 2006, the ACIP extended its recommendation to include children up to the age of five years.[117] Whilst available Australian data suggest a similar significant burden of illness in young children,[118,119] examination of cost-effectiveness, efficacy and feasibility of universal immunisation of healthy children is required before implementing such a population level strategy.[120] Notwithstanding bias due to variable rates of documenting notification or hospitalisation across different age groups, it is noteworthy that, during 2003–2005, notifications in children under 5 years of age were five times greater than in the elderly aged 60 years and over and hospitalisation rates were, similarly, four times higher.

Hepatitis B

At both national and jurisdictional levels, notifications increased between 1993 and 2001 and since then have declined, while hospitalisations have remained steady since 1999. Notification rates in Victoria are disproportionately high compared with other jurisdictions, probably reflecting a more active approach to surveillance. However, hospitalisation rates are also higher suggesting the possibility of a truly higher rate of disease in Victoria. The review period saw a decline in notifications that was most marked in young adults aged 15–24 years. However, it is too soon to expect such a decline as a consequence of universal neonatal vaccination, which commenced nationally in 2000. One potential reason for the considerable reduction in notifications, particularly in 15–24 year olds, is the declining rates of intravenous drug use since 2000, consistent with trends in hepatitis C virus notifications.[90] It is likely that the impact of both the targeted infant and catch-up adolescent vaccination programs will become more evident in the next 5–10 years.

Rare vaccine preventable diseases (tetanus, diphtheria and poliomyelitis)

Tetanus continues to occur at a very low but seemingly declining rate with an average of five cases per year in 2000–2002 compared with 3.7 cases per year in 2003–2005. Tetanus is now largely a disease of older adults, reinforcing the need to check tetanus vaccination status when older adults present for other reasons, such as a routine visit for annual influenza vaccination. 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. Australia and the Western Pacific region have been declared polio free,[303] but the importation and subsequent polio outbreak in Indonesia in 2005[185] highlight the ongoing need for high vaccination coverage and improved active surveillance for acute flaccid paralysis until global certification is achieved. The routine use of IPV, implemented in late 2005, will eliminate the small risk of vaccine-associated paralytic poliomyelitis. With the replacement of OPV with IPV in Australia, incidental detection of polioviruses in faecal specimens should no longer occur. Future poliovirus isolations will, therefore, require full investigation.[179]

Varicella-zoster

With the introduction of varicella-zoster vaccine in late 2005, surveillance of the disease burden due to both varicella and zoster have become greater priorities. Because disease modelling has raised the possibility that an increase in zoster disease may flow on from a reduction in varicella (through reduction of natural 'boosting' of immunity due to intermittent exposure to varicella infection in the community),[287] zoster surveillance will be as important as documenting declines in varicella infection in the coming years. Currently, surveillance data from the USA, where varicella immunisation has been recommended for over a decade, indicates a large reduction in varicella morbidity with no increase in zoster disease yet demonstrated.[291] The availability of a vaccine to prevent zoster may become an important option in the future should surveillance detect an increase in zoster disease.[283]

Varicella-zoster surveillance has been funded as part of the national varicella immunisation program[292] and will include notification to the NNDSS (in five jurisdictions, surveillance will comprise passive notification through GPs and labs; in two, it will include passive notification as well as syndromic surveillance, with specimen collection through sentinel GPs/emergency departments; and in one state, varicella will not be notifiable, with emergency department syndromic surveillance only).[293] Additionally trends in disease burden will continue to be reviewed through hospitalisation data and Australian Paediatric Surveillance Unit data on neonatal and congenital varicella infections as well as severe infections in older children requiring hospitalisation.

Pneumococcal disease

Vaccination with the 23vPPV has been funded nationally since 1999 for Aboriginal and Torres Strait Islander adults aged 50 years and over, and those aged 15–49 years with high-risk conditions.[3] Nationally funded 7vPCV vaccination commenced in 2001 for all Aboriginal and Torres Strait Islander infants and other infants with high-risk conditions.[3] In 2005, the 7vPCV was funded for all Australian infants and the 23vPPV for all aged 65 years and over. Data in this report suggest a noticeable impact in the first year of universal infant vaccination as, in 2005, the IPD notification rate declined by 25% overall and by 66% in those aged less than two years compared to the three previous years, and by 80% in cases with serotypes contained in the vaccine. Smaller decreases occurred in all other age groups, but these should be interpreted with caution due to the smaller numbers of cases in these age groups. Hospitalisation rates were lower in 2004/2005 compared to previous years, although this included only the first six months of the funded vaccination program. There were decreases in all age groups, but the greatest decrease was seen in infants (37%).

Concerns about replacement disease by serotypes not contained in the vaccine have been expressed.[167] In the US, some replacement IPD appears to have occurred, but there is still a substantial overall decrease in IPD notifications.[164] The data presented here for Australia on the first year of the vaccination program suggest the possibility of some replacement disease in unvaccinated age groups, but a substantial overall decline in IPD rates. It will be important to monitor this closely in future.

Disparities between Indigenous and non-Indigenous children may re-emerge following universal vaccination, due to higher rates of non-vaccine type disease in Indigenous people.[163] New conjugate vaccines with greater serotype coverage may be necessary to address this and the persistent high rates in Northern Territory Indigenous adults.

Meningococcal disease

The incidence of meningococcal disease in Australia increased continuously between 1991 and 2002.[3] Following the introduction of the routine and catch-up meningococcal C vaccination programs in January 2003 for those born after 1983 (aged 19 years in 2003),[136] there has been a marked decrease in meningococcal disease notifications, hospitalisations and deaths. The reduction in notifications occurred for both serogroup C disease and those where serogroup information was not available, while notifications for serogroup B and other serogroups, mainly W135 and Y, remained relatively stable. A challenge remains to control serogroup C disease in young adults in whom the catch-up campaign was less comprehensive than for those immunised in schools.

The high burden of meningococcal disease in infants, particularly non-vaccine preventable serogroup B disease, emphasises the importance of early recognition and appropriate clinical management of disease and the need for a vaccine to reduce the significant morbidity and mortality. Several candidate serogroup B vaccines are under investigation in international Phase II clinical trials.[142] However, availability of a universal serogroup B vaccine appropriate for use in Australia is still some way off.

Vaccine preventable diseases not on the Australian Standard Vaccination Schedule in 2003–2005

Hepatitis A

In Australia, as in other industrialised countries, hepatitis A occurs sporadically with periodic epidemic peaks related to point-source and community-wide outbreaks. Notification and hospitalisation rates fell over the last three years. The epidemiology of hepatitis A differs significantly for the Indigenous population, where it has been endemic, compared with the non-Indigenous population. The greater disease burden in Indigenous children has been particularly pronounced in more remote areas. In 1999, an immunisation program commenced for Indigenous children aged 18 months to 6 years living in north Queensland. This was expanded in 2005 to include all Indigenous children aged 12 to 24 months in the Northern Territory, Queensland, South Australia and Western Australia. Continued monitoring should be a priority in Australia, both to assess the impact of these recent changes, and the need for any further expansion of vaccination coverage.[82] In the United States, hepatitis A cases decreased substantially following the recommendation of vaccination of children in communities with high rates of disease in 1996, and for states and counties with high hepatitis A notification rates in 1999. In 2006, this was expanded to include all US infants, as part of a staged implementation of progressively expanded vaccination.[83]

Q fever

Australia is unique in having a Q fever vaccine available and used in a national program. The National Q fever Management Program (NQFMP), implemented in 2001–2002, promoted and provided screening and vaccination services for those at highest risk of Q fever (meat and livestock industry workers and their families and those working on farms). Subsequently, Q fever notification and hospitalisation rates have declined to record low levels. Both because the program is continuing and because it is difficult to evaluate the contribution of non-program factors (e.g. variations in drought conditions or livestock slaughtering) to reductions in disease, continued and enhanced surveillance will be required in the coming years.

Rotavirus

Rotavirus is responsible for a significant number of hospitalisations in Australia each year (around 3,500 hospitalisations were recorded annually with either a principal or non-principal diagnosis of rotavirus). The primary disease burden is in those under five years of age, with hospitalisation most common in the first two years after birth. Two vaccines became available in Australia in 2006 for the prevention of rotavirus gastroenteritis and were recommended for funding under the National Immunisation Program (NIP) for all infants by the Pharmaceutical Benefits Advisory Committee in 2006. The Northern Territory made one of the rotavirus vaccines available free of charge from 1 October 2006 for infants born after 1 August 2006. Universal immunisation of infants in the first six months of life under the NIP should prevent the majority of severe cases of rotavirus and it is anticipated that hospitalisation rates will be substantially reduced. However, it will be important to encourage timeliness of vaccination in order to ensure a maximal impact upon disease. Ongoing analysis of national hospitalisation data will provide valuable data to help assess the impact of a national vaccination program, with laboratory notification in place in the Northern Territory and Queensland and proposed for other jurisdictions.

Vaccine preventable disease notification rates compared with other industrialised countries

The most recent notification rates for five vaccine preventable diseases compared with the rates in New Zealand, the USA, Canada and England, are shown in Table 30. Notifications of invasive Hib disease were low in all countries, reflecting the excellent results of Hib vaccination programs. Australia has moved closer to the situation in North America with respect to measles elimination, with notification rates decreasing from 1.7 per 100,000 in 1998 to 0.1 per 100,000 in 2005. The mumps outbreak in young adults in the UK is clearly evident, with the increasing notifications in Australia mild in comparison. Pertussis notification rates in Australia remain high compared with the USA, Canada and England, although this is attributable at least in part to the availability of serology for diagnosis. New Zealand has experienced a recent epidemic period with high rates in children primarily diagnosed by PCR or culture. Comparisons with other countries are difficult because of differences in case definitions for notification and laboratory diagnosis and notification in Australia. Nevertheless, it is likely that Australia still has a comparatively high pertussis disease burden, as reflected in hospitalisations.

Table 30. Most recent* notification rates per 100,000 population for frequently notified vaccine preventable diseases, by country of residence

Disease	Australia	New Zealand[304]	USA[305]	Canada[306]	England/Wales[307]
Hib disease	0.1	0.2	0.7	0.2	0.3
Measles	0.1	0.5	0	<0.05	4.6 (0.2)†
Mumps	1.2	1.7	0.1	0.1	31.1 (17.7)‡
Pertussis	55.1	72.8	8.9	8.8	1.0
Rubella	0.2	0.3	0	<0.05	2.5 (<0.05)§

* Australia 2005; New Zealand 2005; USA 2004; Canada 2004; England/Wales 2004.

† Incidence corrected for proportion serologically confirmed = 5%.

‡ Incidence corrected for proportion serologically confirmed = 57%.

§ Incidence corrected for proportion serologically confirmed = 0.2%.

Future surveillance priorities

For this 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 again been enhanced by NCIRS' relationship with the AIHW as a collaborating centre. The presentation, for the first time in this report, of some recently enhanced data fields such as immunisation status and typing information from the National Notifiable Diseases Surveillance System (NNDSS), has shown how these fields provide important additional information but that there is still room for improvement in regards to data completeness. The introduction of revised national case definitions in 2004, including those for vaccine preventable diseases, provides increasing consistency to notification data. The 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) provide increasing confidence in notification data. The typing of clinical specimens for measles and rubella is now very important to document transmission of any imported cases and absence of endemic circulating types. The recent additions of varicella, meningococcal C and pneumococcal conjugate vaccines to the NIP in 2005 make close monitoring of the impact of these vaccination programs critical. Enhanced laboratory surveillance is in place for meningococcal and pneumococcal disease, and varicella-zoster surveillance through national notification is imminent. A study involving collaboration between NCIRS and APSU will focus on cases hospitalised with severe varicella disease and collect samples for viral characterisation. A similar collaboration to improve surveillance for acute flaccid paralysis is underway in order to document elimination of polio disease in Australia. A whole of life vaccination register is under consideration and, if implemented, would aid evaluation of vaccine effectiveness across the spectrum.

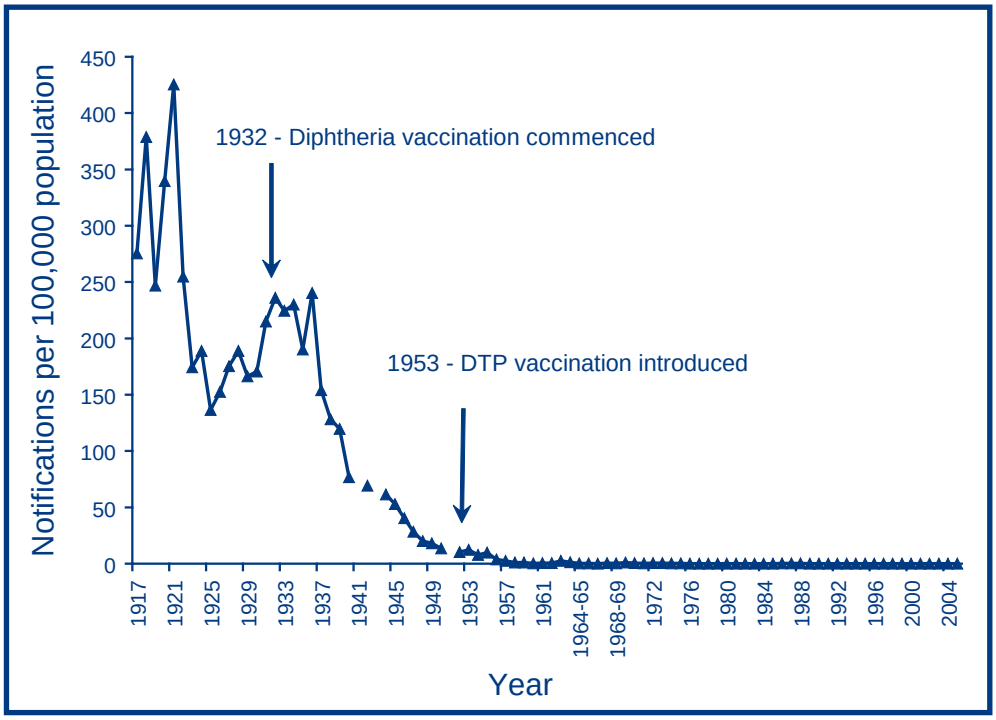
Future vaccination priorities

Table 29 provides measures of morbidity for comparison of disease burden relevant to current general or targeted 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.[41,296,308,309] 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 in young adults, for diseases such as measles, mumps and meningococcal disease.

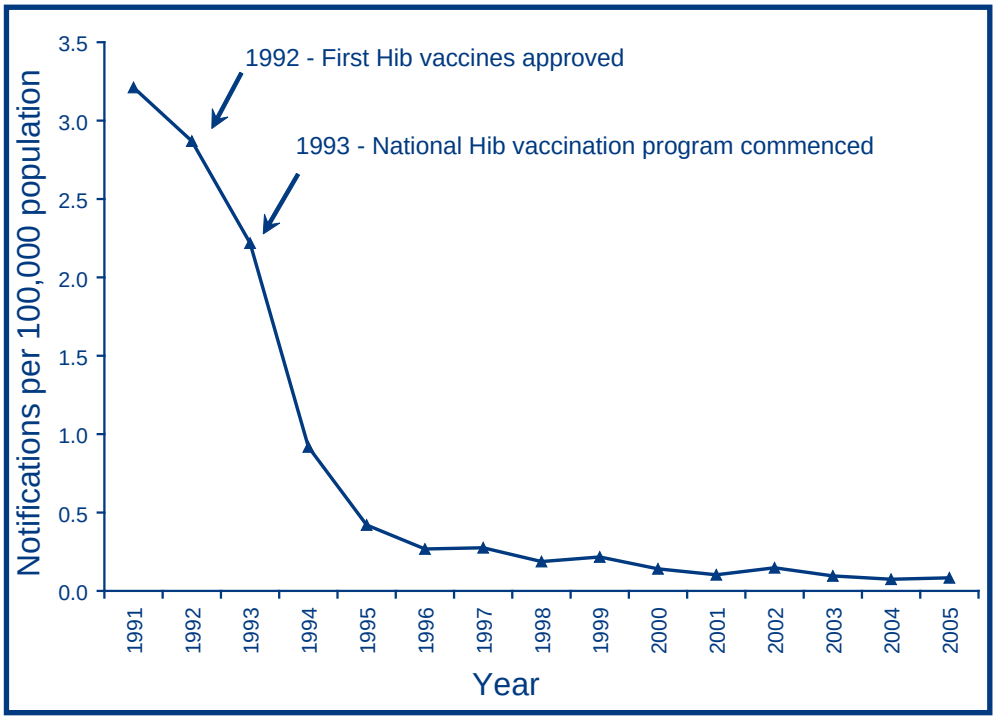
With respect to immunisation programs targeting diseases currently included in the NIP schedule, measles, mumps, meningococcal disease and pertussis in older teenagers and young adults remain priorities. Australia has so far not found a successful means to deliver vaccination programs to older adolescents and young adults no longer attending school, with the approaches adopted for the promotion of MMR and meningococcal C vaccines in this age group having relatively little impact. School-based delivery of conjugate meningococcal C and adult-formulated pertussis vaccines has been a success and provides a model for other interventions in educational institutions. Delivery of vaccines such as MMR, pertussis and meningococcal C to young adults is difficult to implement and it is likely that this group will represent an ongoing challenge for control of measles, mumps, rubella, pertussis and meningococcal C over the next few years. The 2003 addition to the ASVS of dTpa for adolescents in Australia demonstrated an international lead in pertussis control, which should be mirrored in notifications, provided high coverage is maintained. Australia is now in the mature phase of control for many vaccine preventable diseases, where incidence is very low and effectively close to elimination. Vaccines against diseases such as human papillomavirus and rotavirus will be implemented in the near future. Evaluation of the impacts of these new programs, as well as continued efforts to maintain current programs, will be required to sustain the success of immunisation in Australia over the coming years.

Appendix 1. Historical charts of notifications of vaccine preventable diseases

Diphtheria, 1917–2005

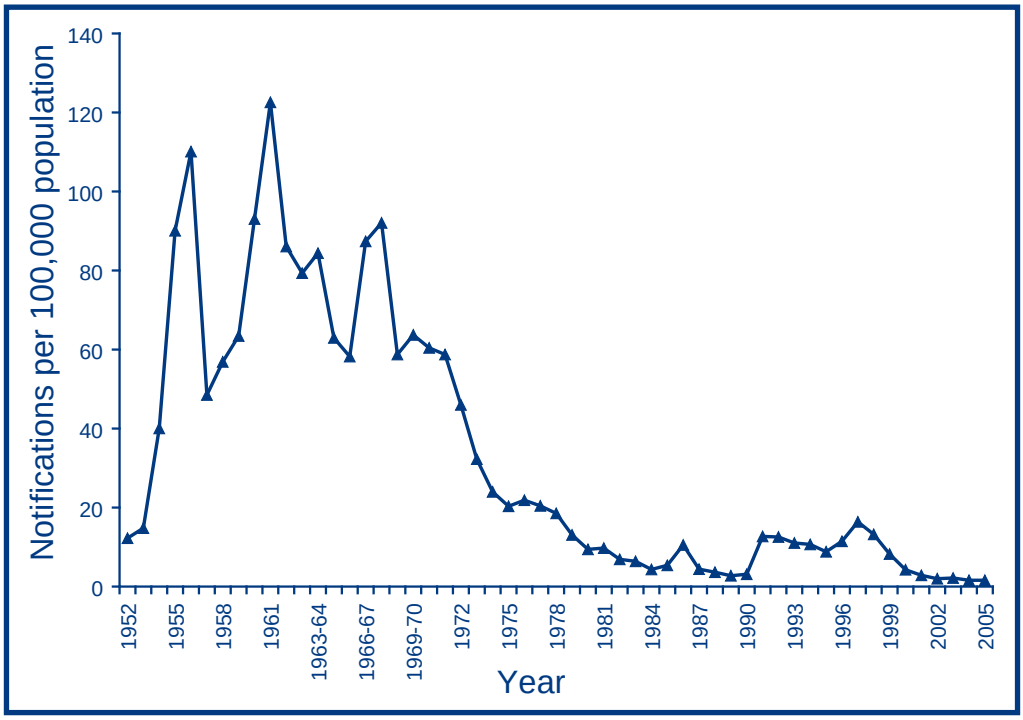


Haemophilus influenzae type b disease, 1991–2005

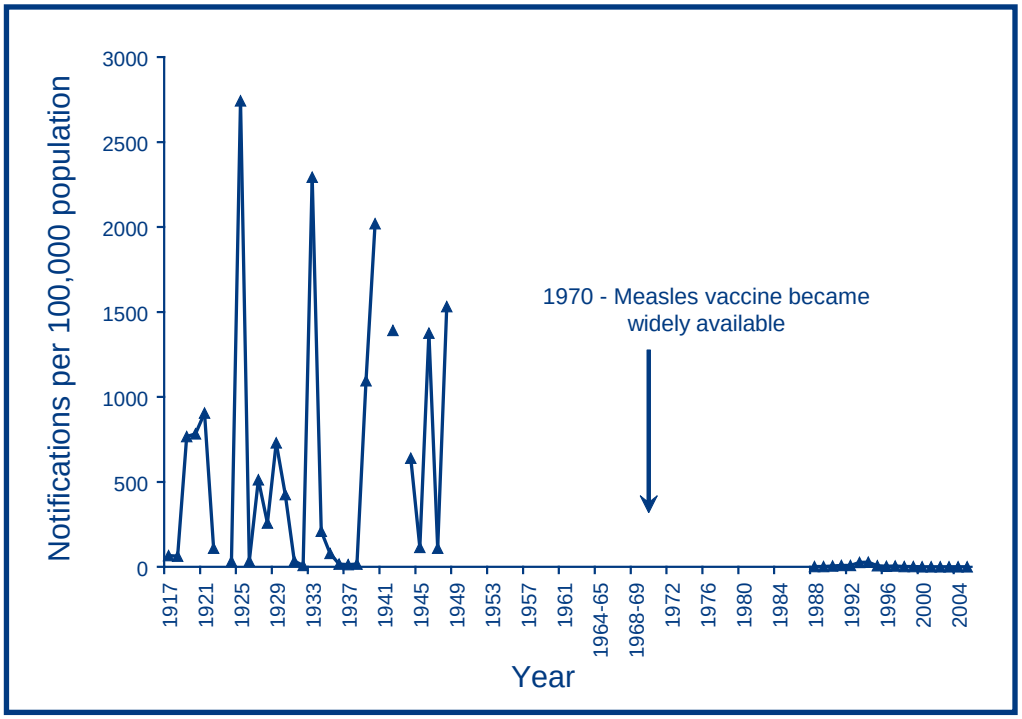


↓ Indicates major change in vaccination policy. Source: Hall R. Notifiable disease surveillance, 1917 to 1991. *Commun Dis Intell* 1993;17:226–36. Updated with NNDSS data 1992–2005

Hepatitis A, 1952–2005

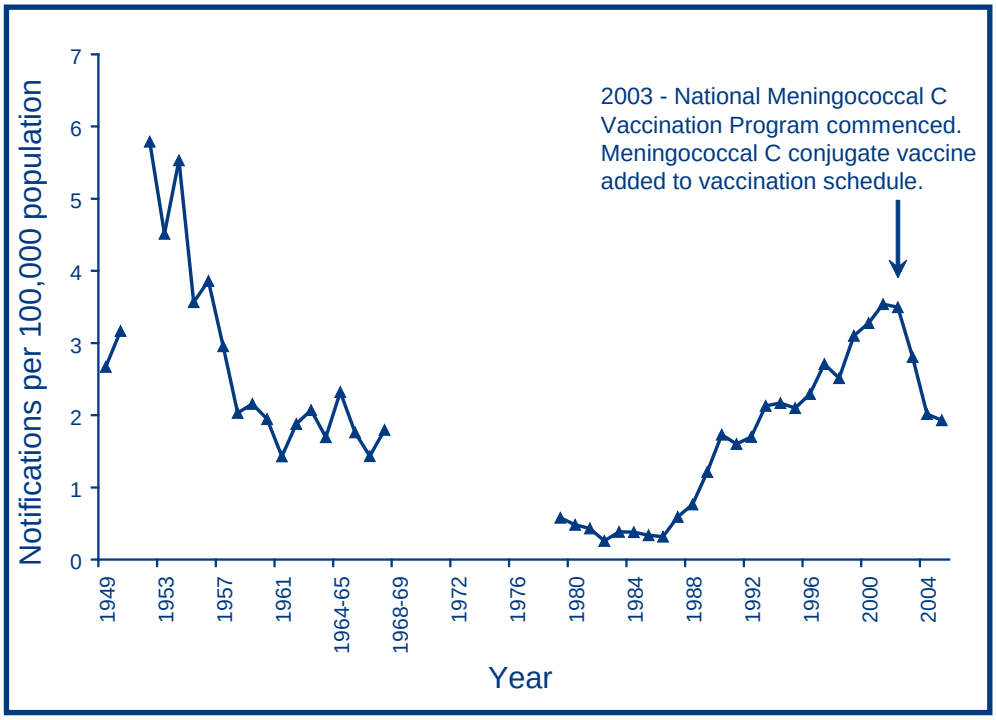


Measles, 1917–2005

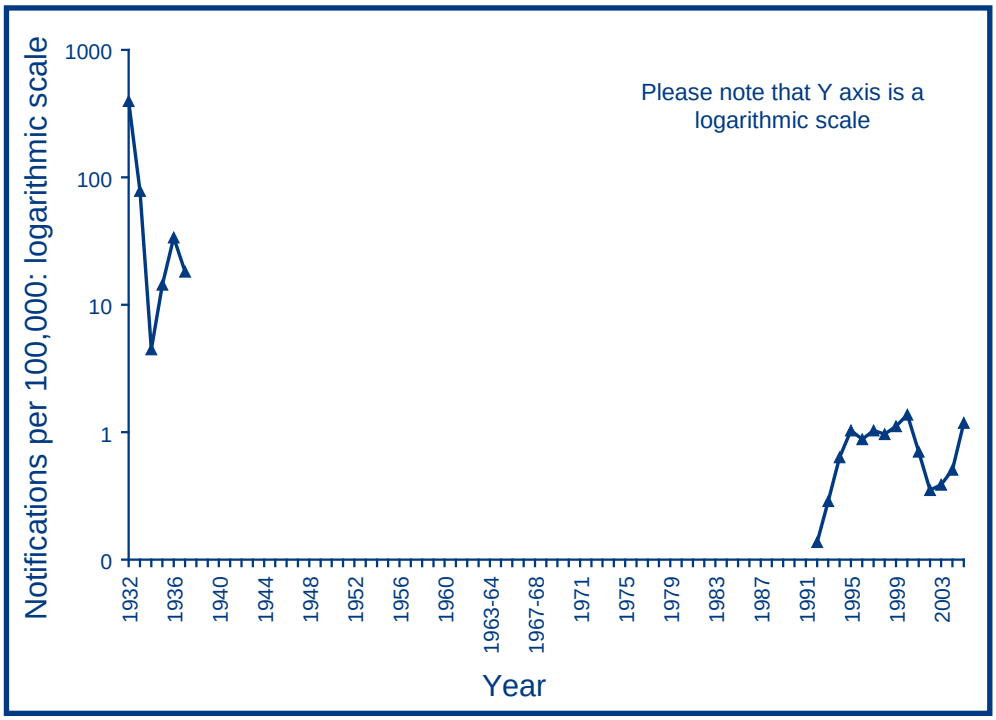


↓ Indicates major change in vaccination policy. Source: Hall R. Notifiable disease surveillance, 1917 to 1991. *Commun Dis Intell* 1993;17:226–36. Updated with NNDSS data 1992–2005

Meningococcal disease (invasive), 1949–2005

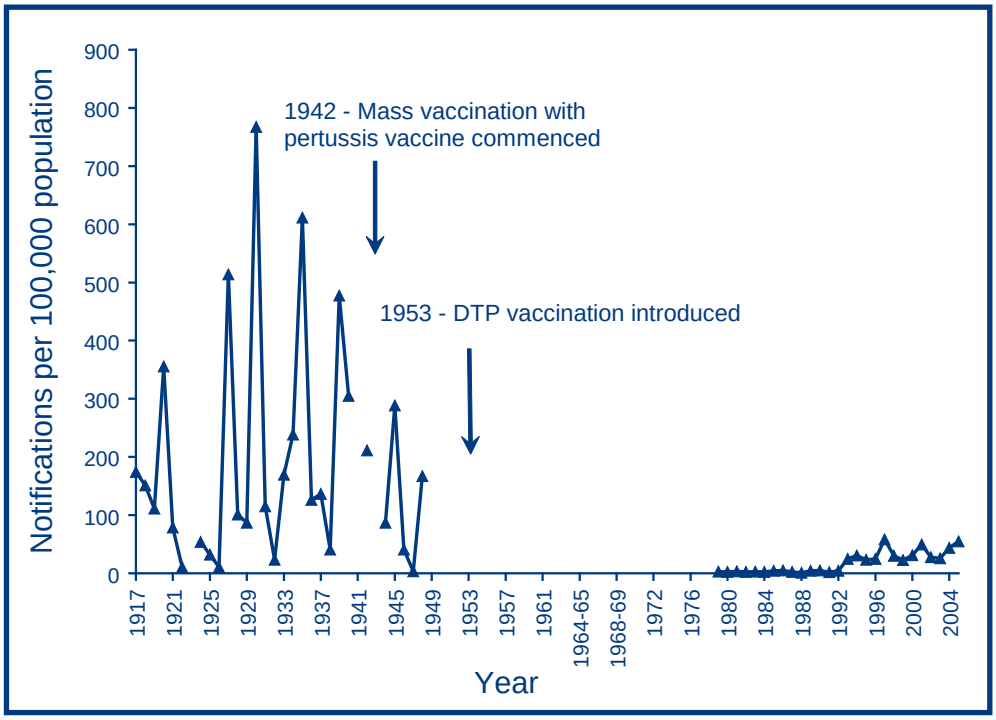


Mumps, 1932–2005

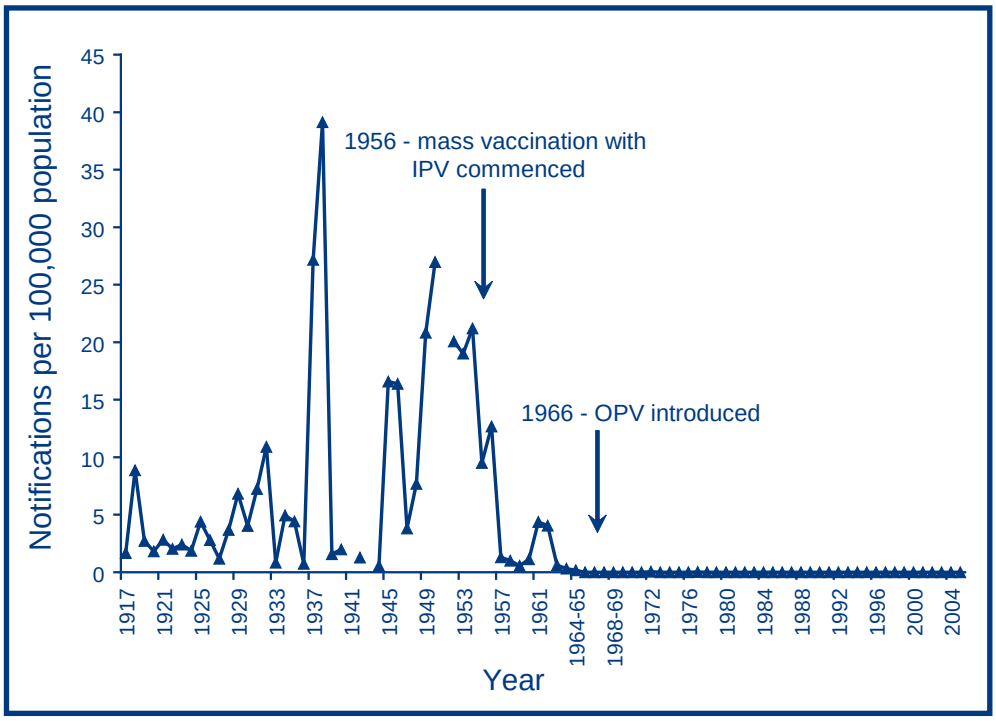


↓ Indicates major change in vaccination policy. Source: Hall R. Notifiable disease surveillance, 1917 to 1991. *Commun Dis Intell* 1993;17:226–36. Updated with NNDSS data 1992–2005

Pertussis, 1917–2005

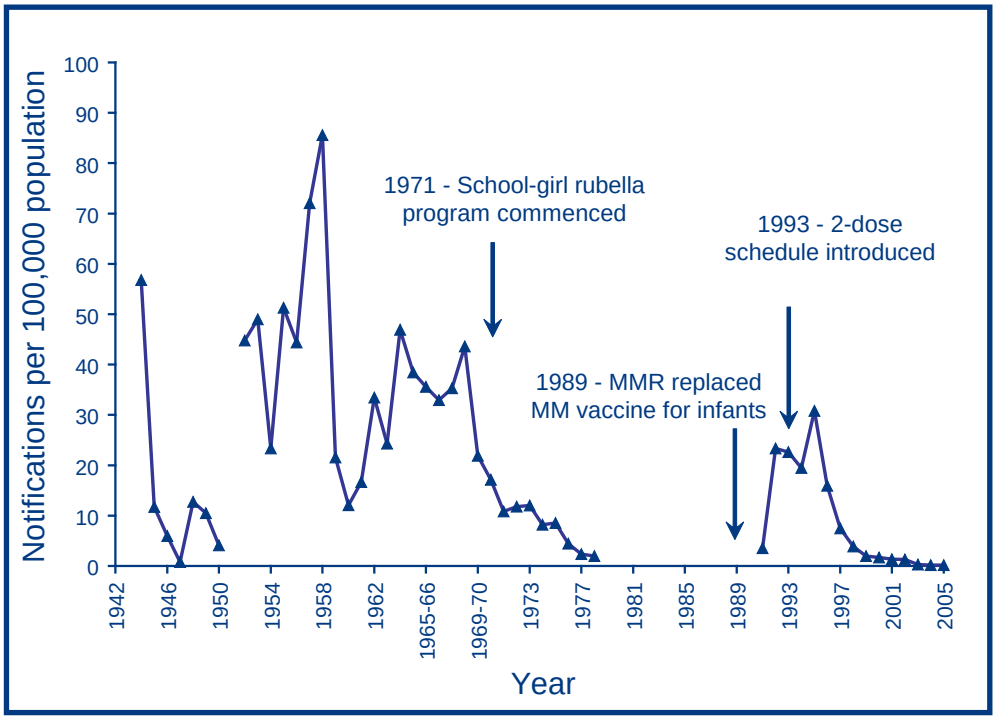


Poliomyelitis, 1917–2005

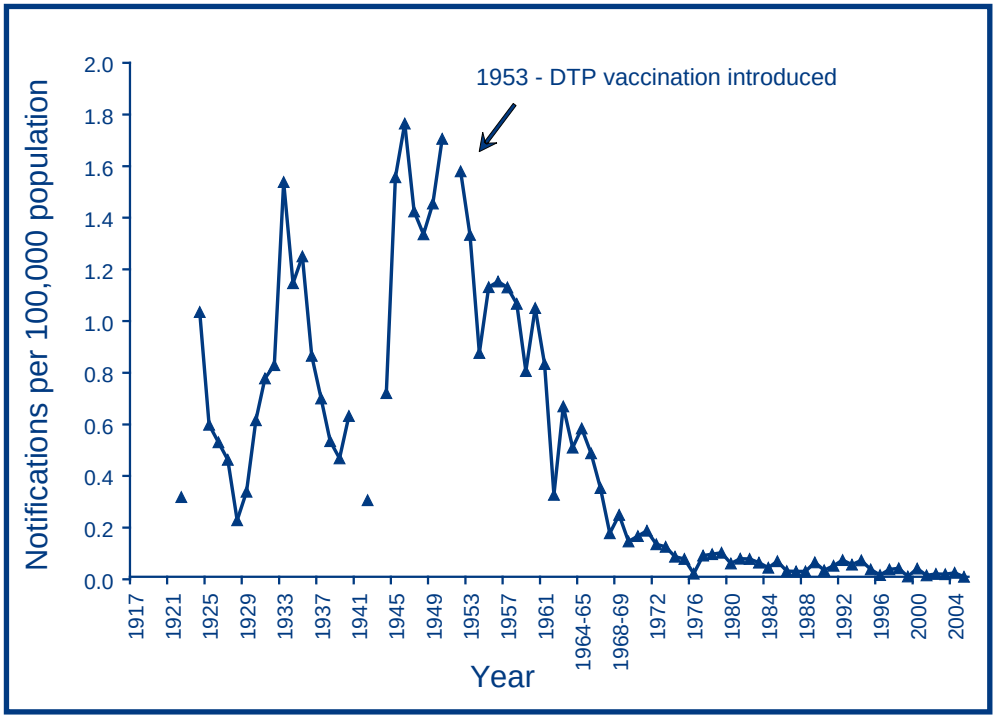


↓ Indicates major change in vaccination policy. Source: Hall R. Notifiable disease surveillance, 1917 to 1991. *Commun Dis Intell* 1993;17:226–36. Updated with NNDSS data 1992–2005

Rubella, 1942–2005



Tetanus, 1917–2005



↓ Indicates major change in vaccination policy. Source: Hall R. Notifiable disease surveillance, 1917 to 1991. *Commun Dis Intell* 1993;17:226–36. Updated with NNDSS data 1992–2005

Appendix 2. Notifications by state or territory (January 2000–December 2005)

Table 31. Notifications by state or territory and year (January 2000–December 2005)

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
Diphtheria	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	–	–	–	–	–	–	–	–	–
	2003	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
	2004	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
	2005	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 meningitis (<15 yrs only)	2000	0	4	0	7	1	0	2	0	14	–	0.3	–	0.9	0.3	–	0.2	–	0.4
	2001	0	6	3	2	2	0	2	1	16	–	0.4	5.9	0.3	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
	2003	0	4	2	3	1	0	1	1	12	–	0.3	4.0	0.4	0.3	–	0.1	0.3	0.3
	2004	0	2	2	3	2	0	1	0	10	–	0.2	4.0	0.4	0.7	–	0.1	–	0.3
	2005	0	4	1	2	0	0	2	0	9	–	0.3	2.0	0.2	–	–	0.2	–	0.2
	Total†	0	24	10	18	8	0	9	8	77	–	0.3	3.3	0.4	0.5	–	0.2	0.3	0.3
Hepatitis A	2000	5	202	45	134	54	3	193	181	817	1.6	3.1	23.0	3.8	3.6	0.6	4.1	9.7	4.3
	2001	27	198	38	120	20	4	105	40	552	8.5	3.0	19.2	3.3	1.3	0.8	2.2	2.1	2.8
	2002	8	145	47	67	16	4	68	37	392	2.5	2.2	23.7	1.8	1.1	0.8	1.4	1.9	2.0
	2003	5	124	43	48	13	14	89	95	431	1.5	1.9	21.7	1.3	0.9	2.9	1.8	4.9	2.2
	2004	1	137	14	27	11	1	71	57	319	0.3	2.0	7.0	0.7	0.7	0.2	1.4	2.9	1.6
	2005	3	83	64	50	10	2	59	54	325	0.9	1.2	31.6	1.3	0.6	0.4	1.2	2.7	1.6
	Total†	49	889	251	446	124	28	585	464	2,836	2.5	2.2	21.0	2.0	1.4	1.0	2.0	4.0	2.4
Hepatitis B (acute)	2000	3	100	6	54	30	18	131	73	415	1.0	1.5	3.1	1.5	2.0	3.8	2.8	3.9	2.2
	2001	4	95	3	49	22	21	192	38	424	1.3	1.4	1.5	1.4	1.5	4.5	4.0	2.0	2.2
	2002	0	79	12	53	11	19	174	35	383	–	1.2	6.0	1.4	0.7	4.0	3.6	1.8	2.0
	2003	0	74	15	40	10	10	152	45	346	–	1.1	7.6	1.1	0.7	2.1	3.1	2.3	1.7
	2004	5	53	8	50	8	17	110	29	280	1.5	0.8	4.0	1.3	0.5	3.5	2.2	1.5	1.4
	2005	3	58	5	59	8	3	78	34	248	0.9	0.9	2.5	1.5	0.5	0.6	1.6	1.7	1.2
	Total†	15	459	49	305	89	88	837	254	2,096	0.8	1.2	4.1	1.4	1.0	3.1	2.9	2.2	1.8

Table 31. Notifications by state or territory and year (January 2000–December 2005), *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
Influenza[§]	2001	26	244	(95)[‖]	(396)[‖]	135	(0)[‖]	(176)[‖]	234	(1,306)[‖]	8.1	3.7	48.0	10.9	8.9	0.0	3.7	12.3	6.7[¶]
	2002	38	996	53	1,142	291	7	593	551	3,671	11.8	15.0	26.7	30.8	19.2	1.5	12.2	28.6	18.7
	2003	7	861	151	890	311	7	640	616	3,483	2.2	12.9	76.1	23.4	20.4	1.5	13.0	31.6	17.5
	2004	1	1,012	39	614	75	3	205	184	2,133	0.3	15.1	19.5	15.8	4.9	0.6	4.1	9.3	10.6
	2005	39	1,415	61	1,698	277	19	594	466	4,569	12.0	20.9	30.1	42.8	18.0	3.9	11.8	23.2	22.5
	Total[‡]	111	4,528	(399)[‖]	(4,740)[‖]	1,089	(36)[‖]	(2,208)[‖]	2,051	(15,162)[‖]	8.6	16.9	49.9	30.9	17.8	1.9	11.2	26.1	19.0[¶]
Measles	2000	3	36	0	28	11	1	21	10	110	1.0	0.6	–	0.8	0.7	0.2	0.4	0.5	0.6
	2001	0	31	0	11	2	2	82	13	141	–	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
	2003	0	18	1	11	24	0	39	0	93	–	0.3	0.5	0.3	1.6	–	0.8	–	0.5
	2004	0	12	3	0	6	0	15	9	45	–	0.2	1.5	–	0.4	–	0.3	0.5	0.2
	2005	0	5	0	1	0	1	2	1	10	–	0.1	–	0.0	–	0.2	0.0	0.0	0.0
	Total[†]	3	110	4	59	45	4	173	33	431	0.2	0.3	0.3	0.3	0.5	0.1	0.6	0.3	0.4
Meningococcal disease	2000	5	254	9	66	32	15	161	86	628	1.6	3.9	4.6	1.9	2.1	3.2	3.4	4.6	3.3
	2001	8	235	13	129	40	24	164	74	687	2.5	3.6	6.6	3.6	2.6	5.1	3.4	3.9	3.5
	2002	12	207	9	124	30	26	211	68	687	3.7	3.1	4.5	3.3	2.0	5.5	4.3	3.5	3.5
	2003	13	202	12	105	33	20	127	46	558	4.0	3.0	6.0	2.8	2.2	4.2	2.6	2.4	2.8
	2004	11	148	12	84	13	18	79	40	405	3.4	2.2	6.0	2.2	0.8	3.7	1.6	2.0	2.0
	2005	8	139	11	62	26	10	89	47	392	2.5	2.1	5.4	1.6	1.7	2.1	1.8	2.3	1.9
	Total[†]	57	1,185	66	570	174	113	831	361	3,357	3.0	3.0	5.5	2.5	1.9	4.0	2.8	3.1	2.8
Mumps	2000	18	92	4	NN	15	2	43	39	(213)[‖]	5.7	1.4	2.0	NA	1.0	0.4	0.9	2.1	1.1[¶]
	2001	2	28	1	(3)[‖]	12	2	40	29	(117)[‖]	0.6	0.4	0.5	NA	0.8	0.4	0.8	1.5	0.6[¶]
	2002	0	27	1	6	10	0	10	13	67	–	0.4	0.5	0.2	0.7	–	0.2	0.7	0.3
	2003	2	35	0	10	13	0	4	13	77	0.6	0.5	–	0.3	0.9	–	0.1	0.7	0.4
	2004	3	65	0	17	4	0	3	10	102	0.9	1.0	–	0.4	0.3	–	0.1	0.5	0.5
	2005	1	110	7	71	8	0	20	23	240	0.3	1.6	3.5	1.8	0.5	–	0.4	1.1	1.2
	Total[†]	26	357	13	(107)[‖]	62	4	120	127	(816)[‖]	1.3	0.9	1.1	0.5	0.7	0.1	0.4	1.1	0.7[¶]

Table 31. Notifications by state or territory and year (January 2000–December 2005), *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
Pertussis	2000	219	3,689	9	540	588	143	715	93	5,996	69.5	56.9	4.6	15.2	39.1	30.3	15.1	5.0	31.3
	2001	162	4,438	150	1,626	2,010	104	863	227	9,580	50.7	67.5	75.8	44.8	133.0	22.0	18.0	11.9	49.3
	2002	110	1,850	37	1,853	473	37	868	234	5,462	34.2	27.9	18.6	49.9	31.1	7.8	17.9	12.2	27.8
	2003	357	2,770	5	716	232	133	626	255	5,094	110.4	41.5	2.5	18.8	15.2	27.9	12.7	13.1	25.6
	2004	124	3,562	27	1,033	994	37	872	2,101	8,750	38.3	53.0	13.5	26.6	64.9	7.7	17.6	106.2	43.6
	2005	315	5,794	92	1,775	1,507	33	1,160	515	11,191	96.9	85.5	45.4	44.8	97.7	6.8	23.1	25.6	55.1
	Total†	1,287	22,103	320	7,543	5,804	487	5,104	3,425	46,073	66.7	55.4	26.8	33.4	63.5	17.0	17.4	29.4	38.9
(<5 yr only)	Total‡	45	1,587	71	550	299	37	399	507	3,495	36.5	61.5	67.6	36.8	55.2	20.1	21.7	67.4	45.9
Pneumococcal disease (invasive)§	2001	33	443	99	430	(118)‖	62	(383)‖	204	(1,772)‖	10.3	6.7	50.1	11.8	NA	13.1	NA	10.7	9.1¶
	2002	66	876	66	438	180	63	552	225	2,466	20.5	13.2	33.2	11.8	11.9	13.3	11.4	11.7	12.6
	2003	41	807	73	468	177	52	469	150	2,237	12.7	12.1	36.8	12.3	11.6	10.9	9.5	7.7	11.3
	2004	55	910	93	476	198	57	389	199	2,377	17.0	13.5	46.5	12.2	12.9	11.8	7.8	10.1	11.8
	2005	30	646	71	325	135	44	299	139	1,689	9.2	9.5	35.0	8.2	8.8	9.1	6.0	6.9	8.3
	Total‡	225	3,682	402	2,137	(808)‖	278	(2,092)‖	917	(10,541)‖	11.7	9.2	33.7	9.5	8.8	9.7	7.1	7.9	8.9¶
Poliomyelitis	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	–	–	–	–	–	–	–	–	–
	2003	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
	2004	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
	2005	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
	Total†	0	0	0	0	0	0	0	0	0	–	–	–	–	–	–	–	–	–
Q fever	2000	0	132	0	393	11	1	24	14	575	–	2.0	–	11.0	0.7	0.2	0.5	0.7	3.0
	2001	5	143	0	444	17	1	65	19	694	1.6	2.2	–	12.2	1.1	0.2	1.4	1.0	3.6
	2002	0	275	1	357	27	0	82	20	762	–	4.1	0.5	9.6	1.8	–	1.7	1.0	3.9
	2003	1	288	1	223	15	1	14	19	562	0.3	4.3	0.5	5.9	1.0	0.2	0.3	1.0	2.8
	2004	2	220	3	160	40	0	29	9	463	0.6	3.3	1.5	4.1	2.6	–	0.6	0.5	2.3
	2005	0	139	3	157	17	0	28	6	350	–	2.1	1.5	4.0	1.1	–	0.6	0.3	1.7
	Total†	8	1,197	8	1,734	127	3	242	87	3,406	0.4	3.0	0.7	7.7	1.4	0.1	0.8	0.7	2.9

Table 31. Notifications by state or territory and year (January 2000–December 2005), *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
Rubella	2000	4	191	0	46	7	1	67	6	322	1.3	2.9	–	1.3	0.5	0.2	1.4	0.3	1.7
	2001	2	58	0	134	5	3	60	3	265	0.6	0.9	–	3.7	0.3	0.6	1.2	0.2	1.4
	2002	6	35	1	189	5	1	16	4	257	1.9	0.5	0.5	5.1	0.3	0.2	0.3	0.2	1.3
	2003	0	23	0	25	1	1	1	3	54	–	0.3	–	0.7	0.1	0.2	0.0	0.2	0.3
	2004	0	17	0	9	2	0	0	3	31	–	0.3	–	0.2	0.1	–	–	0.2	0.2
	2005	0	10	0	9	0	0	6	6	31	–	0.1	–	0.2	–	–	0.1	0.3	0.2
	Total†	12	334	1	412	20	6	150	25	960	0.6	0.8	0.1	1.8	0.2	0.2	0.5	0.2	0.8
Tetanus	2000	0	2	0	2	3	0	1	0	8	–	0.0	–	0.1	0.2	0.0	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	3	0	0	0	1	4	–	–	–	0.1	–	–	–	0.1	0.0
	2003	0	1	0	2	0	0	1	0	4	–	0.0	–	0.1	–	–	0.0	–	0.0
	2004	0	0	0	3	2	0	0	0	5	–	–	–	0.1	0.1	–	–	–	0.0
	2005	0	1	0	0	0	1	0	0	2	–	0.0	–	–	–	0.2	–	–	0.0
	Total†	0	4	0	10	6	2	3	1	26	–	0.0	–	0.0	0.1	0.1	0.0	0.0	0.0

* See Chapter 3 for case definitions.

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

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

§ Disease not notifiable prior to 2001.

|| 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 or territory (1 July 2000–30 June 2005)

Table 32. Hospitalisations by state or territory and financial year of separation (1 July 2000–30 June 2005)*

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
Haemophilus influenzae type b meningitis‡ (<15 yrs only)	00/01	0	1	0	10	1	<5	<5	<5	n.p.	–	0.1	–	1.3	0.3	n.p.	n.p.	n.p.	n.p.
	01/02	0	4	<5	2	4	0	<5	<5	16	–	0.3	n.p.	0.3	1.4	–	n.p.	n.p.	0.4
	02/03	0	3	0	2	2	0	0	7	14	–	0.2	–	0.3	0.7	–	–	1.8	0.4
	03/04	0	2	<5	6	4	0	0	<5	15	–	0.2	n.p.	0.8	1.4	–	–	n.p.	0.4
	04/05	0	2	<5	4	0	<5	<5	0	n.p.	–	0.2	n.p.	0.5	–	n.p.	n.p.	–	n.p.
	Total§	0	12	<15	24	11	<10	<15	<15	70	–	0.2	2.4	0.6	0.8	n.p.	n.p.	0.6	0.4
Hepatitis A	00/01	5	125	13	66	28	7	75	32	351	1.6	1.9	6.6	1.9	1.9	1.5	1.6	1.7	1.8
	01/02	n.p.	145	14	60	28	<5	54	16	325	n.p.	2.2	7.1	1.7	1.9	n.p.	1.1	0.8	1.7
	02/03	<5	112	24	61	29	n.p.	48	26	307	n.p.	1.7	12.1	1.6	1.9	n.p.	1.0	1.4	1.6
	03/04	<5	87	17	25	22	n.p.	42	43	243	n.p.	1.3	8.6	0.7	1.4	n.p.	0.9	2.2	1.2
	04/05	<5	72	9	38	8	<5	57	17	205	n.p.	1.1	4.5	1.0	0.5	n.p.	1.1	0.9	1.0
	Total§	17	541	77	250	115	21	276	134	1,431	1.1	1.6	7.8	1.3	1.5	0.9	1.1	1.4	1.5
Hepatitis B (acute) (principal diagnosis only)	00/01	0	36	<5	14	10	n.p.	65	22	158	–	0.6	n.p.	0.4	0.7	n.p.	1.4	1.2	0.8
	01/02	0	46	n.p.	13	4	<5	61	16	147	–	0.7	n.p.	0.4	0.3	n.p.	1.3	0.8	0.8
	02/03	<5	59	<5	25	4	n.p.	75	20	194	n.p.	0.9	n.p.	0.7	0.3	n.p.	1.5	1.0	1.0
	03/04	0	50	<5	23	8	<5	62	18	169	–	0.7	n.p.	0.6	0.5	n.p.	1.3	0.9	0.9
	04/05	<5	39	<5	15	8	<5	61	22	154	n.p.	0.6	n.p.	0.4	0.5	n.p.	1.2	1.1	0.8
	Total§	<5	230	n.p.	90	34	n.p.	324	98	822	n.p.	0.7	n.p.	0.5	0.4	n.p.	1.3	1.0	0.8
Influenza‖	00/01	17	1,191	38	701	331	54	603	536	3,471	5.4	18.4	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
	02/03	9	997	36	785	211	30	543	530	3,141	2.8	15.0	18.1	21.2	13.9	6.3	11.2	27.5	16.0
	03/04	14	1,241	108	714	386	70	644	779	3,956	4.3	18.6	54.4	18.8	25.3	14.7	13.1	39.9	19.9
	04/05	11	618	35	423	146	14	371	401	2,019	3.4	9.2	17.5	10.9	9.5	2.9	7.5	20.3	10.0
	Total§	62	4,864	241	3,254	1,367	204	2,654	2,748	15,394	3.9	14.7	24.3	17.5	18.0	8.6	10.9	28.5	15.7
Measles	00/01	<5	14	0	6	4	0	36	<5	65	n.p.	0.2	–	0.2	0.3	–	0.8	0.2	0.3
	01/02	0	10	0	2	1	<5	18	n.p.	41	–	0.2	–	0.1	0.1	n.p	0.4	0.4	0.2
	02/03	0	11	0	0	2	<5	16	<5	32	–	0.2	–	–	0.1	n.p	0.3	0.1	0.2
	03/04	<5	13	0	9	4	0	n.p.	<5	39	n.p.	0.2	–	0.2	0.3	–	n.p	0.1	0.2
	04/05	<5	9	n.p	1	3	<5	<5	<5	23	n.p.	0.1	n.p	0.0	0.2	n.p	n.p	0.2	0.1
	Total§	<5	57	<5	18	14	<5	85	17	200	n.p.	0.2	n.p	0.1	0.2	n.p	0.4	0.2	0.2

Table 32. Hospitalisations by state or territory and financial year of separation (1 July 2000–30 June 2005),* *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
Meningococcal disease	00/01	5	323	10	140	51	15	230	96	870	1.6	5.0	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
	02/03	<5	277	10	195	n.p.	19	243	95	883	n.p.	4.2	5.0	5.3	n.p.	4.0	5.0	4.9	4.5
	03/04	19	250	14	152	57	24	189	64	769	5.9	3.7	7.1	4.0	3.7	5.0	3.8	3.3	3.9
	04/05	12	197	12	86	26	18	85	47	483	3.7	2.9	6.0	2.2	1.7	3.7	1.7	2.4	2.4
	Total§	n.p.	1,287	61	751	n.p.	120	1,000	379	2,135	n.p.	3.9	6.2	4.0	n.p.	5.1	4.1	3.9	4.0
Mumps	00/01	<5	15	0	10	4	0	12	<5	48	n.p.	0.2	–	0.3	0.3	–	0.3	n.p	0.3
	01/02	0	18	0	5	4	<5	7	<5	37	–	0.3	–	0.1	0.3	n.p	0.1	n.p	0.2
	02/03	0	19	<5	9	3	0	10	<5	45	–	0.3	n.p	0.2	0.2	–	0.2	n.p	0.2
	03/04	0	16	<5	9	1	<5	11	n.p.	45	–	0.2	n.p	0.2	0.1	n.p	0.2	n.p	0.2
	04/05	0	15	0	9	4	<5	12	n.p.	48	–	0.2	–	0.2	0.3	n.p	0.2	n.p	0.2
	Total§	<5	83	<5	42	16	<5	52	22	223	n.p.	0.3	n.p	0.2	0.2	n.p	0.2	0.2	0.2
Pertussis	00/01	8	267	10	56	78	6	62	19	506	2.5	4.1	5.1	1.6	5.2	1.3	1.3	1.0	2.6
	01/02	<5	260	44	156	134	n.p.	98	68	770	n.p	4.0	22.2	4.3	8.9	n.p.	2.0	3.6	4.0
	02/03	<5.	111	<5	131	29	n.p.	57	22	359	n.p.	1.7	n.p	3.5	1.9	n.p.	1.2	1.1	1.8
	03/04	n.p.	141	<5	76	19	18	73	35	373	n.p	2.1	n.p	2.0	1.2	3.8	1.5	1.8	1.9
	04/05	n.p.	179	13	87	89	<5	81	129	587	n.p.	2.7	6.5	2.2	5.8	n.p.	1.6	6.5	2.9
	Total§	27	958	71	506	349	40	371	273	2,595	1.7	2.9	7.2	2.7	4.6	1.7	1.5	2.8	2.6
(<5 yr only)	Total§	16	605	60	295	172	29	276	200	1,653	15.5	28.0	68.5	23.8	37.9	18.9	18.0	31.9	26.0
Pneumococcal disease (invasive)¶	00/01	14	375	58	170	65	13	218	99	1,012	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
	02/03	25	362	42	175	73	27	281	118	1,103	7.8	5.5	21.1	4.7	4.8	5.7	5.8	6.1	5.6
	03/04	16	390	45	162	78	11	256	85	1,043	4.9	5.8	22.7	4.3	5.1	2.3	5.2	4.4	5.2
	04/05	15	336	45	142	71	24	222	114	969	4.6	5.0	22.5	3.7	4.6	5.0	4.5	5.8	4.8
	Total§	90	1,828	236	838	391	100	1231	514	5,228	5.6	5.5	23.8	4.5	5.1	4.2	5.1	5.3	5.3

Table 32. Hospitalisations by state or territory and financial year of separation (1 July 2000–30 June 2005),* *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
Poliomyelitis**	00/01	0	9	<5	0	3	0	<5	0	n.p.	–	0.1	n.p.	–	0.2	–	n.p.	–	n.p.
	01/02	<5	6	<5	0	2	<5	0	0	14	n.p.	0.1	n.p.	–	0.1	n.p.	–	–	0.1
	02/03	<5	26	<5	3	0	0	n.p.	0	39	n.p.	0.4	n.p.	0.1	–	–	n.p.	–	0.2
	03/04	0	16	0	1	0	0	<5	<5	20	–	0.2	–	0.0	–	–	n.p.	n.p.	0.1
	04/05††	n.p.	n.p.	n.p.	n.p.	n.p.	n.p.	n.p.	n.p.	1	–	–	–	–	–	–	–	–	0.1
	Total§	<5	57	7	n.p.	n.p.	<5	9	<5	88	n.p.	0.2	0.7	0.0	0.1	n.p.	0.0	n.p.	0.1.
(Principal diagnosis only)	Total§	0	6	0	0	1	0	0	0	7	–	0.0	–	–	0.0	–	–	–	0.0
Q fever	00/01	0	62	0	111	3	0	13	<5	n.p.	–	1.0	–	3.1	0.2	–	0.3	n.p.	n.p.
	01/02	0	71	<5	136	11	<5	16	<5	239	–	1.1	n.p.	3.7	0.7	n.p.	0.3	n.p.	1.2
	02/03	<5	109	<5	107	17	<5	13	7	259	n.p.	1.6	n.p.	2.9	1.1	n.p.	0.3	0.4	1.3
	03/04	0	116	<5	86	9	6	13	n.p.	238	–	1.7	n.p.	2.3	0.6	1.3	0.3	n.p.	1.2
	04/05	0	74	<5	51	10	<5	14	<5	155	–	1.1	n.p.	1.3	0.7	n.p.	0.3	n.p.	0.8
	Total§	<5	432	n.p.	491	50	11	69	22	1,084	n.p.	1.3	n.p.	2.6	0.7	0.5	0.3	0.2	1.1
Rotavirus	00/01	147	1,725	298	805	445	44	559	452	4,475	46.6	26.6	152.4	22.6	29.6	9.3	11.8	24.1	23.4
	01/02	142	1,630	296	764	325	95	422	376	4,050	44.5	24.8	149.7	21.1	21.5	20.1	8.8	19.8	20.9
	02/03	91	1,710	199	900	337	49	472	313	4,071	28.3	25.8	100.2	24.3	22.2	10.4	9.7	16.3	20.7
	03/04	39	1,575	231	567	556	53	436	346	3,803	12.1	23.6	116.3	14.9	36.4	11.1	8.9	17.7	19.1
	04/05	45	972	163	571	413	116	344	235	2,859	13.9	14.5	81.6	14.7	26.9	24.1	6.9	11.9	14.2
	Total§	464	7,612	1,187	3,607	2,076	357	2,233	1,722	19,258	28.9	23.0	119.9	19.4	27.3	15.0	9.2	17.9	19.6
Rubella	00/01	0	24	0	3	1	0	<5	<5	33	–	0.4	–	0.1	0.1	–	n.p.	n.p.	0.2
	01/02	0	15	0	1	1	<5	<5	0	21	–	0.2	–	0.0	0.1	n.p.	n.p.	0.0	0.1
	02/03	<5	9	0	2	1	<5	n.p.	<5	21	n.p.	0.1	–	0.1	0.1	n.p.	n.p.	n.p.	0.1
	03/04	0	7	0	2	2	<5	<5	<5	18	–	0.1	–	0.1	0.1	n.p.	n.p.	n.p.	0.1
	04/05	0	1	0	0	0	0	<5	<5	5	–	0.0	–	–	–	–	n.p.	n.p.	0.0
	Total§	<5	56	0	8	5	<5	n.p.	n.p.	98	n.p.	0.2	–	0.0	0.1	n.p.	n.p.	n.p.	0.1

Table 32. Hospitalisations by state or territory and financial year of separation (1 July 2000–30 June 2005),* *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**	00/01	0	17	0	6	3	<5	<5	<5	31	–	0.3	–	0.2	0.2	n.p.	n.p.	n.p.	0.2
	01/02	0	6	0	4	3	0	<5	6	23	–	0.1	–	0.1	0.2	–	n.p.	0.3	0.1
	02/03	0	6	0	7	3	0	5	6	27	–	0.1	–	0.2	0.2	–	0.1	0.3	0.1
	03/04	0	7	0	4	n.p.	<5	<5	0	17	–	0.1	–	0.1	n.p.	n.p.	n.p.	–	0.1
	04/05	0	5	0	10	n.p.	0	<5	0	22	–	0.1	–	0.3	n.p.	–	n.p.	–	0.1
	Total§	0	41	0	31	17	<5	16	n.p.	120	–	0.1	–	0.2	0.2	n.p.	0.1	n.p.	0.1
Varicella	00/01	22	548	14	336	152	30	344	198	1,644	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
	02/03	6	428	18	244	91	34	307	130	1,258	1.9	6.5	9.1	6.6	6.0	7.2	6.3	6.8	6.4
	03/04	8	568	27	354	122	32	290	145	1,546	2.5	8.5	13.6	9.3	8.0	6.7	5.9	7.4	7.8
	04/05	17	492	18	279	113	47	314	197	1,477	5.2	7.3	9.0	7.2	7.4	9.7	6.3	10.0	7.4
	Total§	75	2,606	98	1,521	663	175	1,599	867	7,604	4.7	7.9	9.9	8.2	8.7	7.4	6.6	9.0	7.7
Zoster	00/01	44	1,521	15	838	444	135	1,142	416	4,555	14.0	23.4	7.7	23.5	29.5	28.6	24.1	22.2	23.8
	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
	02/03	57	1,619	26	857	487	176	1,222	448	4,892	17.7	24.4	13.1	23.1	32.1	37.2	25.2	23.3	24.9
	03/04	56	1,545	27	817	456	124	1,246	436	4,707	17.3	23.1	13.6	21.5	29.9	26.0	25.4	22.4	23.7
	04/05	33	1,971	36	951	459	115	1,275	487	5,327	10.2	29.3	18.0	24.5	29.9	23.8	25.7	24.6	26.5
	Total§	246	8,109	125	4,354	2,360	688	6,060	2,167	24,109	15.3	24.5	12.6	23.4	31.1	29.0	25.0	22.5	24.6

* Note that total hospitalisations potentially include readmissions of the same case and inter-hospital transfers. Reported data may also contain possible coding errors.

† See Chapter 3 for defining ICD codes.

‡ *Haemophilus influenzae* type b (Hib) hospitalisations include only hospitalisations coded as G00.0 (Hib meningitis). The ICD-10 code J05.1 (acute epiglottitis) used in previous reports is no longer included due to evidence that the specificity of epiglottitis for Hib infection is now extremely low in Australia.

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

|| Influenza hospitalisations are likely to be a large underestimate since many people will be diagnosed with the complications of influenza, without influenza being reported as the underlying cause.

¶ Pneumococcal meningitis and septicaemia.

** Hospitalisations for rare diseases such as poliomyelitis and tetanus should be interpreted with caution due to possible coding errors. The older age of hospitalisations coded as acute poliomyelitis suggests that these are in fact hospitalisations of adults with a past history of poliomyelitis.

†† All states and territories have been suppressed to allow publication of the national total.

n.p Not published. The n.p. has been used to prevent calculation of the cells marked <5, or to suppress the rate per 100,000 for the cells where hospitalisations have been suppressed either for <5 or for n.p.

Appendix 4. Changes to the Australian Standard Vaccination Schedule (1992–2005)

Table 33. Diphtheria, tetanus and pertussis vaccination practice in Australia, 1992 to 2005

Date	Intervention
1994	Fifth dose of DTP at 4–5 years of age 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 fourth and fifth doses of DTP vaccination (due at 18 months and 4–5 years of age)
1999	DTPa recommended for all five childhood DTP doses Combined DTPa-hepatitis B vaccine approved
2000	Second booster dose of DTPa recommended at 4 years of age instead of 4–5 years NHMRC recommended 10 yearly booster doses of ADT be replaced with a routine booster dose at 50 years 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
2001	Combined DTPa-hepB-IPV and DTPa-hepB-IPV-Hib vaccines approved
2002	Combined DTPa-IPV and DTPa-IPV-Hib vaccines approved
2003	September: Fourth dose of DTPa at 18 months of age no longer recommended. dTpa recommended at 15 years of age, replacing dT.
2004	dTpa funded at 15–17 years of age replacing the diphtheria-tetanus dose

Table 34. *Haemophilus influenzae* type b vaccination practice in Australia, 1992 to 2005

Date	Intervention
1992	First 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 of age for Aboriginal and Torres Strait Islander children and all children in the Northern Territory, HBOC used otherwise
2000	Combined Hib(PRP-OMP)-hepatitis B vaccine approved PRP-OMP recommended for all infants (administered separately or in combination with hepatitis B vaccine)
2001	Combined DTPa-hepB-IPV-Hib vaccine approved
2002	Combined DTPa-IPV-Hib vaccines approved
2005	November: Combined DTPa-IPV-Hib (PRP-T) vaccines used in NSW, ACT, WA and Tasmania; PRP-OMP vaccine continues to be used in other jurisdictions

Table 35. Hepatitis A vaccination practice in Australia, 1992 to 2005

Date	Intervention
1994	Hepatitis A vaccine (formaldehyde inactivated HAV) approved in Australia for at-risk groups, three doses recommended
2005	Hepatitis A vaccination (two doses recommended) recommended for Aboriginal and Torres Strait Islander children aged 12–24 months of age residing in the Northern Territory, Qld, WA and SA.

Table 36. Hepatitis B vaccination practice in Australia, 1992 to 2005

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 Northern Territory 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 Hib(PRP-OMP)-hep B vaccine approved May: Universal infant vaccination included in childhood schedule with a birth dose of monovalent paediatric hepatitis B vaccine, followed by three doses as part of a combination vaccine schedule DTPa-hepB vaccine included on childhood schedule (used in Qld, NSW, ACT, SA, and NT) Hib(PRP-OMP)-hepB vaccine included on childhood schedule (used in Tas, Vic, WA) Preadolescent vaccination recommended at 10–13 years of age rather than 10–16 years of age Booster doses no longer recommended by NHMRC
2001	Combined DTPa-hepB-IPV and DTPa-hepB-IPV-Hib vaccines approved

Table 37. Influenza vaccination practice in Australia, 1992 to 2005

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 38. Measles, mumps and rubella vaccination practice in Australia, 1992 to 2005

Date	Intervention
1992	November: NHMRC recommended second dose of MMR vaccine for both sexes to replace schoolgirl rubella vaccination program
1993	November: Childhood vaccination schedule updated to include second dose of MMR vaccine for 10–16 year olds (replacing schoolgirl rubella vaccination)
1998	Recommended age for first dose of MMR vaccine for Aboriginal and Torres Strait Islander children in the Northern Territory increased to 12 months of age (in line with non-Aboriginal infants) July: Recommended age for second 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 of age not 4–5 years MMR rather than rubella vaccine recommended for non-immune women of child-bearing age
2001	Young adult (18 – 30 years) MMR vaccination campaign conducted

Table 39. Meningococcal C vaccination practice in Australia, 1992 to 2005

Date	Intervention
2001	Meningococcal C conjugate vaccine (Meningitec) approved
2002	Meningococcal C conjugate vaccines (NeisVac-C, Menjugate) approved Funding announced for National Meningococcal C Vaccination Program commencing January 2003 with meningococcal C conjugate vaccine (MenCCV) funded for all children 1–18 years of age, at 12 months of age with a catch-up program for older ages
2003	NHMRC endorses recommended changes. Meningococcal C conjugate vaccine added to childhood vaccination schedule at 12 months of age. National Meningococcal C Vaccination Program commences January 2003.

Table 40. Pneumococcal vaccination practice in Australia, 1992 to 2005

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 15 years and over 7-valent conjugate pneumococcal vaccine licensed in Australia
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)
2003	7-valent conjugate pneumococcal vaccine recommended at 2, 4 and 6 months of age
2005	January: Universal funded 7-valent pneumococcal conjugate vaccine (7vPCV) replaced the previous targeted childhood program Universal 23-valent pneumococcal polysaccharide vaccine (23vPPV) for adults over 65 years replaced previous subsidy through the PBS

Table 41. Polio vaccination practice in Australia, 1992 to 2005

Date	Intervention
1994	Recommendation for reinforcing dose of OPV to 15 year old adolescents
2001	Combined DTPa-hepB-IPV and DTPa-hepB-IPV-Hib vaccines approved
2002	Combined DTPa-IPV and DTPa-IPV-Hib vaccines approved Fifth dose of OPV at 15–17 years of age no longer recommended
2003	IPV recommended to replace OPV at 2, 4 and 6 months and at 4 years of age
2005	November: IPV funded to replace OPV, in combination vaccines

Table 42. Varicella vaccination practice in Australia, 1992 to 2005

Date	Intervention
2003	Varicella vaccine recommended at 18 months of age
2005	Universal funded immunisation against varicella at 18 months of age from November 2005 with a school-based catch-up program for children at 10–12 years of age not previously vaccinated and without a history of varicella infection No funded catch-up for children 2–10 years of age

* See also Gidding HF, Burgess MA, Kempe AE. A brief history of vaccination and childhood vaccination practices in Australia. *Med J Aust* 2001;174:37–40.

Detailed historical tables are available from: http://www.ncirs.usyd.edu.au/publ/publ-79-tbls.html

Appendix 5. Government funding of national immunisation programs in Australia

Government funding of national immunisation programs in Australia

Prior to 1988, the Commonwealth provided childhood vaccines to states and 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 and 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 and territories as part of the Financial Assistance Grants (FAGs) and the Hospital Funding Grants (HFGs). The increase in funding was equivalent to the level of immunisation activity in each jurisdiction in 1988.

As there were increases in vaccination activity above the 1988 levels, states and territories expressed concern about the level of funding provided via the FAGs/HFGs. Details of the funding arrangements were also interpreted differently by the Commonwealth and each state and 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 included the lack of a coordinated scheme for the provision of vaccines, and the wide variation in prices which the states and territories paid for vaccines, with the smaller jurisdictions paying higher prices. The Strategy recommended that the Australian Health Ministers' Advisory Council (AHMAC) consider vaccine funding arrangements.

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 and territories. In July 1993, the Commonwealth provided funds to states and territories for Hib vaccines.

In 1994, the Commonwealth Government agreed to fund the purchase of a number of childhood vaccines (DTP, MMR, OPV) via Specific Purpose Payments (SPPs) to states and territories. Commonwealth funding was conditional on vaccines being provided to all public and private immunisation providers, including medical practitioners, and was formalised in bilateral agreements with each state and territory.

From 1997/1998 to 2003/2004, funding for some vaccines was included in the Public Health Outcome Funding Agreements (PHOFAs). However, a number of vaccines continued to be funded via FAGs (OPV doses 1, 2, 3 and 4 and MMR dose 1) and HFGs (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 from 1999 to 2000, with 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 through the FAGs and HFGs were not adjusted, thereby freeing up state and 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 that it be included on the ASVS.

In 1999 to 2000, the funding provided through the PHOFAs for vaccine allowed the states and territories to purchase enough vaccine for 105% of the eligible cohort for each vaccine. The exception at that time was influenza. Commonwealth funding for vaccines is approved by the Federal Minister for Health and Ageing 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 by the NHMRC and funded by the Commonwealth. In 2001, the 7-valent pneumococcal conjugate vaccine was provided at no cost to children in the following three categories: (1) all Aboriginal and Torres Strait Islander chil-

dren aged up to two years; (2) in the Central Australian region, Indigenous children aged up to five years and non-Indigenous children aged up to two years; and (3) all children under five years with medical risk factors predisposing them to a high incidence or severity of pneumococcal infection.

Meningococcal C conjugate vaccine was recommended and funded at 12 months of age from January 2003. In September 2003, the DTPa booster dose at 18 months of age was no longer recommended.

In September 2003, the recommended schedule was changed to include the universal 7-valent conjugate pneumococcal vaccine at 2, 4 and 6 months of age, the varicella-zoster vaccine at 18 months of age, and the inactivated poliomyelitis vaccine in place of oral vaccine, although these recommendations were not immediately funded. In January 2005, the 7-valent pneumococcal conjugate vaccination program was extended to all children.

In May 2005, the National Immunisation Program (NIP) schedule replaced the Australian Standard Vaccination Schedule (ASVS) for children aged up to 6 years, with all recommended vaccines on the Schedule to be funded, including the varicella-zoster vaccine and inactivated poliomyelitis vaccine from November 2005.

Table 43 summarises the dates when vaccines were provided at no cost in the public sector and through private medical practitioners, as outlined above. There are separate arrangements for the funding of vaccine not provided through the NIP, such as travel-related vaccines, vaccines listed on the Pharmaceutical Benefits Scheme (PBS) and those provided by employers.

Table 43. Dates when childhood vaccines became available in Australia free of charge* in the public and private sectors, up to December 2005

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 Apr		1993 Apr	
Hib vaccines (all infants aged <5 years)	1993 Jul	WA 1993 Jan NT 1993 Apr	1993 Jul	WA 1993 Jan NT 1994
DTPa boosters (infants aged 18 months and 4–5 years)	1997 Sep	Tas 1997 Oct Qld 1997 Dec	1997 Sep	Tas 1997 Oct Qld 1997 Dec
dTpa booster (at 15 years replaces ADT)	2004 Jan		2004 Jan	
DTPa infants aged 2, 4 and 6 months)	1999 Feb	NT 1997 Aug SA 1997 Aug Tas 1999 Feb Qld 1999 Apr	1999 Feb	NT 1997 Aug SA 1997 Aug Tas 1999 Feb Qld 1999 Apr
Hep B (at-risk infants)	1987	NT 1988 Jan SA 1996	Not funded by the Commonwealth	NSW 1987
Hep B (adolescent dose)	1998 Jan	Qld 1998 Mar Tas 1998 Mar NT 1998 Apr NSW 1999 SA 1999	?1998	Qld 1998 Mar Tas 1998 Mar NT 1998 Apr NSW 1999
Hep B (universal infant dose)	2000 May	NT 1990 Aug	2000 May	NT 1994
7vPCV (at-risk children)	2001		2001	
Influenza and pneumococcal for Indigenous at risk (15–49 years)	2003		2003	
Conjugate meningococcal C (infants aged 12 months and catch-up aged 1–18 years)	2003 Jan		2003 Jan	
7vPCV (all children)	2005 Jan		2005 Jan	
VZV (infants aged 18 months and children aged up to 12 years without previous history of varicella)	2005 Nov		2005 Nov	
IPV (replacing OPV)	2005 Nov		2005 Nov	

* 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. Where vaccine is provided by a private medical practitioner, there may be costs associated with the consultation.

† Refers to vaccines provided by private medical practitioners. 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.

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Appendix 6. Notifiable diseases definitions in use prior to 2004

Notifiable diseases definitions in use prior to 2004

In September 2003, new national case definitions for notifications reported to NNDSS were endorsed by the Communicable Diseases Network Australia,[11] with nearly all jurisdictions implementing the new definitions in January 2004 (New South Wales commenced August 2004). Prior to the adoption of the national definitions, some jurisdictions used the 1994 NHMRC case definitions[10] as written (e.g. South Australia and Western Australia) and others used their own definitions (e.g. New South Wales and Victoria). This Appendix describes the definitions in use for notifiable diseases data prior to 2004 (i.e. the first year (2003) of the three year review period of this report). Further detail about definitions previously in use can be found in earlier reports in this series[1–3] and in Skull 2001.[310]

Diphtheria

Notifications prior to 2004

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

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

Hib

Notifications prior to 2004

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

- the isolation of *Haemophilus influenzae* type b (Hib) 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: From 2002 in Victoria, notifications only included cases where Hib was laboratory confirmed.[311]

Hepatitis A

Notifications prior to 2004

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.

Hepatitis B

Notifications prior to 2004

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).

Influenza

Became a notifiable disease in 2001 and the definition has remained unchanged.

Measles

Notifications prior to 2004

a) An illness characterised by all of 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.

Meningococcal disease

Notifications prior to 2004

In jurisdictions apart from New South Wales and the Northern Territory, a notification of meningococcal disease required supportive laboratory evidence, although the nature of this varied. In New South Wales, Queensland and the Northern Territory, a clinical diagnosis of meningococcal disease without laboratory evidence was accepted as a presumptive (New South Wales) or probable (Queensland, Northern Territory) case.

Mumps

Notifications prior to 2004

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)] were 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.

Pertussis

Notifications prior to 2004

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.

Invasive pneumococcal disease

Became a notifiable disease in 2001 and the definition has remained unchanged.

Polio

Notifications prior to 2004

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.

Rubella

Notifications prior to 2004

a) 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

b) Demonstration of rubella-specific IgM antibody, except following vaccination

or

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

or

d) Isolation of rubella virus from a clinical specimen.

Note: From July 2001 to July 2002, enhanced rubella surveillance was undertaken in Victoria leading to an increase in the specificity of notifications.[312]

Tetanus

Notifications prior to 2004

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.

Abbreviations

23vPPV	23-valent pneumococcal polysaccharide vaccine
7vPCV	7-valent pneumococcal conjugate vaccine
ABS	Australian Bureau of Statistics
ACIP	Advisory Committee on Immunization Practices (USA)
ACIR	Australian Childhood Immunisation Register
ADT	Adult diphtheria-tetanus
AFP	Acute flaccid paralysis
AGE	Acute gastroenteritis
AIHW	Australian Institute of Health and Welfare
Anti-HBc	Hepatitis B core antibody
APSU	Australian Paediatric Surveillance Unit
ASVS	Australian Standard Vaccination Schedule
aVDPV	Ambiguous vaccine derived poliovirus
CDT	Combined diphtheria-tetanus
CRS	Congenital rubella syndrome
CSF	Cerebrospinal fluid
cVDPV	Circulating vaccine derived poliovirus
DT (dT)	Diphtheria-tetanus
DTP	Diphtheria-tetanus-pertussis
DTPa	Diphtheria-tetanus-pertussis (acellular)
dTpa	Adolescent/adult diphtheria-tetanus-pertussis (acellular)
DTPw	Diphtheria-tetanus-pertussis (whole cell)
FAG	Finance assistance grant
HAV	Hepatitis A virus
HBOC	*Haemophilus influenzae* type b polysaccharide conjugated to non-toxic diphtheria CRM_{197} protein
HBsAg	Hepatitis B surface antigen
HBV	Hepatitis B virus
HepB	Hepatitis B (vaccine abbreviation)
HFG	Hospital funding grant
Hib	*Haemophilus influenzae* type b
HZ	Herpes zoster
ICD	International Classification of Diseases
ICD-10	International Classification of Diseases, 10th Revision
ICD-10-AM	International Statistical Classification of Diseases and Related Health Problems, 10th Revision, Australian Modification
ICD-9	International Classification of Diseases, 9th Revision
ICD-9-CM	International Classification of Diseases, 9th Revision, Clinical Modification
IgG	Immunoglobulin G
IgM	Immunoglobulin M

IPD	Invasive pneumococcal disease
IPV	Inactivated poliomyelitis vaccine
iVDPV	Immunodeficient vaccine derived poliovirus
LOS	Length of stay (in hospital)
MCC	Measles Control Campaign
MenCCV	Meningococcal C conjugate vaccine
MMR	Measles-mumps-rubella
MR	Measles-rubella
n.p.	Not published
NCCH	National Centre for Classification in Health
NCIRS	National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases
NHMRC	National Health and Medical Research Council
NIP	National Immunisation Program
NIS	National Immunisation Strategy
NNDSS	National Notifiable Diseases Surveillance System
NQFMP	National Q fever Management Program
OPV	Oral poliomyelitis vaccine
PBS	Pharmaceutical Benefits Scheme
PCR	Polymerase chain reaction
PHOFA	Public health outcome funding agreement
PRP-D	*Haemophilus influenzae* type b polysaccharide conjugated to diphtheria toxoid
PRP-OMP	*Haemophilus influenzae* type b polysaccharide conjugated to the outer membrane protein of *Neisseria meningitidis* vaccine
PRP-T	*Haemophilus influenzae* type b polysaccharide conjugated to tetanus toxoid
RSV	Respiratory syncytial virus
SSPE	Subacute sclerosing panencephalitis
VAPP	Vaccine-associated paralytic poliomyelitis
VDPV	Vaccine derived poliovirus
VIDRL	Victorian Infectious Diseases Reference Laboratory
VPD	Vaccine preventable disease
VZV	Varicella-zoster virus
WHO	World Health Organization

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